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THE EFFECTS OF ANTECEDENT FAILURE AND ALCOHOL UPON THE IDENTIFICATION OF CONCEPTS

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"One is not born, but rather becomes a woman." Simone de Beauvoir

TABLE OF CONTENTS

LIST OF TABLES

LIST OF TABLES - Continued

LIST OF TABLES - Continued

LIST OF TABLES - Continued

LIST OF ILLUSTRATIONS

EFFECTS OF ANTECEDENT FAILURE AND ALCOHOL UPON THE IDENTIFICATION OF CONCEPTS

CHAPTER I

INTRODUCTION

Failure orientation appears to be a common characteristic in alcoholics. These individuals tend to drink when faced with problematic situations. One very important goal of this thesis is to examine what happens to normals after drinking when failure has been experimentally induced prior to drinking. This procedure may yield a simple model for alcoholism and the importance and function of drinking in the face of failure.

The task used to induce failure was a concept identification task. The experiment was set up in such a manner that one was able to look at both the effects of alcohol on concept identification and also the effects of alcohol on failure set.

Performance of subjects on the ascending limb of the individual's blood alcohol curve was compared with performance of subjects on the descending limb of the blood alcohol curve to determine if differential effects on concept identification are found on the different limbs of the blood alcohol curve. Inducing failure or success on a concept identification task during Phase I, prior to alcohol intake, was used

to investigate the relationship of motivation and stress to performance on a similar task during Phase II, the portion of the study occurring after alcohol ingestion. The psychophysiological measures of tonic skin conductance (SCL), galvanic skin response (GSR), and muscle tension as indicated by electromyographic measures (EMG), were recorded during the experimental period as measures of arousal under the conditions of stress and alcohol in relation to prior resting levels.

Cognitive, sensory and motor functions are all affected by acute doses of alcohol, usually in a deleterious manner. In general, the effects of alcohol are subtle, complex, and diverse. For example, little or no impairment of performance is seen following moderate doses of alcohol on continuous attention tasks such as color-naming and signal detection (Talland, 1966; Docter, Naitoh, & Smith, 1966). However, if the subject is required to divide his attention between two or more aspects of a task, such as is necessary for dichotic listening (Moskowitz & DePry, 1968), systematic impairment results from moderate doses of alcohol.

Cognitive deficits due to alcohol have often been attributed to the impairment of memory. Not all aspects of memory are affected in the same manner. There is evidence that short-term memory, intermediate-term memory, and long-term memory may be differentially affected by alcohol. Memory impairment is usually not detectable at blood alcohol levels below 0.07% or 0.08% (0.15% is the level of legal intoxication in most states; in Oklahoma a lower level, 0.10%, is considered legal intoxication). A review of the literature by Ryback (1971) indicates that short-term memory is not significantly affected by alcohol.

However, Tarter, Jones, Simpson and Vega (1971) have reported impairment on the WAIS Digit Span, a short-term memory task, at a blood alcohol level of 0.08%, 30 minutes after consumption of drink. Jones (1972) has also reported impairment of short-term verbal memory on both the ascending and descending limbs of the blood alcohol curve at a blood alcohol level of 0.09%. Intermediate memory is adversely affected by alcohol at the 0.10 level on tasks such as paired associates learning (Hutchison, Tuchtie, Gray, & Steinberg, 1964) and recognition of pictures presented earlier (Ryback, Weinert, & Fozard, 1970). Long-term memory for information learned before exposure to alcohol, such as remote family events (Mendelson & LaDou, 1964) and factual information (Cattell, 1930) is not adversely influenced by alcohol. However, long-term memory for information learned after alcohol is administered is adversely affected (Kalin, 1964). Memory deficits are also present in chronic alcoholics and are especially pronounced in Korsakoff patients (Talland, 1965).

Recent evidence (Jones, 1972) suggests that memory deficits produced by alcohol are greater when the blood alcohol level is rising than when it is falling. The deficits in memory may account for performance on cognitive tasks being more adversely affected on the ascending limb of the blood alcohol curve. In 1943 Goldberg had reported finding performance on the Kraepelin subtraction test and the Bourdon cancellation task more impaired on the ascending limb of the blood alcohol curve than on the descending limb. Other studies have confirmed this finding on different psychological tasks. Eggleton (1941) found that performance on the typewriter, the dotting machine, and the 'distraction machine' were more impaired when the

blood alcohol level was rising than when it was falling. On several different measures taken by Ekman, Frankenhaeuser, Goldberg, Hagdahl and Myrsten (1964), nearly all curves obtained in the experiment showed a maximal effect on performance about 30 to 50 minutes after alcohol consumption (ascending limb). In a recent study by Young (1970), reaction time increased as the blood alcohol level increased on the ascending limb. On the descending limb, reaction time fell more rapidly than the blood alcohol concentration, perhaps as the result of adaptation. On the Number Facility Test (an addition test), the variable interval time analyzer task (a time estimation task) and also on the zero input tracking analyzer task (a perceptual motor task) administered by Sidell and Pless (1971), performance was poorest on all three tasks on the ascending limb. On the descending limb, cognitive ability returned to normal rather rapidly, while perceptual motor performance was poor for some time on the descending limb. Jones and Vega (1972) found performance on the Shipley-Hartford abstract scale to be poorer on the ascending limb than on the descending limb of the blood alcohol curve. To summarize briefly, most psychological functions seem to be most impaired during the rising phase of the blood alcohol curve. There is some evidence that cognitive functioning returns to normal on the descending limb before perceptual and motor functioning do. There is a possibility that the improvement that is seen on the descending limb is due to adaptation to the effects of alcohol. In the case of a repeated measures design, practice effects may account for improvement over time. Although subjects in this study were tested twice, they were only tested once under alcohol. Different groups of subjects were used to compare ascending and descending limb effects.

From the above paragraphs, several statements can be made. Alcohol affects memory. Alcohol impairs cognitive functioning. Impairment is usually more pronounced when the level of alcohol in the blood is rising than when it is dropping.

Concept identification (CI) has served as a useful paradigm for investigating information processing, attention, memory and cognitive deficits in relation to psychopathology (Pishkin & Blanchard, 1963; Pishkin & Burn, 1971; Pishkin, Shurley, & Wolfgang, 1967; Pishkin, Fishkin, & Stahl, 1972). In the typical concept identification paradigm, the subject is presented with a number of geometric stimuli that vary on dimensions such as shape, color, size and orientation. It is the task of the subject to learn to classify the stimuli according to certain rules (concepts) chosen in advance by the experimenter. The difficulty of the task can be quantitatively varied by the experimenter by changing the proportion of relevant dimensions in relation to the total number of relevant and irrelevant dimensions. A linear relationship between this proportion and problem solving efficiency has been found by many experimenters. Research in concept identification has been concentrated on the relationship of performance on concept identification tasks to irrelevant information (Archer, Bourne, & Brown, 1955), intertrial interval (Bourne, Dodd, Guy & Justensen, 1968), stimulus redundancy (Bourne & Haygood, 1959), completeness of feedback (Bourne & Pendleton, 1968), and misinformation feedback (Pishkin, 1960). Because stimulus dimensions can be precisely controlled, and performance can be quantified, the concept identification paradigm is a potentially valuable tool for measuring the acute effects

of alcohol on cognitive functioning and will be utilized in the proposed study.

The types of problems used in studying conceptual behavior have two main features, either or both of which may initially be unknown to the subject. First, there are the defining or relevant stimulus attributes which characterize the specific concept that the subject is to learn. Secondly, the rule which determines the function of or the relationship between the attributes may be unknown, and the subject may be required to identify it (Bourne & Guy, 1968). The most common type of problem studied to date has been the first type. Typically, the subject is given the rule in a set of instructions and is required to identify the relevant attribute or set of attributes.

Five types of conceptual rules are commonly studied: affirmation, conjunction, inclusive disjunction, conditional, and biconditional. The simplest type of rule is the unidimensional rule of affirmation, symbolically described by R (an abbreviation for red) and verbally described by the example, all red patterns are examples of the concept (Haygood & Bourne, 1965). The other four rules are bidimensional. The order of difficulty of the four bidimensional rules varies, depending on whether the first stimulus presented is a positive instance of the concept or a negative one. The sequence of difficulty obtained when the initial instance is positive is conjunction, biconditional, inclusive disjunction, and conditional (Laughlin, 1968; Giambra, 1969). If the initial instance is negative, the relative difficulty of the rules is significantly altered in such a manner that the conditional rule becomes the easiest, followed by the inclusive

disjunctive, and the conjunction, with the biconditional being the most difficult (Talpin, 1971). A different order based on the simple, familiarity hypothesis about differences between rules being due to differing familiarity with the rules and their appropriate strategies has also been described (Haygood & Bourne, 1965). Their results yield the following order of difficulty: conjunction, inclusive disjunction, conditional, and biconditional.

Of the possible types of rules that could be utilized in the following experiment, the disjunctive rule has been chosen for several reasons. A unidimensional rule does not require the subject to divide his attention and keep track of two or more attributes of the stimulus array at once. As mentioned earlier, little or no impairment in performance is seen on continuous attention tasks with moderate doses of alcohol. Of the bidimensional rules, the disjunctive ranks second or third in difficulty. Disjunctive concepts are defined by either one or the other or both values. Since positive instances may share no common values, negative instances usually contain more information of value to the subject, because attributes contained within them may be eliminated from the set of possible hypotheses about the solution of the problem (Conant & Trabasso, 1964). Available evidence indicates that as a rule of thumb positive instances are usually more effective than negative instances for providing information that is relevant for problem solution. For conjunctive problems, positive information plays a large part in solution. On the other hand, negative information plays a large part in the solution of disjunctive and conditional problems (Bourne & Guy, 1967). In order to solve a disjunctive problem,

the subject must focus on two stimulus attributes. To solve it most efficiently, he must also pay close attention to the negative as well as the positive instances of the concept.

Another consideration is that not only must a problem used to study alcohol effects be complex, it must also encompass a fairly limited time span. When testing ascending and descending limb effects, one actually tests within a blood alcohol range (in this case, 0.09% to 0.11%). Measuring ascending limb effects is touchy because the time course of absorption is relatively short (30 to 60 minutes to peak) compared with elimination (mean elimination rate in man is 0.015% per hour with a range of 0.006% to 0.04%). If a problem takes very long, the subject may reach peak and start descending during testing and confound results. Results from other studies show that it takes an average of between 14 and 50 trials to solve a disjunctive problem, depending on the instructions, criterion to solution, and the order of stimulus presentation (Haygood & Bourne, 1965; Lovallo, 1970; Taplin & Jeeves, 1972). Depending on the length of the intertrial interval, the subject could solve a disjunctive problem within a couple of minutes and certainly within a five minute period. Even if reaction time is longer for subjects after alcohol, and if it takes more trials than prior to alcohol intake for a subject to solve a problem, it shouldn't take the subject much longer than ten minutes. This would be a reasonable amount of time to complete a problem on the ascending limb.

An important issue related to acute doses of alcohol is what the effects of motivation and stress are upon task performance after administration of moderate doses of alcohol. Misinformation

feedback is a method available from concept identification work by which one can induce stress by manipulating the subject's antecedent set. It is a reliable finding that experimentally induced failure impairs subsequent problem solving (Solley & Stagner, 1956; Goodnow & Pettigrew, 1955; Pishkin, 1961; Feather, 1966; Feather & Saville, 1967). Failure set is usually induced on an initial problem by giving the subject an unsolvable problem (one involving 50% misinformation feedback). The amount of incorrect feedback may also vary between 0% and 50%. The greater percentage of misinformation feedback a subject is given, the poorer the performance and the greater the number of errors produced by the subject (Bourne, 1963). Misinformation feedback on a concept identification task has been used with students (Pishkin, 1960), hospital employees (Pishkin, 1965), psychiatric patients (Pishkin & Shurley, 1969), and chronic alcoholics (Pishkin, Fishkin, & Stahl, 1972). It has recently been used by Pishkin's group to compare the effects of drugs on CI [hydroxyzine (Wolfgang, Pishkin, & Bradshaw, 1963), doxepin and chlordiazepoxide (Unpublished)].

The performance of subjects after administration of alcohol in response to failure is of interest for several reasons. Some alcoholics appear to be failure oriented. Knight (1937) has differentiated between two basic types of alcoholics, the "essential" and the "reactive". He referred to the most common type as "essential alcohol addiction" and stressed the evidence of lifelong fixation at the oral-dependent level of personality functioning. The individuals in the second group, the "reactive alcohol addiction" group, had reached more mature

levels of personality development and had started drinking later in life in response to stressful circumstances. The "essentials" are individuals who have never really been successful people and appear to be oriented toward failure. Evidence presented by Apperson (1965) and Apperson and McAdoo (1965) indicates that when alcoholics were children, their successes were not rewarded by their parents. Thus, the roots of failure orientation may be traced back to the alcoholic's childhood. In a therapeutic situation, the alcoholic is seen as a "help rejector" and a poor treatment risk. In the experimental situation, he may interrupt a series of successful responses with a switch to an unsuccessful mode of responding (Tarter, 1971, Booth, 1969). Indeed, Menninger (1938) regards self-destructiveness, perhaps the greatest form of failure orientation, as a motivating factor in the etiology of alcoholism.

Analysis of reasons given for drinking and abstaining by alcoholics (Ludwig, 1972) reveals that the resumption of drinking was precipitated by failure in 43% of all cases. An individual with an "alcoholic personality" is characteristically unable to tolerate anxiety (Coopersmith, 1964). In the face of undesirable situations, he seeks rapid and undemanding solutions to his problems. Alcohol narcosis provides a way to avoid facing the tensions associated with problems. One of the goals of this study was to confront nonalcoholic individuals with a failure situation prior to drinking alcohol and measure the effect of the failure on subsequent performance on a concept identification task after alcohol consumption.

One way of measuring an individual's reaction to stressful situations such as failure is to use psychophysiological measures. Certain patterns of physiological activity have been correlated with successful performance on cognitive tasks. Pishkin and Shurley (1968), for example, found a positive correlation between CI errors and muscle action potential (MAP), and a negative correlation between spontaneous GSRs and MAP.

Several studies have assessed level of arousal in subjects after alcohol intake by recording psychophysiological activity. Results for galvanic skin response, skin conductance and muscle action potential, the three measures that were employed in the present study, will be discussed briefly. Basal skin conductance (BSC) is thought to reflect a person's chronic tension level, while galvanic skin response is thought to represent reactivity to emotionally arousing stimuli. BSC following low doses of wine (50 ml) was found by Carpenter (1957) to exceed the level observed following a low dose containing the same amount of alcohol plus water; while the reverse was true when the effects of a higher dose were compared. At higher dose levels (350 ml), the evoked GSRs were significantly smaller than baseline levels regardless of the form of the beverage. This study was replicated and a placebo group included in 1959 by McDonnell and Carpenter. Once again, large amounts of alcohol were seen to reduce GSRs by a substantial and significant amount. Lienert and Traxel (1959) also reported that 20 cc of 98% alcohol reduced the amplitude of GSRs elicited to verbal stimuli. Their conclusion was that alcohol is an antianxiety agent. Coopersmith (1964) found that the differences in responsiveness to high and low

affect words was smaller in subjects receiving alcohol. However, he also reported that the absolute GSR to both types of words was greater after 0.86 ml of 43% alcohol per kilogram. McDonnell and Beach (1968) found that the amplitude of a conditioned GSR was also reduced by alcohol. Although it cannot be conclusively stated from GSR data that alcohol reduces anxiety, the majority of studies with GSR and alcohol indicate that at moderate doses alcohol reduces GSR amplitude. Conflicting results may be due to limb effects.

Little work has been done on the effects of alcohol on muscle responses, and what has been done presents controversial results. Alcohol is classified as a depressant, and one would expect to find a reduction in muscle tension following alcohol intake. Results have been reported to the contrary. Doctor and Perkins (1961) reported that after subjects had received doses of 0.5 milliliters of alcohol per kilogram, they maintained the level of resting tonus which was established during the initial rest period. In contrast, Ss who had received no alcohol relaxed as the experiment progressed compared with their initial resting level. Further work needs to be done to clarify the effects of alcohol on EMG activity. The results of Doctor and Perkins might be explained by the fact that the effects of alcohol during the ascending limb phase are those of a stimulant. Subjects are happy and talkative and physiologically activated. Heart rate increases; EEG activation is seen; alcohol diuresis is present. In contrast, on the descending limb, subjects are tired and depressed; EEG returns to baseline; and heart rate begins to slow.

Statement of the Problem

The combined effects of failure and alcohol upon memory and performance on the ascending and descending limbs of the blood alcohol curve are of importance for several reasons. If, in fact, the effects of failure are found to be mitigated by alcohol, then some empirical insight into reasons for drinking may be gained. There are also practical aspects to such a study. Thousands are killed each year on the highways because of drunken drivers. In all probability, they are still on the ascending limb when they leave a party or their favorite drinking spot. Previous research indicates that greater impairment occurs in sensory, motor and cognitive functioning on the ascending limb. If a person gets behind the wheel on the ascending limb, he is in effect becoming more and more impaired as time goes by until he peaks. Even if a law enforcement officer stops such an individual, the individual may not have absorbed enough alcohol at that point to qualify as legally intoxicated. If he is turned loose before he reaches peak, he may go right on absorbing and ascending and becoming more impaired. However, if a person must "pass" a simple test before his car can be started, then he might not get on the road at all. If he does and is stopped, a battery of simple tests or perhaps just one test itself, could be administered to the suspect. If differential effects between ascending and descending limbs could be identified, then an arresting officer could detain the drinking driver until such a point on the descending limb that impairment no longer exists. Concept identification is an example of a simple cognitive task that might be used to establish impairment.

It is hypothesized that:

1) Performance on a concept identification task of normal subjects who receive alcohol will be impaired, following intake of a moderate dose of alcohol.

Since it is known from other studies that the effects of alcohol on cognitive functioning are dependent on both the level of alcohol in the blood and whether the subject is on the ascending or descending limb of the blood alcohol curve, both blood alcohol level and limb of the blood alcohol curve must be taken into account.

1a) It is hypothesized that performance on a concept identification problem will be impaired at a blood alcohol level of 0.09% on the ascending limb of the blood alcohol curve (absorption phase).

1b) It is hypothesized that performance on a concept identification problem will not be impaired at a blood alcohol level of 0.09% on the descending limb of the blood alcohol curve (elimination phase).

1c) It is further hypothesized that performance on a concept identification problem will be more impaired at 0.09% on the ascending limb of the blood alcohol curve than at 0.09% on the descending limb of the blood alcohol curve.

The effects of failure on an initial task have been shown to produce striking decrements on later performance of the same or a similar task. However, the failure set that is to be induced in this study must also take into account state-dependent effects.

2) It is hypothesized that prior failure will produce impairment in performance on a concept identification task.

2a) It is hypothesized that subjects in the placebo group

will be more impaired by the failure set in Phase II relative to the subjects in the success set group.

2b) It is hypothesized that subjects in the alcohol group will be more impaired by the failure set relative to the subjects in the alcohol group in the success set group.

2c) It is also hypothesized that due to the limb effects of alcohol, the failure set will have a greater impact on subjects tested on the ascending limb than on subjects tested on the descending limb.

2d) It is hypothesized that failure under the alcohol condition will have a greater effect on the subjects than failure in the placebo condition.

Several predictions can also be made for the psychophysiological data. Predictions are made on the basis of the widely held theory that alcohol reduces anxiety and tension in humans.

3) It is hypothesized that alcohol will affect electromyographic activity.

3a) On the ascending limb there will be little change in EMG activity. There is, if anything, a slight arousal effect seen in psychophysiological activity on the ascending limb.

3b) On the descending limb, it is hypothesized that there will be a depression of EMG activity in line with the findings that alcohol shows depressant effects on the descending limb.

3c) For those not receiving alcohol, but receiving failure set, EMG activity will increase over time during CI performance.

3d) For those receiving both alcohol and failure set, EMG levels will be not as great as but in the same direction as those receiving a success set.

In regard to SCR activity several hypotheses are also made.

4) It is hypothesized that alcohol will depress SCR activity.

4a) It is hypothesized that a greater depression of SCR activity will occur on the descending limb than on the ascending limb.

4b) Significant increases in SCR will be seen during the concept identification task compared to the rest period.

In summary, this study will test the hypotheses that both alcohol and set will affect performance on a cognitive task. It is expected that greater impairment will be seen on the ascending limb than on the descending limb. It is also predicted that alcohol will reduce tension, a by-product of the failure set, as measured by psychophysiological indices.

CHAPTER II

METHOD

Subjects

Subjects were 40 normal healthy male volunteers, ranging in age from 21 to 35. They were paid $10 each for their participation. They were graduate students and law students from the University of Oklahoma and Oklahoma City University. They were light to moderate social drinkers, and a record was kept of the drinking habits of each subject. At the time each subject signed up for the study, he was told not to drink alcohol, take medications, drugs, or stimulants the night before testing. He was also told to get a regular night's sleep the night before. He was instructed to eat a light meal four hours before testing, but to refrain from eating or drinking anything after that.

Design

The experimental design was a 2 X 2 X 2 factorial design with two drug levels (placebo and alcohol), two misinformation feedback conditions (0% misinformation feedback and 50% misinformation feedback) and two points on the blood alcohol concentration curve (0.09% on the ascending limb and 0.09% on the descending limb) with subjects being randomly assigned to one of the eight cells. The eight equal groups each contained five subjects.

Procedure

When the subject arrived at the laboratory on the day of the study, he was told that the experiment involved alcohol, concept identification, and psychophysiological measures. Then the steps outlined in Table 1 were followed. Each subject first signed a consent form and was weighed. He also completed a personal data sheet containing questions about his individual drinking history such as age of onset of drinking, amount and type of alcohol presently consumed, and frequency of drinking.

Skin surface electrodes were then applied. Two Grass electrodes were attached to the subject's forehead in a standard frontalis muscle placement (one inch up and one inch over from the bridge of the nose) to measure frontalis electromyographic activity (EMG) (Davis, 1952). Two Grass electrodes were also attached to the throat to measure laryngeal EMG. Two Beckman silver silver-chloride skin electrodes were placed on the volar surfaces of the distal phalanx of the first and second fingers of the non-dominant hand (Lykken & Venables, 1971) for measurement of both tonic skin conductance (SCL) and GSR. The ground electrode was applied to the right forearm on the bony prominance near the elbow bend. An 8-channel Beckman Type-R Offner Dynograph paired with an Ampex FM tape recorder was used for recording the psychophysiological data. The electrode hookup was followed by a five-minute rest period to establish each subject's baseline level for EMG, SCL, and GSR.

After the electrode hookup and rest period, each subject then completed both the vocabulary test and abstraction test of the Shipley Institute of Living Scale in order to obtain a baseline level for

TABLE 1

M E T H O D

TESTING ORDER

A. Control Period

1. Sign consent form, weigh, and complete personal data sheet
2. Psychophysiological hookup and rest period (5 mins.)
3. Complete Shipley vocabulary (10 mins.) and abstract (10 mins.)
4. Complete Eysenck Personality Inventory (Form A)
5. Complete Concept Identification Phase I
6. Practice breath before drinking

B. Ascending Limb of the Blood Alcohol Curve

7. Begin Drinking (15 mins.)
8. First Breath (usually invalid)
9. Second Breath (usually valid)
10. Third Breath
11. Fourth Breath
12. Psychophysiological Rest Period (5 mins.) or Concept Identification Phase II
13. Fifth Breath
14. Sixth Breath

C. Descending Limb

15. Seventh Breath
16. Eighth Breath
17. Ninth Breath
18. Psychophysiological Rest Period (5 mins.) or Concept Identification Phase II
19. Tenth Breath

intellectual functioning. These scores can be converted to WAIS-equivalent IQ scores using the tables developed by Paulson and Lin (1970). The Eysenck Personality Inventory (Form A) was also administered to determine baseline levels of the personality variables of extroversion and neuroticism that may interact with alcohol and account for differences in performance between groups. Analyses of variance were performed by Shipley and Eysenck scores to rule out significant differences due to these variables. The groups did not significantly differ on these scores.

After the initial forms, tests and rest period had been completed, Phase I of the concept identification task began and the first problem was presented to the subject. The subject was required to classify a series of stimulus slides containing geometrical designs into two categories, the positive and negative instances of the concept using a disjunctive rule. The stimulus population of 2 X 2 inch, 35 mm. color slides varied along four dimensions, each containing three levels or attributes. They were color (red, yellow, and blue), size (small, medium, and large), shape (square, hexagon, and circle), and number (one, two, or three objects). The total stimulus population included $3^4 = 81$ stimuli.

Before going on to discuss the exact proportions of the composition of the stimulus population, a few points will be discussed about the logic behind the composition. Recent work (Ekstrand, Wallace, & Underwood, 1966) has led to the formation of a frequency theory of verbal discrimination learning. The basic assumption of their theory is that Ss can discriminate subjective differences in the frequencies

of presentation of any two items x and y. The subject is then able to discriminate correct from incorrent items in the verbal discrimination lists. In a concept identification task, discriminations are required and each attribute is presented a certain number of times in relation to other attributes. At least two discriminations are required of the S. He must discriminate between positive and negative stimuli to perform the task successfully. He must also discriminate between relevant and irrelevant stimuli in order to perform correctly. These discriminations lead to solution of the problem.

The natural population of stimuli has a composition bearing the ratio 1:2:2:4 between the TT (both characteristics are present and are positive examples of the concept), TF (the first characteristic is present and is a positive example of the concept; the second is not), FT (the first characteristic is not a positive example of the concept, but the second is), and FF (neither of the characteristics that are examples of the concept are present) classes respectively. The natural population would contain 1 out of 9 TTs, 2 out of nine TFs, 2 out of 9 FTs, and 4 out of 9 FFs. In the case of the disjunctive problems, the natural population yields a 5:4 ratio between positive and negative instances. To compensate for this, slight alterations in the usual ratio were made so that Ss were presented with an equal number of positives and negatives. In a group of 81 stimuli, the natural population contains unequal numbers of stimuli in each category. There are 9 TTs, 18 TFs, 18 FTs, and 36 FFs. If this is altered to 14 TTs, 13 TFs, 13 FTs, and 40 FFs, then there will be an equal number of positive (TT + TF + FT = 40) and negative (FF = 40) instances, and each positive truth table

category will be almost equally represented.

To balance for attribute salience, two sets of relevant attributes were used. The solution for the first problem was one-yellow and for the second problem was small-square. Thus, two attributes from each of the four dimensions was represented in the two solutions. Also, a highly salient attribute was paired with one of low salience. Color and shape are equal in salience as are number and size (Clayton, Merryman & Leonard, 1969; Fishbein, Haygood, & Frieson, 1970).

The stimulus patterns were rear-projected with a 35 mm. Kodak Carousel projector one at a time onto a viewing screen placed directly in front of the subject. A small response box containing two buttons and two feedback lights was placed in front of the subject. The two buttons were labeled "Yes" (positive) and "No" (negative) to indicate whether a pattern could be classified as an example or nonexample of the concept, respectively. If the subject's response was correct, the light came on. If his guess was incorrect the light above the opposite button came on. During Phase I the subjects in the success set received correct feedback 100% of the time. Subjects in the failure set received correct feedback only 50% of the time. The pattern of stimulus presentation was random with the exception that runs of the same category (positive or negative instances) will be limited to five. Misinformation feedback also occurred randomly.

Subjects were allowed as much time as they wished to respond. The subject's responses, the correct responses, and the subject's response time for each trial were recorded by a Hewlett-Packard Digital Recorder Model Number 562A. The sequence of timing for the slides and

feedback for each trial was controlled by a system of BRS Foringer solid-state logic units.

The subject was instructed in detail about the nature of the stimuli, concepts in general, and the disjunctive rule (see Appendix A for exact instructions). It was explained that the experimental task was to identify the two relevant attributes by attempting to choose correctly the positive stimulus on each trial. The test trials then began and continued until the S met a criterion of 16 correct consecutive responses or until trial 160 was reached without solution.

At the termination of the first problem, the subject began to drink. Blood alcohol levels were analyzed by means of repeated breath samples using the Stephenson Model 700 Breathalyzer. Instructions were given to the subjects on the use of the Breathalyzer. A practice breath was taken before drinking and all Ss registered an initial blood alcohol level (BAL) of 0.00%. Alcohol subjects received 1.32 milliliters of 95% USP ethanol per kilogram of body weight mixed with four parts orange juice. This dose is calculated to produce a peak blood alcohol level of 0.11% (Jones & Vega, 1972). Placebo subjects received the same amount of liquid as alcohol subjects but received only five milliliters of alcohol floated on top of the juice to mask the fact that they were not receiving a large dose of alcohol. Subjects were also told that they would receive one of several doses of alcohol. Subjects were given fifteen minutes to consume their drinks. Drinking time was controlled by giving one-third of the total amount every five minutes. Subjects were asked to rinse their mouths immediately after they finished drinking to clear their mouths of any residual alcohol.

Repeated breath samples were taken at intervals of approximately seven minutes until the subject reached a blood alcohol level of 0.08%, which was usually 30 to 45 minutes after drinking. Those subjects in the ascending limb group and their placebo controls were tested again (Phase II of the concept identification task) in the 0.08% to 0.10% range. The subjects and their placebo controls who were to be tested on the descending limb were tested after they had peaked and had two readings at 0.09% and it was established that they were descending. Blood alcohol levels were charted for each subjects. Placebo controls failed to produce readings on the Breathalyzer.

The concept identification problem procedure in Phase II was identical to the problem described for Phase I with the exception that no misinformation feedback was given. The problem solution was changed. The relevant attributes were small-square. Slides were randomized again so that the order of presentation of the slides was not the same. Physiological recordings were made during the period after drinking, during testing and after testing. A schematic of the study is presented in Figure 1.

Statistical Analysis

An analysis of variance approach was taken. Certain of the specific comparisons of the study were made within the same subject and certain were made across different subjects. The influence of alcohol was studied across two independent groups as were the effects of feedback and the effects of limb of the alcohol absorption curve. The effects of the different time periods upon responses were investigated within subjects.

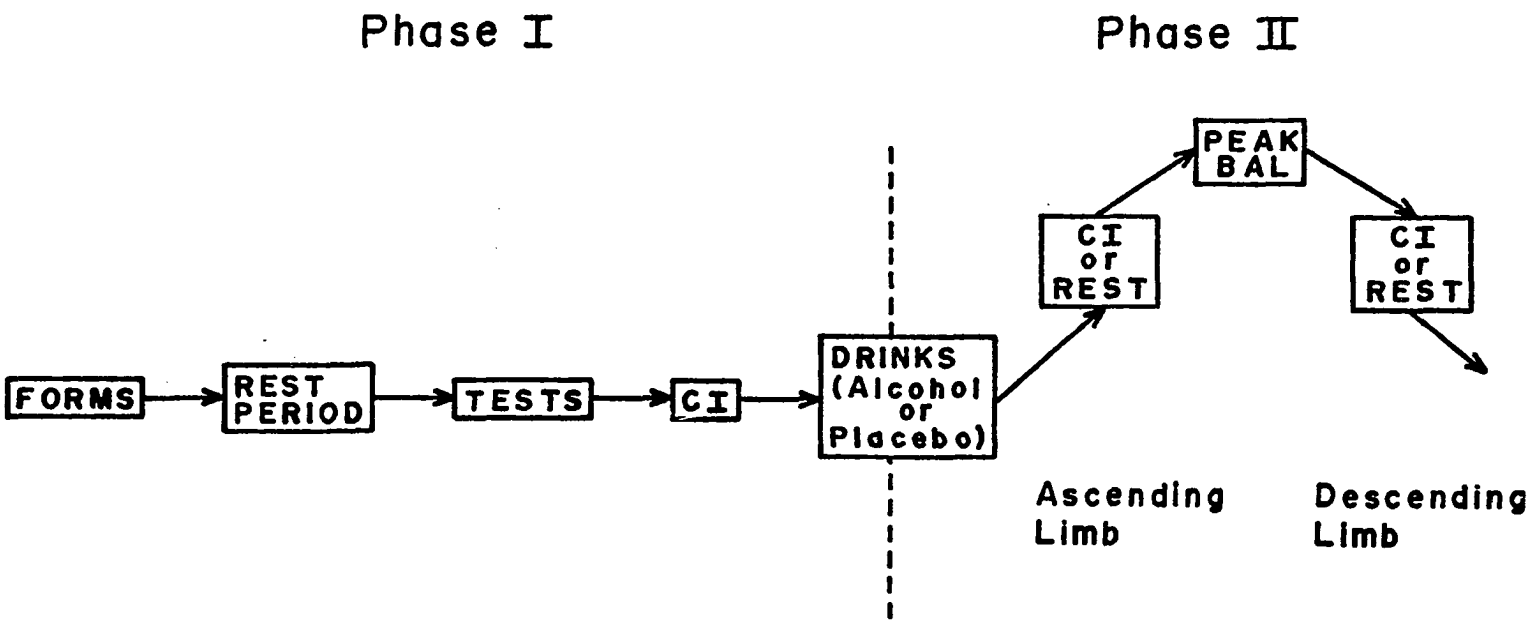

Fig. 1 - Schematic of the study.

CHAPTER III

RESULTS

This section is divided into two parts. The results of the concept identification task are presented first. Then, the results from the psychophysiological analyses follow.

Concept Identification Data

Four measures were used for analysis of the concept identification data. They were trials to criterion, errors to criterion, percentage of errors for number of trials to criterion, and mean response time. Phase I data were omitted from the analyses presented here for two reasons. Firstly, the Phase I concept identification task was the set-inducing task and produced artificial results. Half of the subjects automatically received a score of 160 trials because they received an unsolvable problem (failure set). Secondly, Phase I is traditionally excluded from analysis. Therefore, the analyses presented herein include only Phase II test data. A t test for number of trials to criterion and number of errors to criterion showed that there were no differences for Phase I between alcohol and placebo groups (t = 0.703 and t = 0.640 respectively).

Trial and error scores were both tested for normality and homogeneity of variance. These tests were performed for raw data,

square root transforms, and log transforms. Trial data showed a bimodal distribution. Criterion for solution of the problem was either 16 correct consecutive responses or a total of 160 trials reached without solution. Of the 40 Ss, nine were nonsolvers (two received the alcohol-success treatment, four the alcohol-failure combination, and three the placebo-success treatment). All nonsolvers received a score of 160. The nonsolvers were responsible for the bimodality of the trials data. The means and standard deviations for trials to criterion appear on Table 2. Fmax for trials was not great enough to reject homogeneity of variance ($F = 1.241$). The most appropriate data transform for the trials to criterion data was the log transform. The ANOVA summary appears on Table 3. The alcohol group had significantly more trials to criterion than did the placebo group ($F = 4.249$, $p < .05$). The other treatments and interactions were not significant.

Data for errors to solution showed a positive skew. Means and standard deviations for errors to criterion appear in Table 4. Fmax for errors was not great enough to reject homogeneity of variance ($F = 4.776$). The most appropriate data transform for errors to criterion was the log transform. Table 5 shows a summary of the analysis of variance for errors to criterion. A highly reliable difference was found between the alcohol and placebo groups ($F = 10.366$, $p < .01$) with the alcohol group having more errors. None of the other treatments or interactions produced significant results.

An error rate measure was obtained by dividing the number of errors by the total number of trials each subject had. This procedure

TABLE 2

MEANS AND STANDARD DEVIATIONS FOR TRIALS TO CRITERION ON THE CONCEPT IDENTIFICATION TASK

Group	Mean	S.D.
Drug		
Alcohol	99.2	46.2
Placebo	74.1	43.8
Set		
Success	95.1	43.9
Failure	78.3	48.9
Limb		
Ascending	97.0	47.1
Descending	76.3	44.1

TABLE 3

SUMMARY TABLE FOR ANALYSIS OF VARIANCE
FOR TRIALS TO CRITERION

Source	df	MS	F
Drug	1	0.2132	4.246*
Set	1	0.1196	2.386
Limb	1	0.1366	2.723
Drug x Set	1	0.0057	1.131
Drug x Limb	1	0.0063	1.256
Set x Limb	1	0.0039	0.769
Drug x Set x Limb	1	0.1440	2.869
Error (Within)	32	0.0050	
Total	39		

*$p < .05$

TABLE 4

MEANS AND STANDARD DEVIATIONS FOR ERRORS TO CRITERION ON THE CONCEPT IDENTIFICATION TASK

Group	Mean	S.D.
Drug		
Alcohol	39.6	27.1
Placebo	18.5	12.4
Set		
Success	30.2	20.6
Failure	27.9	26.4
Limb		
Ascending	33.0	24.2
Descending	25.1	22.5

TABLE 5

SUMMARY TABLE FOR ANALYSIS OF VARIANCE
FOR ERRORS TO CRITERION

Source	df	MS	F
Drug	1	1.1003	10.366**
Set	1	0.0079	0.743
Limb	1	0.2382	2.244
Drug x Set	1	0.1017	0.958
Drug x Limb	1	0.0042	0.396
Set x Limb	1	0.0019	0.185
Drug x Set x Limb	1	0.2892	2.725
Error (Within)	32	0.1061	
Total	39		

** $p<.01$

yielded a percentage of errors for each subject. The means and standard deviations for the various treatment groups appear in Table 6. The results from the analysis of variance for percentage of errors are presented in Table 7. A highly reliable difference was detected for the drug condition ($F = 20.987$, $p < .01$) with the alcohol condition showing a higher percentage of errors. There were no significant results for set or limb or for any of the interactions.

For each trial, the amount of time between the presentation of the stimulus and the key press response was recorded by a Hewlett Packard counter and printer. A mean response time was obtained later by averaging all of a subject's response times for a given problem. An analysis of variance was performed on mean response time; however, no significant results were obtained from the ANOVA. The means and standard deviations for mean response time data appear in Table 8. The ANOVA table and all other nonsignificant ANOVA tables are included in Appendix B.

Review of Concept Identification Results

Results of this dissertation indicate that performance on a concept identification task was impaired following alcohol consumption (Hypothesis 1). Mean number of trials and errors to criterion and percentage of errors for the number of trials to criterion were significantly greater for the alcohol group than for the placebo group. Performance of alcohol subjects compared with placebo subjects was impaired at a blood alcohol level of 0.09% on both the ascending and descending limb of the blood alcohol curve (Hypothesis 1a and 1b) for

TABLE 6

MEANS AND STANDARD DEVIATIONS FOR PERCENTAGE OF ERRORS TO CRITERION ON THE CONCEPT IDENTIFICATION TASK

Group	Mean	S.D.
Drug		
Alcohol	36.59	10.29
Placebo	23.82	6.05
Set		
Success	29.41	9.70
Failure	31.00	11.50
Limb		
Ascending	31.01	9.87
Descending	29.40	11.35

TABLE 7

SUMMARY TABLE FOR ANALYSIS OF VARIANCE
FOR PERCENTAGE OF ERRORS TO CRITERION

Source	df	MS	F
Drug	1	0.1631	20.987**
Set	1	0.0026	0.333
Limb	1	0.0026	0.338
Drug x Set	1	0.0090	1.151
Drug x Limb	1	0.0001	0.005
Set x Limb	1	0.0001	0.014
Drug x Set x Limb	1	0.0064	0.821
Error (Within)	32	0.0078	
Total	39		

** $p<.01$

TABLE 8

MEANS AND STANDARD DEVIATIONS FOR MEAN RESPONSE TIME (IN SECONDS) ON THE CONCEPT IDENTIFICATION TASK

Group	Mean	S.D.
Drug		
Alcohol	2.8818	1.3109
Placebo	2.9485	1.6388
Set		
Success	2.5509	1.4178
Failure	3.2794	1.4550
Limb		
Ascending	2.9179	1.2053
Descending	2.9124	1.7186

these three measures. There were no significant drug effects for either trials to criterion or response time, although there was a trend for mean number of trials to be greater after alcohol (99.2 trials) than after placebo (74.1 trials) and mean response time to be slightly shorter after alcohol (2.882 seconds) than after placebo (2.949 seconds).

It was hypothesized that performance on the ascending limb would be significantly poorer than performance on the descending limb, but this hypothesis was not borne out by the data (Hypothesis 1c). There were no significant limb effects, but there were trends in all measures for poorer performance on the ascending limb than on the descending limb. There was a trend for mean number of trials to criterion to be greater on the ascending limb (97.0 trials) than on the descending limb (76.3 trials), for mean number of errors to criterion to be greater on the ascending limb (33.0 errors) than on the descending limb (25.1 errors), for mean percentage of errors to be slightly greater on the ascending limb (31.0) than on the descending limb (29.4) and for mean reaction time to be a few milliseconds greater on the ascending limb (2.918 seconds) than on the descending limb (2.912 seconds).

Prior failure did not produce significantly different impairment in performance on the concept identification task (Hypothesis 2 had predicted that it would). There were no significant differences between alcohol and placebo subjects (Hypothesis 2a) or success and failure set (Hypothesis 2b and 2d) or ascending and descending limbs (Hypothesis 2c). There was a trend for mean number of trials to criterion to be greater

after success set (95.1 trials) than after failure set (78.3 trials), for mean number of errors to be slightly greater after success set (30.2 errors) than after failure set (27.9 errors), for mean percentage of errors to trials to be slightly less after success set (29.4) than after failure set (31.0), and for mean response time to be faster after success set (2.551 seconds) than after failure set (3.279 seconds).

Psychophysiological Data

Psychophysiological data consisted of electromyographic (EMG), basal skin conductance (BSC), and skin conductance response (SCR) data. Both frontal and laryngeal EMG data were recorded and analyzed. SCR data were broken down into number of responses and amplitude of responses. The first minute and the last minute of the initial rest period were scored as were the first and last minute of Phase I of the concept identification task, the first and last minute of the ascending limb measurement period (Phase II CI task for one-half of the Ss and the second rest period for the other one-half of the Ss) and the descending limb measurement period (Phase II CI task for the Ss who had a rest period on the ascending limb and a rest period for those who had CI on the ascending limb). Results for both the first and last minute analyses, as well as for the mean of the two minutes, were almost identical, so only the results from the last minute analyses will be presented at this time. In any case where the data appeared to need transforming, both log transforms and square root transforms were performed. Results were the same for the transforms and the raw data, and so only the results from the raw data will be presented here.

To determine the nature of the alcohol effects, a series of five ANOVAs was performed on each set of data. The first was a comparison of the scores obtained from subjects who rested on the ascending limb of the blood alcohol curve with the scores obtained from the subjects who rested on the descending limb. The second compared scores from the Phase II concept identification task obtained on the ascending limb with those obtained on the descending limb. The third set of analyses compared the psychophysiological data of the subjects who received the Phase II concept identification task on the ascending limb with the data of those who received a rest period during that same time period. Descending limb data were analyzed in the fourth set of ANOVAs. The fifth set of ANOVAs compared the score of each subject on the ascending limb with his score on the descending limb and also took into account whether he had received the CI task first or the rest period first. There were several reasons for performing the various sets of analyses rather than just doing one large ANOVA for each set of data. The first four sets were three-way ANOVAs that provided a relatively simple and conceptually manageable means of analyzing many of the effects of interest. The fifth set, four-way ANOVAs with repeated measures, was included for the purpose of getting a more sensitive look at limb effects. In the fifth set of analyses, the limb effect is a repeated measure variable with a potentially smaller error term. It was felt that this analysis might yield effects that could not be detected by three-way ANOVAs alone. The larger analyses alone are rather complicated with certain effects appearing as higher order interactions and therefore are

conceptually hard to handle. The results of these analyses are presented below for each psychophysiological measure.

Electromyographic Data

Both the frontal and the laryngeal EMG activity were recorded on magnetic tape during the study. The tapes were later run through an integrator that summated all of the electromyographic activity that occurred during any given one-minute period and printed out the total for the minute. Analyses were performed on the integrated print-out.

Frontal EMG. Frontal EMG data yielded the following results. On the analysis of variance for frontal EMG measured during rest periods, there was a significant drug effect ($F = 4.958$, $p<.05$) with the alcohol condition showing a higher level of activity. None of the other main effects or interactions were significant (See Tables 9 and 10). The ANOVA for frontal EMG measured during CI periods yielded no significant results. The means and standard deviations from that analysis are presented in Table 11.

The ANOVA for the comparison of frontal EMG measured during CI with that measured during rest for the ascending limb was also non-significant; however, the analysis for the descending limb during rest had several significant items. Ascending limb data appear on Table 12, and descending limb data are presented in Tables 13 and 14. The drug groups were significantly different ($F = 5.294$, $p<.05$) as were the Drug x Set (DxS) interaction ($F = 4.695$, $p<.05$) and the Set x Period (SxP) interaction ($F = 6.518$, $p<.05$). Simple effects were analyzed for the significant interactions. The DxS interaction is depicted in Figure 2.

TABLE 9

MEANS AND STANDARD DEVIATIONS FOR FRONTAL EMG MEASURED DURING REST PERIODS

Group	Mean	S.D.
Drug		
Alcohol	182.70	83.74
Placebo	135.85	48.66
Set		
Success	174.45	74.93
Failure	144.15	66.63
Limb		
Ascending	167.30	68.65
Descending	151.30	75.44

TABLE 10

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR FRONTAL EMG MEASURED DURING REST PERIODS

Source	df	MS	F
Drug	1	21996.090	4.958*
Set	1	9180.898	2.070
Limb	1	2560.000	0.577
Drug x Set	1	9859.597	2.222
Drug x Limb	1	792.100	0.179
Set x Limb	1	8584.898	1.935
Drug x Set x Limb	1	5290.000	1.192
Error (Within)	32	4436.140	
Total	39		

* $p<.05$

TABLE 11

MEANS AND STANDARD DEVIATIONS FOR FRONTAL EMG MEASURED DURING CI PERIODS

Group	Mean	S.D.
Drug		
Alcohol	175.80	56.52
Placebo	160.30	66.49
Set		
Success	156.50	54.30
Failure	179.60	67.18
Limb		
Ascending	169.70	65.46
Descending	166.40	58.75

TABLE 12

MEANS AND STANDARD DEVIATIONS FOR FRONTAL EMG MEASURED DURING CI AND REST PERIODS ON THE ASCENDING LIMB

Group	Mean	S.D.
Drug		
Alcohol	178.25	70.01
Placebo	159.75	62.43
Set		
Success	166.05	60.96
Failure	170.90	72.54
Period		
CI	169.70	65.46
Rest	167.30	68.65

TABLE 13

MEANS AND STANDARD DEVIATIONS FOR FRONTAL EMG MEASURED DURING CI AND REST PERIODS ON THE DESCENDING LIMB

Group	Mean	S.D.
Drug		
Alcohol	180.3	72.99
Placebo	137.4	54.45
Set		
Success	164.85	70.75
Failure	152.85	64.65
Period		
CI	166.40	58.75
Rest	151.30	75.44

TABLE 14

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR FRONTAL EMG MEASURED DURING CI AND REST PERIODS ON THE DESCENDING LIMB

Source	df	MS	F
Drug	1	18404.090	5.294*
Set	1	1440.000	0.414
Period	1	2280.099	0.656
Drug x Set	1	16321.590	4.695*
Drug x Period	1	1664.099	0.470
Set x Period	1	22657.590	6.518*
Drug x Set x Period	1	1960.000	0.564
Error (Within)	32	3476.357	
Total	39		

*$p<.05$

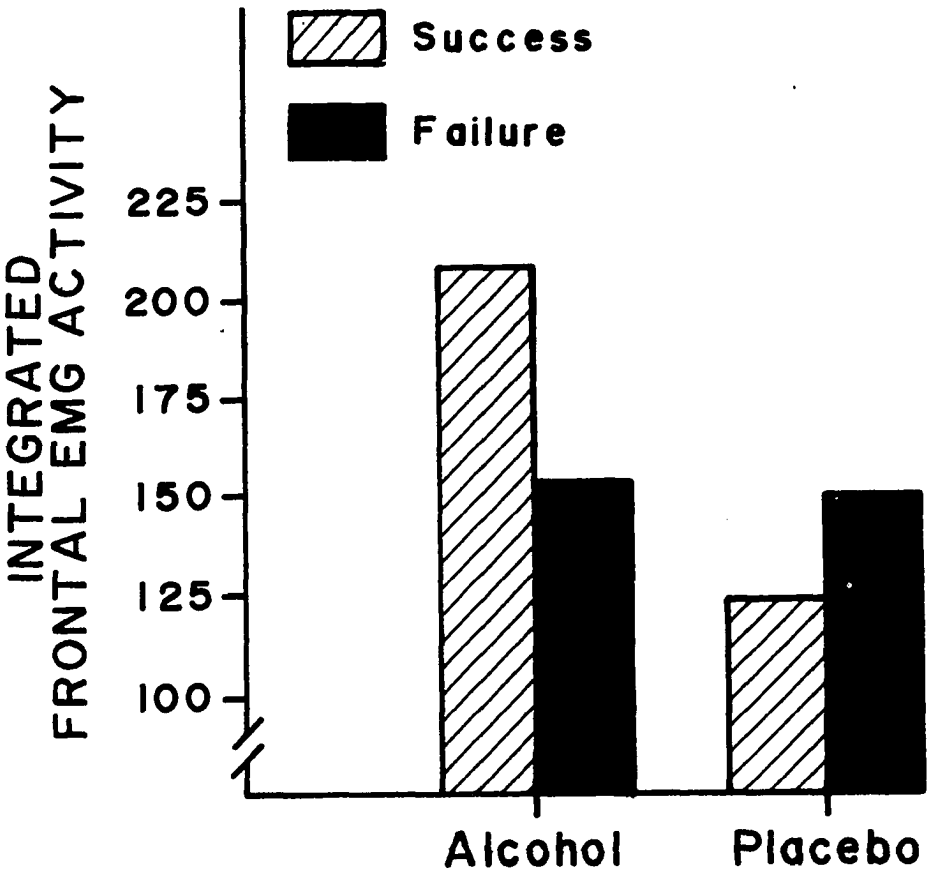

Fig. 2 - Mean frontal EMG activity for each drug group shown separately for success and failure set for the descending limb (Drug x Set interaction).

The success group had a significantly higher level of EMG activity than did the failure group for the alcohol condition ($F = 9.980$, $p < .01$). There was very little difference between the alcohol and placebo groups for failure set. The failure group had a higher level of activity than did the success group for the placebo condition, but the difference was not significant ($F = 3.949$, $p < .05$). The interaction between set and period appears in Figure 3. Success versus failure conditions were not significantly different during the CI task, but subjects in the success group had significantly higher levels of frontal EMG activity during rest periods than did subjects in the failure group ($F = 4.624$, $p < .05$). Levels of frontal EMG activity increased from levels present during CI when Ss rested for those who had received the success set. This increase was not significant. For the failure set, the reverse was true. The failure group showed a significantly higher level of frontal EMG activity during the CI task than during rest ($F = 5.109$, $p < .05$).

For the four-way analysis that compared ascending versus descending limb and order effects, there were two significant triple interactions. They were the drug by set by limb interaction ($F = 5.178$, $p < .05$) and the set by order by limb interaction ($F = 5.357$, $p < .05$). (See Table 15 for means and standard deviations, Table 16 for the ANOVA, and Figures 4 and 5 for illustrations of the interactions). Simple simple effects were analyzed for both of the significant three-factor interactions. For the drug by limb by set interaction, the alcohol group had a significantly higher level of activity for the descending limb by success condition than did the placebo group ($F = 8.263$, $p < .01$). The drug by limb interaction for the success group was significant

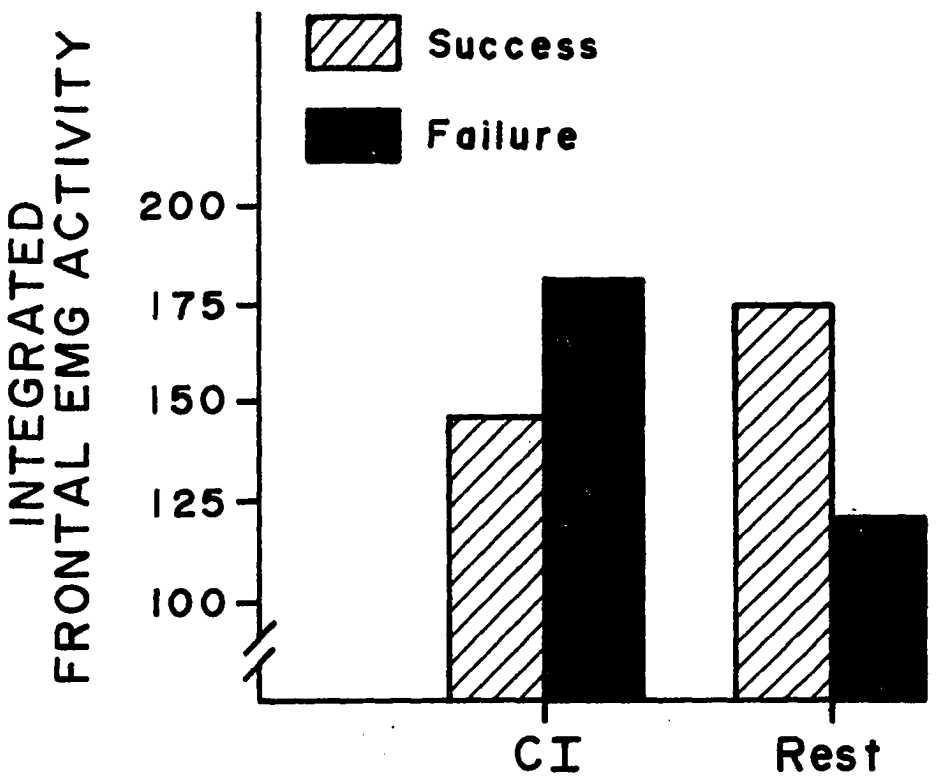

Fig. 3 - Mean frontal EMG activity for each period shown separately for success and failure set for the descending limb (Set x Period interaction).

TABLE 15

MEANS AND STANDARD DEVIATIONS FOR FRONTAL EMG MEASURED ON THE ASCENDING LIMB VS. THE DESCENDING LIMB DURING BOTH CI AND REST PERIODS

Group	Mean	S.D.
Drug		
Alcohol	179.28	70.60
Placebo	148.08	58.00
Set		
Success	165.48	65.22
Failure	161.88	68.44
Order		
CI 1st	160.50	70.33
Rest 1st	166.85	63.07
Limb		
Ascending	168.50	66.22
Descending	158.85	67.17

TABLE 16

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR FRONTAL EMG MEASURED ON THE ASCENDING VS. THE DESCENDING LIMB DURING BOTH CI AND REST PERIODS

Source	df	MS	F
Drug	1	19468.790	3.393
Set	1	259.200	0.045
Order	1	806.450	0.141
Drug x Set	1	4004.500	0.698
Drug x Order	1	156.800	0.027
Set x Order	1	8736.199	1.523
Drug x Set x Order	1	211.250	0.037
Error (Between)	32	5736.566	
Limb	1	1862.499	0.700
Drug x Limb	1	2737.799	1.029
Set x Limb	1	1411.199	0.530
Order x Limb	1	1531.250	0.575
Drug x Set x Limb	1	13781.250	5.178*
Drug x Order x Limb	1	4929.796	1.852
Set x Order x Limb	1	14257.790	5.357*
Drug x Set x Order x Limb	1	5951.250	2.236
Error (Within)	32	2661.000	
Total	79		

* $p < .05$

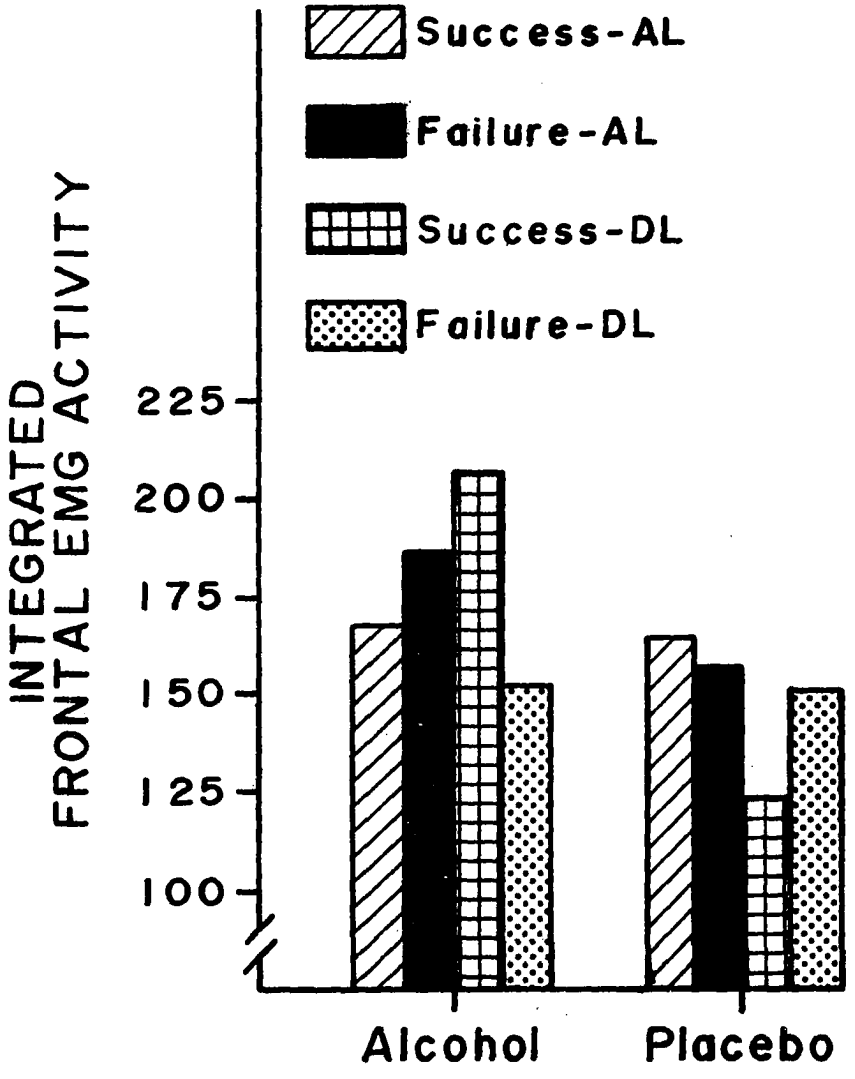

Fig. 4 - Mean frontal EMG activity for each drug group shown separately for success and failure set and ascending and descending limb (Drug x Set x Limb interaction).

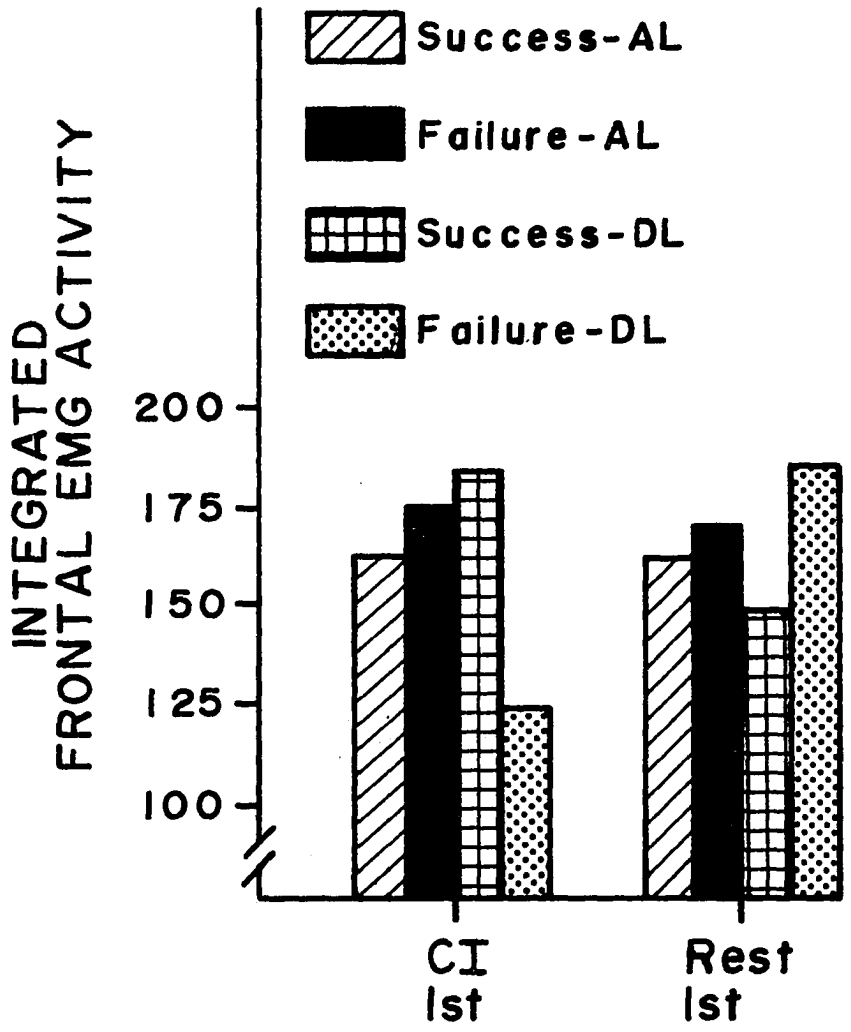

Fig. 5 - Mean frontal EMG activity for each period shown separately for success and failure set and ascending and descending limb (Set x Order x Limb interaction).

(F = 5.412, $p<.05$) as was the same interaction for the failure group (F = 11.274, $p<.01$). Subjects who had success set and who were measured on the descending limb showed considerably higher frontal EMG activity when under the influence of alcohol than when not given alcohol. With other combinations of the set and limb variable, this difference in EMG as a function of drinking or not drinking alcohol was much less marked. The limb by set interaction for the alcohol condition was also significant (F = 5.577, $p<.05$).

Looking at the set by order by limb interaction (Figure 5), the most striking result is that the subjects who had a rest period first had a significantly higher level of frontal EMG activity for the descending limb by failure condition than the subjects who had CI first (F = 4.681, $p<.05$). For the CI first by descending limb condition, the subjects in the success group had a significantly higher level of frontal EMG activity than did the subjects in the failure group (F = 4.230, $p<.05$). The order by limb interaction was significant for the failure condition but not for the success condition (F = 7.192, $p<.05$). The order by set interaction was not significant for the ascending limb, but it was significant for the descending limb (F = 5.396, $p<.05$). The limb by set interactions were significant for both of the order conditions (CI first--F = 4.630, $p<.05$; rest first--F = 10.050, $p<.01$).

Frontal EMG activity theoretically is illustrative of general overall arousal. In this study alcohol produced a significant increase in this activity during rest periods and across the concept identification task and the rest period on the descending limb. Frontal EMG was not significantly greater for CI or ascending limb analysis.

Laryngeal EMG. Whereas frontal EMG can be thought to represent general tonic arousal level, laryngeal EMG is an indicator of phasic activity such as subvocal activity or swallowing that may go on during cognitive activity. For this reason, both were included for analysis in this study. Laryngeal EMG did not show the significant differences during rest periods (see Table 17) that frontal EMG did, but did show a very significant increase after alcohol during the CI task ($F = 7.598$, $p<.01$). The means and standard deviations are presented in Table 18 and the ANOVA is summarized on Table 19. No significant differences were found for the ascending limb (Table 20). For the descending limb there was a significant drug effect ($F = 5.898$, $p<.05$) and a significant drug by set by period interaction which is depicted in Figure 6 (see Tables 21 and 22 for means and ANOVA). Simple effects analysis of the drug by set by period interaction indicated a significantly higher level of laryngeal EMG for the alcohol group compared with the placebo group for the success by rest condition ($F = 4.455$, $p<.05$) and for the failure by CI condition ($F = 5.605$, $p<.05$). The rest group had a significantly higher level of laryngeal EMG activity than the CI group for the alcohol by failure condition ($F = 4.562$, $p<.05$). The drug by period interaction was significant for the success condition ($F = 4.488$, $p<.05$) but not for the failure condition. None of the other factors were significant. The overall analysis (Tables 23 and 24) showed a significant drug effect ($F = 7.387$, $p<.01$) but no other significant main effects or interactions. Laryngeal EMG activity increased after alcohol during the CI task itself and was significantly increased on the descending limb after alcohol. The combined results from frontal and laryngeal EMG show greater overall

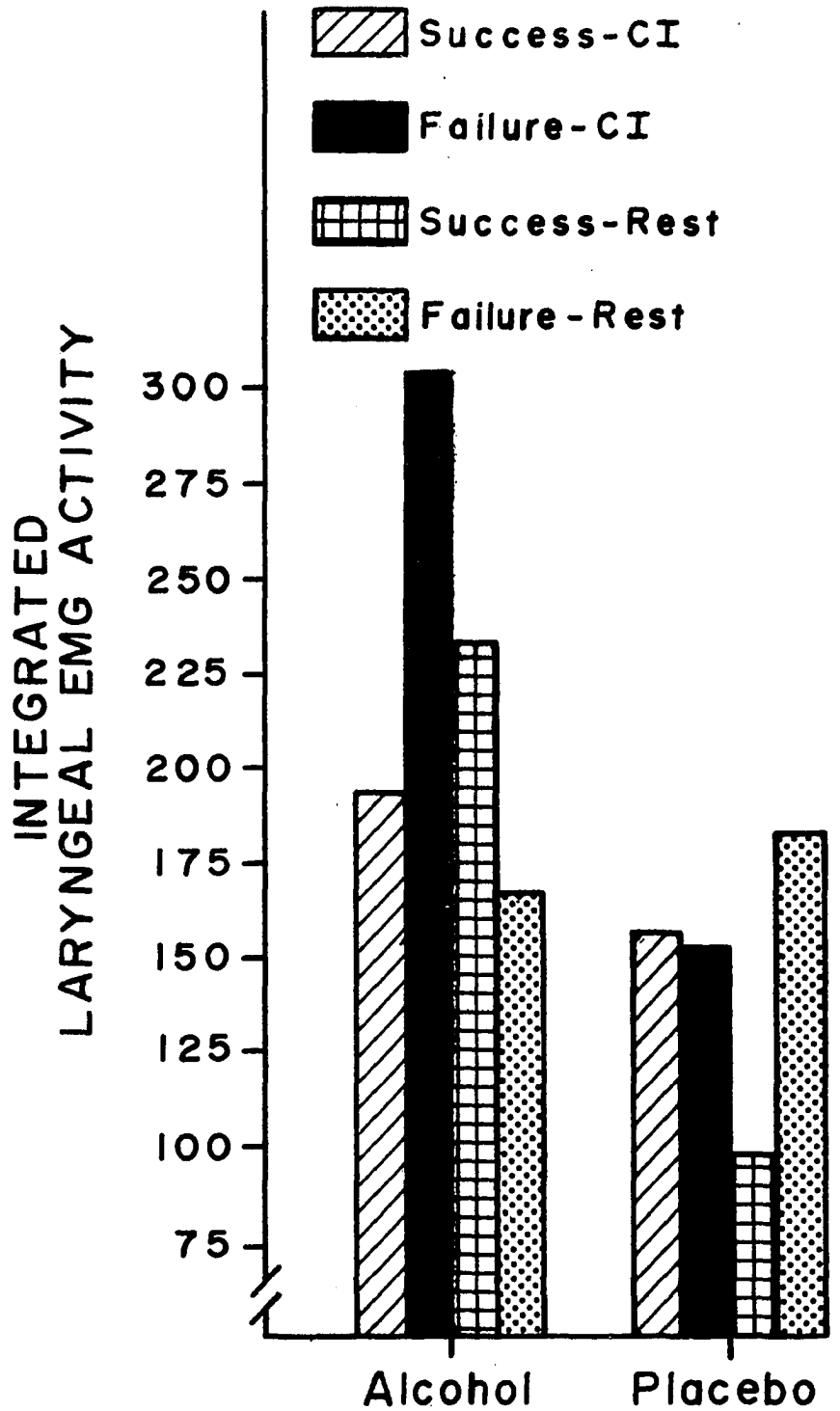

Fig. 6 - Mean laryngeal EMG activity for each drug group shown separately for success and failure set and concept identification task and rest periods for the descending limb (Drug x Set x Period interaction).

TABLE 17

MEANS AND STANDARD DEVIATIONS FOR LARYNGEAL EMG MEASURED DURING REST PERIODS

Group	Mean	S.D.
Drug		
Alcohol	197.85	100.08
Placebo	153.45	74.50
Set		
Success	170.55	96.14
Failure	180.75	85.48
Limb		
Ascending	183.30	88.52
Descending	170.30	92.06

TABLE 18

MEANS AND STANDARD DEVIATIONS FOR LARYNGEAL EMG MEASURED DURING CI PERIODS

Group	Mean	S.D.
Drug		
Alcohol	231.90	124.91
Placebo	149.70	38.98
Set		
Success	169.30	56.21
Failure	212.30	128.62
Limb		
Ascending	181.55	73.11
Descending	250.05	247.07

TABLE 19

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR LARYNGEAL EMG MEASURED DURING CI PERIODS

Source	df	MS	F
Drug	1	67568.370	7.598**
Set	1	18490.000	2.079
Limb	1	3422.500	0.385
Drug x Set	1	5290.000	0.595
Drug x Limb	1	1932.099	0.217
Set x Limb	1	1276.899	0.144
Drug x Set x Limb	1	10304.090	1.159
Error (Within)	32	8893.066	
Total	39		

**p<.01

TABLE 20

MEANS AND STANDARD DEVIATIONS FOR LARYNGEAL EMG MEASURED DURING CI AND REST PERIODS ON THE ASCENDING LIMB

Group	Mean	S.D.
Drug		
Alcohol	205.80	92.14
Placebo	156.75	60.47
Set		
Success	170.50	75.90
Failure	174.28	86.48
Period		
CI	181.55	73.11
Rest	183.30	88.52

TABLE 21

MEANS AND STANDARD DEVIATIONS FOR LARYNGEAL EMG MEASURED DURING CI AND REST PERIODS ON THE DESCENDING LIMB

Group	Mean	S.D.
Drug		
Alcohol	223.95	32.53
Placebo	146.40	57.99
Set		
Success	169.35	81.50
Failure	201.00	130.09
Period		
CI	250.05	247.07
Rest	170.30	92.06

TABLE 22

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR LARYNGEAL EMG MEASURED DURING CI AND REST PERIODS ON THE DESCENDING LIMB

Source	df	MS	F
Drug	1	60140.020	5.898*
Set	1	10017.220	0.982
Period	1	8850.625	0.868
Drug x Set	1	1071.224	0.105
Drug x Period	1	3441.024	0.337
Set x Period	1	5130.222	0.503
Drug x Set x Period	1	42837.020	4.201*
Error (Within)	32	10196.490	
Total	39		

*$p < .05$

TABLE 23

MEANS AND STANDARD DEVIATIONS FOR LARYNGEAL EMG MEASURED ON THE ASCENDING LIMB VS. THE DESCENDING LIMB DURING BOTH CI AND REST PERIODS

Group	Mean	S.D.
Drug		
Alcohol	214.88	113.04
Placebo	151.58	58.71
Set		
Success	169.93	77.73
Failure	196.53	108.97
Order		
CI 1st	175.93	82.25
Rest 1st	190.53	106.80
Limb		
Ascending	181.28	80.84
Descending	185.18	108.34

TABLE 24

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR LARYNGEAL EMG MEASURED ON THE ASCENDING VS. DESCENDING LIMB DURING BOTH CI AND REST PERIODS

Source	df	MS	F
Drug	1	80137.750	7.387**
Set	1	14151.190	1.305
Order	1	4263.199	0.743
Drug x Set	1	84.050	0.008
Drug x Order	1	2.450	0.001
Set x Order	1	781.250	0.072
Drug x Set x Order	1	32643.190	3.009
Error (Between)	32	10847.910	
Limb	1	304.200	0.049
Drug x Limb	1	4061.250	0.657
Set x Limb	1	510.050	0.083
Order x Limb	1	4590.500	0.743
Drug x Set x Limb	1	1377.799	0.223
Drug x Order x Limb	1	7144.199	1.156
Set x Order x Limb	1	5379.199	0.870
Drug x Set x Order x Limb	1	12550.040	2.030
Error (Within)	32	6181.792	
Total	79		

** $p < .01$

arousal after alcohol during rest and more laryngeal activity during actual problem solving.

Skin Conductance Data

Skin conductance results will be presented next. Skin conductance level analyses appear first and are followed by the skin conductance response data. For the purpose of these analyses, a response was defined as being one-fourth micromho or greater. Both the frequency of skin conductance responses and the amplitude of responses were analyzed.

Skin conductance level. For skin conductance level, there were no significant main effects on the ANOVA comparing measurements taken during rest (Tables 25 and 26). There was a significant drug by set interaction ($F = 5.116$, $p<.05$). Simple effects analysis indicated a significantly higher mean skin conductance level for the alcohol group than the placebo group for the failure condition ($F = 4.314$, $p<.05$). The success group had a significantly higher mean skin conductance level than the failure group for the placebo condition ($F = 4.373$, $p<.05$). The interaction is graphed in Figure 7. There were no significant results for the ANOVA of CI periods, for the ascending limb, or for the descending limb for skin conductance levels. The means and standard deviations for these three analyses appear in Tables 27, 28, and 29 respectively. The ANOVA for skin conductance levels measured on the ascending versus the descending limb during both CI and rest periods (Tables 30 and 31) showed a highly significant limb effect ($F = 19.967$, $p<.01$). There was also a highly significant order by limb interaction, which is illustrated in Figure 8. Simple effects analysis showed a very

TABLE 25

MEANS AND STANDARD DEVIATIONS FOR SKIN CONDUCTANCE LEVEL MEASURED DURING REST PERIODS

Group	Mean	S.D.
Drug		
Alcohol	8.78	3.78
Placebo	8.20	3.79
Set		
Success	8.70	3.30
Failure	8.28	4.22
Limb		
Ascending	8.58	4.13
Descending	8.40	3.42

TABLE 26

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR SKIN CONDUCTANCE LEVELS MEASURED DURING REST PERIODS

Source	df	MS	F
Drug	1	10.506	0.445
Set	1	0.006	0.001
Limb	1	6.006	0.254
Drug x Set	1	120.756	5.116*
Drug x Limb	1	29.756	1.261
Set x Limb	1	9.506	0.403
Drug x Set x Limb	1	0.156	0.007
Error (Within)	32	23.603	
Total	39		

*$p < .05$

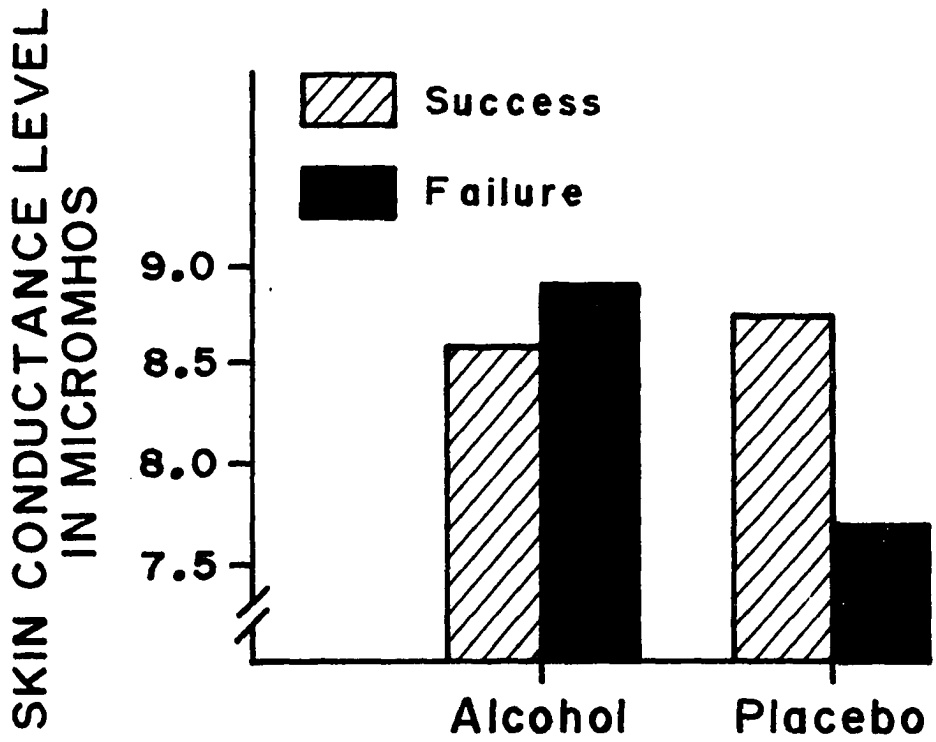

Fig. 7 - Mean skin conductance level for each drug group shown separately for success and failure set for rest periods (Drug x Set interaction).

TABLE 27

MEANS AND STANDARD DEVIATIONS FOR SKIN CONDUCTANCE LEVEL MEASURED DURING CI PERIODS

Group	Mean	S.D.
Drug		
Alcohol	10.58	4.66
Placebo	9.38	5.03
Set		
Success	10.20	4.50
Failure	9.75	5.24
Limb		
Ascending	10.93	4.40
Descending	9.03	5.14

TABLE 28

MEANS AND STANDARD DEVIATIONS FOR SKIN CONDUCTANCE LEVEL MEASURED DURING CI AND REST PERIODS ON THE ASCENDING LIMB

Group	Mean	S.D.
Drug		
Alcohol	10.45	4.34
Placebo	9.05	4.42
Set		
Success	10.00	4.03
Failure	9.50	4.79
Period		
CI	10.93	4.40
Rest	8.58	4.13

TABLE 29

MEANS AND STANDARD DEVIATIONS FOR SKIN CONDUCTANCE LEVEL MEASURED DURING CI AND REST PERIODS ON THE DESCENDING LIMB

Group	Mean	S.D.
Drug		
Alcohol	8.90	4.19
Placebo	8.53	4.55
Set		
Success	8.90	3.92
Failure	8.53	4.78
Period		
CI	9.03	5.14
Rest	8.40	3.42

TABLE 30

MEANS AND STANDARD DEVIATIONS FOR SKIN CONDUCTANCE LEVEL MEASURED ON THE ASCENDING LIMB VS. THE DESCENDING LIMB DURING BOTH CI AND REST PERIODS

Group	Mean	S.D.
Drug		
Alcohol	9.68	4.28
Placebo	8.76	4.41
Set		
Success	9.45	3.97
Failure	9.01	4.76
Order		
CI 1st	9.66	4.10
Rest 1st	8.80	4.61
Limb		
Ascending	9.75	4.38
Descending	8.71	4.32

TABLE 31

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR SKIN CONDUCTANCE LEVELS MEASURED ON THE ASCENDING VS. DESCENDING LIMB DURING BOTH CI AND REST PERIODS

Source	df	MS	F
Drug	1	15.753	0.388
Set	1	3.828	0.094
Order	1	14.878	0.367
Drug x Set	1	6.903	0.170
Drug x Order	1	0.078	0.002
Set x Order	1	35.778	0.882
Drug x Set x Order	1	3.828	0.094
Error (Between)	32	40.556	
Limb	1	28.203	19.960**
Drug x Limb	1	2.628	1.860
Set x Limb	1	0.153	0.108
Order x Limb	1	35.778	25.330**
Drug x Set x Limb	1	2.278	1.613
Drug x Order x Limb	1	4.278	3.029
Set x Order x Limb	1	0.528	0.373
Drug x Set x Order x Limb	1	0.078	0.055
Error (Within)	32	1.413	
Total	79		

** $p < .01$

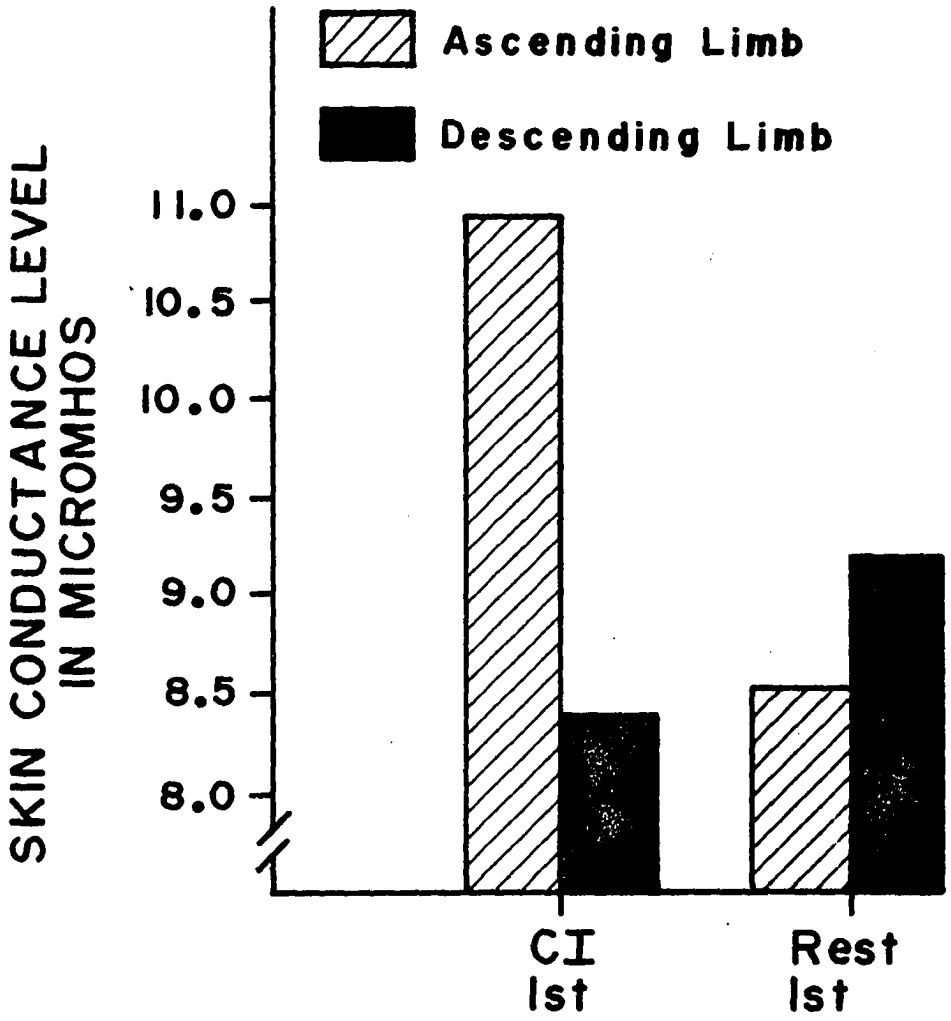

Fig. 8 - Mean skin conductance level for each order shown separately for the ascending and descending limb (Order x Limb interaction).

significantly higher mean skin conductance level for the ascending limb than the descending limb for the CI first condition ($F = 39.084$, $p<.01$). In summary, skin conductance levels were increased after alcohol in the failure set. Mean BSC level was greater on the ascending limb than on the descending limb. Mean BSC level was also greater on the ascending limb than for the descending limb for subjects who had CI on the ascending limb.

Skin conductance response frequency. Skin conductance response data will be presented next. There were a significantly greater number of skin conductance responses recorded during rest periods for the alcohol group ($F = 5.202$, $p<.05$). The means appear in Table 32 and the ANOVA in Table 33. There were no significant main effects or interactions during CI periods. Those means are presented in Table 34. On both the ascending and descending limb ANOVAs, there was a significantly greater number of skin conductance responses during CI than during rest periods ($F = 5.389$, $p<.05$ and $F = 4.514$, $p<.05$ respectively). Data are presented in Tables 35 through 38. There were also significantly more skin conductance responses occurring on the ascending limb as revealed by the analysis comparing ascending limb with descending limb during both CI and rest periods ($F = 4.951$, $p<.05$). Data are summarized in Tables 39 and 40. There was also a very highly significant order by limb interaction ($F = 15.823$, $p<.01$) which is illustrated by Figure 9. Simple effects analysis indicated a significantly larger number of skin conductance responses for the ascending limb than for the descending limb for the CI first condition ($F = 20.039$, $p<.01$). There was not a significant difference for the rest first condition. The CI first group

TABLE 32

MEANS AND STANDARD DEVIATIONS FOR NUMBER OF SCRs MEASURED DURING REST PERIODS

Group	Mean	S.D.
Drug		
Alcohol	1.50	2.09
Placebo	0.50	0.89
Set		
Success	1.15	1.98
Failure	0.85	1.31
Limb		
Ascending	1.35	2.03
Descending	0.65	1.14

TABLE 33

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR NUMBER OF SCRs MEASURED DURING REST PERIODS

Source	df	MS	F
Drug	1	30.625	5.202*
Set	1	0.225	0.038
Limb	1	1.225	0.208
Drug x Set	1	13.225	2.246
Drug x Limb	1	18.225	3.096
Set x Limb	1	0.225	0.038
Drug x Set x Limb	1	15.625	2.654
Error (Within)	32	5.888	
Total	39		

*p< .05

TABLE 34

MEANS AND STANDARD DEVIATIONS FOR NUMBER OF SCRs MEASURED DURING CI PERIODS

Group	Mean	S.D.
Drug		
Alcohol	2.50	2.42
Placebo	2.45	2.67
Set		
Success	2.85	2.68
Failure	2.10	2.34
Limb		
Ascending	2.95	2.54
Descending	1.40	2.06

TABLE 35

MEANS AND STANDARD DEVIATIONS FOR NUMBER OF SCRs MEASURED DURING CI AND REST PERIODS ON THE ASCENDING LIMB

Group	Mean	S.D.
Drug		
Alcohol	2.55	2.39
Placebo	1.75	2.43
Set		
Success	2.70	2.72
Failure	1.60	1.98
Period		
CI	2.95	2.54
Rest	1.35	2.03

TABLE 36

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR NUMBER OF SCRs MEASURED DURING CI AND REST PERIODS ON THE ASCENDING LIMB

Source	df	MS	F
Drug	1	6.400	1.347
Set	1	12.100	2.547
Period	1	25.600	5.389*
Drug x Set	1	0.400	0.084
Drug x Period	1	12.100	2.547
Set x Period	1	1.600	0.337
Drug x Set x Period	1	16.900	3.560
Error (Within)	32	4.750	
Total	39		

* $p < .05$

TABLE 37

MEANS AND STANDARD DEVIATIONS FOR NUMBER OF SCRs MEASURED DURING CI AND REST PERIODS ON THE DESCENDING LIMB

Group	Mean	S.D.
Drug		
Alcohol	1.45	2.09
Placebo	1.20	1.96
Set		
Success	1.30	2.06
Failure	1.35	2.01
Period		
CI	1.40	2.06
Rest	0.65	1.14

TABLE 38

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR NUMBER OF SCRs MEASURED DURING CI AND REST PERIODS ON THE DESCENDING LIMB

Source	df	MS	F
Drug	1	0.625	0.155
Set	1	0.025	0.006
Period	1	18.225	4.514*
Drug x Set	1	7.225	1.789
Drug x Period	1	0.225	0.056
Set x Period	1	0.025	0.006
Drug x Set x Period	1	1.225	0.303
Error (Within)	32	4.038	
Total	39		

* $p < .05$

TABLE 39

MEANS AND STANDARD DEVIATIONS FOR NUMBER OF SCRs MEASURED ON THE ASCENDING LIMB VS. THE DESCENDING LIMB DURING BOTH CI AND REST PERIODS

Group	Mean	S.D.
Drug		
Alcohol	2.00	2.29
Placebo	1.48	2.20
Set		
Success	2.00	2.48
Failure	1.48	1.97
Order		
CI 1st	1.80	2.27
Rest 1st	1.68	2.25
Limb		
Ascending	2.15	2.41
Descending	1.58	2.42

TABLE 40

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR NUMBER OF SCRs MEASURED ON THE ASCENDING VS. DESCENDING LIMB DURING BOTH CI AND REST PERIODS

Source	df	MS	F
Drug	1	5.513	0,913
Set	1	5.513	0.913
Order	1	0.313	0.052
Drug x Set	1	5.513	0.913
Drug x Order	1	7.813	1.294
Set x Order	1	0.613	0.101
Drug x Set x Order	1	4.513	0.747
Error (Between)	32	6.038	
Limb	1	13.613	4.950*
Drug x Limb	1	1.513	0.550
Set x Limb	1	6.613	2.405
Order x Limb	1	43.513	15.822**
Drug x Set x Limb	1	2.113	0.768
Drug x Order x Limb	1	4.513	1.641
Set x Order x Limb	1	1.013	0.368
Drug x Set x Order x Limb	1	13.613	4.950*
Error (Within)	32	2.750	
Total	79		

* $p < .05$
** $p < .01$

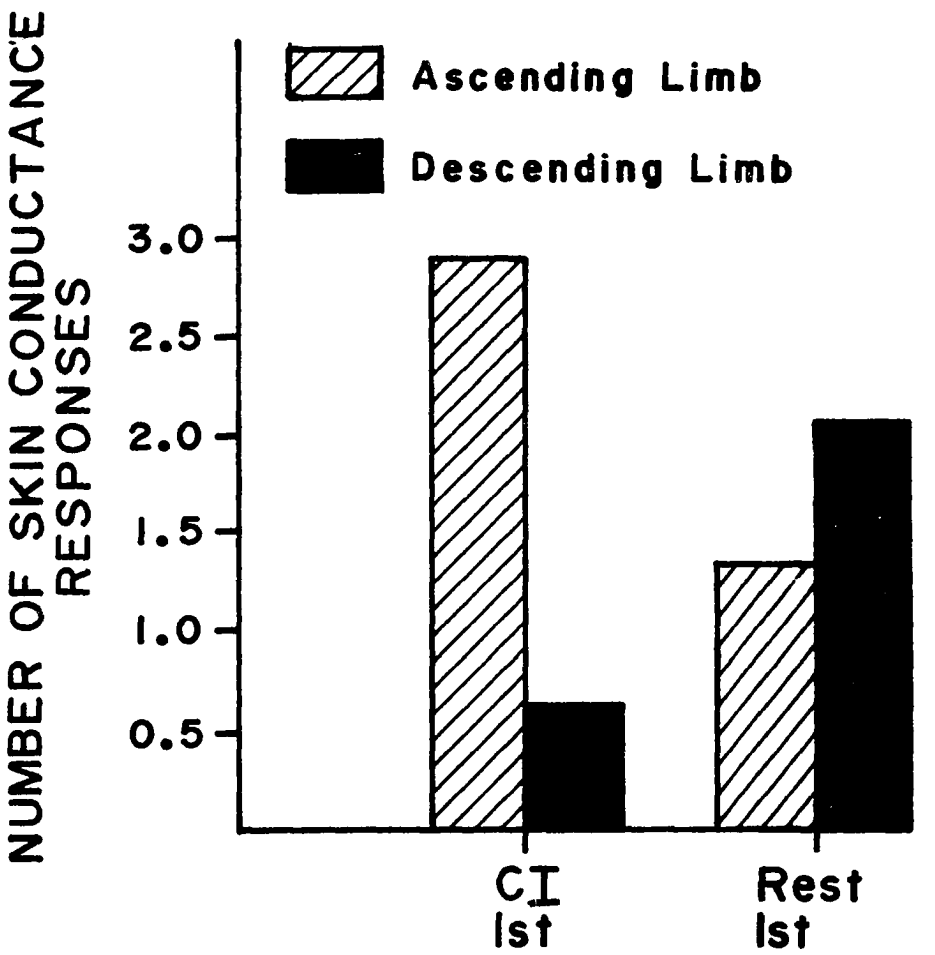

Fig. 9 - Mean number of skin conductance responses for each order shown separately for the ascending and descending limb (Order x Limb interaction).

showed significantly more SCRs for the ascending limb condition than the rest first group (F = 9.309, $p<.01$). The rest first group showed significantly more SCRs for the descending limb condition than the CI first group (F = 6.627, $p<.05$). There was also a significant drug by set by order by limb interaction (F = 4.950, $p<.01$).

In summary, skin conductance responses showed a greater increase in number after alcohol during rest. There was also a greater number of responses during CI than during rest on both the ascending and descending limbs. Significantly more responses appeared on the ascending limb than on the descending limb period.

Skin conductance response amplitude. Amplitude of skin conductance responses was also significantly greater after alcohol during rest periods (F = 8.463, $p<.01$; see Tables 41 and 42) but not during CI (Table 43). There were significantly larger responses on the ascending limb after alcohol (F = 4.276, $p<.05$). Responses were also significantly larger during CI than during rest (F = 4.276, $p<.05$). The means appear in Table 44 and the ANOVA summary in Table 45. There were no significant main effects or interactions on the descending limb analysis. Those means are presented in Table 46. The analysis of ascending versus descending limb during both CI and rest periods showed a highly significant order by limb interaction. The mean table is Table 47 and the ANOVA table is Table 48. The depiction of the interaction is presented in Figure 10. Simple effects analysis indicated a significantly greater response amplitude for the descending limb condition than for the ascending limb for the rest first condition (F = 4.435, $p<.05$). None of the other conditions was significant. To briefly summarize,

TABLE 41

MEANS AND STANDARD DEVIATIONS FOR AMPLITUDE OF SCRs MEASURED DURING REST PERIODS

Group	Mean	S.D.
Drug		
Alcohol	1.06	1.18
Placebo	0.400	0.73
Set		
Success	0.69	0.99
Failure	1.28	2.63
Limb		
Ascending	0.79	1.06
Descending	0.68	1.02

TABLE 42

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR AMPLITUDE OF SCRs MEASURED DURING REST PERIODS

Source	df	MS	F
Drug	1	9.216	8.463**
Set	1	0.130	0.119
Limb	1	0.036	0.033
Drug x Set	1	2.470	2.268
Drug x Limb	1	1.962	1.802
Set x Limb	1	0.610	0.560
Drug x Set x Limb	1	2.362	2.169
Error (Within)	32	1.089	
Total	39		

** $p < .01$

TABLE 43

MEANS AND STANDARD DEVIATIONS FOR AMPLITUDE OF SCRs MEASURED DURING CI PERIODS

Group	Mean	S.D.
Drug		
Alcohol	1.55	1.34
Placebo	1.49	1.79
Set		
Success	1.39	1.27
Failure	1.65	1.83
Limb		
Ascending	1.52	1.18
Descending	1.52	1.90

TABLE 44

MEANS AND STANDARD DEVIATIONS FOR AMPLITUDE OF SCRs MEASURED DURING CI AND REST PERIODS ON THE ASCENDING LIMB

Group	Mean	S.D.
Drug		
Alcohol	1.52	1.29
Placebo	0.79	0.93
Set		
Success	1.22	1.33
Failure	1.09	1.02
Period		
CI	1.52	1.18
Rest	0.79	1.06

TABLE 45

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR AMPLITUDE OF SCRs MEASURED DURING CI AND REST PERIODS ON THE ASCENDING LIMB

Source	df	MS	F
Drug	1	5.336	4.276*
Set	1	0.173	0.139
Period	1	5.322	4.276*
Drug x Set	1	0.352	0.282
Drug x Period	1	0.915	0.735
Set x Period	1	1.040	0.836
Drug x Set x Period	1	0.294	0.236
Error (Within)	32	1.245	
Total	39		

*P < .05

TABLE 46

MEANS AND STANDARD DEVIATIONS FOR AMPLITUDE OF SCRs MEASURED DURING CI AND REST PERIODS ON THE DESCENDING LIMB

Group	Mean	S.D.
Drug		
Alcohol	1.10	1.26
Placebo	1.10	1.86
Set		
Success	0.85	1.02
Failure	1.34	1.97
Period		
CI	1.52	1.90
Rest	0.68	1.02

TABLE 47

MEANS AND STANDARD DEVIATIONS FOR AMPLITUDE OF SCRs MEASURED ON THE ASCENDING LIMB VS. THE DESCENDING LIMB DURING BOTH CI AND REST PERIODS

Group	Mean	S.D.
Drug		
Alcohol	1.31	1.27
Placebo	0.94	1.46
Set		
Success	1.04	1.18
Failure	1.22	1.55
Order		
CI 1st	1.10	1.17
Rest 1st	1.15	1.56
Limb		
Ascending	1.56	1.17
Descending	1.10	1.56

TABLE 48

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR AMPLITUDE OF SCRs MEASURED ON THE ASCENDING VS. DESCENDING LIMB DURING BOTH CI AND REST PERIODS

Source	df	MS	F
Drug	1	2.679	1.219
Set	1	0.648	0.295
Order	1	0.067	0.030
Drug x Set	1	0.005	0.002
Drug x Order	1	0.001	0.001
Set x Order	1	3.313	1.507
Drug x Set x Order	1	0.673	0.306
Error (Between)	32	2.198	
Limb	1	0.069	0.043
Drug x Limb	1	2.657	1.652
Set x Limb	1	1.941	1.206
Order x Limb	1	12.387	7.702**
Drug x Set x Limb	1	0.585	0.364
Drug x Order x Limb	1	1.806	1.123
Set x Order x Limb	1	0.143	0.089
Drug x Set x Order x Limb	1	0.003	0.002
Errors (Within)	32	1.608	
Total	79		

** $p < .01$

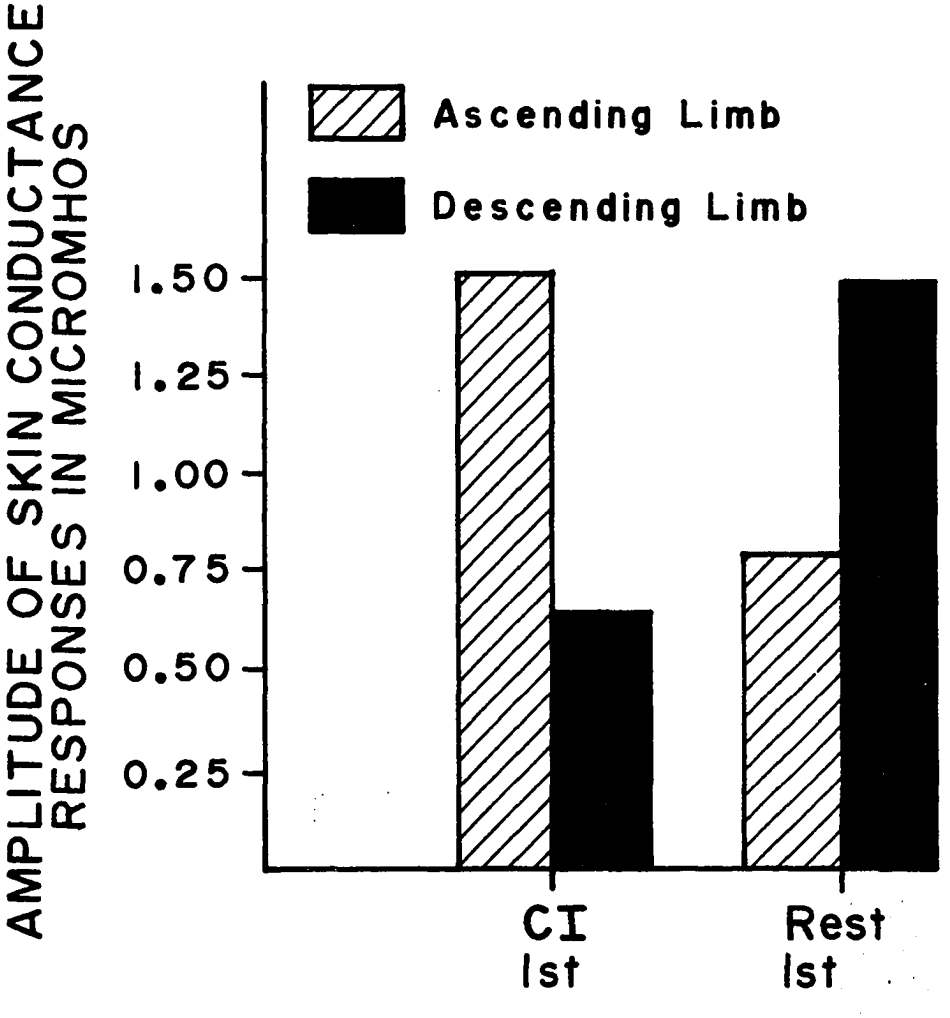

Fig. 10 - Mean skin conductance response amplitude for each order shown separately for the ascending and descending limb (Order x Limb interaction).

amplitude of skin conductance responses increased after alcohol. The amplitude was also larger during CI than during rest. The skin conductance response is also indicative of general arousal. There is an increase in both number and amplitude of responses after alcohol. These responses are larger during task (CI) than rest. There are a greater number of responses on the ascending limb than on the descending limb.

Review of Psychophysiological Results

Alcohol affected electromyographic activity, as had been hypothesized (Hypothesis 3). There was a significantly greater level of frontal EMG activity for the alcohol group during rest periods but not during concept identification periods; whereas, there was a significantly greater level of laryngeal EMG activity for the alcohol group during concept identification periods but no significant difference during rest. The alcohol group had a significantly higher level of activity than did the placebo group for the ascending limb by success condition (Hypothesis 3a). On the ascending limb, the alcohol group had higher levels of both frontal and laryngeal EMG activity than the placebo group, but these differences were not significant. On the descending limb, a depression in EMG activity had been predicted for the alcohol group (Hypothesis 3b). Instead, there was a significantly higher level of both frontal and laryngeal EMG activity for the descending limb period for alcohol subjects compared with placebo subjects. There was no significant difference in either frontal or laryngeal EMG activity during concept identification for placebo subjects who had received the

failure set, which is counter to Hypothesis 3c. Those who received alcohol and success set had a significantly higher mean level of frontal EMG than those who received alcohol and failure set (Hypothesis 3d).

There were several additional significant findings. Subjects who received success set showed higher mean levels of frontal EMG during rest periods than did subjects in the failure set groups. For laryngeal EMG, subjects in the success by rest group who received alcohol had a significantly higher mean level of EMG activity than did placebo subjects. Subjects in the failure by concept identification group who received alcohol had a significantly higher mean level of laryngeal EMG activity than did subjects in the placebo group. Mean frontal EMG level was greater for the alcohol subjects for the ascending limb by success condition than the mean level for the placebo subjects. Subjects who received the concept identification task first and failure set had a higher mean level of frontal EMG activity on the ascending limb than they did on the descending limb when they had a rest period. Frontal EMG activity was greater for subjects who had the CI task on the descending limb and also failure set than was activity for subjects who had a rest period and failure set on the descending limb. On the descending limb subjects who had success set had a significantly greater level of frontal EMG activity during the rest period than subjects in the failure group. The rest group had a significantly higher level of laryngeal activity than did the CI group for the alcohol by failure condition.

There was no significant main drug effect for BSC activity. It has been hypothesized that alcohol would decrease SCR activity

(Hypothesis 4). Skin conductance level was significantly higher for alcohol subjects than placebo subjects for the failure condition. There were both a significantly greater number and greater amplitude of skin conductance responses for alcohol than placebo subjects during rest periods. Skin conductance level, number of responses and response amplitude were all significantly greater on the ascending limb than they were on the descending limb of the blood alcohol curve (Hypothesis 4a). For both the ascending and the descending limb, there were significantly more SCRs during CI than during rest. There were also significantly larger SCRs during CI than during rest for the ascending limb analysis (Hypothesis 4b). There were both a significantly higher mean skin conductance level and a greater number of SCRs for the ascending limb (CI task) than the descending limb (rest period) for the CI first condition. The number of SCRs was greater during the CI period than during the rest period for the ascending limb. Both the number and amplitude of SCRs were greater for the rest first (CI task) than the CI first rest period on the descending limb. SCL was greater during success than during failure for the placebo condition.

CHAPTER IV

DISCUSSION

Relationship of Concept Identification Data to Previous Research

The results of the present study demonstrate impairment in information processing after acute alcohol ingestion. Mean number of trials and errors and percentage of errors were significantly greater in the alcohol group than in the placebo group. Mean reaction time was not significantly different. Decreased efficiency was observed in the alcohol group, as manifested by a significant increase in number of trials and both absolute number and percentage of errors. Previous research is consistent with this finding. Kastl (1969) found logical thinking to remain largely intact on a problem-solving task (hypothesis testing) after 0.33, 0.67, or 1.00 ml. of absolute alcohol per kg. Fourteen out of 23 measures showed no change after alcohol. The nine that did show a decline in performance were related to decreased efficiency and loss of motivation rather than a shift in mode of thought. Carpenter, Moore, Snyder, and Lisansky (1961) employed the calculus method to examine the effects of 0.00, 0.33, 0.67, and 1.00 ml. of absolute alcohol per kg. on higher-order problem solving. Their results indicated that problem solving efficiency was curvilinearly related to

dose with the 0.33 ml./kg. dose showing increased efficiency and the 1.0 ml./kg. dose showing decreased efficiency. Subjects were able to apply the rules of the calculus correctly, regardless of the dose. The results of both Kastl and Carpenter, as well as the results of this study, indicate that alcohol subjects are still able to solve complex problems at relatively high dose levels but with a decrease in efficiency.

The findings of Moskowitz and DePry (1968) can also be interpreted in terms of decreased efficiency. The study involved both a vigilance (tone detection) and divided attention task (tone detection plus digit reporting). Their Ss maintained prealcohol performance levels on the vigilance task at the expense of reduced performance on the divided attention task after receiving 0.52 g. of alcohol per kg. of body weight. Loomis and West (1958) also demonstrated a reduction in available attention after alcohol on a driver simulator task that required the subject to divide his attention effectively between the road and the signal lights. Huntley (1973) has referred to the decreased efficiency in information processing after alcohol as a reduction in attentional bandwidth.

In his 1962 review of alcohol effects on psychological processes related to automobile driving skill, Carpenter offers two hypotheses as to why some investigators find that cognitive or intellectual processes are not adversely affected by alcohol. They are as follows:

> (1) Intellectual processes, considered in the evolutionary scheme of things, are advantageous only if they are relatively resistant to adverse and unusual conditions. If this were true, the higher processes would be expected to continue to function, within limits, despite increased BAL's. (2) Intellectual functions are not more complex but simpler. Since they are simpler, there is less to go wrong, hence they are

more resistant to adverse conditions such as alcohol or other forms of intoxication, high altitude, extreme temperatures, etc.

It has been customarily assumed in the past that higher processes are more affected by alcohol than lower ones; however, it appears that intellectual functions may be more resistant to alcohol than are sensory and motor functions. Efficiency of information processing may be reduced after alcohol intake, but at least with moderate doses of alcohol, subjects are still able to solve complex problems after drinking. Impairment seen in subjects in this study may have been due to perceptual or motor deficits. Visual information about the color and shape of slides may have been distorted and have been a source of errors.

Wells (1963) has pointed out that differential familiarity with the rules may also contribute to the orderings of rule difficulty. Pretraining on the less familiar disjunctive rule reduces its difficulty to a level comparable to that of the conjunctive rule. Training on the use of the rule was given in the instructions because it was felt that without training and rule familiarity, subjects would attribute failure to the fact that the rule was unfamiliar, and also that subjects would have an extremely difficult time solving the problem after alcohol in any condition without prior familiarity with the rule. As it is, there exists a possibility that even with pretraining subjects still are not able to use the disjunctive rule as indicated by the fact that some placebo subjects did not solve the problem.

Several studies have just been reviewed that indicate that acute doses of alcohol can decrease problem solving efficiency. Studies with alcoholics also indicate impairment in performance similar to but greater

than those seen in nonalcoholic subjects given moderate doses of alcohol. Tarter and Parsons (1971) reported that on the Wisconsin Card Sorting Test chronic alcoholics showed significantly more trials to criterion, total number of errors, perseverative errors, and errors of other kinds than did either hospital controls or college student controls. Alcoholics with longer drinking histories were significantly more impaired than those with short drinking histories. These deficits were further explored by Tarter (1973) who concluded that alcoholics are impaired in their ability to maintain a cognitive set, as indicated by interruption of a correct sequence of responses with an error. Subjects who had been alcoholics for ten or more years were deficient in set persistence, set shifting, and error utilization; whereas subjects who had been alcoholics for less than ten years were deficient only in set persistence when compared with controls. Pishkin, Fishkin, and Stahl (1972) did not find an overall difference between alcoholics and controls on a CI paradigm and stated that cognitive deficit associated with alcoholism is limited to aspects of cognitive functioning not tapped by analysis of CI errors, at least not for the type of paradigm they were using. The pattern of decreased efficiency and impairment of cognitive functioning, which is present in acute alcohol intake studies, is also present in studies using alcoholics as subjects.

In alcohol research, investigators have tended to be dismayed with any results other than those indicating impairment after alcohol ingestion. In fact, results indicating no change or improvement after alcohol consumption may be of great practical and theoretical importance. An employer has no problem deciding that the heavily intoxicated employee

should not work, but he may be in a quandry as to whether or not an employee who has had two or three drinks can effectively perform his assigned duties. Results indicating no change or improvement after low to moderate doses of alcohol may provide answers to industrial questions pertaining to employee competency after drinking. Automobile driving or airplane flying may be severely affected by alcohol. Alcohol is related to 25,000 deaths and at least 800,000 automobile crashes in the United States each year [Alcohol and Highway Safety Report (90-34), 1968]. A study by Mohler, Berner, and Goldbaum (1968) reported that 23% of all aircraft accidents investigated in 1967 toxicologically presented blood alcohol levels in excess of 150 mg. per cent. Another study on 158 fatal general aviation accidents in 1963 reported that tests for 35.4% of the pilots were positive for blood and/or tissue alcohol upon toxicological examination. With the high number of both airplane and automobile accidents and fatalities attributable to alcohol, it is of extreme practical importance to know at what level improvement or no change occurs in critical skills and at what level impairment is first seen.

Limb effects were not significant for the concept identification task although the trend was for both trials and errors to be greater on the ascending limb than on the descending limb. Although most studies that have looked at both ascending and descending limb effects have found deficits for the ascending limb condition, some have not. Loomis and West (1958) found no evidence for adaptation on a simulated driving task that measured both tracking ability and reaction time. Goldberg (1943) found differential limb effects for various tasks, i.e., no impairment of sensory functions (flicker frequency and corneal

sensitivity), some impairment of perceptual motor functioning on the ascending limb (finger to finger and modified Romberg), and greater impairment of cognitive functioning on the ascending limb (subtraction and Bourdon test). The control procedure used in this study may have contributed to the lack of significance. There would have been less variance if each subject had served as his own control; however, that procedure was not utilized here for several reasons. It would have been impossible to induce failure on more than one occasion. Subjects would have become suspicious of the manipulation. The concept identification problem is a relatively simple one. Subjects become highly trained and make few mistakes per problem very quickly. To administer set before drinking, test on the ascending limb, then again on the descending limb and repeat the process for each drug condition would result in highly trained subjects. Such a procedure would make it difficult to differentiate between drug and limb effects and practice effects. Perhaps the design employed was not the best for elucidating limb effects, but it was deemed to be the design of choice given the task being used and the goals of the study.

The set variable did not produce any significant differences, which is somewhat surprising since failure set usually produces deficits in subsequent performance. Carpenter (1968) has pointed out that expectations produced by an alcohol placebo may in fact produce symptom relief and relaxation. Set itself is a form of expectation that may in fact be cancelled by another expectation (placebo alcohol narcosis and escape). Williams (1966) pointed out that comparison of alcohol and placebo subjects does not isolate the effects of alcohol per se: it

isolates the effect of alcohol in the anxiety-arousing atmosphere of the alcohol research laboratory, an atmosphere which is likely to produce controlled, inhibited behavior in subjects.

It has been suggested by Korman, Knopf, and Austin (1960) that alcohol ingested during periods of stress may facilitate functioning by permitting increased control over the disrupting effect of intruding emotional factors. Inhibitory mechanisms may be released that permit a subject to function more effectively and overcome the effects of a prior failure set. Risk-taking behavior has been shown experimentally to increase after alcohol consumption (Hurst, Bagley, and Ross, 1972; Hurst, Radlow, Chubb, and Bagley, 1969; Teger, Katkin, and Pruitt, 1969). Subjects may not have been as cautious as they normally would have been when responding. Being drunk or thinking that one is drunk brings about a relaxation of obligations. Subjects who have received a success set may not feel the need to perform as well as they possibly can. Williams (1968) concluded that one reason for college student drinking may be to "attain a state where they can be themselves without being so subject to criticism or accountability." This benefit may be an appreciable one that could lead to heavy and frequent drinking. Another reason that no significant differences between success and failure subjects were found may have been that all of the subjects in this study were bright. Failure set may have been easily overcome by this population and may not have provided the block that it might have for populations other than graduate students.

Response time measures were not significantly different for drug groups. Response time was included as a measure of the effect of alcohol on the speed of processing the complex task of pattern classification.

Moskowitz and Roth (1971) have pointed out that whereas 150 to 200 milliseconds are required for executing simple responses to stimuli, 500 to 1400 milliseconds are required for naming an object. The task used in this study of classifying stimuli according to significant parameters, a much more complex task, produced response latencies of about three seconds. Frankenhaeuser, Myrsten, and Jarpe (1962) found that verbal and inductive test performance was unaffected by alcohol. Their results showed that performance speed was less affected than accuracy, which is the same type of result that was found in this study.

A possible explanation for the lack of significant results for reaction time is discussed below. An experiment of this type is a game which the subject concentrates on beating. With such motivation, he is often able to perform as well with an amount of alcohol as without it. This strong motivation to perform well does not usually obtain in most life situations encountered after the consumption of alcohol. In common usage, the action of ethanol is not mitigated by a strong desire to antagonize it, but rather is augmented by the welcome anticipation of its effect.

The data presented in this section emphasize that alcohol effects are complex and that to speak in terms of only deteriorative effects of alcohol is a gross oversimplification. Alcohol may decrease efficiency but not all functioning is equally impaired by alcohol. In fact, performance on some measures may not be affected by alcohol or improvement may even be seen. Moskowitz (1973) points out that at this point in time little is known about what specific aspects of information processing are affected by alcohol. Specific knowledge about which

central processes are affected by alcohol and which are not may lead to theories about causation of alcohol problems and offer new ideas for their solution.

Relationship of Psychophysiological Data to Previous Studies

As stated in the introduction, not much work has been done on the effects of alcohol on psychophysiological activity. Several studies have looked at the effects of alcohol on skin conductance level and skin conductance responses. There have only been a couple of studies which have examined the effects of alcohol on EMG activity. This study examined SCL, number and amplitude of SCRs, and frontal and laryngeal EMG during both performance (CI) and rest, on both the ascending and descending limbs of the BAC, after success set and after failure set, and after alcohol and after placebo. Studies dealing with EMG will be discussed first; then, studies on skin conductance will follow.

Docter and Perkins (1961) reported that levels of resting muscle tonus for the right forearm were significantly increased in alcohol subjects relative to the control subjects. Docter's study is the only study known of in the literature that has looked at resting muscle tension changes associated with alcohol. Abnormal levels of muscle tension have been reported for alcoholics by Kissin, Schenker, and Schenker (1959). It appears that alcohol may influence cortical or subcortical centers associated with the regulation of muscle tension and produce an increment in muscle tonus in the acute state with normals and a chronic increase in the alcoholic that may be slightly decreased by drinking alcohol. Frontal EMG was significantly higher for the alcohol group

than for the placebo group during rest in this present study, which agrees with the results of Docter, et al. (1961). Frontal EMG was significantly greater for alcohol subjects than for placebo subjects on the descending limb. This difference was not significant on the ascending limb.

Laryngeal EMG was significantly greater for the alcohol group than for the placebo group during the CI task. Covert oral behavior increases during cognitive tasks but does not typically increase during the performance of nonlanguage tasks (McGuigan, 1970). Verbalization has been shown to facilitate performance (Davis, Carey, Foxman, and Tarr, 1968; Gagne and Smith, 1962). Increased subvocal activity after alcohol may have been a compensatory attempt to overcome impairment produced by the drug by additional iteration of the concept and classification of each slide. It may also be that the release of inhibitory mechanisms by alcohol produces additional random EMG activity that is not necessarily goal-oriented and may even be disruptive to performance.

There was not the relationship between EMG activity and failure set that has been reported previously (Pishkin and Shurley, 1968; Pishkin, 1973). These studies have also reported a greater number of errors for the failure condition than for the success condition, and the positive correlation was between increased errors and electromyographic activity. In the present study there were slightly more errors for the success condition, although this difference was not significant. The EMG conditions that show significantly higher levels for set conditions indicate the success condition as having higher levels of EMG activity (i.e., alcohol x success--higher frontal EMG than alcohol x failure set; success--greater frontal EMG during rest than failure set).

Most of the research done with alcohol and electrodermal activity has been directed towards showing that alcohol reduces tension and anxiety and providing support for the tension reduction hypothesis (Carpenter, 1957; McDonnel and Carpenter, 1959; Lienert and Traxel, 1959; Coopersmith, 1964a; McGonnell and Beach, 1968; Cappell and Herman, 1972). The fallacy in such an interpretation is that a response does not indicate whether the affect experienced by the subject was negative or positive. To label a response as an indicator of anxiety is an error. Instead, the response can be viewed as an indicator of reactivity or responsiveness, and results can be examined in terms of the effects of alcohol on reactivity. Likewise, basal skin conductance can be viewed as a measure of arousal level and motivation, i.e., alertness and attention instead of prevailing anxiety (Coopersmith and Woodrow, 1967).

A significant drug effect for skin conductance activity was not found in this study. Powell, Goodwin, Janes, and Hoine (1971) have reported a similar finding. The number of skin potential responses was not significantly different for their alcohol and nonalcohol subjects. Docter and Perkins (1961) found no skin resistance changes associated with alcohol. In this present study there were both a significantly greater number and greater amplitude of skin conductance responses for alcohol subjects compared with placebo subjects during rest periods. Skin conductance level, number of SCRs, and amplitude of SCRs were all significantly greater on the ascending limb than on the descending limb of the blood alcohol curve. Skin conductance on the palms of the hands and the soles of the feet is controlled exclusively by the sympathetic

nervous system. Evidence indicates that subjects are generally more activated on the ascending limb. They are happy and talkative. EEG activation and heart rate increases have been reported for this period. This may be due to sympathetic activation during this period. On the descending limb, subjects are tired and depressed. Heart rate decreases, and EEG returns to baseline. The issue of whether alcohol is a stimulant or a depressant will be discussed later.

When rest and task were compared, there were generally significantly more SCRs, and their amplitude was greater during CI than rest. In other words, the general level of responsiveness was increased during the concept identification task. There was not a general set effect, but skin conductance level was significantly higher for alcohol subjects than for placebo subjects for the failure condition. Even though there was not a significant effect of set on performance, the alcohol subjects' level of arousal was greater than that of the placebo subjects. Increased arousal and motivation of the failure group may be part of the explanation for no impairment in performance on the set variable.

Alcohol was not found to reduce skin conductance level as has been reported by other investigators under some other conditions (Carpenter, 1957; McDonnell and Carpenter, 1959; Coopersmith and Woodrow, 1967). Studies in the past have tended to yield mixed results, have been methodologically unsound, and have used different (usually lower) amounts of alcohol than this study. It may be that small doses of alcohol minimize arousal, and moderate doses are somewhat more arousing or at least not suppressing. In this study, the alcohol group showed slightly higher, although not significantly different, levels of arousal for the

alcohol group. A study needs to be done comparing dose and skin conductance level to establish the exact relationship of the two. Since individual differences are so important, it is suggested that such a study use repeated measures. It would also be of interest to use alcoholics with and without alcohol. Coopersmith and Woodrow (1967) demonstrated no differences in basal skin conductance (BSC) levels between alcoholics and normals without alcohol. These alcoholic subjects were not placed in a stressful arousing situation. The BSC levels may be entirely different in alcoholics under stress. Extremely high levels of BSC are associated with low levels of distress. The dose of alcohol used here produced a slightly higher SCL than did the placebo. It may be that high doses of alcohol produce much higher skin conductance levels and less reactivity (relief from noxious stimuli) and provide an explanation for drinking in face of distressing events.

One of the questions raised by this section is whether alcohol is a stimulant or a depressant. Mello (1968) has stated that "alcohol acts as a stimulant at low doses and a depressant only at higher doses." It is pharmacologically classified as a central nervous system depressant. Carpenter (1968) discussed some of the problems involved with using this classification as an explanatory principle. Instead of invoking disinhibition as an explanation of improvement after alcohol, he considers alcohol to be neither a stimulant nor a depressant, but to be both, depending on the dose given and the function being examined. In the present study, frontal EMG was increased during rest after alcohol as were the number and amplitude of skin conductance responses. Murphree (1973) reported finding EEG arousal (beta activity) and heart rate

increases shortly after alcohol ingestion. He discussed the possibility that such findings may result from catecholamine release. Frankenhaeuser, Dunne, Bjurstrom, and Lundberg (1973) have recently reported that stress (shock) significantly reduced the impairment in performance produced by alcohol. They concluded that cognitive evaluation of the situation and its accompanying threat were responsible for attenuating the effect of alcohol on behavior.

The picture is indeed a complicated one. Much work remains to be done. So many structures have been implicated in interpreting alcohol effects. The reticular activating system is involved with arousal and attention, the limbic system with motivation and emotion, and the cortex with higher functions. Any or all can be involved with a given improvement or deficit. To quote Carpenter (1968),

> Such a paucity of research on so many structures suggests that very little is known about how alcohol causes behavioral changes by influencing the CNS. The "depressant" idea is sheer assumption. It may be correct, but there is insufficient evidence for a sweeping generalization.

Future Studies

The need for more research to increase understanding of the pharmacological effects of alcohol is apparent. Research needs to be done both on the primary effects of alcohol on the central nervous system and the secondary effects of alcohol on behavior. It is surprising how little is now known about the antecedents of excessive uncontrollable drinking of alcohol, especially considering how widespread its occurrence is. Several lines of research are suggested on the basis of the results of this study.

Some performance variables were found to be affected by alcohol; some were not. In some ways negative or biphasic results may provide more answers about mechanisms than results that show only improvement. Dose-response studies give an indication of which mechanisms are functioning and which are not within a given system at different drug levels. A dose-response study is currently being conducted by the author using the latency operating characteristic (LOC) analysis to study the effects of alcohol on discriminative reaction time. This analysis permits a separation of sensitivity and decision criterion effects on discriminative reaction time.

This study looked at effects of alcohol on normals during performance of a disjunctive concept identification task. It would be interesting to administer the same task to short and long term alcoholics to see if there is a continuum of deficits present for this particular task with increasing impairment and inefficiency for chronic alcoholics. A systematic program could be carried out using attribute identification with different rules and different doses of alcohol and looking at performance. Rule-learning studies could be conducted as well.

Another study presently being conducted by the author is continuing along the lines developed in this study. The effects of alcohol as a mild stressor on attention and memory processes are being examined using a verbal learning paradigm with reaction time probes being interjected at various points in time after stimulus presentation. Some of the words will be perceptually isolated, a manipulation which increases the likelihood of recall of a given word (Von Restorf effect). Limb effects will also be examined. This task should give additional

information about mechanisms impaired by alcohol. Examining the amounts of time required for the different processing tasks using the same probe signal will enable a comparison of the central capacity required for each task to be made.

Relatively little basic psychophysiological work has been done with alcohol. Autonomic measures could be useful for settling some issues evolving around the stimulant-depressant question and mechanisms and possible sites of action of alcohol. Autonomic measures could also be useful in examining the effects of alcohol on cognitive activity during information processing. A study is proposed that will examine cardiac reactions to the memory task with probes which was described above. Such a study will provide information about the cardiac-performance relationship in the presence of stress. Much of the early psychophysiological work with alcohol has methodological limitations. Additional studies done in a sophisticated manner could provide answers about alcohol effects and mechanisms which are presently unavailable or are only now beginning to bud.

CHAPTER V

SUMMARY

Effects of alcohol on tension have been studied in an attempt to determine if tension reduction plays a part in the etiology of excessive drinking. Alcoholics tend to be individuals who drink when faced with a problematic situation and/or failure. One of the goals of this study was to confront nonalcoholic individuals with a failure situation prior to drinking alcohol and then to determine if alcohol mitigated the effects of the prior failure. In order to implement this goal, failure or success set was experimentally induced in subjects prior to drinking by giving either a solvable or unsolvable disjunctive concept identification problem. Subjects then drank either a moderate dose of alcohol (1.32 ml./kg.) or a placebo and were tested again. Subjects were forty healthy male graduate and law students who were light to moderate social drinkers. Previous research shows that acute alcohol ingestion produces greater impairment during absorption (ascending limb of the blood alcohol curve) than during elimination (descending limb). A comparison of limb effects was made in this study at a blood alcohol level of 0.09% on both limbs of the blood alcohol curve. Four task measures were made: (1) trials to criterion; (2) errors to criterion; (3) percentage of errors for trials to criterion; and (4) mean response time. Arousal during the different conditions was measured by

psychophysiological indices. Measures included were mean frontal electromyographic activity (EMG), laryngeal EMG, skin conductance level (SCL), and number and amplitude of skin conductance responses (SCRs).

The following testing procedure was employed. Subjects completed consent forms and answered questions about their drinking histories. Then, the baseline psychophysiological measures were made during a rest period. A test period followed during which each subject completed the Shipley Institute of Living Scale and the Eysenck Personality Inventory. These tests were administered to determine if any significant differences in groups on intellectual functioning or personality variable which might account for experimental differences existed. No significant differences between groups were found. The initial concept identification task was then administered. Half of the subjects received an unsolvable problem (failure set), and the other half received a solvable problem (success set). Drinking followed with half of the subjects receiving alcohol and half a placebo. Breathalyzer readings were taken until alcohol subjects reached a BAL of 0.09% on the ascending limb. Half of the alcohol subjects rested at this point, and half received a concept identification task. Controls received corresponding treatment. At 0.09% of the descending limb of the blood alcohol curve, subjects were tested again in reverse order. Those who had rested on the ascending limb received the concept identification task and vice versa.

Efficiency on the concept identification task was significantly impaired after alcohol consumption, as indicated by significantly greater number of trials and errors and percentage of errors to trials for alcohol

Ss compared to placebo Ss. Mean response time was not significantly affected by alcohol. Although all measures showed a trend toward greater impairment on the ascending limb than on the descending limb, none of these differences was statistically significant. Failure set did not produce any significant differences from success set after drinking. The lack of set effects may possibly be due to state-dependent effects, task unfamiliarity, or placebo effects.

General arousal was significantly greater after alcohol than placebo as indicated by increased frontal EMG activity, and both a greater number and amplitude of SCRs during rest. Laryngeal EMG (a measure of covert oral activity), SCL, and number and amplitude of SCRs (indicators of increased arousal and reactivity) were all greater during CI than during rest. SCL, number and amplitude of responses were all significantly greater on the ascending limb than on the descending limb.

It was concluded that alcohol decreased the efficiency with which a disjunctive concept identification task was solved. Increased arousal and reactivity were seen after alcohol consumption. The results of this study do not support the tension reduction hypothesis.

Results were discussed in relation to other studies which have found decreased efficiency or no change after alcohol. The issue of alcohol being a stimulant or a depressant was discussed in relation to the psychophysiological findings indicating increased arousal and reactivity. Suggestions were made for future research.

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APPENDIX A

INSTRUCTIONS FOR SUBJECTS

INSTRUCTIONS FOR SUBJECTS

This is a study of the effects of alcohol on concept learning. The word concept is used to mean a rule for classifying a group of objects by their physical attributes. For example, a concept based on one attribute would be "face card". A deck of cards could be sorted into two piles: a) face cards, which belong to the concept, and b) non-face cards, which do not belong to the concept. A concept based on two attributes would be "red face card." In the same manner, the deck can be sorted into two piles: a) red face cards, and b) non-red face cards. In this study, we will not be using face cards but will be using slides containing geometric patterns instead. (The S will be shown two initial sample slides. The S will then be shown three demonstration slides and asked to name the attributes himself. The S will also be given an index card with dimensions and attributes on it.) There are 4 dimensions present in the slides being used. They are size, shape, number and color. Each dimension in turn has three attributes:

Size - Large, medium, or small objects
Shape - Squares, triangles, or hexagons
Number - 1, 2, or 3 objects
Color - Red, yellow, or blue

Now let's practice using the attributes. Suppose the two attributes, large and blue, are the ones of interest. A given slide can have both attributes, neither attribute, or one attribute but not another. (The S will be shown a set of practice slides and asked to tell if both attributes, large and blue, are present, neither is

present, or one attribute but not the other is present.) Note that a slide will fit into one of four categories: 1) Both present, 2) neither present, 3) A but not B, or 4) B but not A.

The concept you will be working with in this study will be based on two attributes. You need to have a rule which relates the attributes to each other. A simple rule would be A and B. Thus only slides which have both attributes fit the rule. Slides with neither or only one attribute would not fit the rule. Slides which fit the rule belong to the concept, and slides which do not fit the rule do not belong to the concept. Thus, to classify an object as belonging or not belonging to the concept, you need to know two things: the rule and the two attributes.

The rule you will be using in this study works this way. Slides which have both attributes and slides which have one attribute but not the other fit the rule and thus belong to the concept. Slides which have neither attribute do not fit the rule and do not belong to the concept. Note that we are always interested in two attributes. Some other attributes which we are not interested in are present, but we never use them to learn how to classify a slide. Here is a card with the rule listed on it. The subject will be given a card with the following information on it:

Rule

YES

Both A and B present

Either one but not the other present (A or B present)

NO

Neither A nor B present.)

You can see that slides which do not fit the rule will always have no relevant attributes. Slides which fit the rule have both relevant attributes or one relevant attribute. (The subject will then be given a set of practice slides. He will be told that the relevant attributes are medium and red. He will be asked to answer yes if the slide belongs to the concept and tell why or no if the slide does not belong to the concept and why.)

Now I am going to give you your first problem. A series of slides will appear on the screen in front of you. I am going to be using the same rule you have just been practicing and that also appears on your card. This time, however, I am not going to tell you the two attributes that I am using to classify the slides. Your job will be to guess what they are. The lights will tell you whether or not your guess is correct. You will begin by guessing which of these two buttons to press. If the light above the button you press comes on, you are correct. If the opposite light comes on, your guess is wrong. Soon you will be able to figure out which attributes are relevant and which are not. Then you should be able to tell correctly which slides belong to the concept and which slides do not belong to the concept. Try to be right as often as you can. Remember that slides that belong to the concept may or may not contain two relevant attributes. They may only have one or another of the relevant attributes. Slides that do not belong to the concept do not contain any relevant attributes.

By now you should understand the rule and how to gain information about the attributes. It is important to remember that slides belonging to the concept and slides not belonging to the concept all have valuable information. Are there any questions?

I am now going to begin the problem. You will not know which attribute I am using to classify the slides. The rule will be the same. When you think you know to which category the slide belongs, press the button. If you think that the slide is an example of the concept, press the button marked "Yes". If you think that the slide is not an example of the concept, press the button marked "No". If the slide contains the attribute, the "yes" light will come on. If it doesn't, the "no" light will come on. Then a new slide will appear. Eventually you should be able to figure out which of the two attributes you need to know to classify each slide.

APPENDIX B

ANALYSES OF VARIANCE SUMMARY TABLES

CONCEPT IDENTIFICATION TASK

SUMMARY TABLE FOR ANALYSIS OF VARIANCE
FOR RESPONSE TIME

Source	df	MS	F
Drug	1	0.0445	0.0192
Set	1	5.3079	2.2955
Limb	1	0.0003	0.0001
Drug x Set	1	1.3612	0.5887
Drug x Limb	1	0.7689	0.3325
Set x Limb	1	0.4289	0.1855
Drug x Set x Limb	1	1.8197	0.7870
Error (Within)	32	2.3123	
Total	39		

APPENDIX C

ANALYSES OF VARIANCE SUMMARY TABLES

PSYCHOPHYSIOLOGICAL DATA

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR FRONTAL EMG MEASURED DURING CI PERIODS

Source	df	MS	F
Drug	1	2402.500	0.606
Set	1	5336.097	1.347
Limb	1	108.900	0.027
Drug x Set	1	96.099	0.024
Drug x Limb	1	2102.500	0.531
Set x Limb	1	1562.500	0.394
Drug x Set x Limb	1	8702.500	2.197
Error (Within	32	3961.708	
Total	39		

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR
FRONTAL EMG MEASURED DURING CI AND REST
PERIODS ON THE ASCENDING LIMB

Source	df	MS	F
Drug	1	3802.500	0.773
Set	1	230.400	0.047
Period	1	57.600	0.012
Drug x Set	1	1464.099	0.297
Drug x Period	1	3422.500	0.695
Set x Period	1	336.400	0.068
Drug x Set x Period	1	4202.500	0.854
Error (Within)	32	4921.492	
Total	39		

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR
LARYNGEAL EMG MEASURED DURING REST PERIODS

Source	df	MS	F
Drug	1	19713.590	2.423
Set	1	1040.399	0.128
Limb	1	1144.899	0.141
Drug x Set	1	7344.097	0.903
Drug x Limb	1	2131.599	0.262
Set x Limb	1	14.400	0.002
Drug x Set x Limb	1	23716.890	2.915
Error (Within)	32	8136.652	
Total	39		

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR
LARYNGEAL EMG MEASURED DURING CI AND
REST PERIODS ON THE ASCENDING LIMB

Source	df	MS	F
Drug	1	24059.020	3.521
Set	1	4644.023	0.680
Period	1	3.025	0.001
Drug x Set	1	390.625	0.057
Drug x Period	1	3705.625	0.542
Set x Period	1	1030.224	0.151
Drug x Set x Period	1	2356.224	0.345
Error (Within)	32	6833.213	
Total	39		

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR
BASAL GSR MEASURED DURING CI PERIODS

Source	df	MS	F
Drug	1	15.625	0.675
Set	1	1.225	0.053
Limb	1	48.400	2.092
Drug x Set	1	0.625	0.027
Drug x Limb	1	1.600	0.069
Set x Limb	1	22.500	0.973
Drug x Set x Limb	1	2.500	0.108
Error (Within)	32	23.134	
Total	39		

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR
BASAL GSR MEASURED DURING CI AND REST
PERIODS ON THE ASCENDING LIMB

Source	df	MS	F
Drug	1	18.225	0.789
Set	1	3.600	0.156
Period	1	42.025	1.820
Drug x Set	1	4.225	0.183
Drug x Period	1	0.900	0.039
Set x Period	1	15.625	0.677
Drug x Set x Period	1	0.100	0.004
Error (Within)	32	23.091	
Total	39		

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR
BASAL GSR MEASURED DURING CI AND REST
PERIODS ON THE DESCENDING LIMB

Source	df	MS	F
Drug	1	2.756	0.146
Set	1	2.756	0.146
Period	1	2.256	0.120
Drug x Set	1	8.556	0.454
Drug x Period	1	2.756	0.146
Set x Period	1	13.806	0.733
Drug x Set x Period	1	1.406	0.075
Error (Within)	32	18.834	
Total	39		

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR
NUMBER OF GSRs DURING CI PERIODS

Source	df	MS	F
Drug	1	0.025	0.004
Set	1	5.625	0.874
Limb	1	9.025	1.402
Drug x Set	1	18.225	2.831
Drug x Limb	1	1.225	0.190
Set x Limb	1	5.625	0.874
Drug x Set x Limb	1	0.225	0.035
Error (Within)	32	6.438	
Total	39		

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR
AMPLITUDE OF GSRs MEASURED DURING
CI PERIODS

Source	df	MS	F
Drug	1	0.043	0.016
Set	1	0.700	0.255
Limb	1	0.002	0.001
Drug x Set	1	0.008	0.003
Drug x Limb	1	1.314	0.479
Set x Limb	1	5.162	1.882
Drug x Set x Limb	1	0.002	0.001
Error (Within)	32	2.743	
Total	39		

SUMMARY TABLE FOR ANALYSIS OF VARIANCE FOR AMPLITUDE OF GSRs MEASURED DURING CI AND REST PERIODS ON THE DESCENDING LIMB

Source	df	MS	F
Drug	1	0.001	0.001
Set	1	2.416	0.943
Period	1	7.132	2.784
Drug x Set	1	0.239	0.093
Drug x Period	1	0.891	0.354
Set x Period	1	2.416	0.943
Drug x Set x Period	1	2.416	0.943
Error (Within)	32	2.562	
Total	39		

APPENDIX D

DEMOGRAPHIC AND TEST DATA FOR EXPERIMENTAL SUBJECTS

DEMOGRAPHIC AND TEST DATA FOR ALCOHOL GROUP

Subjects	Age	Education	Weight (lbs.)	Height (in.)	Shipley-Hartford			Eysenck Personality Inventory		
					Vocab-ulary	Abstract	WAIS	E	N	L
1	23	17	185	69	32	38	121	16	2	2
2	25	19	160	73	29	30	108	16	10	1
3	22	16	229	72	32	40	123	9	12	2
4	27	20	157	72	39	40	125	14	4	3
5	23	17	157	72	32	28	111	12	6	1
6	29	19	162	71	35	20	105	12	13	3
7	27	21	144	66	33	32	113	15	0	0
8	32	18	218	70	34	24	108	18	12	0
9	25	20	177	70	38	32	117	15	4	2
10	31	19	168	71	36	30	114	16	4	2
11	26	19	157	70	33	30	112	20	5	1
12	25	19	177	70	35	30	113	10	11	3
13	32	19	268	76	33	36	117	14	15	2
14	25	19	166	68	32	32	113	14	7	3
15	35	20	265	72	32	32	113	11	15	2
16	31	18	189	70	33	32	113	15	5	1
17	26	20	194	72	36	38	121	14	21	0
18	29	18	220	72	32	38	117	16	16	0
19	21	17	145	66	30	36	117	15	18	0
20	25	17	137	70	27	30	107	18	11	1
Mean	26.95	18.60	183.75	70.60	33.15	32.40	114.40	14.50	9.55	1.45
S.D.	3.76	1.31	38.03	2.30	2.85	5.17	5.39	2.72	5.77	1.10

DEMOGRAPHIC AND TEST DATA FOR PLACEBO GROUP

Subjects	Age	Education	Weight (lbs.)	Height (in.)	Shipley-Hartford			Eysenck Personality Inventory		
					Vocab-ulary	Abstract	WAIS	E	N	L
1	34	19	216	74	32	36	116	14	5	2
2	24	19	147	66	31	36	118	9	10	1
3	24	16	164	74	38	40	128	17	9	1
4	29	20	168	70	24	34	108	12	9	3
5	35	20	186	70	38	40	124	9	10	0
6	25	18	143	68	36	36	119	11	8	0
7	25	19	190	74	34	30	113	15	7	2
8	28	19	203	70	34	36	117	7	8	1
9	28	18	160	68	32	31	112	14	8	1
10	25	18	184	72	32	35	115	15	9	2
11	24	19	167	68	34	32	117	14	12	1
12	23	17	152	68	31	36	118	14	12	2
13	28	20	174	73	32	34	114	9	20	0
14	32	19	187	72	32	28	109	14	17	2
15	25	19	151	72	31	36	115	13	10	3
16	25	19	151	69	32	28	109	13	3	2
17	27	18	184	71	27	28	105	15	6	5
18	26	20	137	69	33	40	120	16	9	0
19	25	18	147	68	40	40	125	17	7	0
20	33	16	246	79	34	38	119	10	8	2
Mean	27.25	18.55	172.85	70.75	32.85	34.70	116.05	12.90	9.35	1.50
S.D.	3.61	1.19	27.38	3.06	3.60	4.05	5.84	2.86	3.81	1.28

CONCEPT IDENTIFICATION DATA FOR ALCOHOL GROUP

Subjects	Condition	Number of Trials	Number of Errors	Percentage of Errors	Mean Reaction Time
1	S x DL	71	17	23.9	1.922
2	F x AL	160	77	48.1	5.281
3	F x DL	29	5	17.2	3.250
4	S x AL	65	19	29.2	1.302
5	F x DL	50	19	38.0	3.480
6	F x AL	111	45	40.5	2.708
7	S x DL	160	87	54.4	1.194
8	S x DL	65	20	30.8	1.869
9	F x DL	160	78	48.8	2.811
10	F x DL	48	18	37.5	3.658
11	S x AL	85	24	28.2	1.839
12	F x AL	160	81	50.6	4.098
13	S x AL	82	31	37.8	1.617
14	S x DL	122	44	36.1	2.614
15	S x AL	98	27	27.6	3.980
16	S x AL	160	72	45.0	5.217
17	F x AL	86	19	22.1	2.904
18	F x AL	160	70	43.8	1.284
19	F x DL	45	19	42.2	4.830
20	S x DL	67	20	29.9	1.775
Mean		99.2	39.6	36.59	2.882
S.D.		28.2	13.1	10.29	1.311

CONCEPT IDENTIFICATION DATA FOR PLACEBO GROUP

Subjects	Condition	Number of Trials	Number of Errors	Percentage of Errors	Mean Reaction Time
1	S x DL	22	2	9.1	1.603
2	S x AL	63	19	30.2	2.412
3	F x DL	30	7	23.3	2.463
4	F x AL	33	10	30.3	3.227
5	S x DL	160	40	25.0	0.921
6	F x AL	48	12	25.0	2.903
7	F x AL	64	13	20.3	1.825
8	F x DL	65	13	20.0	1.981
9	S x DL	77	25	32.5	3.743
10	F x DL	36	10	20.0	2.304
11	F x AL	92	22	23.9	3.845
12	S x AL	160	40	25.0	2.853
13	S x AL	38	9	23.7	2.710
14	S x DL	105	34	32.4	2.343
15	F x DL	99	22	22.2	7.609
16	F x DL	56	13	23.2	1.415
17	S x AL	160	43	26.9	3.376
18	S x DL	59	8	13.6	6.462
19	F x AL	33	5	15.2	3.711
20	S x AL	82	22	26.8	1.263
Mean		74.1	18.5	23.82	2.949
S.D.		22.4	6.9	6.05	1.639

PSYCHOPHYSIOLOGICAL DATA FOR ALCOHOL GROUP

Sub-jects	Condition	Frontal EMG		Laryngeal EMG		Basal SCL		Number of SCRs		Amplitude of SCRs	
		AL	DL	AL	DL	AL	DL	AL	DL	AL	DL
1	S x DL	172	145	192	162	8.0	7.0	6	0	1.83	0.00
2	F x AL	289	91	307	94	14.0	9.5	0	0	0.00	0.00
3	F x DL	121	182	130	116	6.5	6.0	1	0	2.25	0.00
4	S x AL	92	173	165	165	16.0	12.0	2	1	4.75	1.50
5	F x DL	157	303	195	678	13.5	14.5	4	6	2.37	2.16
6	F x AL	137	114	359	290	4.0	2.0	1	0	1.00	0.00
7	S x DL	186	204	106	265	5.5	4.5	0	0	0.00	0.00
8	S x DL	141	166	121	184	5.0	4.5	0	0	0.00	0.00
9	F x DL	156	146	132	189	3.0	4.0	0	1	0.00	1.00
10	F x DL	374	177	428	270	14.0	13.5	2	5	1.50	3.30
11	S x AL	232	402	307	443	11.0	7.0	4	0	2.25	0.00
12	F x AL	117	79	185	150	12.0	9.5	5	1	2.00	1.50
13	S x AL	125	201	112	201	10.0	6.0	0	0	0.00	0.00
14	S x DL	263	226	329	172	10.0	10.0	6	6	3.00	2.00
15	S x AL	140	169	203	138	15.0	11.0	5	0	1.35	0.00
16	S x AL	154	236	235	219	9.5	7.0	3	1	1.50	1.75
17	F x AL	229	159	132	114	7.5	6.0	1	0	1.50	0.00
18	F x AL	187	168	152	184	18.5	14.0	7	4	3.00	3.50
19	F x DL	100	122	177	261	11.0	12.0	0	2	0.00	2.75
20	S x DL	193	143	149	184	15.0	18.0	4	2	2.13	2.50
Mean		178.3	180.3	205.8	223.9	10.5	8.9	2.6	1.5	1.52	1.10
S.D.		70.0	73.0	92.1	32.5	4.3	4.2	2.4	2.1	1.29	1.26

PSYCHOPHYSIOLOGICAL DATA FOR PLACEBO GROUP

Sub-jects	Condition	Frontal EMG		Laryngeal EMG		Basal SCL		Number of SCRs		Amplitude of SCRs	
		AL	DL	AL	DL	AL	DL	AL	DL	AL	DL
1	S x DL	88	95	138	160	9.5	10.0	0	6	0.00	2.33
2	F x AL	153	84	111	89	4.0	5.0	0	0	0.00	0.00
3	F x DL	186	205	209	121	6.0	6.0	1	0	1.00	0.00
4	S x AL	146	66	148	95	7.0	6.0	0	0	0.00	0.00
5	F x DL	150	170	223	180	4.0	5.0	0	1	0.00	1.00
6	F x AL	205	150	239	290	2.0	2.0	0	0	0.00	0.00
7	S x DL	67	63	180	176	9.0	6.5	2	0	2.25	0.00
8	S x DL	117	87	129	145	5.0	5.0	0	1	0.00	7.50
9	F x DL	259	108	313	212	7.0	7.0	1	0	1.00	0.00
10	F x DL	195	255	238	161	5.0	4.0	2	0	1.75	0.00
11	S x DL	122	174	129	251	11.0	9.0	2	2	1.38	1.75
12	F x AL	186	89	106	74	16.0	10.5	7	0	1.68	0.00
13	S x AL	119	142	161	126	12.0	14.0	6	3	1.88	1.75
14	S x DL	91	120	91	114	4.5	4.0	0	0	0.00	0.00
15	S x AL	152	246	83	126	17.0	21.0	0	5	0.00	3.40
16	S x AL	110	119	146	205	9.0	9.0	0	0	0.00	0.00
17	F x AL	313	164	155	109	12.0	9.0	3	0	1.92	0.00
18	F x AL	135	109	91	96	13.0	15.5	0	5	0.00	2.45
19	F x DL	251	151	143	104	15.0	11.0	4	0	1.81	0.00
20	S x DL	130	151	102	94	13.0	11.0	7	1	2.15	1.75
Mean		159.8	137.4	156.8	146.4	9.1	8.5	1.8	1.2	0.79	1.10
S.D.		62.4	54.5	60.5	58.0	4.4	4.6	2.4	2.0	0.93	1.86