A DOUBLE-BLIND INVESTIGATION OF COGNITIVE FACTORS IN MUSCULAR BIOFEEDBACK

By

NANCY HALL MCSWAIN

Bachelor of Arts
Huntingdon College
Montgomery, Alabama
1969

Master of Science
Oklahoma State University
Stillwater, Oklahoma
1971

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of the Oklahoma State University
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Thesis Approved:

Thesis Adviser

Dean of the Graduate College

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TABLE OF CONTENTS

Chapter Page

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

Biofeedback is a process through which one learns voluntary control over automatic, reflexly regulated body functions. The term "biofeedback" was conceived to describe the process of feeding back physiological information to the individual generating the information. This technique is essentially one in which a selected physiologic activity is monitored by an instrument which detects, by electrodes or transducers, physiological signals such as heart rate, blood pressure, muscle tension or brain waves. These signals are amplified to activate a display that reflects changes in the physiologic activity.

The basic elements of the biofeedback process are as follows:

(1) the selection of a physiologic function,

(2) an instrument recording the activity of this function,

(3) presentation of this biological information to the individual in the form of auditory or visual signals, and

(4) an implicit intention to change this physiologic activity and utilization of the information for this purpose.

(5) However, the actual change which occurs in the biologic function is due to an as yet unexplained mechanism (Brown, 1977).

As Budzynski (1973) states, biofeedback training has three major goals: (1) the development of increased awareness of the relevant internal physiological functions; (2) the establishment of control over

those functions; and (3) the transfer or generalization of that control to situations outside the experimental setting.

Several studies have supported the effectiveness of veridical (true) electromyographic (EMG) biofeedback in the attainment of deep relaxation (Budzynski and Stoyva, 1969; Canter, Kondo, and Knott, 1975; Townsend, House, and Addairo, 1975; Le Boeuf, 1977; Green, Walters, Green, and Murphy, 1969; Raskin, Johnson, and Rondestveldt, 1972; and Coursey, 1975) and an improvement in anxiety symptoms (Canter, Kondo, and Knott, 1975; Townsend, House, and Addario, 1975; Le Boeuf, 1977; and Coursey, 1975). Others have reported a reduction in tension headaches through the utilization of EMG feedback (Philips, 1977; Budzynski, Stoyva, and Adler, 1970; and Budzynski, Stoyva, Adler, and Mullaney, 1973). However, Alexander (1975) reported no change in subjective feelings of relaxation and no generalization of EMG reductions from one muscle to the other muscles.

Both Brown (1977) and Budzynski (1973) emphasize the essential role of veridical feedback of frontalis muscle activity to achieve a reduction in tension and/or anxiety symptoms. In many studies, the utilization of irrelevant or pseudofeedback has effected no significant changes in the frontalis muscle, either positive or negative, or in self-reported symptoms (Budzynski and Stoyova, 1969; Philips, 1977; and Budzynski, Stoyva, Adler, and Mullaney, 1973).

Although these researchers have reported insignificant results with false EMG feedback, it is possible that their findings were due to variables other than the irrelevant feedback. Perhaps the subjects became less motivated or irritated as they perceived no change in the rate of clicks or in their level of relaxation. Or, in studies utilizing a

low constant tone for pseudofeedback, perhaps they became bored and uninterested. Thus, it is possible that these subjects were manifesting a lack of motivation rather than responding to nonveridical feedback. A more powerful control would have been a manipulation of the subject's cognitions so that they perceived the EMG feedback to be veridical feedback of an increasingly more relaxed physiological state over sessions. A study utilizing double-blind procedures by Cohen, Graham, Fotopoulos, and Cook (1977) appears to have implemented this procedure. The subjects were 29 opiate addicts who received 14 sessions of contingent or non-contingent EMG biofeedback training for symptom reduction during detoxification. For the non-contingent feedback, these experimenters used tape recordings of biofeedback signals generated by four previously successful subjects who received contingent feedback. Analysis of the results indicated that the manipulation was successful. Subjects in the non-contingent as well as the contingent feedback groups experienced subjective feelings of control over the feedback variables. No differences in therapeutic outcome were discovered. However, the contingent subjects demonstrated more control over EMG activity. This study would seem to support the important contribution of cognitive factors in the successful outcome of biofeedback therapy.

Miller and Dollard (1941), in their description of a mediational view of emotional arousal, state that fear or anxiety reactions may often be elicited by an individual's cue-producing response, i.e., his label to a given situation rather than the objective stimulus properties of the situation itself. Furthermore, they contend that modifying the label that the individual attaches to a situation, then one should be able to change his emotional reaction.

Schachter and Singer (1962), in an ingenuous experiment, manipulated states of physiological arousal along with an individual's cognitions about those states. Subjects were injected with either epinephrine or a saline solution and placed in a room with either a euphoric or an angry stooge. Those individuals injected with epinephrine were then divided into three separate groups. One group was given veridical information, another was given false information and the third group was told that no side effects from the injection of epinephrine would be experienced. The placebo subjects were also told that no side effects would occur. Subjects who were informed about the specific effects of epinephrine were significantly less euphoric or angry than those who were either misinformed or ignorant about the effects of the drug. These researchers concluded that an emotional state may be considered a function of a state of physiological arousal and cognitions appropriate to this state of arousal.

Although Schachter and Singer's (1962) study supported their hypotheses, the differences between the placebo and epinephrine subjects were barely statistically significant. Perhaps, Schachter and Wheeler (1962) theorized, these results were due to the self-arousal of the sympathetic nervous system by the placebo subjects. To test this hypothesis, they compared subjects who received either an injection of epinephrine, a placebo or chlorpromazine. If sympathetic nervous system activity is a necessary component of an emotional experience, Schachter and Wheeler (1962) anticipated the following: Whatever the experimentally manipulated emotional state, it should be most intensely experienced by subjects who have received epinephrine, next by placebo subjects and least of all by those injected with chlorpromazine. Ratings of amusement

for all subjects were made during a funny movie. As predicted, epinephrine subjects were more amused than chlorpromazine subjects and chloropromazine subjects were more amused than placebo subjects. Therefore, these results support the assumption that a state of sympathetic arousal is a necessary component of an emotional experience.

Other investigators have examined the influence of cognitive patterns upon physiological reactivity. Sternbach (1962) discovered that manipulation of various instructional sets altered subject's reports of autonomic activity. In another experiment looking at the effect of self-verbalizations upon emotional arousal, Rimm and Litvak (1969) found that subjects tended to show greater emotional responsiveness to sentences of affective nature than to neutral sentences. May and Johnson (1973) using inhibitory, neutral and arousal thoughts discovered that these internally evoked stimuli produce physiological changes. Furthermore, the direction of the change is partially dependent upon the affective nature of the cognitions. Schwartz (1975) reported that self-induced thoughts are not only capable of acting as stimuli for heart rate changes but also have response characteristics. Therefore, this research seems to support a conclusion that changing the individual's cognitions or set of self-instructions can have direct physiological effects.

Valins (1966) investigated the effects of false heart rate feedback upon rated attractiveness of semi-nude females. When the male subjects were shown slides of the females, half of them heard their heart rates increase to some of the slides while the other subjects heard their heart rates decrease to half of the presented slides. Valins hypothesized that if cognitive representations of internal events are important

for emotional behavior, then these "bogus" heart rates or nonveridical representations of physiological changes should have the same effects as true heart rate or veridical representations. The results supported Valins' hypothesis: The slides to which the subjects heard a definite change in their supposed heart rate, whether increased or decreased, were rated significantly more attractive in two post-tests and these slides were chosen as remuneration for experimental participation significantly more often than the other slides. In a replication of this experiment with emotional and unemotional subjects, Valins (1966) obtained similar results utilizing, in addition to the post-test, a two month follow-up.

In a more stringent test of the hypothesis that cognitive representations of internal events, whether veridical or nonveridical, should have similar effects upon emotional states, Valins and Ray (1967) presented slides of snakes and slides with the word "shock." Also, he shocked the subjects at the same time a "shock" slide was presented. This group also received false heart rate feedback which increased to the slides of shock and decreased to the slides of snakes. For the control subjects, the procedure was the same except they were told that the sounds they heard were meaningless sounds. Valins hypothesized that cognitions concerning one's physiological reactions will affect avoidance behavior. Therefore, those subjects who believe that snake stimuli do not affect them internally will consider their fear of snakes unfounded. Consequently, they should manifest more approach behavior toward a live snake than controls. All subjects were then given an avoidance task and experimental subjects showed a nonsignificant trend for greater approach behavior. However, when those individuals with previous experience with snakes were eliminated from the analyses,

Valins found significantly more approach behavior by experimental subjects. From this study, Valins and Ray (1967) concluded that avoidance behavior can be modified by information concerning internal reactions. Subjects who thought that snake stimuli did not affect them internally were more likely to hold a live snake than those individuals who had no information about their internal reactions.

Although most of the research in biofeedback emphasizes the essential role of veridical feedback to achieve a state of relaxation, reduction in anxiety symptoms and/or tension headaches, other studies point out the importance of cognitive factors in one's physiological pattern of reactions. In fact, some researchers (Sternbach, 1964; Rimm and Litvak, 1969; May and Johnson, 1973; Schwartz, 1975) have found that altering one's cognitions affects one's physiological and emotional reactivity. Valins (1966) and Valins and Ray (1967) manipulated subjects' perceptions of internal physiological reactions and found significant differences between experimentals and controls in behavior and attractiveness ratings of semi-nude females. Therefore, in any emotional state, either arousal or relaxation, there appear to be two primary components, i.e., a physiological pattern of reactivity and cognitions about one's physiological state. In previous feedback studies, the role of cognitive factors has been largely ignored. This study is an attempt to manipulate cognitions about one's physiological state in an effort to learn more about the role of cognitive factors in the process of biofeedback.

The Present Study

In this study, it is hypothesized that a reduction in levels of

muscle tension may be irrelevant to the experience of relaxation. As long as an individual feels or thinks that he/she is relaxed, then his/her actual level of muscle tension may be unimportant. In other words, a low level of muscle tension may not be essential to a subjective experience of calm and relaxation.

To test this hypothesis, three separate conditions were examined:

(1) true EMG feedback with relaxation instructions,

(2) true EMG feedback without relaxation instructions, and

(3) false decreasing tone EMG feedback with relaxation instructions.

This experimental design will enable one to separate the effects of expectancy from the effects of biofeedback training in the reduction of muscle tension and attainment of relaxation. With the false decreasing tone group, manipulation of a strong expectancy effect is anticipated as this group will be receiving non-veridical information about their level of muscle tension. If belief of the false EMG signal occurs, then a decrease in muscle tension will be acknowledged which may facilitate a state of calmness and relaxation. The first group will receive true EMG feedback plus an expectation of increased calmness and deep relaxation. Thus, this group will receive both manipulations. For the true biofeedback group without relaxation instructions, the expectancy effect will be attenuated. Although they will receive veridical EMG feedback, the expectation of increased relaxation and decreased tension and anxiety will be eliminated.

Consequently, it is hypothesized that feedback of a combined measure of muscle tension from the frontalis and the forearm flexor muscles as well as expectancies about the treatment are both important factors in biofeedback training. Furthermore, it is postulated that a state of

deep relaxation may be experienced without a significant learned reduction in EMG measures. In other words, one may experience relaxation as a result of expectations to relax with no significant reduction in EMG levels, as, with the false decreasing tone group, no opportunity is provided to learn to change one's levels of muscle tension. Another measure of one's general level of sympathetic arousal, the galvanic skin response, was utilized to examine the strength of the false decreasing tone manipulation in its physiologically relaxing effects. In other words, one's belief in a lower level of muscle tension should decrease the level of GSR, if the generalization hypothesis that a change in one physiological system tends to spread to other systems, is supported. It should also facilitate the experience of relaxation, but would not necessarily produce a linear decrease in EMG levels of muscle tension.

Hypotheses

It is hypothesized that the true EMG feedback group with relaxation instructions will show a significantly more efficient tension reduction in EMG levels, i.e., a greater linear trend across time than the other two groups plus a significant reduction in GSR levels and State-Trait Anxiety Inventory-A-State (STAI-A-State) scores.

The second hypothesis is that the true EMG feedback group without relaxation instructions will show a significantly greater reduction in muscle tension than the false decreasing tone group, but no significant reduction in GSR levels or in STAI-A-State scores.

The third hypothesis is that the false decreasing tone group with relaxation instructions will show a significant reduction in the

STAI-A-State scores and GSR levels, but the least amount of learned EMG reduction.

CHAPTER II

METHOD

Subjects

Twenty-four female subjects were selected from introductory psychology courses on the basis of their scores on the Fenz-Epstein Modified Anxiety Scale. The students who attained scores which indicate low levels of muscle tension (mean score of 1.25 or less) were chosen to participate in the study. This is minus one standard deviation below the mean for females (Fenz and Epstein, 1965). Previous research indicates that females are generally more compliant than males in their interactions with authority figures (Macoby and Jacklin, 1974). Therefore, females were selected for this study to facilitate belief in and consequent compliance with the experimental procedures in order to significantly differentiate among the three treatment groups. The subjects ranged in age from approximately 18 to 40 years. Due to the loss of four subjects during the first run of the experiment, it was necessary to schedule a second run. For this run, eight subjects were trained from which four were randomly selected. For the GSR data, the number of subjects was reduced to 18 due to the breakdown of the Autogen 3400 Feedback Dermograph.

Instruments

The Fenz-Epstein Modified Anxiety Scale (Fenz and Epstein, 1965) was given to all subjects participating in the experiment. This instrument has three subscales: One scale contains 16 items related to symptoms of autonomic arousal which refers to visceral symptoms associated with activation of the autonomic nervous system. Some of these items refer to tachycardia, vasomotor reactions, emotionally induced sweating, failure of body temperature control and digestive disorders. The second scale of 18 items is concerned with symptoms of muscular tension which refer to the effects of sustained contraction of striated or voluntary muscle. Items include references to tremor, motor incoordination, backache, rapid breathing, pressure headaches and skin sensitivity. The last scale of 19 items involves subjective feelings of fear and insecurity which refer to the inability to concentrate or relax, the tendency to worry excessively over trifles, unexplained feelings of fear and panic, fitful sleep, compulsive mannerisms and stated feelings of insecurity.

The scale was given to 52 female and 46 male undergraduates at the University of Massachusetts. Odd-even reliability coefficients were computed independently for each scale and corrected for attenuation. A reliability coefficient of .83 was obtained for autonomic arousal; .84 for striated muscle tension; and .85 for feelings of anxiety (Fenz and Epstein, 1965).

Pre- and post-treatment measures of subjective states of anxiety were examined with the State-Trait Anxiety Inventory, A-State (STAI-A-State) scale (Spielberger, Gorsuch, and Lushene, 1970). The STAI-A-State

scale consists of 20 statements to which subjects respond with their particular feelings at a specific moment in time. The authors define state anxiety as a transitory emotional state characterized by subjective, consciously perceived feelings of tension and apprehension and heightened autonomic system activity. Furthermore, A-States may vary in intensity and fluctuate over time. According to Spielberger, Gorsuch, and Lushene (1970), scores on the A-State scale increase in response to various kinds of stress and decrease after relaxation training. Test-retest reliability coefficients, obtained from a sample of undergraduate college students, ranged from .16 to .54 with a median correlation coefficient of .32. However, these low coefficients were anticipated with the A-State of the STAI as it reflects various situational factors present at the time of testing as well as subjective states. Because of the transitory nature of anxiety states, measures of internal consistency such as the alpha coefficient would most likely produce a more meaningful index of the reliability of the A-State scale than test-retest correlations. Internal reliability coefficients ranged from .83 to .92 in a sample of high school and college students. A measure of the construct validity for the A-State scale was computed after more than 900 college students were administered the scale under two different conditions, NORM (with standard instructions) and EXAM (before exam). The mean score for the A-State scale was considerably higher in the EXAM (57.75) condition than the NORM (39.69) condition (Spielberger, Gorsuch, and Lushene, 1970). In another validation study, 197 college students were given the STAI-A-State scale under four experimental conditions: Normal, Relax, Exam, and Stressful Movie. The mean score for the STAI-A-State scale was lowest in the Relax condition and highest

after the stressful film (32.70 to 50.03), respectively (Lazarus and Opton, 1966).

Apparatus

Electromyographic (EMG) measures were recorded from an Autogen 1700 Feedback Myograph using standard frontalis placement two inches on either side of center forehead and one inch above each eyebrow. Midway between these electrodes, a ground electrode was placed upon the forehead. Two other electrodes for the standard forearm flexor placements were also attached (Venables and Martin, 1970). The subject received auditory feedback of muscular tension on an interval schedule through headphones which are connected to the Autogen unit. The feedback is in the form of clicks which are logarithmically proportional to the level of EMG activity being monitored.

GSR (galvanic skin response) measures were recorded from an Autogen 3400 Feedback Dermograph. The two active silver/silver chloride electrodes were placed on the second and third fingertips of the non-dominant hand. The ground electrode was placed on the index finger of the non-dominant hand. The electrodes were held in place with the use of velcro fasteners.

Procedure

In an attempt to separate the active and placebo components of this study, a double-blind design was used. This design controlled for the expectations and biases of both the subject and the experimenter as transmitted overtly or covertly through the procedures, interactions, and experimental setting. Before the experimental training sessions,

all subjects were informed, verbally and in writing, that this was a biofeedback experiment in which they would be gaining control over their particular physiological pattern. No information was given about the type of biofeedback training to be utilized in the study or the specific physiological responses monitored. The lack of male subjects was explained as due to the differences in patterns of physiological activity for males and females. Furthermore, subjects were requested not to question their experimenter during the training sessions, and were given blank sheets to write down any questions at the end of each session. These questions were forwarded to an individual not participating in the experimental procedures.

The experimenter utilized identical procedures for all subjects. An individual not actively involved in the experiment randomly assigned each subject to one of the three treatment conditions: EMG with relaxation instructions (Group 1), EMG without relaxation instructions (Group 2), or the false decreasing tone group with relaxation instructions (Group 3). The experimenter was given only the subject's name and the tape code to be used with that subject.

Three sets of tapes, one for each of the three groups, were made. Each set of tapes included four separate tapes. The first tape was used for Sessions 1 and 2, the second tape for Sessions 3 and 4, the third tape for Sessions 5 and 6, and the fourth tape for Sessions 7 and 8. The instructions for Groups 1 and 3 were identical. The instructions for Group 2 did not include relaxation expectancies. After the instructions, the tapes for Groups 1 and 2 were blank as they were receiving true EMG feedback. The tapes for Group 3, after the instructions, included decreasing tone feedback at the rate of 180-120, 150-90,

120-60, 90-30 clicks per minute for tapes 1, 2, 3, and 4, respectively.

At the beginning of the first experimental session, subjects were asked to complete the State-Trait Anxiety Inventory, A-State scale. Then they were seated in a comfortable chair and asked to relax themselves with both legs and arms uncrossed. EMG electrodes were attached at the standard frontalis and forearm flexor placements. GSR electrodes were placed on the first three fingertips of the non-dominant hand. Subjects were instructed to sit quietly while baseline data was recorded. Levels of muscle tension in microvolts were recorded from the frontalis and forearm flexor muscles in combination and individually. A baseline GSR in average ohms resistance was also recorded. The earphones were then placed on the subject's head at which time the appropriate tape recorded instructions were initiated. Instructions for the true EMG feedback group with relaxation instructions and the false decreasing tone feedback group were as follows:

> This is an experiment on the effects of biofeedback upon an individual's physiological pattern of responses. Through the earphones, you will hear a series of clicks. As you decrease the number of clicks, you will be gaining control over your particular psychological pattern which will facilitate your becoming more relaxed. We have found that the following procedures generally produce the most relaxation. Let yourself begin to feel quite relaxed. Close your eyes. Try not to blink, swallow or move your face but let it feel heavy and sagging. Breathe deeply and rhythmically. Try to settle into a daydreamy type of state. Let relaxing images come into your mind. These machines are quite sensitive and often record not only your physiological pattern, but also bodily movements. To control for these movements, we have placed electronic filters on the machines which screen them out. However, occasionally, the bodily movements will override the filters. At this time, you will hear an increase in the clicks. Periodically, throughout the session, there will be silent periods in which we will be recording different physiological measurements. Therefore, try to remain as still as possible during the session. The session will last approximately 21 minutes.

The instructions for the true EMG feedback group without relaxation instructions were as follows:

> This is an experiment on the effects of biofeedback upon an individual's physiological pattern of responses. Through the earphones, you will hear a series of clicks. As you decrease the number of clicks, you will be gaining control over your particular physiological pattern. These machines are quite sensitive and often record, not only your physiological pattern, but also bodily movements. To control for these movements, we have placed electronic filters on the machines which screen them out. However, occasionally the bodily movements will override the filters. At this time, you will hear an increase in the clicks. Periodically, throughout the session, there will be silent periods in which we will be recording different physiological measurements. Therefore, try to remain as still as possible during the session. The session will last approximately 21 minutes.

After the electrodes were attached and the baseline measurements for the EMG levels for the forearm flexor and frontalis muscles, individually and in combination, along with the GSR levels, were recorded, the experimenter placed the correct tape in the tape recorder, set the switch for instructions, turned the sound switch on and turned on the tape recorder for the pre-recorded instructions. After the instructions, the experimenter activated one of three combinations of switches, either BC, CD, or AD and the switch for training. Two of these combinations, BC and CD, initiated EMG feedback from the Autogen 1700 Feedback Myograph for Groups 1 and 2. The third combination activated the taped decreasing tone feedback for Group 3. Each subject, in the EMG feedback group, received six two-minute periods of feedback with one-minute rest intervals between them. The taped false feedback was presented on a similar schedule.

The sound from the tape recorder and the Autogen 1700 Feedback Myograph were both wired into a volume control switch. This was done

so that if a subject stated that the sound was too low, the volume control switch could be changed and the experimenter would not know whether the sound was being regulated on the tape recorder or the Autogen 1700. The tape recorder played continuously. Thus, the experimenter was unaware as to whether the subject was receiving false feedback from the tape recorder or EMG feedback from the Autogen 1700.

The experimenter monitored four physiological measures for all subjects throughout the training sessions. Each physiological measure was recorded during each of the seven three-minute trials. The Autogen 5100 Digital Integrator was used during the first two minutes of each trial to reflect the average amplitude of the EMG levels in microvolts. The 5100 Integrator combined the frontalis and forearm flexor EMG signals and, thus, reflected the average level of muscle tension for them over a two-minute period.

Three other measures were taken during the third minute of each trial. The average EMG levels in microvolts from the frontalis and forearm flexor muscles were recorded separately over a 15-second period. The GSR, in average ohms resistance, was also recorded. Therefore, the experimental procedures for each subject in each condition were identical in order to implement the double-blind design.

At the conclusion of the initial session, all subjects were scheduled for seven experimental sessions of 21 minutes each to extend over a four-week period. Sessions were scheduled with at least one day intervening between them. Subjects were also asked to complete a short questionnaire about strategies utilized to decrease the number of clicks during each session. At the end of the four-week period, only 20 subjects had completed the eight training sessions. Therefore, a

second run of the experiment was scheduled. Eight subjects were trained from which four were selected, one subject for two of the treatment groups and two subjects for the third group.

After the subjects had completed all training sessions, they were asked to respond to the State-Trait Anxiety Inventory, A-State scale again. In addition, information about the nature of the experiment was provided. Any questions the subjects had about their performance were also answered.

With the completion of all the experimental sessions, the cognitive strategies utilized by the subjects were tallied and a frequency distribution made. Suspicion of false feedback by the subjects was determined and a X^2 analysis of this data was performed.

Four of the six trainers knew nothing about the use of false feedback for one of the treatment conditions. Furthermore, no details about the hypotheses or the experimental design were known. Two of the six trainers knew about the experimental design and hypotheses but were uninformed about the meaning of the codes. Questioning of the experimenters revealed only one of the two who knew of the experimental design had decided which group was given false feedback. Another X^2 analysis of this data was computed.

Design

Independent Variables

The independent between subjects variable is treatment groups. The EMG feedback plus relaxation instructions provided the condition of cognitive expectancy as well as physiological learning. The EMG

feedback without relaxation instructions provided the condition of physiological learning alone. The false decreasing tone placebo group provided the condition of cognitive expectancy alone.

Two within subjects independent variables were eight sessions and seven trials within each session for the physiological data. Another within subjects variable was pre- and post-scores on the STAI-A-State scale.

Dependent Variables

Five dependent variables were utilized in this study. EMG levels of muscle tension in average integral microvolts were recorded from the frontalis and forearm flexor, individually and in combination. GSR measures in average ohms resistance and change scores from the State-Trait Anxiety Inventory, A-State scale were also analyzed.

The data were analyzed in three separate analyses of variance (ANOVA). The first ANOVA, on EMG levels of muscle tension (one for each of the three EMG dependent measures), involved one between subjects variable (Groups-3) and two within subjects variables (Sessions-8 and Trials-7). The ANOVA computed on the GSR levels had the same design as the one for the EMG levels of tension. The third major ANOVA, on the State-Trait Anxiety Inventory, A-State scores, was a one between subjects variable (Groups-3) and a one within subjects variable (pre- and post-scores-2).

CHAPTER III

RESULTS

Introduction

Results will be presented in six separate sections. The first section will present the combined EMG results which include the forearm flexor muscle (EMG-A), and the frontalis muscle (EMG-F). The second and third sections will discuss the EMG-F and EMG-A data respectively. The fourth is a presentation of the GSR results. In the fifth section, an analysis of the STAI-A-State scores will be presented. The sixth section examines the subject's cognitive strategies.

EMG

A mixed ANOVA on Treatments (3) x Sessions (8) x Trials (7) was performed on the EMG combined measures which included the forearm flexor and the frontalis muscle. The between subjects variable was the treatment groups of false feedback with relaxation instructions, EMG feedback with relaxation instructions and EMG feedback without relaxation instructions, and the within subjects variables were the eight treatment sessions and the seven trials within each session.

There was no significant main group effect on the EMG combined data indicating that the different treatments did not significantly effect the muscle tension levels of the three groups ($\underline{F}$ (2, 21) = 1.11).

However, there was a significant main effect for sessions, F (7, 147) = 3.17, $p < .003$, and trials, F (6, 126) = 23.62, $p < .0001$. The main sessions effect was based on a general reduction in combined muscle tension levels from session 1 at 1.99 microvolts (mv) to session 4 at 1.48 mv. The main trials effect is based upon the very large drop in muscle tension from trial 1 at 2.24 mv to trial 2 at 1.59 mv. From trial 2 to trial 7, the combined muscle tension levels changed only .07 mv. These significant main effects indicate that change was occurring across trials within sessions and from session to session across time. None of the interaction effects for the EMG combined measures were significant although the interaction of treatments with sessions and with trials did approach significance, F (84, 882) = 1.26, $p < .06$. Because of the interest in the cognitive as well as the physiological aspects of biofeedback and separate performance of the three treatment groups, planned simple effects tests were performed.

For the EMG feedback group with relaxation instructions, significant sessions (F (7, 49) = 3.35, $p < .005$), and trials (F (6, 42) = 9.88, $p < .0001$) effects were found. A look at either the graph for Session means (Figure 1) or at the Table of Trial and Session Means for this group (Table XXI), it is apparent that there is a rather consistent drop in muscle tension levels across both trials and sessions. Furthermore, Figure 1 shows that much of the reduction occurs within the first two or three sessions. Any other changes in levels of muscle tension are minimal and predominantly help to stabilize the change that occurs rather early in the process of biofeedback. Figure 2 depicts a tremendous reduction in muscle tension levels from trial 1 to

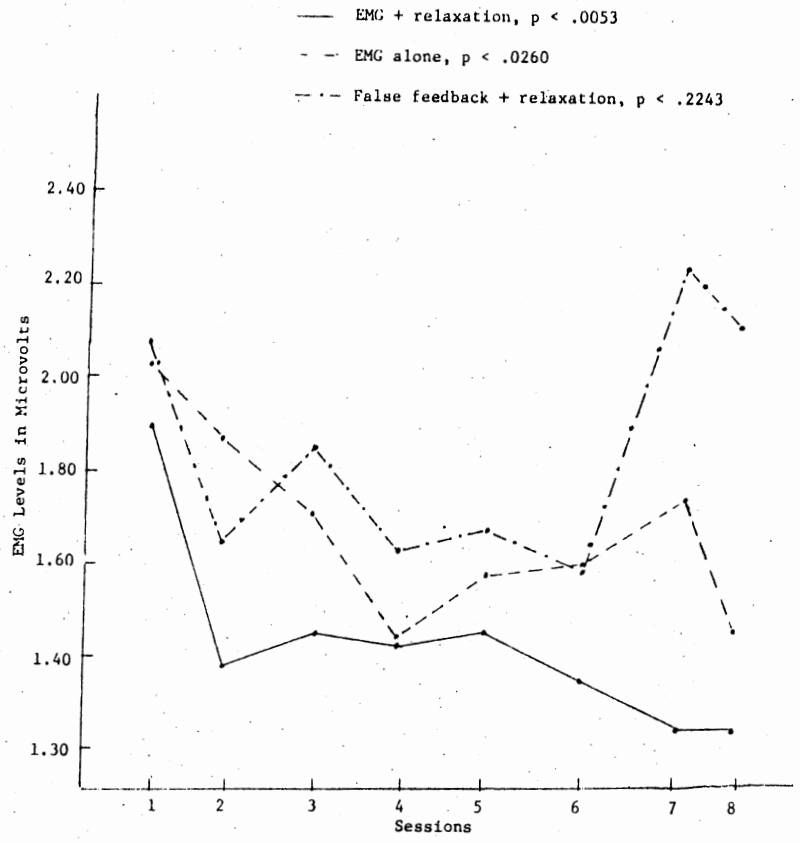

Figure 1. Combined EMG Levels for Sessions

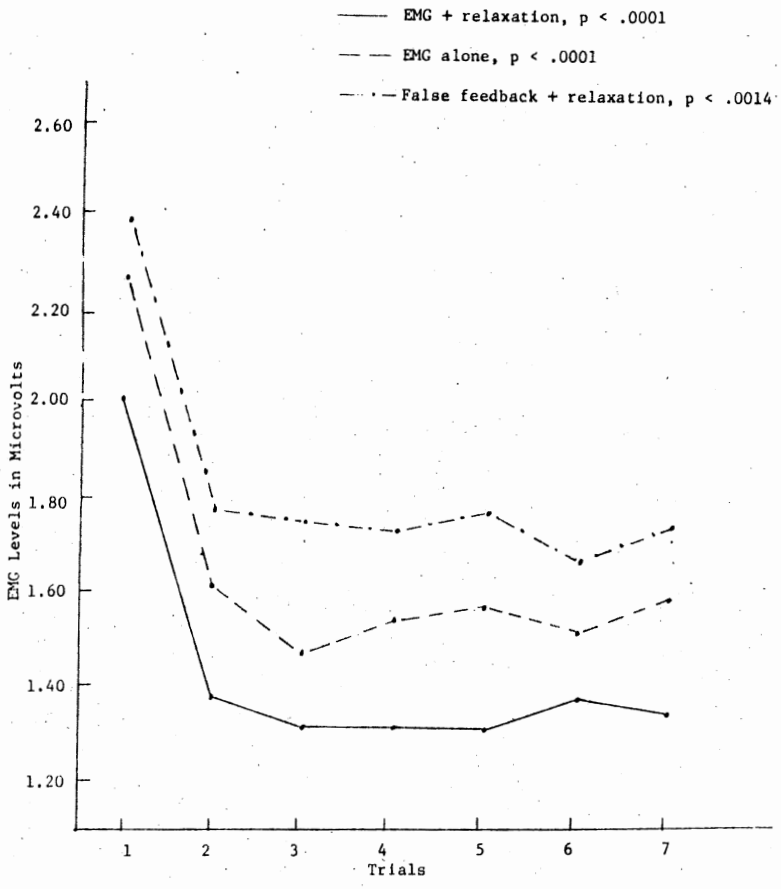

Figure 2. Combined EMG Levels for Trials

to trial 2 of .63 mv. The range of trial means is only .74 mv. Therefore, the change in the EMG levels of muscle tension occurs predominantly from trial 1 to trial 2.

Planned simple effects tests were also performed on the treatment data for the EMG without relaxation instructions group. Significant effects for sessions (Figure 1), $\underline{F}$ (7, 49) = 2.54, $p < .02$, and trials (Figure 2), $\underline{F}$ (6, 42) = 14.56, $p < .0001$, were found for this treatment group. A significant session x trial interaction, $\underline{F}$ (42, 294) = 1.45, $p < .04$, was also revealed. A Table of Means across Trials and Sessions is presented in Table XXII for this group. Although there is a drop in muscle tension levels across sessions, Figure 1 shows that the change is rather variable. Thus, the change across trials within sessions, although decreasing in levels of muscle tension, is rather variable from session to session. Again, as with the EMG feedback with relaxation instructions group, much of the change in muscle tension levels occurs from trial 1 to trial 2 with a reduction of .67 mv which indicates a large reduction from trial 1 to trial 2 with minimal change from trial 2 to trial 7, 1.62 mv to 1.60 mv, respectively.

For the false feedback group with relaxation instructions, planned simple effects tests revealed only a significant trials, $\underline{F}$ (6, 42) = 4.48, $p < .001$, effect. The graph of trial means (Figure 2) shows that the subjects were performing inconsistently across trials beginning with a trial 1 mean of 2.40 mv, dropping to 1.74 mv in trial 4 and then rising to 1.78 mv, down to 1.68 mv and up to 1.78 mv in trials 5, 6 and 7, respectively. The lack of a statistically significant difference across sessions for this group appears to be due to the extremely variable performance of this group.

EMG-F

Further analysis of the EMG data with a mixed ANOVA on Treatments (3) x Sessions (8) x Trials (7) utilizing the separate measures from the frontalis muscle (EMG-F) revealed significant main effects for sessions, F (7, 147) = 4.35, $p < .0002$, and trials, F (6, 126) = 6.42, $p < .0001$. This indicates that the lowering of muscle tension levels of the frontalis muscle was occurring across trials within sessions and across sessions. These main effects for trials and sessions are consistent with the significant main effects discovered with the measures of combined muscle tension. This is partially due to the frontalis tension levels being generally much higher than those obtained from the forearm flexor. Thus, the combined measure of EMG was weighted with higher levels of tension from the frontalis muscle.

Although there were no main treatment or interaction treatment effects, simple effects tests were computed to look at the differential performance of the three treatment groups. For the EMG feedback with relaxation instructions group, significant main effects were revealed on sessions, F (7, 49) = 3.93, $p < .001$, and trials, F (6, 42) = 2.73, $p < .02$. These effects are consistent with the previous results for this group on the combined EMG measure. Figure 3 reveals a rather consistent drop across sessions from 2.71 mv in session 1 to 1.87 mv in session 3. On session 4, however, there is a large increase to 2.12 mv and then a general reduction in muscle tension levels to 1.65 mv in session 8. In Figure 4, the trials effect seems more inconsistent with a drop from 2.36 mv on trial 1 to 1.70 mv on trial 3. Then there is a rise to 2.03 mv on trial 5 and finally another drop to 1.92 mv on trial 7.

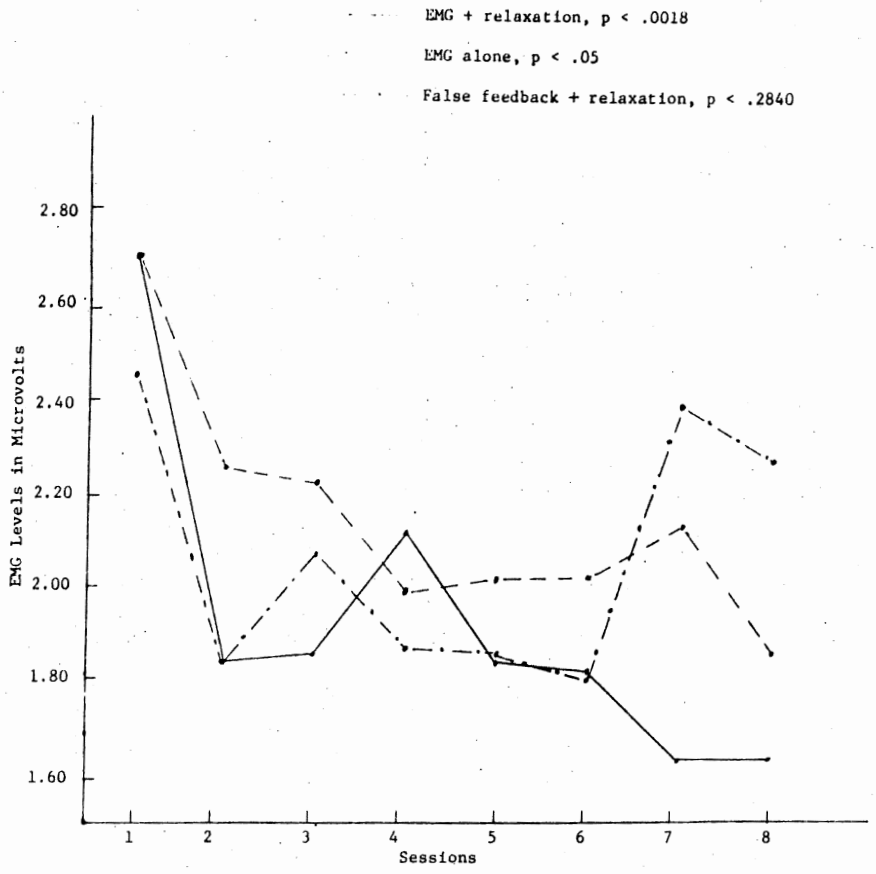

Figure 3. Frontalis EMG Levels for Sessions

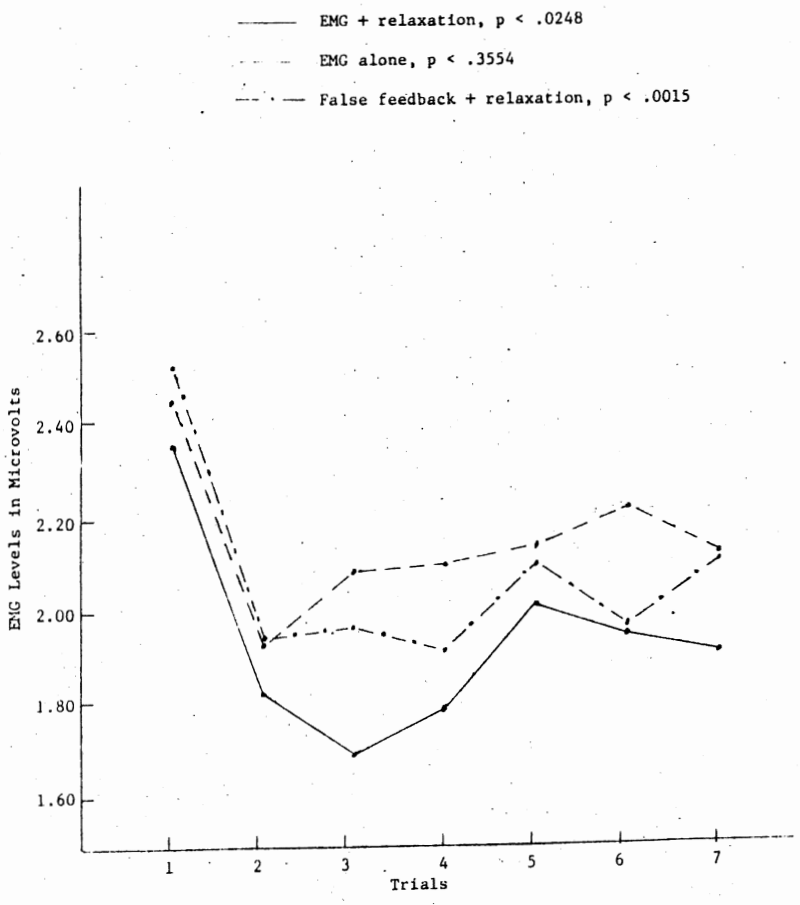

Figure 4. Frontalis EMG Levels for Trials

Analysis of simple effects for the EMG feedback without relaxation instructions group revealed only a significant sessions effect, F (7, 49) = 2.11, $p < .05$, which indicates that biofeedback without relaxation instructions produced learned reductions in EMG frontalis tension levels across time from session to session. Analysis of the session means, as displayed in Figure 3, shows a gradual consistent drop from 2.71 mv in session 1 to 2.03 mv in session 6. Session 7 rises to 2.15 mv and finally drops to 1.87 mv in session 8.

For the false feedback group with relaxation instructions, a planned simple effects test on the EMG-F data revealed only a significant trials effect, F (6, 42) = 4.43, $p < .001$. This significant difference for trials indicates that the individuals were lowering their levels of muscle tension across trials within any one session but no significant reductions across sessions occurred. Figure 4 reveals a large drop in muscle tension levels for the frontalis muscle of .59 mv from trial 1 to trial 2. The range among the other trials is only .20 mv indicating that most of the change for this group on the frontalis muscle occurred from trial 1 to trial 2.

EMG-A

Another mixed analysis of variance on Treatments (3) x Sessions (8) x Trials (7) on the forearm flexor EMG (EMG-A) data showed a significant trials effect, F (6, 126) = 27.58, $p < .0001$, and a significant interaction of treatment with sessions and with trials, F (84, 882) = 1.51, $p < .002$.

In order to understand the significant three-way interaction on the EMG-A data, planned simple effects tests were computed to analyze

more completely the differential performance of the three treatment groups. These tests revealed a significant effect for trials, F (6, 42) = 11.70, $p < .0001$, and a significant session x trial interaction, F (42, 294) = 1.41, $p < .05$, for the EMG feedback group with relaxation instructions. Figure 6 indicates that this group was lowering muscle tension levels across trials. Planned Newman-Keuls comparisons on the EMG-A data for them also showed that trial 1 was significantly different from all other trials indicating that most of the change in EMG-A muscle tension levels occurred from trial 1 to trial 2. This interpretation is supported by a .69 mv change from trial 1 (1.42 mv) to trial 2 (.73 mv) with the range among the other trials being .25 mv. Figure 5 indicates that that this group was performing differentially across trials from session to session in changing levels of muscle tension for the forearm flexor. A comparison of the trial means shows that there is a consistent and cumulative efficient reduction in EMG levels for all sessions except session 6. In this session, the group drops from .94 mv on trial 1 to .46 mv on trial 3. A rise to .54 mv on trial 4, a drop to .40 mv on trial 5, with a subsequent rise to 1.01 mv on trial 7 describes their variable performance.

Analysis of the simple effects tests for the EMG feedback group without relaxation instructions on the EMG-A data revealed a significant trials effect, F (6, 42) = 7.71, $p < .0001$. Figure 6 shows that this group was reducing their EMG levels in the forearm flexor across trials within sessions. However, this group demonstrated no significant change in forearm EMG across sessions. Independent

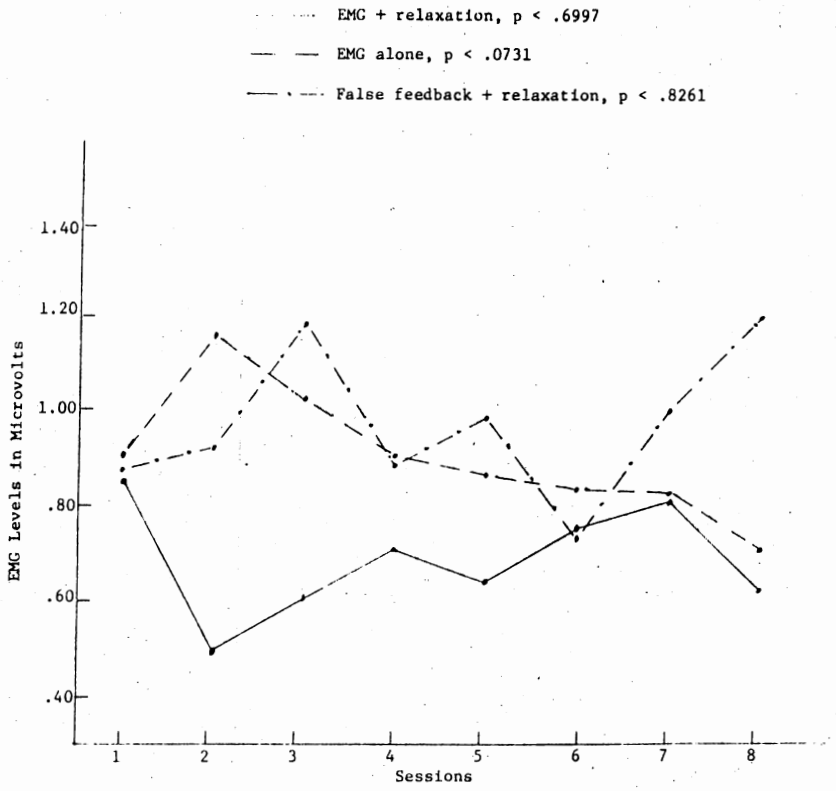

Figure 5. Forearm EMG Levels for Sessions

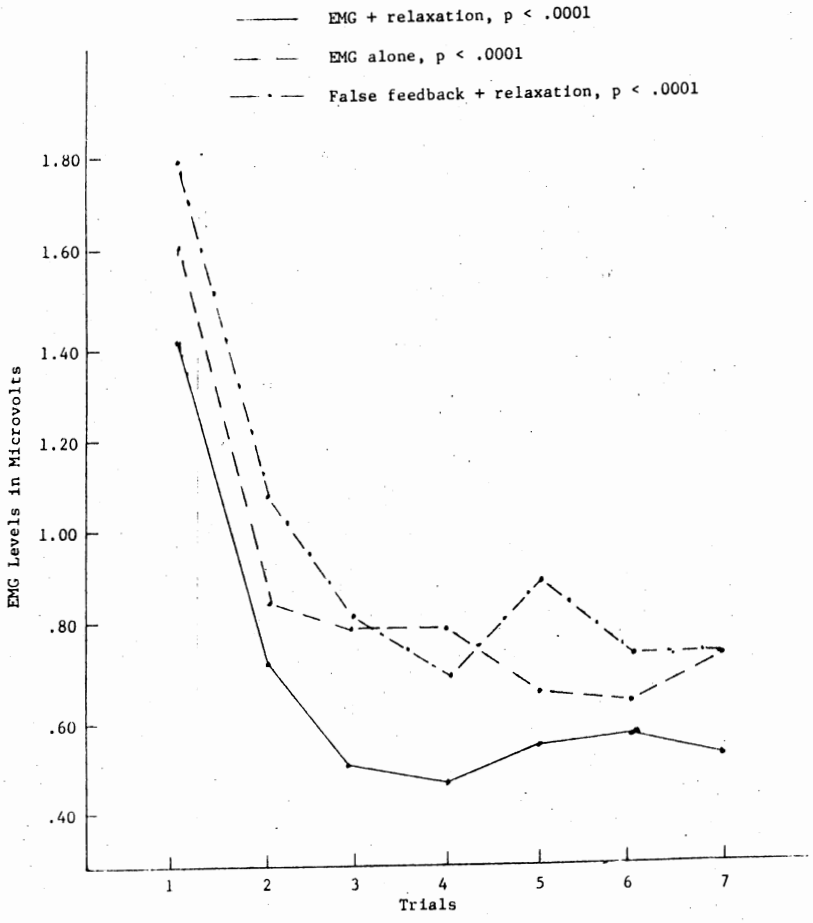

Figure 6. Forearm EMG Levels for Trials

Newman-Keuls comparisons of the trial means for the EMG-A data revealed that trial 1 was significantly different from all other trials. From trial 1 at 1.63 mv there was a .76 mv drop to trial 2 at .87 mv. Among the trials 2 through 7, there was only a .21 mv change. Therefore, most of the reduction in EMG-A levels of muscle tension for this group occurred from trial 1 to trial 2.

For the false feedback group with relaxation instructions, the simple effects test on the forearm flexor revealed a significant trials effect, F (6, 42) = 9.81, $p < .0001$, and a significant session by trial interaction, F (42, 294) = 1.42, $p < .05$. Figure 6 shows that this group was reducing their levels of muscle tension across trials within sessions. Planned Newman-Keuls comparisons of the trial means on the EMG-A data demonstrated significant differences between trial 1 and all other trials. The group began on trial 1 with a mean of 1.81 mv and lowered their EMG levels to .76 mv on trial 7. Again, the largest change occurred from trial 1 to trial 2 with a .66 mv drop on the forearm flexor muscle tension levels. A comparison of the trial means within each session revealed this group to be changing in an inconsistent and disorderly manner in some sessions. Although a regular and consistent reduction in EMG levels was revealed in sessions 1, 2, 5 and 6, other sessions were quite variable. For example, in session 3, from a mean of 1.50 mv on trial 1, the group increased muscle tension to 1.86 mv on trial 2, then dropped to .93 mv on trial 4, changed to 1.43 mv on trial 5, dropped to .70 mv on trial 6 and rose to 1.06 mv on trial 7. Similar kinds of changes occurred in other sessions, especially session 7, with 1.65 mv on trial 1, a drop to .64 mv on trial 3,

and a rise to 1.05 mv on trial 4 which variability continues throughout the session.

GSR

A mixed analysis of variance on the GSR data utilizing a Treatments (3) x Sessions (6) x Trials (7) was computed. The between subjects variable was the three treatment groups of false feedback with relaxation instructions, true EMG feedback with relaxation instructions, and true EMG feedback without relaxation instructions. The within subjects variables were the six treatment sessions and seven trials within each session. Due to the failure of the Autogen 3400 Feedback Dermograph, GSR data was obtained on only 18 subjects for six sessions.

There was no significant main treatment effect on the GSR data indicating that the GSR measures obtained in the three groups did not significantly differ. However, there was a significant main effect for sessions, $\underline{F}$ (5, 75) = 9.84, $p < .0001$, which indicates that the subjects within each group were changing their GSR responses across sessions in a direction toward greater relaxation. Planned Newman-Keuls comparisons of the session means revealed significant differences between session 1 and session 5 and between session 1 and session 6. The data demonstrated a consistent and orderly increase in GSR levels across sessions with a mean of 99.42 K ohms in session 1 to 123.46 K ohms in session 6. Another significant main effect for trials, $\underline{F}$ (6, 90) = 19.76, $p < .0001$, was discovered in the GSR data indicating that changes toward a more relaxed state were occurring across trials within each session.

Independent Newman-Keuls comparisons of the trial means demonstrated significant differences between trial 1 and trial 5 and trial 6 as well as trial 2 and trial 5 and trial 6. The GSR trial means show a regular increase across trials from a mean of 102.44 K ohms on trial 1 to 123.02 K ohms on trial 6. This overall ANOVA on the GSR treatment data is consistent with the overall ANOVA on the EMG combined and EMG frontalis data which supports the hypothesis that lower tension levels and greater relaxation were occurring within these three treatment groups. The fact that the GSR data changed in a direction indicating relaxation supports the generalization hypothesis that learning to relax in one physiological system will tend to generalize to other systems.

In order to better understand the differential performance of the three treatment groups, planned simple effects tests were computed for each group. For the EMG feedback group with relaxation instructions, significant sessions, F (5, 25) = 6.90, $p < .0004$, and trials effects, F (6, 30) = 5.70, $p < .0004$, were discovered which indicates that this group was changing their GSR responses across trials within sessions and across sessions in a manner consistent with increased relaxation. It also indicates that this group was changing in a regular and consistent manner across sessions with a mean of 79.45 K ohms in session 1 to 118.47 K ohms in session 6. A similar pattern emerged with a mean of 90.83 K ohms for trial 1 and 109.25 K ohms for trial 7.

Analysis of the GSR data for the EMG feedback group without relaxation instructions revealed only a significant trials effect, F (6, 30) = 8.45, $p < .0001$. This indicates that this group was changing GSR responses across trials in a direction indicating greater

relaxation but this change was not retained across time from session to session. This interpretation is supported by a comparison of the trial means which changed from 106.61 K ohms on trial 1 to 125.66 K ohms on trial 7.

Analysis of the GSR treatment data for the false feedback group with relaxation instructions revealed a significant sessions, $\underline{F}$ (5, 25) = 3.18, $p < .02$, and trials effect, $\underline{F}$ (6, 30) = 8.29, $p < .0001$, which indicates a change in GSR responses across trials and sessions indicating greater relaxation. Most of the change, however, appears to occur from session 1 to session 2 with a mean of 113.00 K ohms to 125.97 K ohms, respectively. The range of scores from session 2 to session 6 is only 2.95 K ohms. A comparison of the trial means for this group on the GSR data indicates a regular and consistent increase in GSR responses from 109.88 K ohms on trial 1 to 129.72 K ohms on trial 7.

Interrelationships Among the Physiological Measures for Each Treatment Group

A four x four matrix of Pearson product moment correlations for each treatment group by sessions on the EMG combined (EMG-I), EMG forearm flexor (EMG-A), EMG frontalis (EMG-F) measures and the GSR scores were computed.

For the true EMG feedback group with relaxation instructions, EMG-I (Appendix E) correlated significantly with both EMG-F, r (6) = +.98, $p < .0001$, and GSR, r (6) = -.82, $p < .01$. EMG-F also correlated significantly with GSR, r (6) = -.80, $p < .01$. These correlations support the hypothesis that this group was changing in a direction indicating

greater relaxation and the generalization theory that relaxation of one physiological system will tend to spread to other systems.

Significant correlations for the EMG feedback without relaxation instructions group by sessions (Appendix F) include EMG-I with EMG-A, $r\ (6) = +.77$, $p < .02$, and EMG-I with EMG-F, $r\ (6) = +.89$, $p < .002$. The correlations for all the EMG dependent measures were significant indicating that they were all changing in a similar manner across sessions. No other significant correlations were found for this group except the EMG-F and GSR measures approached significance, $r\ (6) = -.67$, $p < .06$.

For the false feedback group with relaxation instructions (Appendix G) only the EMG-F and EMG-I measures correlated significantly with each other, $r\ (6) = +.95$, $p < .002$. The high correlation of EMG-I with EMG-F is anticipated in view of the extreme contribution of the EMG-F measures to the EMG-I measures. For this group, the EMG-F and EMG-A measures approached significance, $r\ (6) = +.66$, $p < .07$.

In addition, a four x four matrix of Pearson product moment correlations for each treatment group by trials on the EMG-I, EMG-F, EMG-A and GSR data was computed.

Significant correlations across trials for the EMG feedback group with relaxation instructions (Appendix H) included EMG-I measures with EMG-A, $r\ (7) = +.98$, $p < .0001$, EMG-F, $r\ (7) = +.80$, $p < .03$, and GSR, $r\ (7) = -.89$, $p < .007$. EMG-A also correlated significantly with GSR, $r\ (7) = -.93$, $p < .001$, and EMG-A approached significance with EMG-F measures, $r\ (7) = +.71$, $p < .07$. This indicates that the combined EMG, forearm EMG and GSR measures were changing together in a more consistent

manner than the EMG frontalis. This may be a function of the degree of difficulty experienced in relaxing the frontalis muscle.

For the EMG feedback group without relaxation instructions, significant correlations across trials (Appendix I) were EMG-I with EMG-A, $r\ (7) = +.93$, $p < .001$, and EMG-I with GSR, $r\ (7) = -.84$, $p < .01$. The only other significant correlation was the EMG-A measure with GSR, $r\ (7) = -.92$, $p < .002$. These significant correlations seem to support the previous conclusion for the EMG feedback group with relaxation instructions, i.e., these physiological dependent measures are changing in a more consistent and linear manner than the EMG frontalis. This may again be due to the difficulty of relaxing the frontalis muscle.

For the false feedback group with relaxation instructions (Appendix J) each physiological dependent measure correlated significantly with every other physiological measure. EMG-I correlated significantly with EMG-A, $r\ (7) = +.96$, $p < .0003$, with EMG-F, $r\ (7) = +.96$, $p < .0004$, and GSR, $r\ (7) = -.87$, $p < .01$. EMG-A correlated significantly with EMG-F, $r\ (7) = +.91$, $p < .003$, and with GSR, $r\ (7) = -.94$, $p < .001$. EMG-F also correlated significantly with GSR, $r\ (7) = -.77$, $p < .04$. These significant correlations indicate that all of the physiological measures for this group across trials were changing in a consistent and linear direction which would support the efficacy of cognitive strategies over short time periods. Most (five of six) of the significant correlations support the extreme contribution of the EMG frontalis measures to the combined EMG measures. The significant correlations of the EMG forearm and EMG frontalis support the hypothesis that relaxation in one muscle of the body will tend to generalize to other muscles in the body. The GSR correlations with the EMG measures support the

generalization hypothesis that relaxation of one physiological system tends to spread to other physiological systems.

It is interesting to note that the GSR measures, for the session correlations, are significant only for the EMG feedback group with relaxation instructions on the EMG-I and EMG-F data. On the correlations by trials, however, GSR correlates with all three EMG measures for the false feedback group with relaxation instructions and with EMG-I for the EMG feedback group with relaxation instructions and the EMG feedback group without relaxation instructions, respectively. This data supports the contention that the generalization of relaxation from one physiological system to another may be quickly learned across trials but is not easily retained across sessions. Furthermore, it appears that mere cognitive strategies are extremely effective across trials within sessions in changing the physiological dependent measures in a direction indicating greater relaxation.

State-Trait Anxiety Inventory

An analysis of variance with Groups (3) x Pre- and Post-measures (2) was performed on the State-Trait Anxiety Inventory, A-State (STAI-A-State) scores. The between subjects variable was the three treatment groups of false feedback with relaxation instructions, true EMG feedback with relaxation instructions, and true EMG feedback without relaxation instructions. The within subjects variable was the pre- and post-treatment scores on the STAI-A-State.

No significant main effect for groups was revealed in the STAI-A-State scores indicating that the subjective reports of the three groups regarding their state of anxiety before and after treatment did not

differ. The main effect for the pre- and post-treatment scores, however, was significant, $\underline{F}$ (1, 21) = 8.84, $p < .007$, indicating a reduction in tension and anxiety for all three groups.

Cognitive Strategies

Most subjects, 21 of the 24, thought of the various activities and people with which they were involved, i.e., parties, sororities, friends, concerts, dates, etc. Many, 19 of the 24 subjects, mentioned different classes, exams and grades. They also thought about their relationships with their boyfriends, friends, and relatives. Others, four of the 24 individuals, planned their day's activities during the experimental session. Approximately eight of the 24 mentioned trying to think relaxing thoughts.

A chi-square analysis was performed to detect any relationship between treatment groups and suspicion of false feedback. A X^2 of 2.08 was computed which indicates that the treatment groups did not suspect false feedback was one of the treatment conditions.

Another X^2 analysis was performed to detect any relationship between experimenters and suspicion of the specific group being given pseudo-feedback. A X^2 of 2.40 was computed which indicates that the experimenters did not guess correctly the placebo group.

CHAPTER IV

DISCUSSION

The primary purpose of this study was to examine the role of cognitive factors in the process of biofeedback. More specifically, an attempt was made to look at the effects of instructions, verbal and coded (clicks indicating increasing levels of relaxation), upon one's psychological level of muscle tension and one's physiological state of relaxation. It was hypothesized that those individuals receiving true EMG feedback with specific relaxation instructions would show the most consistent and efficient reduction in muscle tension levels of the three treatment groups. It was also predicted that this group would show a significant reduction in GSR levels, a measure of one's general level of arousal, and STAI-A-State scores, a measure of subjective anxiety. It was further hypothesized that the true biofeedback group without relaxation instructions would show a significantly greater reduction in muscle tension than the false decreasing tone group but no significant reduction in GSR levels or STAI-A-State scores. These hypotheses were made because this group would be receiving true EMG feedback and, consequently, would experience a reduction in EMG levels. However, they would receive no cognitive instructions to relax or feel less tense. Therefore, no significant reductions in the general level of arousal, GSR, or subjective state of anxiety would occur.

It was also hypothesized that the false decreasing tone group which received relaxation instructions along with decreasing pre-recorded clicks which indicated increasing levels of relaxation across sessions, would show a significant reduction in GSR levels and STAI-A-State scores. However, this group would show the least amount of learned EMG reduction as they would be receiving false feedback.

Results of the data analysis indicate that the three treatment groups did not significantly differ from each other on their EMG frontalis levels, GSR measures or STAI-A-State scores. However, a treatment x session x trial interaction effect was significant for EMG forearm data and marginally significant for the combined levels of muscle tension. In order to have a better understanding and describe the differences among the three groups, simple effects tests were computed.

From these analyses, it appears that both EMG groups were lowering their levels of muscle tension as they displayed a significant sessions and trials main effect. However, the false feedback group showed only a significant trials main effect on the EMG combined measures. This indicates that this group was changing across trials within sessions in a manner consistent with greater relaxation. However, the data suggests that these changes were more likely due to habituation rather than learning as they were not changing EMG levels from session to session. Furthermore, it appears that cognitive instructions to relax are effective in changing muscle tension levels across trials within sessions but not across time from session to session. The lack of the EMG feedback and consequent lack of knowledge of one's level of muscle tension seems

to lead to habituation across trials and a minimal level of learned EMG reduction as hypothesized.

A close examination of the graph of session means for the EMG feedback group with relaxation instructions (Figure 1) on the EMG combined data indicates a rather consistent and significant reduction in EMG levels across trials within sessions and from session to session across time. Thus, it appears that the combined impact of true EMG feedback with the explicit message of relaxation, i.e., information about one's level of muscle tension plus the cognitive strategy to relax led to consistent and efficient reduction in muscle tension.

A comparison of the graph of session means for the EMG feedback group without relaxation instructions (Figure 1) shows a reduction in EMG levels across trials and sessions but in a more inconsistent manner than the EMG feedback group with relaxation instructions. These inconsistencies of performance across trials in various sessions for this group resulted in a significant session by trial interaction on the EMG combined data. Although this group also showed a reduction in EMG levels across trials and sessions, it appears that the lack of a relaxation message resulted in more variable and inconsistent performances. For an orderly and efficient drop in muscle tension, it seems that not only is true EMG feedback necessary but also appropriate instructions so that the individuals may develop the essential idiosyncratic cognitive strategies to attain a state of relaxation. It appears that the inconsistencies in EMG reduction increase as a result of the lack of relaxation instructions across trials within sessions and across sessions.

Analysis of the overall EMG-F data revealed a significant trials and sessions effect which is also found with the combined EMG measures.

These findings are not surprising in view of the large contribution of the frontalis levels of muscle tension to the EMG combined measures.

A significant trials and sessions main effect for the EMG-F data was also found in the simple effects tests for the EMG feedback group with relaxation instructions. This, also, is anticipated in view of the large contribution of the EMG-F measures to the EMG combined measures and the significant sessions and trials effect on EMG combined for this group. Moreover, it is not surprising in view of their consistent and regular changes in EMG levels across both trials and sessions.

For the EMG feedback group without relaxation instructions, a significant sessions effect was discovered on the EMG-F data. This indicates a reduction in EMG-F levels from session to session but little, if any, consistent decrease in EMG-F levels of muscle tension across trials.

Analysis of the EMG-F data for the false feedback group with relaxation instructions revealed only a significant trials effect indicating change across trials in a direction consonant with greater relaxation which is probably due to habituation to the experimental procedures. However, this habituation does not lead to any retention or change in behavior from session to session.

Analysis of the overall EMG-A data revealed a significant trials effect and a significant treatment by session by trial interaction. The interaction indicates that within at least one treatment group, the individuals are performing differentially across trials in different sessions.

Further analysis of the EMG-A data revealed a significant trials effect for all three treatment groups. Although the forearm flexor

muscle is an easier muscle to relax than the frontalis muscle, the measures from the forearm flexor were contributing a much smaller amount to the combined EMG levels than the frontalis. Consequently, since the feedback to the subjects was based upon the EMG combined measures, then this feedback was not always precisely accurate in regard to the forearm flexor muscle. Thus, a situation was created by the experimental procedures which could easily result in a non-significant sessions main effect. Inaccurate information about one's level of muscle tension does not facilitate learned EMG reduction. Another possible explanation is that the forearm EMG reached such low levels of muscle tension so early that a basement effect was operating which did not permit any further reduction from session 1.

Thus, it appears that the mere cognitive message of relaxation is sufficient to effect a decrease in muscle tension levels over short periods of time as supported by the significant trials effect on all measures of EMG for both groups receiving relaxation instructions. This is further supported by the significant correlations of all physiological dependent measures computed for the false feedback group with relaxation instructions by trials and most of the physiological dependent measures for the EMG feedback group with relaxation instructions by trials.

However, learning to reduce one's level of muscle tension and retention of this learning across time appears to require veridical feedback of one's changing levels of muscle tension as evidenced by the significant sessions effect for all EMG dependent measures for both groups receiving EMG feedback except EMG-A. This is supported by the

sparse number of significant correlations of the physiological dependent measures for the false feedback group by sessions, in addition to the absence of significant sessions effects for this group.

Analysis of the GSR data appears to support the generalization hypothesis that a change in one physiological system in a specific direction facilitates similar kinds of changes in another physiological system. For example, a change in the EMG levels toward greater relaxation appears to have facilitated similar changes in the GSR levels as evidenced by the significant sessions and trials main effects for the GSR data. However, there was no significant main effect for treatment indicating that the three treatment groups did not significantly differ in their GSR levels. Although all three groups changed their GSR levels in a direction indicating greater relaxation, no one group showed a significantly greater change than the other groups. This, again, supports the generalization hypothesis as there was no significant main effect for treatment on the EMG data, either.

The simple effects tests showed a significant trials and sessions main effects for the EMG feedback group with relaxation instructions and the false feedback group with relaxation instructions on the GSR responses. The relaxation instructions as well as the lowered EMG levels appeared to facilitate the change in GSR levels, a measure of one's general level of arousal. The GSR levels seemed to be significantly affected by the cognitive instructions as the EMG feedback group without relaxation instructions attained only a significant trials effect. This indicates a change in GSR levels across short time periods but no retention of this change from session to session.

The STAI-A-State data are quite similar to the results obtained from the overall EMG combined ANOVA and the overall GSR ANOVA. As with the EMG combined and GSR data, the STAI-A-State scores show no significant main treatment effect. However, a significant main effect for pre- and post-scores indicate a significant reduction in subjective states of anxiety for all three groups before and after treatment.

Although the treatment x session x trial interaction was not significant on the frontalis EMG measures, $\underline{F}$ (84, 822) = .63, $p < .969$, and only marginally significant on the combined EMG measures, $\underline{F}$ (84, 882) = 1.26, $p < .06$, this interaction was significant on the forearm flexor measures, $\underline{F}$ (84, 882) = 1.51, $p < .002$. This marginal interaction effect on the combined EMG data and the significant interaction on the forearm flexor EMG data led to the decision to compute the simple effects tests, although this interaction was only a small proportion of the total variance. Without these tests, the differential performance of the three treatment groups could not have been explored and explicated. Thus, the decision to analyze the data more thoroughly with the simple effects tests was made without following precise statistical procedures so that a better understanding of the differential performance of the three groups could be obtained.

This study supports the research of Budzynski and Stoyva (1969), Canter, Kondo, and Knott (1975), Le Boeuf (1977), and others that veridical feedback of EMG muscle tension levels faciltiates reductions in EMG levels and consequent states of relaxation. It does not, however, support Alexander's (1975) conclusion that EMG reduction does not lead to increased feelings of relaxation. It is possible that Alexander's results were merely an artifact due to his experimental

procedures. For example, subjects were only trained for three sessions for approximately 17 minutes each session. Even more important were his procedures for obtaining reports of relaxed states. Following each 4.5 minutes within each session, the experimenter would ask the subject to verbalize his physical and mental feelings of relaxation. The subject then rated himself on a scale from -2 to +2 aloud. This procedure of having the subject respond verbally, at specific intervals, throughout the training sessions, may have interrupted the process of relaxation and led to the insignificant results obtained by Alexander.

Although most studies, utilizing pseudofeedback, have reported no significant changes in frontalis muscle tension levels or in self-reported symptoms (Budzynski and Stoyva, 1969; Philips, 1977; and Budzynski, Stoyva, Adler, and Mullaney, 1973), this study does not support those conclusions. However, this is the first experiment in which pre-recorded decreasing clicks were used as false feedback indicating to the subjects that they were becoming more and more relaxed. This type of false feedback facilitated significant changes in forearm and frontalis EMG levels, separately and in combination, across trials. It also resulted in increased feelings of relaxation. It is possible that the decreasing clicks indicating more relaxed states may have increased the subjects' motivation and maintained their interest better than previous irrelevant feedback with a consequent significant reduction in EMG levels across trials. The double-blind procedures may have also faciltiated these significant changes for the placebo group as there were similar expectations by the experimenter for all subjects in all groups.

This double-blind study supports the only other double-blind biofeedback experiment in the literature by Cohen, Graham, Fotopoulos, and Cook (1977) in which they reported no differences in therapeutic outcome. In the present study, there were no differences in reported states of relaxation among the three treatment groups. Furthermore, those subjects who received true EMG feedback demonstrated more control over EMG levels than those who received false feedback. This was also found in the previous double-blind study. It appears that the results obtained with previous false feedback groups may have been significantly affected by the lack of motivation and interest of the subjects and the expectations of the experimenters which were covertly communicated to the subjects.

Alexander's (1975) conclusion that EMG reduction in one muscle does not generalize to other muscles is not supported. Significant results were obtained for both the frontalis and the forearm flexor muscles although training was primarily on the frontalis muscle. These results are in direct contradiction to Alexander's conclusion. However, previous normative research (Greenfield and Sternbach, 1972) has found the forearm extensor muscle to be an unreliable measure in the resting state (.117). This was the muscle Alexander chose as his generalization site. In this study, the forearm flexor muscle was utilized as a measure of generalization. This particular muscle was chosen due to its reliability as a measure of muscle tension during the resting state (.460). Not only did we obtain generalization from one muscle to another, we also found significant results with the GSR response, which is also in contradiction to Alexander's findings. This supports the generalization hypothesis that a change in one physiological system in a specific direction facilitates similar changes in other

physiological systems which Alexander disputed.

Furthermore, this study supports the conclusion of other research that manipulation of a subject's cognitions can alter subjective reports of autonomic activity (Sternbach, 1962) and emotional responsiveness (Rimm and Litvak, 1969) as the false feedback group reported changes in states of relaxation. Valins' (1966) and Valins and Ray's (1967) conclusion that non-veridical cognitive representations of physiological events affect subjective reports is validated by the reported changes in relaxation for the false feedback group. The significant reduction across trials in EMG and GSR levels for the false feedback group also supports May and Johnson's (1973) conclusion that internal cognitive stimuli produce physiological changes.

In conclusion, it appears that cognitive factors are effective in producing EMG reductions in muscle tension levels for short periods of time (trials). Furthermore, changes in reported states of relaxation also appear to be significantly affected by cognitions. There is also some evidence to support the role of muscles as mediators in the process of biofeedback. For example, only with the true EMG feedback groups did we obtain significant EMG reductions across time (sessions). There were also significant reductions in GSR levels and STAI-A-State scores for these groups. Thus, accurate information about one's EMG levels seems to be essential to attain a significant reduction in levels of muscle tension across sessions. It appears that not only are cognitive factors important in the process of biofeedback as critical mediating variables but so are muscular levels of tension.

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APPENDIXES

APPENDIX A

LITERATURE REVIEW

A Literature Review of Studies Investigating the Role of Cognitive Factors in Behavior

Although biofeedback refers to the process of feeding back to an individual information about the functioning of a specific physiological reaction for the purpose of altering its responsivity, it is actually part of a much broader area of research called self-control. This area refers not only to biofeedback but also to hypnosis, yoga, meditation and autogenic training which have also been found to alter psychophysiological functions.

The current preliminary successes of biofeedback training in the treatment of various physiological disturbances such as tension headaches, anxiety symptoms, etc., have led to renewed interest in the methods of Zen, yoga, progressive relaxation and autogenic training. In many ways, biofeedback techniques represent a modern electronic version of these other approaches. All of them, however, teach the subject to be aware of subtle internal cues and to use these cues to bring about desired psychophysiological states (Raskin, Johnson, and Rondestvedt, 1973).

For hundreds of years, stories about Indian yogis who have learned to control heart beat, skin temperature and respiration rate have been known. Furthermore, practitioners of hypnotic trances have stated that suggestions given during a deep trance are effective in producing blisters, removing warts, and altering such physiological functions as salivation, heart rate and sensory perceptions.

Although these reports suggested that certain individuals could learn to control specific physiological functions formerly believed to

be involuntary, there was no technique by which ordinary persons could do so until the advent of biofeedback. With biofeedback, numerous possibilities emerged for the control of various physiological functions and subsequent treatment of psychophysiological disorders.

Biofeedback

Biofeedback is a process through which one learns voluntary control over automatic, reflexly regulated body functions. The technique of biofeedback is based on the fundamental learning principle of "shaping" or "approximation." In utilizing the principle of approximation, individuals are reinforced for responses similar to the desired behavior.

Biofeedback involves selection of a specific bodily function which is monitored by an instrument that detects physiological signals such as heart rate, blood pressure, muscle tension or brain waves. These signals are amplified to activate a display, either visual or auditory, that reflects changes in the physiologic activity.

The basic elements of the biofeedback process are as follows:

1. the selection of a physiologic function,
2. an instrument recording the activity of this function,
3. presentation of this information to the individual in the form of auditory or visual signals, and
4. an implicit intention to change this physiologic activity utilizing the biologic information.
5. The change which occurs in the physiologic activity is due to an as yet unexplained mechanism (Brown, 1977).

All of the above elements also occur in hypnosis, yoga and autogenic training except for recording of the biologic function and electronic presentation of this information to the individual. As stated above,

these techniques are subsumed under the broader rubric of self-control.

Self-control can be viewed as the process through which the individual becomes the primary agent in directing, guiding and regulating those particular features of his own behavior that may lead to more positive outcomes. In self-control, the individual makes a conscious decision to achieve certain desirable goals. It is also a functionally defined concept. Whether or not one reaches specified outcomes demonstrates the process of self-control, not the specific techniques involved. In the area of self-control, we are talking about the entire repertoire of responses by which one changes behavior, i.e., changing contingencies of behavior, self-reinforcement, self-punishment, self-relaxation and cognitive relabeling (Goldfried and Merbaum, 1973).

Research in hypnosis has intensified during the past two decades. During this time, it has become increasingly clear that a wide variety of bodily functions can be influenced by suggestions or instructions given with or without hypnotic induction procedures. Researchers seem to be reaching a consensus that mere suggestions are effective in altering psychophysiological processes when the subject is actively involved and believes the suggestions (Spanos and Barber, 1974).

For example, remarkable control over skin responses has been demonstrated in hypnotized subjects. Much of the accumulated evidence from this research suggests that the critical mediating variable can be more appropriately conceptualized as acceptance of, involvement in or belief of the suggestions.

In a review of Ikemi and Nakagava's work with 13 subjects who were allergic to the leaves of two trees found in Japan, Barber (1971) explicated the following results. Five subjects were hypnotized and told

that they were being touched by the allergic-reactive leaves while the other subjects were merely blindfolded and told the same thing. All were presented with harmless leaves. These harmless leaves produced a slight to marked degree of the skin allergic reaction in both hypnotized and non-hypnotized subjects. Then the experimental procedure was reversed with all subjects touched by allergy-producing leaves but told they were harmless leaves. Four of the five hypnotic subjects and seven of the eight control subjects did not experience the allergic response. Therefore, it appears that the critical factor in this study was the subjects' belief that the allergy-producing substance was actually harmless or that the harmless leaves will produce an allergic reaction.

Thus, it is possible that researchers in hypnosis and biofeedback may have underestimated the abilities and potentialities of normal individuals. For example, it was found that deeply hypnotized subjects showed an increase in heart rate when given the suggestion that their hearts were accelerating (Van Pelt, 1954). Although these results appeared astounding, it was pointed out by Barber (1961) that several documented cases existed of individuals who could accelerate and also decelerate their heart rate whenever desired. Some studies have also been published in which subjects who received cardiac biofeedback learned to accelerate their heart rate (Blanchard, Scott, Young, and Edmundson, 1974; Bergman and Johnson, 1971; Bergman and Johnson, 1972).

Although Bergman and Johnson (1972) attempted to differentiate the effects of specific versus no specific heart rate information and external versus no external reinforcement on one's ability to increase heart rate, equivocal results were obtained. However, they did find that specific heart rate information (feedback) given to the subjects

facilitated their ability to increase their heart rate. Those subjects who received no specific heart rate information evidenced no increases in heart rate which led these investigators to conclude that awareness of the criterion response plays an important role in accelerating heart rate. No differences were found between the reinforcement conditions.

In another study of the effects of feedback upon one's ability to control heart rate, Blanchard, Scott, Young, and Edmundson (1974) utilized four different conditions. One group was given feedback and informed of the response to be controlled (heart rate), another group was given no feedback and correctly informed, a third group was given feedback and incorrectly informed of the response to be controlled, while a fourth group was not informed of the correct response but was given feedback of heart rate. They found that when subjects were given feedback about their heart rates, knowledge of the response (heart rate) to be changed facilitated learning to lower heart rate. Non-significant trends for the subjects' ability to raise heart rate were found in the group correctly informed of the response and given feedback about their heart rates.

However, other investigators have found that subjects can perform in a similar fashion when simply asked to increase their heart rate. Bergman and Johnson (1971) concluded that most of the studies which concerned one's ability to control cardiovascular responses, the contribution of instructional set alone had been obscured by the use of external feedback. Consequently, to separate the effects of instructional set and external reinforcement of cardiac responses, they utilized three different instructional groups. One group was asked to increase heart rate at the presentation of a tone, another group was

asked to decrease heart rate and a third group was not instructed to change heart rate in any direction. No feedback was provided. The results suggest that subjects can decrease or increase heart rate without external feedback. Analyses of respiration and skin resistance levels show that the heart rate changes were not mediated by variations in these physiological processes. Thus, the authors conclude that instructional sets alone can account for heart rate changes.

In another study assessing the relative effectiveness of biofeedback techniques on the voluntary control of heart rate, Manuck, Levenson, Hinrichsen, and Gryll (1975) randomly assigned 32 subjects to one of four feedback conditions. One group was given no feedback, another was given binary feedback, another was given proportional feedback and the fourth group was given numerical, proportional feedback which indicated the relationship of the inter-beat interval to the pre-trial mean in direction and magnitude. They discovered that the type of feedback had no consistent effect upon heart rate changes. Thus, these investigators concluded that feedback does not necessarily facilitate voluntary heart rate control.

From the above results, what could one postulate as the essential relationship or critical mediating variable between electromyographic (EMG) feedback and relaxation? Is it possible that subjects without EMG feedback can learn to achieve a state of deep relaxation as well as those with EMG feedback? Are simple self-instructions to relax sufficient to attain a significant degree of relaxation? Or, is one's belief that he is lowering his level of muscular tension sufficient to attain a significant degree of relaxation? If so, what is the role of EMG feedback in attaining a state of deep relaxation?

Several studies have supported the hypothesis that electromyographic feedback aids in the attainment of a state of deep relaxation. Two pioneer investigations by Budzynski and Stoyva (1969) as well as Green, Waters, Green, and Murphy (1969) both reported the technique and instruments of EMG feedback which had successfully produced deep muscle relaxation. Other investigators have replicated those experiments (Canter, Kondo, and Knott, 1975; Townsend, House, and Addario, 1975; LeBoeuf, 1977; Reinking and Kohl, 1975; and Haynes, Moseley, and McGowan, 1975). However, Alexander (1975) utilizing three biofeedback training sessions, concluded that lowered EMG levels of muscle tension did not result in increased feelings of relaxation. Furthermore, he stated that there was no evidence that EMG reductions in muscle tension for the frontalis muscle generalized to other muscles.

Once a state of deep relaxation has been achieved through the use of electromyographic techniques, then a reduction in anxiety symptoms should occur according to the principle of reciprocal inhibition. In a test of this assumption, Canter, Kondo, and Knott (1975), Townsend, House, and Addario (1975), LeBoeuf (1977), and Coursey (1975) all reported a significant reduction in anxiety symptoms utilizing EMG techniques to achieve a relaxed state.

Another possibility for the utilization of biofeedback techniques exists in the area of tension headaches which are caused by sustained contraction of the muscles of the head and neck. Thus, procedures which reduce muscle tension of the head and neck should result in a decrease in measures of tension headache activity.

C. Philips (1977) found that training in electromyographic techniques produced decrements in resting muscle tension levels, headache

activity, medication frequency and a slight decrement in headache frequency. Budzynski, Stoyva, and Adler (1970), in a pilot study with five subjects, concluded that "chronic tension headache sufferers can be trained to voluntarily lower their striate muscle tension in the face of daily life stresses and to reduce the incidence of tension headaches" (p. 210). In a later study, Budzynski, Stoyva, Adler, and Mullaney (1973) and Raskin, Johnson, and Rondestvedt (1973) both found EMG training to be useful in reducing frontalis muscle tension levels and the intensity and severity of tension headaches. Thus, it appears that veridical electromyographic techniques result in not only a reduction in frontalis muscle tension levels but also a reduction in anxiety symptoms and tension headaches.

Most studies have emphasized the utilization of veridical feedback to achieve either lowered muscle tension, change in heart rate, skin temperature, etc. Budzynski and Stoyva (1969) found that the use of irrelevant feedback, a steady low tone, actually resulted in a 28 percent mean increase in muscle action potential levels while the true feedback group showed a mean decrease of 50 percent. In other research utilizing individuals with tension headaches, Budzynski and colleagues (Budzynski, Stoyva, Adler, and Mullaney, 1973) found no significant decreases in frontalis muscle tension or headache activity for the pseudofeedback group. Philips (1977) discovered that the pseudofeedback group retained no ability to lower muscle tension, whereas the true biofeedback group reduced the level and variability of muscle tension. In another study utilizing false feedback, Haynes, Moseley, and McGowan (1975) found veridical EMG feedback more effective in reducing frontalis muscle tension than false feedback. From these studies, it appears that false

feedback, in the form of a constant low tone or noncontingent feedback, is ineffective in producing low levels of muscular tension and consequent reduction in anxiety or headache symptoms.

However, it is possible that these studies were not maintaining a constant level of motivation between the experimental and control subjects. Perhaps some subjects simply became bored and disinterested when the feedback was a constant low tone. Others receiving noncontingent feedback may have perceived no difference in the rate of clicks or their level of muscle tension after a few sessions and simply "turned off." Some controls may have experienced irritation thus resulting in an increase in muscle action potentials. Therefore, this biofeedback research may not have been evaluating the effects of relevant versus irrelevant feedback but rather the effects of an inconsistent level of motivation between the groups. A more powerful control for the effects of noncontingent feedback would be a manipulation of the subjects' cognitions so that they perceived the EMG signals to be an accurate measure of their level of muscle tension. This particular manipulation apparently was implemented in a double-blind study by Cohen, Graham, Fotopoulos, and Cook (1977). The subjects were 29 opiate addicts who received 14 sessions of contingent or noncontingent EMG biofeedback training for symptom reduction during detoxification. No differences in therapeutic outcome were discovered although the contingent subjects demonstrated more control over EMG activity.

Much research has been done looking at cognitive factors as mediating variables. Miller and Dollard (1941) theorize that fear or anxiety reactions may often be elicited by an individual's cue-producing response, i.e., his or her perception and labeling of a given situation

rather than the actual situation itself. In this mediational view of emotional arousal, Dollard and Miller believe that by changing the individual's cue-producing responses, then the emotional reaction will be modified, also.

In a later experiment, Schachter and Singer (1962) investigated in a more precise manner the mediational view of emotional arousal. They manipulated states of physiological arousal plus an individual's cognitions about those states. One-half of the subjects were injected with epinephrine, while the other half received an injection of saline solution. All subjects were placed with either an angry or euphoric stooge. Those individuals injected with epinephrine were then divided into three separate groups: One group was given veridical information, another was given false information and the third group was informed that no side effects from the injection of epinephrine would occur. The placebo subjects were also told that no side effects would be experienced. As a result, subjects who were informed about the specific effects of epinephrine were significantly less euphoric or angry than those who were either misinformed or ignorant about the effects of the drug. Therefore, these researchers concluded that an emotional reaction may be considered a function of a state of physiological arousal and cognitions appropriate to this state of arousal. This definition supports the mediational view of emotional arousal.

Although Schachter and Singer's (1962) study supported the hypothesis that an emotion is a function of a state of physiological arousal and cognitions about that aroused state, the differences between the placebo and epinephrine subjects were barely statistically significant. Schachter and Wheeler (1962) subsequently theorized that these results

may have been due to the self-arousal of the sympathetic nervous system by the placebo subjects which allowed them to feel more angry or euphoric than anticipated. Thus, they compared subjects who were injected with either epinephrine, chlorpromazine or placebo. If sympathetic nervous system activation is an essential component of an emotional experience, then epinephrine, which facilitates arousal of sympathetic nervous system, should intensify the emotional reaction, whereas chlorpromazine, a blocking agent of sympathetic arousal, should lower the intensity of emotional experience. The placebo subjects should display an emotional reaction between the epinephrine and chlorpromazine subjects. Therefore, Schachter and Wheeler predicted the following results: whatever the experimentally manipulated emotional state, it should be most intensely experienced by epinephrine subjects, next by placebo subjects, and least of all by those subjects injected with chlorpromazine. Ratings of amusement for all subjects were made during a funny movie. Results were as predicted with epinephrine subjects more amused than placebo subjects more amused than chlorpromazine subjects. These results support the assumption that a state of sympathetic arousal is an essential component of an emotional experience as well as the cognitions appropriate for that aroused state.

Other investigators have also been interested in the mediational view of emotional arousal and have examined the effects of cognitive patterns upon physiological reactivity. Sternbach (1964), with six subjects, recorded various autonomic responses such as gastric motility, respiration rate, palmar skin resistance, finger pulse volume and heart rate. In three different experimental conditions, the subjects were told that they were receiving either a stimulant drug, a relaxant or a

placebo. Actually, each subject received, in each condition, a magnet which was used to measure the gastric peristaltic rate. Only the results for gastric motility were reported but these measurements indicated that the instructions did have a significant effect on stomach motility with the "stimulant" instructional set producing more peristaltic contractions than the "relaxant" instructions. The "placebo" instructions led to gastric motility measurements between those for stimulant and relaxant instructional sets.

Rimm and Litvak (1969), in another study of verbal mediational constructs examined the effects of self-verbalizations upon emotional responses. Experimental subjects were instructed to read triads of sentences which culminated in negative affective conclusions while control subjects read affectively neutral sentences with no evaluative conclusions. Galvanic skin responses and respiration rate and depth were continuously monitored. Clearly significant differences were found between experimental and control subjects for respiration rate and depth. For the galvanic skin response, experimental subjects demonstrated greater reactivity although the differences were not statistically significant. In conclusion, Rimm and Litvak state that self-verbalizations do have a direct affect on emotional arousal.

May and Johnson (1973), in an attempt to demonstrate divergent autonomic responding to different affective experiences, asked one group of subjects to recall either inhibitory or neutral words and another group to recall either arousal or neutral words. These experimenters utilized a time-locked procedure in which the specific words were remembered only upon the presentation of a tone. Dependent measures were heart rate, skin conductance level, galvanic skin response and

respiration rate. Heart rate and respiration rate both demonstrated significant differences between the two groups. However, the skin conductance levels and the galvanic skin responses were only significantly different between conditions. Therefore, it seems that internally evoked thoughts produce physiological changes and the direction of the change is partially dependent upon the affective nature of the cognitions. Furthermore, this supports the possible importance of cognitive events as significant factors in operant autonomic nervous system conditioning and the mediational view of emotional arousal.

Schwartz (1971) examined autonomic responsivity (heart rate) to three specific thought sequences consisting of numbers, letters and affect-laden words. Heart rate significantly differentiated between the numbers condition and the affect-laden words condition. Schwartz concluded that specific thoughts can act as potential stimuli of autonomic responses.

From the above research, it appears that changing an individual's cognitions or changing the instructional set has direct effects upon autonomic nervous system reactivity. Therefore, if an emotional experience is a function of a specific physiological state and cognitions appropriate to that state, then changing one's cognitions should also affect the nature and/or intensity of the emotional responses.

In a test of this hypothesis, Valins (1966) manipulated male subjects' cognitive representations about their physiological reactions (change of heart rate) and then analyzed their ratings of ten slides of semi-nude females. One-half of the experimental subjects heard their heart rates increase to some slides while the rest of the subjects heard their heart rates decrease to other slides. All subjects were presented

with bogus heart sounds which was a tape recording of square wave pulses produced by a Hewlett-Packard low frequency generator. Valins hypothesized that if cognitive representations about internal events are important in an emotional experience, then these nonveridical representations of physiological changes (bogus heart rates) should have the same effects as veridical representations of true heart rate feedback. The controls were subjected to the same experimental procedures except they were told the audible sounds were meaningless sounds. Dependent measures were attractiveness ratings of slides of semi-nude females made immediately after the experimental procedure, choice of photographs as remuneration for experimental participation and attractiveness ratings made four to five weeks later. Valins' hypothesis was supported: Experimental subjects rated those slides to which they heard their heart rates change as significantly more attractive on two separate occasions. Furthermore, they chose these same slides significantly more often as remuneration for participation in the experiment. When the sounds were not considered their heart beats, as in the control condition, they had virtually no effect upon subjects' ratings.

Valins (1967) in another experiment with emotional and unemotional subjects, replicated his previous results.

Valins and Ray (1967) assumed that cognitive representations of internal events, either veridical or non-veridical, will not only affect one's emotional experience but also overt behavior. To test this hypothesis, all subjects were presented with slides of snakes and slides with the word "shock." When shock slides were presented, subjects were given a mild electric shock. As previously described, experimental subjects "heard" their heart rates increase to the shock slides but not to the

slides of snakes. Control subjects were presented with the same fear stimuli and tape recording but told that these were meaningless sounds. Valins and Ray predicted that experimental subjects would manifest more approach behaviors to a live snake than would controls as they believed their heart rates were affected only by the shock slides but not the snake slides. In the behavioral avoidance task, experimental subjects showed a non-significant trend for greater approach behavior. However, when the subjects with previous experience with snakes were eliminated from the study, the manipulation appeared to have the predicted effect. Upon analysis of the data of those subjects who had never previously touched a snake, significantly closer approach behavior was demonstrated. Therefore, Valins and Ray concluded that avoidance behavior can be modified by information concerning internal reactions. Those subjects who believed that the snake stimuli did not affect them internally were more likely to hold a live snake than those who received no information about their internal reactions.

In summary, it appears that these studies are supporting the hypothesis that the critical mediating variable in one's level of physiological reactivity or overt behavior is cognitive representations of internal or external events or Miller and Dollard's "cue-producing response." In the process of biofeedback, what are the cue-producing responses that lead to a state of deep relaxation and a reduction in anxiety and headache symptoms? In other words, what are the critical mediating variables in a process which allows an individual to change or alter certain psychophysiological responses? No study has yet attempted to answer these questions. Before we can begin to understand the potential possibilities

of biofeedback, we must first attempt to explain the nature of the process in biofeedback.

APPENDIX B

LIST OF ITEMS ON THE FENZ-EPSTEIN MODIFIED ANXIETY SCALE

Autonomic Arousal Items

I am troubled by discomfort in the pit of my stomach.
I have pounding headaches in which I can feel a definite beat.
I am bothered by dizziness.
I notice my heart pounding.
I am afraid I am going to blush.
I feel chilly at temperatures that are comfortable for others.
I suddenly feel hot all over, without apparent cause.
My finger tips or other extremities become cold.
In the absence of physical action my heart beats wildly.
I am either too hot or too cold and cannot get comfortable at a constant room temperature setting.
My mouth feels dry.
I am bothered with blushing.
When embarrassed, I break out in a sweat which annoys me greatly.
I have stomach trouble.
I break out in a sweat, which is not the result of heat of physical exertion.
I am troubled with diarrhea.

Muscle Tension Items

I am troubled with backaches.
The muscles in my neck ache as if they were tied in knots.
The top of my head feels tender.
I have a hard time swallowing.
I have trouble with my hand shaking while I write.
I clench my teeth when anxious.
I am troubled by tension interfering with my speech.
I have trouble with muscles twitching and jumping.
My hands shake when I try to do something.
My skin becomes painfully sensitive.
I have pains in the back of my neck.
I am short of breath without knowing why.
I have sensations of burning, tingling, or crawling in certain parts of my body.
I have enduring headaches that last over several days.
My head feels tender to the point that it hurts when I comb my hair or put on a hat.
I have trouble getting my breath, for no special reason.
I grind my teeth in my sleep.
I have pressure headaches in which my head feels as if it were caught in a vise or as if there was a tight band around it.

Feelings of Insecurity Items

My feelings are easily hurt.
(R) I am an easy going person.
I have a tendency to worry.
I am a nervous person.
I have frightening dreams.
I do not think I am as happy as others.
I have feelings of panic for no special reason.
(R) I am a relaxed person.
I am easily frightened.
(R) I go to sleep without thoughts or ideas bothering me.
I take things hard.
(R) I take things in stride.
Life is a strain for me.
I become upset when I have to wait.
My sleep is fitful and disturbed.
I feel that I am about to go to pieces.
I worry about little things.
I have periods of such restlessness that I cannot sit still.
I become irritable about little things.

APPENDIX C

THE FENZ-EPSTEIN MODIFIED

ANXIETY SCALE

Name:_____ Phone Number:_____

Instructor:_____

THE FOLLOWING ARE SOME STATEMENTS ON FEELINGS, DAYDREAMS, ATTITUDES AND BEHAVIOR. READ EACH STATEMENT AND DECIDE HOW OFTEN IT APPLIES TO YOU. CIRCLE "1" IF THE STATEMENT NEVER APPLIES TO YOU; "5" IF YOU EXPERIENCE IT ALMOST ALL THE TIME; USE "2," "3" AND "4" FOR IN BETWEEN RATINGS. BE HONEST BUT DO NOT SPEND TOO MUCH TIME OVER ANY ONE STATEMENT. AS A RULE, FIRST IMPRESSIONS ARE AS ACCURATE AS ANY.

	Never				Always
I am troubled by discomfort in the pit of my stomach.	1	2	3	4	5
I am troubled with backaches.	1	2	3	4	5
My feelings are easily hurt.	1	2	3	4	5
I have pounding headaches in which I can feel a definite beat.	1	2	3	4	5
The muscles in my neck ache as if they were tied in knots.	1	2	3	4	5
I am an easy-going person.	1	2	3	4	5
I am bothered by dizziness.	1	2	3	4	5
I notice my heart pounding.	1	2	3	4	5
The top of my head feels tender.	1	2	3	4	5
I have a tendency to worry.	1	2	3	4	5
I have a hard time swallowing.	1	2	3	4	5
I am a nervous person.	1	2	3	4	5
I am afraid I am going to blush.	1	2	3	4	5
I have trouble with my hand shaking while I write.	1	2	3	4	5
I have frightening dreams.	1	2	3	4	5
I feel chilly at temperatures that are comfortable for others.	1	2	3	4	5
I clench my teeth when anxious.	1	2	3	4	5
I do not think I am as happy as others.	1	2	3	4	5
I suddenly feel hot all over, without apparent cause.	1	2	3	4	5
I am troubled by tension interfering with my speech.	1	2	3	4	5
I have feelings of panic for no special reason.	1	2	3	4	5
My finger tips or other extremities become cold.	1	2	3	4	5
I have trouble with muscles twitching and jumping.	1	2	3	4	5
I am a relaxed person.	1	2	3	4	5
In the absence of physical action my heart beats wildly.	1	2	3	4	5
My hand shakes when I try to do something.	1	2	3	4	5
I am easily frightened.	1	2	3	4	5
My mouth feels dry.	1	2	3	4	5
My skin becomes painfully sensitive.	1	2	3	4	5

	Never				Always
I go to sleep without thoughts or ideas bothering me.	1	2	3	4	5
I am either too hot or too cold and cannot get comfortable at a constant temperature setting.	1	2	3	4	5
I have pains in the back of my neck.	1	2	3	4	5
I take things hard.	1	2	3	4	5
I am bothered with blushing.	1	2	3	4	5
I am short of breath without knowing why.	1	2	3	4	5
I take things in stride.	1	2	3	4	5
When embarrassed, I break out in a sweat which annoys me greatly.	1	2	3	4	5
I have sensations of burning, tingling, or crawling in certain parts of my body.	1	2	3	4	5
Life is a strain for me.	1	2	3	4	5
I have stomach trouble.	1	2	3	4	5
I have enduring headaches that last over several days.	1	2	3	4	5
I become upset when I have to wait.	1	2	3	4	5
I break out in sweat, which is not the result of heat or physical exertion.	1	2	3	4	5
My sleep is fitful and disturbed.	1	2	3	4	5
I am troubled with diarrhea.	1	2	3	4	5
My head feels tender to the point that it hurts when I comb my hair or put on a hat.	1	2	3	4	5
I feel that I am about to go to pieces.	1	2	3	4	5
I have trouble getting my breath, for no special reason.	1	2	3	4	5
I worry about little things.	1	2	3	4	5
I grind my teeth in my sleep.	1	2	3	4	5
I have periods of such restlessness that I cannot sit still.	1	2	3	4	5
I have pressure headaches in which my head feels as if it were caught in a vise or as if there were a tight band around it.	1	2	3	4	5
I become irritable about little things.	1	2	3	4	5

APPENDIX D

THE STATE-TRAIT ANXIETY INVENTORY-A-STATE SCALE

SELF-EVALUATION QUESTIONNAIRE

Developed by C. D. Spielberger, R. L. Gorsuch and R. Lushene

STAI FORM X-1

Name: _____ Date: _____

Directions: A number of statements which people have used to describe themselves are given below. Read each statement and then blacken in the appropriate circle to the right of the statement to indicate how you feel right now, that is, at this moment. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to best describe your present feelings best.

		NOT AT ALL	SOMEWHAT	MODERATELY SO	VERY MUCH SO
1.	I feel calm	(1)	(2)	(3)	(4)
2.	I feel secure	(1)	(2)	(3)	(4)
3.	I am tense	(1)	(2)	(3)	(4)
4.	I am regretful	(1)	(2)	(3)	(4)
5.	I feel at ease	(1)	(2)	(3)	(4)
6.	I feel upset	(1)	(2)	(3)	(4)
7.	I am presently worrying over possible misfortunes	(1)	(2)	(3)	(4)
8.	I feel rested	(1)	(2)	(3)	(4)
9.	I feel anxious	(1)	(2)	(3)	(4)
10.	I feel comfortable	(1)	(2)	(3)	(4)
11.	I feel self-confident	(1)	(2)	(3)	(4)
12.	I feel nervous	(1)	(2)	(3)	(4)
13.	I am jittery	(1)	(2)	(3)	(4)
14.	I feel "high strung"	(1)	(2)	(3)	(4)
15.	I am relaxed	(1)	(2)	(3)	(4)

	NOT AT ALL	SOMEWHAT	MODERATELY SO	VERY MUCH SO
16. I feel content	(1)	(2)	(3)	(4)
17. I am worried	(1)	(2)	(3)	(4)
18. I feel over-excited and "rattled"	(1)	(2)	(3)	(4)
19. I feel joyful	(1)	(2)	(3)	(4)
20. I feel pleasant	(1)	(2)	(3)	(4)

APPENDIX E

CORRELATION MATRIX FOR THE EMG FEEDBACK

WITH RELAXATION INSTRUCTIONS

GROUP BY SESSIONS

TABLE I

CORRELATION MATRIX FOR THE EMG FEEDBACK WITH RELAXATION INSTRUCTIONS GROUP BY SESSIONS

	EMG	EMG-A	EMG-F	GSR
EMG		0.51311 S=0.1935	0.98051 S=0.0001	-0.82547 S=0.0116
EMG-A			0.51325 S=0.1933	-0.42082 S=0.2992
EMG-F				-0.80358 S=0.0163
GSR				

EMG = EMG Combined, EMG-A = EMG Forearm Flexor, EMG-F = EMG Frontalis, GSR = Galvanic Skin Response.

APPENDIX F

CORRELATION MATRIX FOR THE EMG FEEDBACK WITHOUT RELAXATION INSTRUCTIONS GROUP BY SESSIONS

TABLE II

CORRELATION MATRIX FOR THE EMG FEEDBACK
WITHOUT RELAXATION INSTRUCTIONS
GROUP BY SESSIONS

	EMG	EMG-A	EMG-F	GSR
EMG		0.77348 S=0.0243	0.89638 S=0.0026	-0.34370 S=0.4045
EMG-A			0.61844 S=0.1022	-0.19861 S=0.6373
EMG-F				-0.67218 S=0.0678
GSR				

APPENDIX G

CORRELATION MATRIX FOR THE FALSE FEEDBACK WITH RELAXATION INSTRUCTIONS GROUP BY SESSIONS

TABLE III

CORRELATION MATRIX FOR THE FALSE FEEDBACK
WITH RELAXATION INSTRUCTIONS
GROUP BY SESSIONS

	GSR	EMG-A	EMG-F	GSR
EMG		0.60419 S=0.1126	0.95989 S=0.0002	-0.30630 S=0.4606
EMG-A			0.66218 S=0.0736	-0.54474 S=0.1627
EMG-F				-0.50406 S=0.2028
GSR				

APPENDIX H

CORRELATION MATRIX FOR THE EMG FEEDBACK

WITH RELAXATION INSTRUCTIONS

GROUP BY TRIALS

TABLE IV

CORRELATION MATRIX FOR THE EMG FEEDBACK
WITH RELAXATION INSTRUCTIONS
GROUP BY TRIALS

	EMG	EMG-A	EMG-F	GSR
EMG		0.98140 S=0.0001	0.80049 S=0.0306	-0.89037 S=0.0072
EMG-A			0.71618 S=0.0702	-0.93838 S=0.0018
EMG-F				-0.52537 S-0.2259
GSR				

APPENDIX I

CORRELATION MATRIX FOR THE EMG FEEDBACK WITHOUT RELAXATION INSTRUCTIONS GROUP BY TRIALS

TABLE V

CORRELATION MATRIX FOR THE EMG FEEDBACK
WITHOUT RELAXATION INSTRUCTIONS
GROUP BY TRIALS

	EMG	EMG-A	EMG-F	GSR
EMG		0.93583 S=0.0019	0.65013 S=0.1139	-0.84569 S=0.0165
EMG-A			0.48042 S=0.2752	-0.92808 S=0.0026
EMG-F				-0.22015 S=0.6353
GSR				

APPENDIX J

CORRELATION MATRIX FOR THE FALSE FEEDBACK

WITH RELAXATION INSTRUCTIONS

GROUP BY TRIALS

TABLE VI

CORRELATION MATRIX FOR THE FALSE FEEDBACK
WITH RELAXATION INSTRUCTIONS
GROUP BY TRIALS

	EMG	EMG-A	EMG-F	GSR
EMG		0.96836 S=0.0003	0.96691 S=0.0004	-0.87036 S=0.0108
EMG-A			0.91960 S=0.0034	-0.94997 S=0.0010
EMG-F				-0.77605 S=0.0402
GSR				

APPENDIX K

ANALYSES OF VARIANCE TABLES

TABLE VII

ANALYSIS OF VARIANCE SUMMARY TABLE FOR THE COMBINED EMG MEASURES FOR THE THREE TREATMENT GROUPS

Source	df	M.S.	F	p value
Between Groups				
Treatment (Trt)	2	18.8140	1.11	.3496
Subject (Trt)	21	17.0187		
Within Groups				
Session	7	4.2977	3.17	.0038
Subject x Session (Trt)	147	1.3538		
Trial	6	13.1902	23.62	.0001
Subject x Trial (Trt)	126	.5585		
Trt x Session	14	1.8846	1.39	.1636
Trt x Trial	12	.1061	.19	.9987
Session x Trial	42	.1960	.78	.8473
Trt x Session x Trial	84	.3178	1.26	.0655
Subject x Session x Trial (Trt)	882	.2526		

TABLE VIII

ANALYSIS OF VARIANCE SUMMARY TABLE FOR THE COMBINED EMG MEASURES FOR THE EMG FEEDBACK GROUP WITH RELAXATION INSTRUCTIONS

Source	df	M.S.	F	p value
Within Groups				
Session	7	2.31	3.35	.0053
Subject x Session	49	.68		
Trial	6	4.45	9.88	.0001
Subject x Trial	42	.45		
Session x Trial	42	.22	1.21	.1885
Subject x Session x Trial	294	.18		

TABLE IX

ANALYSIS OF VARIANCE SUMMARY TABLE FOR THE COMBINED EMG MEASURES FOR THE EMG FEEDBACK GROUP WITHOUT RELAXATION INSTRUCTIONS

Source	df	M.S.	F	p value
Within Groups				
Session	7	2.27	2.54	.0260
Subject x Session	49	.86		
Trial	6	5.00	14.56	.0001
Subject x Trial	42	.34		
Session x Trial	42	.30	1.45	.0423
Subject x Session x Trial	294	.20		

TABLE X

ANALYSIS OF VARIANCE SUMMARY TABLE FOR THE COMBINED EMG MEASURES FOR THE FALSE FEEDBACK GROUP WITH RELAXATION INSTRUCTIONS

Source	df	M.S.	F	p value
Within Groups				
Session	7	3.48	1.41	.2243
Subject x Session	49	2.47		
Trial	6	3.94	4.48	.0014
Subject x Trial	42	.88		
Session x Trial	42	.31	.84	.7427
Subject x Session x Trial	294	.36		

TABLE XI

ANALYSIS OF VARIANCE SUMMARY TABLE FOR THE FRONTALIS EMG MEASURES FOR THE THREE TREATMENT GROUPS

Source	df	M.S.	F	p value
Between Groups				
Treatment (Trt)	2	5.4371	.18	.8373
Subject (Trt)	21	30.3665		
Within Groups				
Session	7	9.4028	4.35	.0002
Subject x Session (Trt)	147	2.1609		
Trial	6	6.6532	6.42	.0001
Subject x Trial (Trt)	126	1.0359		
Trt x Session	14	2.4157	1.12	.3469
Trt x Trial	12	.3578	.35	.9787
Session x Trial	42	.3794	.63	.9697
Trt x Session x Trial	84	.4647	.77	.9364
Subject x Session x Trial (Trt)	882	.6045		

TABLE XII

ANALYSIS OF VARIANCE SUMMARY TABLE FOR THE FRONTALIS EMG MEASURES FOR THE EMG FEEDBACK GROUP WITH RELAXATION INSTRUCTIONS

Source	df	M.S.	F	p value
Within Groups				
Session	7	6.64	3.93	.0018
Subject x Session	49	1.69		
Trial	6	2.95	2.73	.0248
Subject x Trial	42	1.08		
Session x Trial	42	.46	.67	.9404
Subject x Session x Trial	294	.69		

TABLE XIII

ANALYSIS OF VARIANCE SUMMARY TABLE FOR THE FRONTALIS EMG MEASURES FOR THE EMG FEEDBACK GROUP WITHOUT RELAXATION INSTRUCTIONS

Source	df	M.S.	F	p value
Within Groups				
Session	7	3.76	2.11	.0598
Subject x Session	49	1.78		
Trial	6	1.58	1.14	.3554
Subject x Trial	42	1.38		
Session x Trial	42	.55	.77	.8478
Subject x Session x Trial	294	.71		

TABLE XIV

ANALYSIS OF VARIANCE SUMMARY TABLE FOR THE FRONTALIS EMG MEASURES FOR THE FALSE FEEDBACK GROUP WITH RELAXATION INSTRUCTIONS

Source	df	M.S.	F	p value
Within Groups				
Session	7	3.82	1.27	.2840
Subject x Session	49	3.01		
Trial	6	2.82	4.43	.0015
Subject x Trial	42	.63		
Session x Trial	42	.28	.72	.8989
Subject x Session x Trial	294	.39		

TABLE XV

ANALYSIS OF VARIANCE SUMMARY TABLE FOR THE FOREARM FLEXOR EMG MEASURES FOR THE THREE TREATMENT GROUPS

Source	df	M.S.	F	p value
Between Groups				
Treatment (Trt)	2	10.5870	1.80	1.8930
Subject (Trt)	21	5.8707		
Within Groups				
Session	7	.4970	.33	.9375
Subject x Session (Trt)	147	1.4940		
Trial	6	23.0472	27.58	.0001
Subject x Trial (Trt)	126	.8356		
Trt x Session	14	1.5156	1.01	.4421
Trt x Trial	12	.3558	.43	.9508
Session x Trial	42	.4519	1.11	.2920
Trt x Session x Trial	84	.6151	1.51	.0029
Subject x Session x Trial (Trt)	882	.4061		

TABLE XVI

ANALYSIS OF VARIANCE SUMMARY TABLE FOR THE FOREARM FLEXOR EMG MEASURES FOR THE EMG FEEDBACK GROUP WITH RELAXATION INSTRUCTIONS

Source	df	M.S.	F	p value
Within Groups				
Session	7	.77	.67	.6997
Subject x Session	49	1.16		
Trial	6	6.87	11.70	.0001
Subject x Trial	42	.58		
Session x Trial	42	.40	1.41	.0566
Subject x Session x Trial	294	.28		

TABLE XVII

ANALYSIS OF VARIANCE SUMMARY TABLE FOR THE FOREARM FLEXOR EMG MEASURES FOR THE EMG FEEDBACK GROUP WITHOUT RELAXATION INSTRUCTIONS

Source	df	M.S.	F	p value
Within Groups				
Session	7	1.44	2.01	.0731
Subject x Session	49	.71		
Trial	6	7.18	7.71	.0001
Subject x Trial	42	.93		
Session x Trial	42	.40	1.27	.1331
Subject x Session x Trial	294	.31		

TABLE XVIII

ANALYSIS OF VARIANCE SUMMARY TABLE FOR THE FOREARM FLEXOR EMG MEASURES FOR THE FALSE FEEDBACK GROUP WITH RELAXATION INSTRUCTIONS

Source	df	M.S.	F	p value
Within Groups				
Session	7	1.31	.50	.8266
Subject x Session	49	2.59		
Trial	6	9.69	9.81	.0001
Subject x Trial	42	.98		
Session x Trial	42	.87	1.42	.0510
Subject x Session x Trial	294	.61		

TABLE XIX

ANALYSIS OF VARIANCE SUMMARY TABLE ON GSR MEASURES FOR THE THREE TREATMENT GROUPS

Source	df	M.S.	df	p value
Between Groups				
Treatment (Trt)	2	14735.01	1.03	.3801
Subject (Trt)	15	14272.06		
Within Groups				
Session	5	10789.00	9.84	.0001
Subject x Session (Trt)	75	1096.22		
Trial	6	6023.50	19.76	.0001
Subject x Trial (Trt)	90	304.79		
Trt x Session	10	1326.65	1.21	.2985
Trt x Trial	12	172.72	.57	.8633
Session x Trial	30	92.79	1.03	.4210
Trt x Session x Trial	60	109.57	1.22	.1367
Subject x Session x Trial (Trt)	449	89.84		

TABLE XX

ANALYSIS OF VARIANCE SUMMARY TABLE ON THE PRE- AND POST-SCORES OF THE STATE-TRAIT ANXIETY INVENTORY-A-STATE

Source	df	M.S.	F	p value
Between Groups				
Treatments	2	125.687	1.43	.2605
Subject (Trt)	21	87.592		
Within Groups				
Pre-Post	1	475.020	8.84	.0073
Subject x Test (Trt)	21	53.735		

APPENDIX L

TABLES OF TRIAL AND SESSION MEANS FOR THE EMG MEASURES AND THE GSR RESPONSES

TABLE XXI

TABLE OF TRIAL AND SESSION MEANS ON THE COMBINED EMG MEASURES FOR EMG FEEDBACK WITH RELAXATION INSTRUCTIONS GROUP

T_1	T_2	T_3	T_4	T_5	T_6	T_7	
2.19	1.79	1.88	1.93	1.74	1.89	1.85	$= 1.90\ S_{1_{\bar{X}}}$
2.49	1.41	1.19	1.20	1.18	1.17	1.10	$= 1.39\ S_{2_{\bar{X}}}$
2.08	1.33	1.53	1.33	1.25	1.43	1.23	$= 1.45\ S_{3_{\bar{X}}}$
1.75	1.33	1.29	1.40	1.36	1.37	1.39	$= 1.41\ S_{4_{\bar{X}}}$
2.15	1.52	1.26	1.32	1.35	1.34	1.19	$= 1.45\ S_{5_{\bar{X}}}$
1.45	1.25	1.27	1.21	1.35	1.44	1.46	$= 1.35\ S_{6_{\bar{X}}}$
2.04	1.15	1.06	1.07	1.20	1.24	1.03	$= 1.25\ S_{7_{\bar{X}}}$
2.04	1.31	1.06	1.10	1.08	1.18	.98	$= 1.25\ S_{8_{\bar{X}}}$
$2.02_{T_{1_{\bar{X}}}}$	$1.39_{T_{2_{\bar{X}}}}$	$1.32_{T_{3_{\bar{X}}}}$	$1.32_{T_{4_{\bar{X}}}}$	$1.31_{T_{5_{\bar{X}}}}$	$1.38_{T_{6_{\bar{X}}}}$	$1.28_{T_{7_{\bar{X}}}}$	

TABLE XXII

TABLE OF TRIAL AND SESSION MEANS ON THE COMBINED EMG MEASURES FOR EMG FEEDBACK WITHOUT RELAXATION INSTRUCTIONS GROUP

T_1	T_2	T_3	T_4	T_5	T_6	T_7	
2.51	2.08	1.86	2.11	1.98	1.67	1.91	$= 2.02\ S_{1_{\bar{X}}}$
2.49	1.57	1.45	1.78	1.86	1.91	1.97	$= 1.86\ S_{2_{\bar{X}}}$
2.74	1.78	1.58	1.43	1.52	1.36	1.48	$= 1.70\ S_{3_{\bar{X}}}$
1.87	1.52	1.27	1.24	1.35	1.34	1.32	$= 1.42\ S_{4_{\bar{X}}}$
2.38	1.41	1.40	1.45	1.46	1.46	1.49	$= 1.58\ S_{5_{\bar{X}}}$
2.66	1.53	1.44	1.50	1.46	1.26	1.26	$= 1.59\ S_{6_{\bar{X}}}$
2.18	1.52	1.55	1.50	1.61	1.86	1.84	$= 1.72\ S_{7_{\bar{X}}}$
1.54	1.55	1.37	1.42	1.32	1.46	1.57	$= 1.46\ S_{8_{\bar{X}}}$
$2.29_{T_{1_{\bar{X}}}}$	$1.62_{T_{2_{\bar{X}}}}$	$1.49_{T_{3_{\bar{X}}}}$	$1.55_{T_{4_{\bar{X}}}}$	$1.57_{T_{5_{\bar{X}}}}$	$1.54_{T_{6_{\bar{X}}}}$	$1.60_{T_{7_{\bar{X}}}}$	

TABLE XXIII

TABLE OF TRIAL AND SESSION MEANS ON THE COMBINED EMG MEASURES FOR FALSE FEEDBACK WITH RELAXATION INSTRUCTIONS GROUP

T_1	T_2	T_3	T_4	T_5	T_6	T_7	
3.00	1.91	1.79	1.90	2.15	1.93	1.67	= 2.05 $S_{1_{\bar{X}}}$
1.89	1.79	1.62	1.64	1.43	1.44	1.65	= 1.64 $S_{2_{\bar{X}}}$
2.43	2.14	1.88	1.70	1.56	1.61	1.57	= 1.84 $S_{3_{\bar{X}}}$
2.33	1.59	1.45	1.37	1.61	1.45	1.56	= 1.62 $S_{4_{\bar{X}}}$
2.27	1.62	1.65	1.71	1.46	1.48	1.46	= 1.66 $S_{5_{\bar{X}}}$
2.21	1.45	1.66	1.46	1.32	1.40	1.63	= 1.59 $S_{6_{\bar{X}}}$
2.59	1.81	2.03	2.23	2.17	2.10	2.59	= 2.22 $S_{7_{\bar{X}}}$
2.46	1.91	1.95	1.89	2.51	1.99	1.97	= 2.10 $S_{8_{\bar{X}}}$
$2.40_{T_{1_{\bar{X}}}}$	$1.78_{T_{2_{\bar{X}}}}$	$1.75_{T_{3_{\bar{X}}}}$	$1.74_{T_{4_{\bar{X}}}}$	$1.78_{T_{5_{\bar{X}}}}$	$1.68_{T_{6_{\bar{X}}}}$	$1.76_{T_{7_{\bar{X}}}}$	

TABLE XXIV

TABLE OF TRIAL AND SESSION MEANS ON THE FRONTALIS EMG MEASURES FOR EMG FEEDBACK WITH RELAXATION INSTRUCTIONS GROUP

T_1	T_2	T_3	T_4	T_5	T_6	T_7	
3.28	2.47	2.48	2.67	3.01	2.62	2.46	= 2.71 $S_{1_{\bar{X}}}$
2.60	1.76	1.44	1.87	2.05	1.69	1.57	= 1.86 $S_{2_{\bar{X}}}$
2.43	1.54	1.84	1.62	1.97	1.84	1.88	= 1.87 $S_{3_{\bar{X}}}$
2.25	2.28	1.66	1.98	1.91	2.51	2.29	= 2.12 $S_{4_{\bar{X}}}$
2.54	1.67	1.67	1.96	1.68	1.46	2.02	= 1.86 $S_{5_{\bar{X}}}$
1.81	2.02	1.56	1.60	1.76	1.86	2.22	= 1.83 $S_{6_{\bar{X}}}$
1.75	1.54	1.54	1.33	1.81	2.06	1.49	= 1.65 $S_{7_{\bar{X}}}$
2.25	1.37	1.43	1.36	2.06	1.66	1.45	= 1.65 $S_{8_{\bar{X}}}$
$2.36_{T_{1_{\bar{X}}}}$	$1.83_{T_{2_{\bar{X}}}}$	$1.70_{T_{3_{\bar{X}}}}$	$1.80_{T_{4_{\bar{X}}}}$	$2.03_{T_{5_{\bar{X}}}}$	$1.96_{T_{6_{\bar{X}}}}$	$1.92_{T_{7_{\bar{X}}}}$	

TABLE XXV

TABLE OF TRIAL AND SESSION MEANS ON THE FRONTALIS EMG MEASURES FOR EMG FEEDBACK WITHOUT RELAXATION INSTRUCTIONS GROUP

T_1	T_2	T_3	T_4	T_5	T_6	T_7	
3.09	2.34	2.70	2.58	2.58	3.32	2.39	= 2.71 $S_{1_{\bar{X}}}$
2.23	1.82	3.00	2.36	2.27	2.12	2.08	= 2.27 $S_{2_{\bar{X}}}$
2.90	2.13	2.03	1.85	2.45	2.20	2.10	= 2.24 $S_{3_{\bar{X}}}$
2.38	1.85	1.66	1.93	1.99	1.92	2.27	= 2.00 $S_{4_{\bar{X}}}$
2.41	1.65	1.64	1.75	2.03	2.58	2.14	= 2.03 $S_{5_{\bar{X}}}$
2.57	1.94	1.81	2.20	1.92	1.84	1.92	= 2.03 $S_{6_{\bar{X}}}$
2.18	2.06	2.11	2.35	2.14	2.08	2.12	= 2.15 $S_{7_{\bar{X}}}$
1.88	1.72	1.81	1.90	1.81	1.82	2.11	= 1.87 $S_{8_{\bar{X}}}$
$2.46_{T_{1_{\bar{X}}}}$	$1.94_{T_{2_{\bar{X}}}}$	$2.10_{T_{3_{\bar{X}}}}$	$2.11_{T_{4_{\bar{X}}}}$	$2.15_{T_{5_{\bar{X}}}}$	$2.23_{T_{6_{\bar{X}}}}$	$2.14_{T_{7_{\bar{X}}}}$	

TABLE XXVI

TABLE OF TRIAL AND SESSION MEANS ON THE FRONTALIS EMG MEASURES FOR FALSE FEEDBACK WITH RELAXATION INSTRUCTIONS GROUP

T_1	T_2	T_3	T_4	T_5	T_6	T_7	
3.01	2.04	2.16	2.70	2.57	2.26	2.46	$= 2.46\ S_{1_{\bar{X}}}$
2.59	1.92	1.74	1.62	1.62	1.72	1.80	$= 1.86\ S_{2_{\bar{X}}}$
2.73	1.95	2.03	1.80	1.94	2.03	2.18	$= 2.09\ S_{3_{\bar{X}}}$
2.15	1.89	1.95	1.62	1.80	1.95	1.91	$= 1.89\ S_{4_{\bar{X}}}$
1.97	1.75	1.84	1.80	1.89	2.08	1.86	$= 1.88\ S_{5_{\bar{X}}}$
2.35	1.57	1.73	1.64	1.84	1.79	1.84	$= 1.82\ S_{6_{\bar{X}}}$
2.60	2.34	2.19	2.30	2.76	1.91	2.68	$= 2.40\ S_{7_{\bar{X}}}$
2.84	2.08	2.21	1.94	2.49	2.15	2.32	$= 2.29\ S_{8_{\bar{X}}}$
$2.53_{T_{1_{\bar{X}}}}$	$1.94_{T_{2_{\bar{X}}}}$	$1.98_{T_{3_{\bar{X}}}}$	$1.93_{T_{4_{\bar{X}}}}$	$2.11_{T_{5_{\bar{X}}}}$	$1.98_{T_{6_{\bar{X}}}}$	$2.13_{T_{7_{\bar{X}}}}$	

TABLE XXVII

TABLE OF TRIAL AND SESSION MEANS ON THE FOREARM FLEXOR EMG MEASURES FOR EMG FEEDBACK WITH RELAXATION INSTRUCTIONS GROUP

T_1	T_2	T_3	T_4	T_5	T_6	T_7		
1.31	1.11	.70	.68	.81	.55	.87	=	.85 $S_{1_{\bar{X}}}$
1.25	.40	.31	.29	.44	.43	.36	=	.50 $S_{2_{\bar{X}}}$
1.27	.94	.60	.35	.37	.40	.32	=	.61 $S_{3_{\bar{X}}}$
1.48	.66	.51	.54	.89	.45	.43	=	.71 $S_{4_{\bar{X}}}$
1.78	.67	.47	.34	.29	.46	.53	=	.65 $S_{5_{\bar{X}}}$
.94	.69	.46	.54	.40	1.33	1.01	=	.77 $S_{6_{\bar{X}}}$
1.99	.64	.61	.65	.67	.67	.44	=	.81 $S_{7_{\bar{X}}}$
1.31	.72	.52	.48	.60	.42	.35	=	.63 $S_{8_{\bar{X}}}$
1.42 $T_{1_{\bar{X}}}$	.73 $T_{2_{\bar{X}}}$	.52 $T_{3_{\bar{X}}}$	.48 $T_{4_{\bar{X}}}$	.56 $T_{5_{\bar{X}}}$	.59 $T_{6_{\bar{X}}}$	.54 $T_{7_{\bar{X}}}$		

TABLE XXVIII

TABLE OF TRIAL AND SESSION MEANS ON THE FOREARM FLEXOR EMG MEASURES FOR EMG FEEDBACK WITHOUT RELAXATION INSTRUCTIONS GROUP

T_1	T_2	T_3	T_4	T_5	T_6	T_7	
1.37	1.16	.92	1.00	.58	.56	.75	= .91 $S_{1_{\bar{X}}}$
1.75	.84	1.14	1.42	.99	.73	1.48	= 1.19 $S_{2_{\bar{X}}}$
2.43	1.10	.83	.84	.70	.78	.62	= 1.04 $S_{3_{\bar{X}}}$
1.40	.68	.64	.64	.40	.60	.66	= .71 $S_{4_{\bar{X}}}$
1.71	.99	.94	.71	.52	.70	.64	= .89 $S_{5_{\bar{X}}}$
1.49	.86	1.06	.79	.80	.51	.44	= .85 $S_{6_{\bar{X}}}$
1.55	.73	.60	.60	.90	.90	.52	= .83 $S_{7_{\bar{X}}}$
1.38	.64	.39	.52	.61	.50	1.03	= .72 $S_{8_{\bar{X}}}$
$1.63_{T_{1_{\bar{X}}}}$	$.87_{T_{2_{\bar{X}}}}$	$.81_{T_{3_{\bar{X}}}}$	$.81_{T_{4_{\bar{X}}}}$	$.69_{T_{5_{\bar{X}}}}$	$.66_{T_{6_{\bar{X}}}}$	$.77_{T_{7_{\bar{X}}}}$	

TABLE XXIX

TABLE OF TRIAL AND SESSION MEANS ON THE FOREARM FLEXOR EMG MEASURES FOR FALSE FEEDBACK WITH RELAXATION INSTRUCTIONS GROUP

T_1	T_2	T_3	T_4	T_5	T_6	T_7	
2.64	.80	.55	.41	.73	.57	.55	= .89 $S_{1_{\bar{X}}}$
1.60	.81	.91	.68	.62	1.05	.83	= .93 $S_{2_{\bar{X}}}$
1.50	1.86	.92	.93	1.43	.70	1.06	= 1.20 $S_{3_{\bar{X}}}$
1.95	1.18	.65	.37	.45	.76	.97	.90 $S_{4_{\bar{X}}}$
1.65	1.33	1.24	.94	.76	.59	.47	= 1.00 $S_{5_{\bar{X}}}$
1.89	1.05	.49	.42	.62	.42	.54	= .77 $S_{6_{\bar{X}}}$
1.65	.81	.64	1.05	.74	1.39	.83	= 1.01 $S_{7_{\bar{X}}}$
1.62	1.36	1.21	.91	1.90	.70	.84	= 1.22 $S_{8_{\bar{X}}}$
$1.81_{T_{1_{\bar{X}}}}$	$1.15_{T_{2_{\bar{X}}}}$	$.83_{T_{3_{\bar{X}}}}$	$.71_{T_{4_{\bar{X}}}}$	$.91_{T_{5_{\bar{X}}}}$	$.77_{T_{6_{\bar{X}}}}$	$.76_{T_{7_{\bar{X}}}}$	

TABLE XXX

TABLE OF TRIAL AND SESSION MEANS ON THE GSR RESPONSES FOR EMG FEEDBACK WITH RELAXATION INSTRUCTIONS GROUP

T_1	T_2	T_3	T_4	T_5	T_6	T_7	
66.50	68.83	75.00	83.33	85.00	89.16	88.33	= 79.45 $S_{1_{\bar{X}}}$
81.50	105.83	115.83	116.66	122.83	112.00	106.40	= 108.78 $S_{2_{\bar{X}}}$
98.16	120.00	115.00	120.16	120.00	120.00	109.50	= 114.69 $S_{3_{\bar{X}}}$
96.16	118.16	122.50	116.66	123.50	121.66	115.83	= 116.35 $S_{4_{\bar{X}}}$
96.66	115.66	120.00	125.83	127.66	126.66	127.00	= 119.92 $S_{5_{\bar{X}}}$
106.00	119.00	123.50	124.66	123.50	124.66	108.00	= 118.47 $S_{6_{\bar{X}}}$
$90.83_{T_{1_{\bar{X}}}}$	$107.91_{T_{2_{\bar{X}}}}$	$111.97_{T_{3_{\bar{X}}}}$	$114.55_{T_{4_{\bar{X}}}}$	$117.08_{T_{5_{\bar{X}}}}$	$115.69_{T_{6_{\bar{X}}}}$	$109.25_{T_{7_{\bar{X}}}}$	

TABLE XXXI

TABLE OF TRIAL AND SESSION MEANS ON THE GSR RESPONSES FOR EMG FEEDBACK WITHOUT RELAXATION INSTRUCTIONS GROUP

T_1	T_2	T_3	T_4	T_5	T_6	T_7	
101.66	100.00	107.50	106.66	109.16	106.16	109.50	$= 105.80\ S_{1_{\bar{X}}}$
107.16	117.50	122.00	125.16	127.50	131.33	132.16	$= 123.26\ S_{2_{\bar{X}}}$
105.00	115.83	122.83	124.16	126.66	127.00	128.33	$= 121.40\ S_{3_{\bar{X}}}$
105.83	115.33	121.66	123.16	116.33	123.33	123.00	$= 118.42\ S_{4_{\bar{X}}}$
115.00	124.33	124.33	126.50	128.00	129.83	130.50	$= 125.50\ S_{5_{\bar{X}}}$
105.00	120.00	125.00	127.50	131.00	131.00	130.67	$= 124.23\ S_{6_{\bar{X}}}$
$106.61_{T_{1_{\bar{X}}}}$	$115.50_{T_{2_{\bar{X}}}}$	$120.55_{T_{3_{\bar{X}}}}$	$122.19_{T_{4_{\bar{X}}}}$	$123.11_{T_{5_{\bar{X}}}}$	$124.77_{T_{6_{\bar{X}}}}$	$125.66_{T_{7_{\bar{X}}}}$	

TABLE XXXII

TABLE OF TRIAL AND SESSION MEANS ON THE GSR RESPONSES FOR FALSE FEEDBACK WITH RELAXATION INSTRUCTIONS GROUP

T_1	T_2	T_3	T_4	T_5	T_6	T_7	
91.66	101.66	119.16	121.66	122.50	112.00	122.33	= 113.00 $S_{1_{\bar{X}}}$
109.66	123.33	129.16	130.00	130.16	131.83	127.66	= 125.97 $S_{2_{\bar{X}}}$
106.83	122.50	124.16	131.00	133.33	133.33	134.16	= 126.47 $S_{3_{\bar{X}}}$
114.83	124.33	130.66	131.33	132.83	134.33	134.16	= 128.92 $S_{4_{\bar{X}}}$
120.16	122.16	124.66	126.66	127.16	130.00	128.66	= 125.64 $S_{5_{\bar{X}}}$
116.16	128.83	127.83	129.50	130.00	130.16	131.33	= 127.69 $S_{6_{\bar{X}}}$
$109.88_{T_{1_{\bar{X}}}}$	$120.47_{T_{2_{\bar{X}}}}$	$125.94_{T_{3_{\bar{X}}}}$	$128.36_{T_{4_{\bar{X}}}}$	$129.33_{T_{5_{\bar{X}}}}$	$128.61_{T_{6_{\bar{X}}}}$	$129.72_{T_{7_{\bar{X}}}}$	

APPENDIX M

CHI-SQUARE TABLES

TABLE XXXIII

SUSPICION OF FALSE FEEDBACK BY THE TREATMENT GROUPS

	BC	CD	AD
Suspect	0	0	1
No suspect	8	8	7

$X^2 = 2.08$, $p < .50$.

TABLE XXIV

SUSPICION OF FALSE FEEDBACK GROUP BY EXPERIMENTERS

	Suspect	No Suspect
BC	1	1
CD	0	2
AD	0	2

$X^2 = 2.40$, $p < .50$.

VITA

Nancy Hall McSwain

Candidate for the Degree of

Doctor of Philosophy

Thesis: A DOUBLE-BLIND INVESTIGATION OF COGNITIVE FACTORS IN MUSCULAR BIOFEEDBACK

Major Field: Psychology

Biographical:

Personal Data: Born in Montgomery, Alabama, September 15, 1943, the daughter of Mr. and Mrs. Marshall Shelby Hall.

Education: Graduated from Sidney Lanier High School, Montgomery, Alabama, in May, 1961; received the Bachelor of Arts degree in Psychology from Huntingdon College, Montgomery, Alabama, in May, 1969; received the Master of Science degree in Psychology from Oklahoma State University in July, 1971; completed a Clinical Internship at the University of Oklahoma Health Sciences Center, Oklahoma City, Oklahoma, August, 1977; completed the requirements for the Doctor of Philosophy degree in Psychology at Oklahoma State University in July, 1978.

Professional Experience: Practicum student at the Payne County Guidance Center, 1971 and 1974-1975; practicum student at the Psychological Services Center in Stillwater, Oklahoma, 1975-1976; completed Clinical Psychology Internship at University of Oklahoma Health Sciences Center in Oklahoma City, Oklahoma, 1976-1977; psychological assistant at the Psychological Services Center in Stillwater, Oklahoma, 1977-1978, graduate teaching assistant, 1969-1970; instructor of undergraduate psychology courses, 1971-1973.

Professional Organizations and Honors: Recipient of the National Institute of Health Grant, 1975-1976; member of American Psychological Association, Oklahoma Psychological Association, Association of Women in Psychology, and Southwestern Psychological Association.