53468

National Library of Canada | Bibliothèque nationale du Canada

CANADIAN THESES ON MICROFICHE | *THÈSES CANADIENNES SUR MICROFICHE*

NAME OF AUTHOR/*NOM DE L'AUTEUR* Erik Petersen

TITLE OF THESIS/*TITRE DE LA THÈSE* Reactive Modality Biofeedback Training: A Pilot Investigation

UNIVERSITY/*UNIVERSITÉ* Simon Fraser University

DEGREE FOR WHICH THESIS WAS PRESENTED/*GRADE POUR LEQUEL CETTE THÈSE FUT PRÉSENTÉE* M.A. (Education)

YEAR THIS DEGREE CONFERRED/*ANNÉE D'OBTENTION DE CE DEGRÉ* 1982

NAME OF SUPERVISOR/*NOM DU DIRECTEUR DE THÈSE* Dr. Bryan Hiebert

Permission is hereby granted to the NATIONAL LIBRARY OF CANADA to microfilm this thesis and to lend or sell copies of the film.

The author reserves other publication rights, and neither the thesis nor extensive extracts from it may be printed or otherwise reproduced without the author's written permission.

L'autorisation est, par la présente, accordée à la BIBLIOTHÈQUE NATIONALE DU CANADA de microfilmer cette thèse et de prêter ou de vendre des exemplaires du film.

L'auteur se réserve les autres droits de publication; ni la thèse ni de longs extraits de celle-ci ne doivent être imprimés ou autrement reproduits sans l'autorisation écrite de l'auteur.

DATED/*DATÉ* Dec 21/81 SIGNED/*SIGNÉ* [redacted]

PERMANENT ADDRESS/*RÉSIDENCE FIXÉ* [redacted]

NOTICE

The quality of this microfiche is heavily dependent upon the quality of the original thesis submitted for microfilming. Every effort has been made to ensure the highest quality of reproduction possible.

If pages are missing, contact the university which granted the degree.

Some pages may have indistinct print especially if the original pages were typed with a poor typewriter ribbon or if the university sent us a poor photocopy.

Previously copyrighted materials (journal articles, published tests, etc.) are not filmed.

Reproduction in full or in part of this film is governed by the Canadian Copyright Act, R.S.C. 1970, c. C-30. Please read the authorization forms which accompany this thesis.

THIS DISSERTATION
HAS BEEN MICROFILMED
EXACTLY AS RECEIVED

AVIS

La qualité de cette microfiche dépend grandement de la qualité de la thèse soumise au microfilmage. Nous avons tout fait pour assurer une qualité supérieure de reproduction.

S'il manque des pages, veuillez communiquer avec l'université qui a conféré le grade.

La qualité d'impression de certaines pages peut laisser à désirer, surtout si les pages originales ont été dactylographiées à l'aide d'un ruban usé ou si l'université nous a fait parvenir une photocopie de mauvaise qualité.

Les documents qui font déjà l'objet d'un droit d'auteur (articles de revue, examens publiés, etc.) ne sont pas microfilmés.

La reproduction, même partielle, de ce microfilm est soumise à la Loi canadienne sur le droit d'auteur, SRC 1970, c. C-30. Veuillez prendre connaissance des formules d'autorisation qui accompagnent cette thèse.

LA THÈSE A ÉTÉ
MICROFILMÉE TELLE QUE
NOUS L'AVONS REÇUE

REACTIVE MODALITY BIOFEEDBACK TRAINING:

A PILOT INVESTIGATION

by

Erik Petersen

B.A., Carleton University, 1971

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF

THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF ARTS (EDUCATION)

in the Faculty

of

Education

SIMON FRASER UNIVERSITY

December 1981

PARTIAL COPYRIGHT LICENSE

I hereby grant to Simon Fraser University the right to lend my thesis, project or extended essay (the title of which is shown below) to users of the Simon Fraser University Library, and to make partial or single copies only for such users or in response to a request from the Library of any other university, or other educational institution, on its own behalf or for one of its users. I further agree that permission for multiple copying of this work for scholarly purposes may be granted by me or the Dean of Graduate Studies. It is understood that copying or publication of this work for financial gain shall not be allowed without my written permission.

Title of Thesis/Project/Extended Essay

REACTIVE MODALITY BIOFEEDBACK TRAINING:

A PILOT INVESTIGATION

Author: _____

(signature)

Erik Petersen

(name)

Dec 21/81

(date)

APPROVAL

Name: Erik Petersen

Degree: M.A. (Education)

Title of Thesis: Reactive Modality Biofeedback Training: A Pilot Investigation

Examing Committee

Chairman: P. Winne

B. Hiebert
Senior Supervisor

A. Horvath
Graduate Faculty Associate

A. Hayduk
Assistant Professor
Department of Instructional Design
Athabasca University
14515 - 122 Avenue
Edmonton, Alberta
T5L 2W4

December 21, 1981
Date Approved

ABSTRACT

The biofeedback treatment of anxiety using a common target training response for all subjects has proven to be effective only for a subset of the treatment population. Researchers have suggested that a way to increase the size of this population is to match the biofeedback target training behaviour to the physiological modality that constitutes the major component of the physiological anxiety reaction for the individual.

This study was a preliminary analogue investigation of biofeedback treatment for anxiety where the target training behaviour was matched to the subjects' physiological modality that was most reactive to a cognitive stressor.

Twenty undergraduate students with normal anxiety levels were alternately assigned to either a most reactive modality biofeedback training group, or to a delayed treatment control group. Frontal EMG, peripheral skin temperature, skin resistance and heart rate were monitored during pre- and posttest administration of a psychophysiological stress profile.

The data were analyzed by comparing groups with mean levels of physiological activity under stress and nonstress conditions, and groups by scores on the State-Trait-Anxiety Inventory (STAI). Alternate analyses were performed using subject responsiveness as a dependent measure.

A disproportionately high number of heart rate reactive subjects precluded a full test of most reactive modality training as a generic

treatment. Results indicated, however, that heart rate reactive subjects receiving heart rate biofeedback training experienced heart rates that were significantly lower than control subjects during stress conditions. Treatment subjects also maintained frontal EMG levels under stress whereas control subjects did not. Analyses using subject responsiveness to stress revealed that mean level reductions in frontal EMG were accompanied by decreases in frontal EMG variability, but that decreases in heart rate mean levels could occur despite increases in heart rate variability.

Implications are discussed, namely that reactive modality training may be more beneficial than uniform target training biofeedback, and that response variability may provide new information about self-control of physiological responses. The use of a stress profile and considerations concerning adequate controls in biofeedback research are also discussed.

TABLE OF CONTENTS

TABLE OF CONTENTS (continued)

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

Biofeedback training has frequently been used in the treatment of anxiety. The results of these efforts have not been without success yet, after almost ten years of usage, the best conclusion is that biofeedback remains an effective treatment only for an unspecified subset of the population (Schwartz, 1981).

Gatchel (1979) has argued that a crucial variable for the effective use of biofeedback in anxiety may be the choice of physiological response trained. Previous work has almost exclusively employed a uniform physiological target training behaviour across all subjects without first determining whether that target behaviour constituted a major physiological component of anxiety for that specific individual (p. 141).

This thesis was conceived as a pilot study to determine whether biofeedback training in a subject's most reactive physiological modality constituted an effective strategy in the biofeedback treatment of anxiety. To test this question, a psychophysiological assessment procedure using a stress induction procedure (i.e. a nonstress-stress-nonstress design) was developed to preassess a subject's most reactive physiological modality. Frontal electromyography (EMG), peripheral skin temperature, skin resistance and heart rate were monitored during the nonstress-stress-nonstress sequence and the results compared to determine most reactive modality. Twenty undergraduate students with

normal anxiety levels were assigned to treatment and control conditions. The treatment condition provided biofeedback training in the subject's assessed most reactive modality. The control condition involved contact with biofeedback apparatus and the promise of receiving instruction in progressive relaxation training which subjects were told had previously been shown to be an effective stress management procedure. Postscore comparisons were made on both stress and nonstress indices of physiological arousal and on self-report measures of anxiety.

In the remainder of this chapter, the reasons for undertaking the study will be reviewed. Subsequent chapters will outline the theoretical rationale of the experiment (Chapter two); the methodology employed (Chapter three), and the results of the experiment (Chapter four). Chapter five will provide a discussion of the results and present suggestions for further research.

Overview

The theoretical justification for the use of biofeedback in the treatment of anxiety is derived from the principles derived by Wolpe (1958) in systematic desensitization. Wolpe's theory of anxiety follows the classical conditioning model whereby repeated pairing of a conditioned stimulus (CS) with an unconditioned stimulus (UCS) leads to the elicitation of a conditioned response (CR) by the CS alone. This conditioned (anxiety) response is marked by excessive levels of sympathetic nervous system arousal which the therapist treats by teaching the client a competing response involving decreased sympathetic arousal and/or parasympathetic dominance. This competing response

which is thought to be incompatible with sympathetic arousal weakens the bond between the stimulus and the anxiety response CR leading to less anxious responding. The process, in Wolpe's formation, is referred to as reciprocal inhibition.

When biofeedback has been used to induce the competing behavior, frontal EMG has typically been the physiological target training response. This practice hinges on the assumption that training in frontal EMG reduction will result in reduced arousal of other physiological response systems. It is this state of low physiological arousal, often referred to as cultivated low arousal (Stoyva & Budzynski, 1974), that serves as the competing response incompatible with anxiety. The evidence for cultivated low arousal is, however, contradictory. While there is empirical support for physiological generalization of low arousal as a result of frontal EMG biofeedback training (Stoyva & Budzynski, 1974; Glaus & Kostes, 1977; DeGood & Chisholm, 1977), there is also alternate evidence that suggests when frontal EMG biofeedback without supplementary autogenic training and/or progressive relaxation instructions is used, control of the trained frontal EMG response is the sole result and generalization to other physiological responses does not occur (Alexander & White, 1979; Alexander, 1975; Fridlund, Fowler & Pritchard, 1980). Recent evidence is also unanimous in indicating that even when cultivated low arousal does occur in nonstress conditions, the generalized low arousal is not maintained under stress induction conditions. Subjects under stress are able to maintain their learned physiological reductions only in

the frontal EMG training target response modality (Burish, Hendrix & Frost, 1981; Gatchel, Korman, Weis, Smith & Clark, 1978; De Good & Adams, 1976). These results provide empirical support for Gatchel's (1979) suggestion that the inadequacy of biofeedback in the treatment of anxiety for the population at large may reflect the fact frontal EMG biofeedback is an effective treatment only for that subset of the population for whom frontal EMG constitutes the major physiological component of anxiety. Employing this rationale it follows that by tailoring biofeedback training target response to the subject's dominant physiological component of anxiety, biofeedback training may become an effective treatment for anxiety for the entire population.

This Study

To date, no systematic studies have been conducted which matched biofeedback training target response with the dominant physiological component of anxiety for the treatment population. Moreover, no studies which have used a training target response other than frontal EMG have monitored other physiological response modalities to assess the physiological generalization effects associated with providing biofeedback training in that modality. This study was a preliminary analogue investigation of matched training target response biofeedback training while simultaneously monitoring other physiological response modalities. A subject's most reactive physiological response when exposed to cognitive stressors was used to determine which physiological response system constituted the major component of physiological anxiety for a

subject. The decision to use most reactive physiological modality was developed from Malmo's (Malmo, 1975) hypothesis of symptom specificity. According to Malmo, each individual has a unique and idiosyncratic response to stress in which one physiological response is maximally reactive to stress. Persistent exposure to stress results in over-responsiveness to stress and greater recovery periods required to return to prestressor physiological baseline levels. The ultimate consequence of persistent exposure to stress is overelevated physiological baselines leading to the development of clinically significant symptomatology (Malmo, 1975; Malmo, Wallerstein & Shagass, 1953; Malmo, Shagass & Davis, 1950).

In this study, the subjects employed had normal anxiety levels so that overelevated physiological baselines and/or clinically significant symptomatology were not expected. A subject's responsiveness to stressors or reactivity was, therefore chosen to determine the subject's dominant physiological component of anxiety. Moreover, since the treatment condition involved the providion of biofeedback training in one physiological response modality a method for determining a subject's most reactive physiological modality was required. Since no work has been done in determining a subject's most reactive modality, a second purpose of this study was to develop and assess a procedure for determining the greatest reactivity between different physiological response modalities in reaction to a stressor.

A third purpose of the study was to investigate the use of an alternate physiological dependent measure. In the past, mean levels

of physiological activity during resting conditions and, less frequently during stress conditions, have been used to evaluate the outcome of biofeedback treatment. Historically there are two reasons for this. One is the obvious face validity which increasing and decreasing physiological baselines have in demonstrating that self-control of visceral and autonomic responses was possible. The second reason was the clinical utility which overelevated baselines have for the treatment of clients with fully developed symptomatology. The use of subjects with normal levels of anxiety eliminated the clinical utility argument in favor of mean levels of physiological activity as a dependent measure. Additionally the use of a stress induction procedure implies that it is the subject's responsiveness or response variability as one experiences the nonstress-stress-nonstress sequence that is the appropriate dependent measure. For this reason, a comparison of response variability versus mean levels of phhsiological activity was included as a goal of this study.

Summary

This thesis was designed as a preliminary analogue investigation of the effectiveness of biofeedback training in the reduction of anxiety when the biofeedback training target response was matched to the subject's major physiological component of anxiety. A subject's major physiological anxiety response was defined as the subject's most reactive physiological modality to a cognitive stressor. Additional issues of interest were outlined. These included the

question of to what extent physiological generalization to other response modalities (cultivated low arousal) occurs as a result of providing feedback in a training target response other than frontal EMG; the development and assessment of a procedure for determing a subject's most reactive modality; and the comparison of response variability with mean levels of physiological activity as a physiological dependent measure.

In the next chapter, the theoretical rationale underlying this thesis will be presented.

CHAPTER II

THEORETICAL RATIONALE

Introduction

The preceding chapter presented the position that providing biofeedback training in a physiological modality constituting a major physiological component of anxiety for a given individual might be important for the effective use of biofeedback in the treatment of anxiety. In part, this claim was derived from the conflicting evidence surrounding the assumption of cultivated low arousal as a result of frontalis EMG reduction training. In this chapter the concept of anxiety will be outlined, the evidence cited on both sides of the cultivated low arousal dispute will be reviewed, and conclusions drawn. This will be followed by a consideration of the physiological theory of human emotion and the concepts response specificity, response stereotyping and symptom specificity which support the use of reactivity in determining the major physiological component of anxiety. Next, the evidence suggesting differential results as a consequence of physiological dependent measure will be outlined and a review of the literature surrounding the psychophysiological stress profile will be presented. Finally, hypotheses will be developed.

Anxiety

The consensual view of anxiety in psychological research is that it is an unobservable construct inferred to account for some sort of

behavior (Gatchel, 1979). The construct consists of cognitive, behavioral and physiological components which interact to give anxiety its meaning. The alternate events that can potentially denote each of these components results in innumerable permutations of the actual representation of anxiety. For example, although attempts have been made to specify the conditions under which certain stimuli are more likely to be perceived as threatening, the actual number of objective stimuli which lead to perceived threat are unlimited. Similarly, the behavioral and physiological representations of anxiety are highly variable. The behavioral symptoms of anxiety may be comprised of fidgeting, stuttering, sleeplessness or flight to mention several examples. The physiological component may involve increased muscle tension, increased heart rate and/or blood pressure, peripheral vasoconstriction, decreased skin resistance or any singular or combinatory indices of physiological arousal.

Given this conceptual complexity of anxiety it is unlikely that any successful treatment of anxiety will focus exclusively on one of these components of anxiety. Moreover, when approaches emphasizing one component of anxiety are employed, other researchers whose primary approach focuses on one of the other two components of anxiety are apt to provide an alternate interpretation in keeping with their own orientation. This is true also of biofeedback. The biofeedback literature tends to view biofeedback as a physiological process in which cultivated low physiological arousal is incompatible with cognitive and behavioral components of anxiety. Biofeedback has also

been considered primarily a cognitive process (eg., Lazarus, 1975; Meichenbaum, 1976), or a behavioral process (eg., Black, Cott & Pavloski, 1977). Meichenbaum's (1976) approach is illustrative of the cognitive interpretation. Meichenbaum (1976) argues that cultivated low physiological arousal as a result of biofeedback training is important only insofar as client recognition of low arousal serves as a cue to emit incompatible cognitions and behaviors. It is the change in a client's internal dialogue which is influencable through positive self-statements that is the crucial factor in the biofeedback treatment of anxiety. In Meichenbaum's formulation we see a change in the role of cultivated low arousal from that of the anxiety incompatible response per se, to that of the cue for emitting incompatible cognitive and behavioral responses.

While it is recognized here that there is dissent in interpreting the role of biofeedback in the treatment of anxiety, the main concern of this thesis is with the physiological mechanisms involved in establishing cultivated low arousal. Aside from the important problem of the role of biofeedback in the treatment of anxiety, a more basic issue is whether cultivated low physiological arousal is possible using biofeedback treatment alone. It is this cultivated low arousal controversy that will be dealt with next.

The Cultivated Low Arousal Controversy

Frontal EMG reduction is the most frequently chosen training target response in the treatment of anxiety. Explicit in this choice is the assumption that training in frontal EMG reduction leads to a cultivated low arousal state. The most often cited research in support of this assumption is a set of parameter studies by Stoyva and Budzynski (1974). Although the numbers are small (N = 5 per group), Stoyva and Budzynski found that training in frontalis EMG reduction was accompanied by decreases in forearm tension, heart and respiration rates, and cortical changes in the direction of lower arousal. Verbal reports of "thoroughly relaxed" subjects in this study also revealed sensations of heaviness, warmth and drowsiness which the authors took to indicate generalization of effects to other autonomic responses.

Other studies have provided supportive evidence. Glaus and Kostes (1979) trained 30 undergraduate students in either frontalis EMG increase, frontalis EMG decrease or frontalis EMG noncontingent (ie. unrelated to facial EMG) conditions. Results indicated that covariation between frontalis and other facial EMG measures decreased for both frontalis conditions but remained the same for the noncontingent group.

DeGood and Chisholm (1977) found that a group (N = 10) trained in frontalis EMG reduction exhibited decreases in heart and respiration rates and increases in pareital alpha density indicative of low arousal, though it was also reported that the contradictory result of increased peripheral vasoconstriction occurred. Blanchard, Haynes, Kallman

and Harkey (1976) found that training in reduction of frontalis EMG was equally as effective as training in reduction of systolic blood pressure in producing decreases in systolic blood pressure in normotensive subjects.

Finally, clinical studies are often cited in support of the generalization hypothesis. Townsend, House and Addario's (1975) study which employed a biofeedback mediated relaxation group versus a psychotherapy group in chronic anxiety patients serves as an example. Although Townsend et al. found significant decreases in frontalis EMG, mood disturbance and trait anxiety in the biofeedback mediated relaxation group relative to the psychotherapy group, their results are confounded by the conjunctive use of relaxation training. Other clinical reports similarly employ relaxation or autogenic training-like instructions which pertain to bodily functions other than those for which biofeedback training is provided. When it is considered that even such closely related functions as heart rate and blood pressure have been shown to be controlled in independent and opposite directions as the result of instructions alone (Schwartz, 1972), it is possible to see that such results cannot be taken as evidence in support of multi-response autonomic generalization as a result of biofeedback training alone.

In contradiction to these generalization arguments, there is recent and accumulating evidence suggesting fractionalization or specificity of physiological responses due to biofeedback training.

Alexander (1975) tested directly the assumptions that frontalis EMG training generalized to untrained muscles (forearm and lower leg)

and that subjective feelings of relaxation were related to frontalis EMG reduction. Using a no feedback control (N=14), no support was found for either assumption. In two subsequent studies, Alexander and White (1979) instructed both treatment and control subjects to relax a particular area (either frontalis or forearm) as much as possible prior to EMG biofeedback training. After three sessions in the first study and five sessions in the second study, the controls displayed a slight albeit nonsignificant advantage over the biofeedback subjects in frontal EMG reductions. The authors concluded that frontal EMG reduction does not lead to generalization to other muscular sites.

Fridlund, Fowler, and Pritchard (1980) also tested the assumption of frontalis EMG reduction generalization to other muscular sites. Using a computer controlled scanning electromycrograph, Fridlund et al. exposed four normal male subjcts to five alternate day sessions of frontalis EMG reduction while continuously monitoring EMG activity from the frontalis and seven adjunctive muscle groups. No support was found for the generalization of frontal EMG reduction.

Perhaps the most significant study in relation to this thesis is that by Gatchel, Korman, Weis, Smith and Clarke (1978). The study departs from others cited in that it employed a stress induction procedure (threat of electric shock) to evaluate treatment while simultaneously monitoring frontalis EMG, heart rate, respiration rate and skin conductance. Using 12 undergraduate volunteers as subjects, Gatchel et. al found that in the absence of external threat, training in frontalis

EMG reduction was accompanied by decreases in heart and respiration rates but an increase in skin conductance indicative of sympathetic arousal. Most importantly, however, they found that while subjects were able to maintain their EMG decreases (which they were trained in) in the presence of the external threat, they were unable to maintain their heart rate decreases under this condition. Heart rate, skin conductance and self-report of anxiety all increased in the face of the external threat. Gatchel et al. conclude the results clearly demonstrate specificity of learned physiological self control under stress inducing conditions.

Summary

As can be seen, the conclusion of cultivated low arousal via frontalis EMG reduction is controversial. The most optimistic conclusion in favor of the generalization hypothesis seems to be that in the absence of external threats, the frontalis response and viscerally mediated responses of respiration rate and heart rate are more closely related than the other autonomic responses of peripheral vasoconstriction (DeGood & Chisholm, 1977) and skin conductance (Gatchel et al., 1978) though even this position is contentious (Alexander, 1975; Alexander & White, 1979; Fridlund et al., 1980). In the presence of external threat, however, there is evidence that suggests this relationship is untenable and that there is fractionalization of specificity of the learned physiological response. It is interesting to note that even Budsynski whose work is universally cited in support of the culti-

vated low arousal as a result of frontal EMG training typically uses, in his clinical work, a physiological target response other than frontal EMG. The target response chosen is one that has previously been determined to have over-elevated baseline levels for that individual. Frontal EMG training is initially used solely to facilitate training in the over-elevated target response (Budzynski, 1978).

The conclusions drawn from this evidence support two broad points. First, they suggest that only the trained modality remains under self-control during stress conditions. It follows from this point that maximum clinical utility would be derived by training clients in their most responsive modality rather than a uniform frontalis EMG modality which may not constitute a component of their anxiety. Training in a physiological response system that is not a physiological component of anxiety will not lead to control of the dominant physiological anxiety response during stress conditions.

Second, the evidence underscores the position that the effectiveness of biofeedback training must be evaluated under stress induction conditions. The failure of previous biofeedback researchers to assess training under these conditions has greatly limited the application of their results for stress management purposes.

In the next section, the theoretical physiological basis for most reactive response modality as the determinant of appropriate response will be given.

Physiological Theory of Emotion

The general physiological theory of human emotion is that during normal activities there is a homeostasis between the sympathetic and parasympathetic branches of the autonomic nervous system. Heightened arousal, including anxiety, results in the activation and dominance of the sympathetic system marked by increases in respiration, heart rate, muscular tension and peripheral vasoconstriction; decreases in skin resistance; intestinal contraction, secretion of epinephrine (adrenalin), pupillary dilation, and cessation of salivary gland secretion. During periods of rest or relaxation, the parasympthatic system becomes dominant with the converse physiological reactions. The initial conception of activation was that these physiological responses acted in concert. Different patterns of activity, where they existed, were of little practical or theoretical importance (Cannon, 1929).

Further investigations, however, have shown that individual response variations or patterns are important. There are both unique intraindividual patterns of physiological response across all stimuli, and unique physiological reactions to different types of stimuli that are common across persons. These response patterns are known respectively as individual response sterotypy and stimulus-response specificity. Below, a brief summary of the evidence for the two concepts will be presented, and the implications of each for the present study given.

Individual Response Stereotypy

Individual response stereotypy refers to the tendency of an individual to exhibit a consistent but idiosyncratic pattern of autonomic response in response to the presentation of a stimulus or stimuli (Lacey, Bateman & Van Lehn, 1953). The demonstration of sterotypy involves the concordance in rankings of changes in sympathetic activity from nonstress to stress conditions, and/or the rankings of absolute level of sympathetic activity. Lacey et al. (1953) used a lability score defined as the maximum displacement a physiological function exhibits during stress (and measured by a normalized T score) to provide comparisons between modalities. Monitoring the three autonomic functions of palmar conductance, heart rate and heart rate variability, evidence of sterotypy was found across four different stimuli. Using an autonomic tension score (a normalized T score to compare absolute activity across stressors), Lacey and Lacey (1958) replicated their findings across six standard stressors. Fully 93% of their subjects demonstrated stereotypy under these conditions.

Other support for stereotypy has come from Patton (1969). Patton (1969) exposed 24 U. S. Navy enlisted men to physical, perceptual (movies of surgical procedures), and cognitive tasks while monitoring pulse rate, systolic blood pressure, skin conductance, and instep temperature. He reported that "subjects responded consistently under varying stress conditions both in terms of change in sympathetic nervous system (SNS) activity from nonstress to stress conditions and in terms of absolute level of SNS activity displayed during stress" (p. 207).

Knight and Borden (1979) monitored heart rate, skin conductance, finger pulse volume (analogous to peripheral skin temperature), and self-reported anxiety in undergraduate students anticipating social speaking. They found that high and low socially anxious subjects were discriminable on the basis of finger pulse volume indicative of stereotypy within these two subject groups. Finally, Roessler, Greenfield and Alexander (1964) found a 72 percent incidence of stereotypic responses in 36 undergraduate students exposed to six different intensities of sound administered two different times, although this evidence of stereotypy is mitigated by the fact that only skin resistance was monitored.

The implication of individual response stereotypy for the present thesis lies in the evidence that individuals do have a unique and idiosyncratic response to stress and that this unique physiological response may be indicative of the major physiological component of anxiety for the individual. An important consideration in demonstrating stereotypy however, is the concept of stimulus-response specificity.

Stimulus-Response Specificity

As defined by Sternbach (1966) stimulus-response specificity states that stressful stimuli will produce a characteristic pattern of response across individuals. For example, Averill (1969) was able to distinguish unique patterns of sympathetic activation produced from stimuli producing sadness or mirth. Monitoring systolic and diastolic blood pressures, heart rate, face and finger tip temperatures, skin resistance and respiration, Averill (1969) found the sadness group was character-

ized by increased blood pressure while the mirth group was denoted by increased respiration and heart rates, increased facial temperature and decreased finger temperature. Ax (1953) measured subject's heart rate, respiration rate, face and finger temperatures, skin conductance and frontal muscle tension in response to stimuli representing fear or anger. He found that diastolic blood pressure increases, heart rate decreases, number of rises in skin conductance, and frontal muscle tension increases were greater for anger than for fear, whereas skin conductance, frontal muscle tension and respiration rate increases were greater for fear than for anger. The study by Knight and Borden (1979) previously cited in support of individual response stereotypy also provides support for stimulus-response specificity. Although high and low socially anxious subjects were discriminable on the basis of finger pulse volume, both groups demonstrated increased heart rate and skin conductance levels in anticipation of social speaking. After reviewing the literature on response specificity, Sternbach (1966) concludes that it is clear that depending on different stimuli or stimulus conditions, different patterns of physiological activation which are indicative of stimulus-response specificity also exist (pp. 89-90).

The importance of stimulus-response specificity in demonstrating individual response stereotypy has to do with stimulus similarity. Roessler et al. (1964) have found that stimulus similarity has a positive effect on the degree of stereotypy exhibited. Lacey and Lacey (1959, 1962) have also found support for this relationship. Additionally, they provide the finding that physiological response patterns could be

classified according to the functional significance of the stimuli that elicit them -- either perceptual (eg. tracking); motor (eg. cold pressor test) or cognitive (eg. mental arithmetic). In order to have the most sensitive measure of stereotypy, it follows then, that stimuli employed should be of the same functional set.

Most Reactive Physiological Modality

Despite the evidence cited in support of response stereotypy, there is some cause for caution in using stereotypy in psychophysiological research. This caution concerns the reproducibility of stereotypy over time (Lawler, 1980: Roessler & Engel, 1977). Tests on the reliability of the concept have not been promising.

Lacey and Lacey (1962) tested 37 children ages 6 to 17 in reaction to a cold pressor test four years apart. Using rank orders of response magnitudes to systolic and diastolic blood pressures, palmar conductance, heart rate and heart rate variability, good reproducbility was found. Yet to show reproducbility of stereotypy, reactivity to more than one stressor is required. The other two studies that have asked this question directly have yielded negative results. Oken, Grinker, Heath, Hertz, Korchin, Sabshin and Schwartz (1962) monitored nine autonomic variables in 18 college students exposed to three stimulus conditions over a period of one week. Regardless of whether the specific variable showing maximum change, or the hierarchy of responses, was used, reproducibility was not found. Similarly, Johnson, Hord and Labin (reported in Sternbach, 1966) measured four autonomic responses 48 hours

apart in 24 Navy men exposed to six stimuli. Although analysis of variance yielded supportive evidence for stereotypy in two thirds of the subjects, those subjects who demonstrated stereotypy the first day did not necessarily do so the second day.

This lack of intraindividual reliability has caused recent work to shift away from the concept of complete response patterning to the concept of most reactive response system or modality (Lawler, 1980). Insofar as reactivity is only one physiological modality or in a most reactive modality requires the reproducibility of one physiological response rather than a pattern of responses, it has greater a priori reliability. Moreover, studies have been conducted in which empirical reliability of reactivity have been demonstrated, at least for cardiovascular indices of arousal (Manuck & Schaefer, 1978). What remains to be shown, however, is that high reactivity leads to a developed symptomatology. This relationship relies on the concept of symptom specificity.

Symptom Specificity

The hypothesis of symptom specificity states that somatic complaints are the end result of persistent exposure to stress of physiological functions specifically susceptible to activation by stressful experience (Malmo, Wallerstein & Shagass, 1953; Malmo, Shagass & Davis, 1950). Although conclusive evidence in support of the hypothesis awaits the arrival of longitudinal studies, the research in this area is highly supportive.

Most recently, evidence comes from the work in cardiovascular disorders. For example, Light and Obrist (1980) have demonstrated a relationship between high heart rate reactivity and a family history of hypertension. Light (1981) has replicated this finding with both heart rate and heart pressure reactors, and added the important finding that reactive subjects were indistinguishable from nonreactive ones at resting baseline levels. Manuck, Corse and Winkelman (1979) have shown that high blood pressure reactors exhibit the coronary prone type A behavior characteristics of time urgency and loud and emphatic speech patterns (Friedman & Rosenman, 1979).

Further support for the link between reactivity and both behavioral and psychosomatic complaints comes from Malmo (1953) who found unique EMG stereotypy among headache prone subjects versus subjects with other psychsomatic complaints; Goldstein, Grinker, Heath, Oken, and Shipman (1964) who were able to demonstrate individual response stereotypy on separate occasions among depressive female patients; and Walker and Sandman (1977) who employed an electrogastogram (slides) to show that gastric activity in duodenal ulcer and rheumatoid arthritis patients could be differentiated in stressful situations.

Summary

Although not mutually exclusive, the concepts of stimulus-response specificity and individual response stereotypy are statistically independent and demonstrable simultaneously within the same subject (Sersen, Clausen, & Lidskey, 1978; Roessler & Engel, 1977). The

suspect reliability of response stereotypy has led to the use of reactivity as the independent variable. Evidence strongly supports a relationship between reactivity and specific somatic complaints and forms the basis for using most reactive modality to identify the appropriate response system for training.

Up to this point the reasons for not using the most appropriate response system and the theoretical justification for choosing the most reactive modality have been dealt with. Next, it is necessary to consider some of the evidence surrounding the psychophysiological stress profile used to identify the most reactive modality and to assess the efficacy of training.

The Stress Profile

The Procedure

Corson, Schneider, Biondi and Myers (1980), Fair (1979) and Almy (1978) have each pointed out the necessity of a standardized diagnostic and evaluation procedure when employing psychophysiological treatments. While generalized arousal is characteristic of physiological responses to stress, individuals vary in their specific patterns of physiological arousal to stress, and individuals with anxiety of psychosomatic symptoms are distinguished from normal subjects by their experience of greater and more frequent arousal. In order to assess these differences objectively, a standardized procedure is required.

The normal procedure of such a psychophysiological assessment

procedure or stress profile, follows an A-B-A design. The subject is comfortably seated and requested to relax for a preliminary baseline period. He/she is then exposed to a moderate stressor and asked to relax again for a poststress baseline period (Budsynzki, 1976; Fair, 1979). Throughout this period multi-channel physiological responses are monitored.

The inclusion of a stress condition is a requisite of this procedure. Whereas previous clinical work focussed exclusively on elevated baselines, the necessity of deriving a measure of reactivity demands a stressor episode. Light's (1981) finding that reactive and nonreactive subjects are indistinguishable during resting conditions, and DeGood and Adam's (1976) finding that posttest comparisons of baseline levels differentiated biofeedback training, muscle relaxation, and no feedback groups only under stress conditions clearly demonstrate the need for a stress condition in both diagnosis and evaluation. Moreover, as Gatchel et al. (1978) point out, the clinical concern in biofeedback training is not merely whether subjects can maintain control over physiological functions relative to control groups, but whether they are able to do so under stressful conditions.

Factors Affecting the Stress Profile

There are a number of internal and external factors that influence the results of a stress profile. Internally, the two most important considerations are the type of stimuli, and the standardization procedure employed. Externally, a number of environmental and personal

factors must be considered. The next section will deal first with the type of stimuli.

Type of Stimuli. The type of stimulus employed in a stress profile has important implications for identifying the most reactive physiological modality. Almy (1978) has argued that the stimuli used in a true clinical setting should, as nearly as possible, have perceived stress value for the individual. While conceding the difficulty of individually selecting such stimuli, and the risk of inducing a traumatic experience for the client, Almy (1978) argues that individual-specific stimuli will be more likely to detect reactivity than standardized and more neutral stimuli.

An alternate approach is suggested by Roessler et al. (1964), Lacy (1959), and Lacey, Kagan, Lacey and Moss (1963). Their findings of a positive effect for stimulus similarity in demonstrating response stereotypy and hence reactivity, and Lacey et al.'s (1963) stimulus classification system based on functional significance suggests that the most sensitive measure of reactivity would be the one that utilizes a number of stimuli from the same functional set. Although Almy's point may be well taken in a purely clinical setting, in an experimental procedure stimuli which are standardized across individuals and which can be used to make objective independent assessments is preferable. In accord with this logic, three cognitive stressors were employed in this thesis. It is important to note, of course, that this choice limits the

generalizability of findings about reactivity to reactivity as a result of cognitive stressors.

Method of Standardization. A matter of crucial importance is the method by which most reactive physiological modality will be assessed. The difficulty arises in the need to make meaningful intraindividual comparisons between response modalities with different units of measurement. For example, is a heart rate increase of 10 beats per minute (bpm) in response to a stressor more reactive than a frontal EMG increase of 1 microvolt, or a decrease in peripheral skin temperature of 2 C°. In the past, the identification of dominant physiological responses was made by clinical judgement of overelevated baselines (Pelletier, Gladman & Mikuriya, 1976). When reactivity is used to determine dominant physiological response, however, these discriminations are more difficult and a more objective procedure is required.

The only attempt to date of determining reactivity has been the split-half (Lawler, 1980) or median-split (Carney, 1981) method. Using this method, differences between resting physiological baselines and a physiological stress reaction are rank ordered from highest to lowest scores. The sample is then divided into two equal groups according to the median value with the top half being identified as high reactive and the bottom half being labeled as low reactive. There are two difficulties with this method. First, the approach does not allow intraindividual comparisons of reactivity to be made between different physiological response systems. Although one within subject physiological response may be exceptionally more reactive than

other physiological responses, this overreactivity will not be identified. The second difficulty with the median-split method is that depending upon the sampling distribution, individual differences that may be clinically significant are obviated. For example, the method would have limited utility if a sample were drawn from a clinical population in which all subjects might be expected to be highly reactive. By definition this method would still label half of the sample as low reactive subjects. There is, therefore, a need for a standardized procedure which would make meaningful comparisons between physiological responses within subjects and which would be independent of the population from which the sample was drawn.

To date, four standardization procedures have been employed. Lacey et al. (1953) have used autonomic tension and autonomic lability scores (normalized T scores across stressors, and across subjects respectively) to make these comparisons with the autonomic tension scores displaying the most dramatic results. Engel (1960) has employed a variation on the standardized Z score transformation with the difference between the individual's response and the group's mean response to all stimuli as the numerator, and the within subject error of a covariance analysis as the denominator. Sersen et al. (1978), in response to Engel, argue that use of this numerator does not adjust for differences in response as a function of mean level, and propose the alternate numerator of deviation between the actual and predicted score plus the mean response of the group to that particular physiological function.

Each of these previously used methods are, however, subject to the

criticism that they employ group means to detect what are in fact intraindividual differences (Hiebert, 1980). A more appropriate procedure would seem to be a standardized difference score with the difference between baseline and stimulus scores as the numerator and the baseline standard deviation as the denominator.

The formula for such a standardized reactivity transformation or ZR transformation would be as follows:

$$ZR = \frac{\bar{X}_s - \bar{X}_{1B}}{SD_{1B}}$$

where $\bar{X}_s$ = the mean for stressor 1.

$\bar{X}_{1B}$ = the initial baseline mean

SD_{1B} = the standard deviation for the initial baseline

This method would take into account a subject's change from baseline in response to a stressor as well as variations in baseline that could exaggerate reactivity. As a diagnostic instrument it would also provide a measure of susceptibility to stress (reactivity) prior to the development of chronic symptoms marked by overelevated baselines (Malmo, 1975).

External factors. There are also external factors that affect physiological measurement and hence reliability of the stress profile. Time of day (Corson et al., 1980) and semester (Fisher & Winkel, 1979), season and meteorological changes (Waters, Koresko, Rossie & Hackley, 1979), fatigue (Roessler & Engel, 1977), caffeine (Asterita, Smolnicky & Iatridis, 1981) and post prandial effects (Fair, 1979; Patton, 1969)

all have a documented relationship with physiological measurement. Insofar as possible, these factors should be controlled to avoid spurious results.

Summary

The psychophysiological stress profile is a necessary procedure in order to provide an objective and quantitative assessment of most reactive modality and evaluation of treatment under stress conditions. The stress profile follows an A-B-A relaxation-stress-relaxation design while monitoring physiological modalities. The nature of stimuli chosen, the standardization procedure between modalities, and external variables are all important considerations in the employment of the stress profile.

Standard Deviation as a Physiological Dependent Measure

The final issue to be dealt with here is the choice of physiological dependent measure. The stress profile strongly implies that response variability throughout the relaxation-stress-relaxation procedure is the appropriate dependent measure. It is, however, only very recently that standard deviation (SD) has been called for as an important and sensitive measure of self-regulation (Schwartz, 1981; Shein & Mandel, 1981). As a result, only little research has been performed using this measure.

Shein and Mandel (1981) employed an A-B-A design in a single case study of a cerebral palsy subject. Sampling frontalis EMG 60 times per minute over 16 sessions of frontalis EMG reduction training,

Shein and Mandel found that although there were no clear indications of mean frontalis muscle decreases over sessions, there were SD decreases across sessions.

Hnatiow and Lang (1965) incidentally reported evidence of differential conclusions using the two dependent measures in a controlled outcome study of heart rate variability. Subjects receiving visual analogue feedback were requested to maintain their heart rate within one beat of their average resting level while control subjects simply tracked the feedback signal. It was demonstrated that experimental subjects were able to reduce their cardiac variability successfully, but this stabilization effect was unrelated to coincident changes in mean heart rate. This incidental finding has also been reported in later studies (Lang, Sroufe & Hastings, 1967; Harrison & Raskin, 1976), although at least one study (Sroufe, 1969) has found that experimental subjects maintained both mean heart rate and variability decreases over a control group.

Although all of these studies simply sampled SD over the duration of training procedures, and none of the studies employed a stress induction procedure, they offer the suggestion that using a subject's response variability as a physiological dependent measure may lead to different conclusions than are achieved by using mean levels of physiological activity as a physiological dependent measure. Moreover, when a stress induction procedure is employed, it is a subject's response variability as one goes from a nonstress to stress and returning to a nonstress situation that is of primary interest. A

method of measuring this response variability is to calculate a subject's standard deviation over the duration of the nonstress-stress-nonstress sequence for each physiological modality. The question that arises is whether using this derived measure of response variability, referred to hereafter as a subject's profile SD, yields the same results as when mean levels of physiological activity are used as the physiological dependent measure.

The Development of Hypotheses

This thesis was designed as a preliminary investigation of the biofeedback treatment of anxiety when the biofeedback training target response was matched to the subject's major physiological component of anxiety. Specific issues of interest were whether biofeedback training in physiological response modalities other than frontal EMG would yield different patterns of physiological generalization, and whether profile SD, used as a measure of a subject's response variability, would lead to different patterns of physiological generalization.

These issues were stated in the form of hypotheses as follows:

1. Subjects receiving reactive modality feedback training will as a group, exhibit significant anxiety decrements on treatment-control postscore comparisons as measured by frontal EMG, peripheral skin temperature, skin resistance, heart rate and the State-Trait-Anxiety Inventory (STAI).

2. Subjects receiving reactive modality biofeedback training on the same modality will experience significant anxiety decrements on treatment-control postscore comparisons for the physiological modality on which they received training.

3. Subjects receiving reactive modality biofeedback training on the same modality will not experience significant anxiety increments on treatment-control postscore comparisons on modalities in which they did not receive training.

4. Subjects receiving reactive modality biofeedback training will, as a group, experience significant anxiety decrements on a greater number of physiological indices of anxiety when profile SD is used as a dependent measure than when mean level of physiological activity is used as a dependent measure.

5. Subjects receiving reactive modality biofeedback training on the same physiological modality will experience significant anxiety decrements on a greater number of physiological indices of anxiety when profile SD is used as a dependent measure than when mean levels of physiological activity is used as a dependent measure.

A word of caution should be introduced here. The statement of hypotheses above, rely upon the central assumption that biofeedback training in a specific modality will lead to physiological reductions

only on the target training modality. The statement in hypothesis one that subjects will, as a group, experience physiological reductions on all physiological indices of anxiety does not refer to the generalization of physiological reductions within subjects. Rather it connotes that with equal subgroups of subjects receiving biofeedback training in each modality, each subgroup should experience sufficient reductions in the trained modality to influence the mean for that modality when the subgroups are considered together.

The next chapter will outline the research design, provide descriptive statistics of the research sample, and describe the assessment, treatment and analysis procedures used in testing these hypotheses.

CHAPTER III

PROCEDURE AND DESIGN

In the previous chapter, the hypotheses for this study were developed. This chapter outlines the methodology employed to test the hypotheses. First, the research design is outlined, This is followed by a discussion of the subject sample and the physical facilities in which the experiment took place. Next the assessment procedures and dependent measures are briefly presented. Finally, the treatment procedure is described.

Research Design

The research design employed in this study was a 2x2 factorial design with repeated measures on one factor. The design is illustrated in Figure 1 below:

		Index of Anxiety	
		Pretest Stress Profile (T_1)	Posttest Stress Profile (T_2)
Treatment	Reactive Modality Biofeedback		
	No Contact Biofeedback Exposed Control		

Figure 1. Experimental Design for Data Analysis

In Figure 1, the treatment variable (reactive modality biofeedback training or no contact biofeedback exposed control) is the nonrepeated factor, and the index of anxiety factor (T_1, T_2) is the repeated factor. Repeated measures analyses of variance were replicated for each of the four monitored physiological modalities using first, mean levels of physiological recording and the STAI results as dependent measures and second, response variability for each physiological modality as the dependent measure.

Choice of Statistical Procedure

Due to the number of dependent measures employed in this study, a logical argument could be made that a multivariate analysis of variance would be the most appropriate statistical procedure. This is especially true considering the nonzero intercorrelations between the physiological indices of anxiety, and between the physiological and self-report measures of anxiety. A multivariate analysis of variance would permit statements to be made about the joint occurrence of physiological reductions in one physiological modality independent of the effects of reductions in another physiological modality. For example, if one wished to make statements to the effect that frontal EMG training resulted both in frontal EMG reductions and in decreased heart rate independent of frontal EMG, a multivariate analysis would be necessary.

As an exploratory investigation, however, this study was most interested in identifying the dependent measures most affected by reactive modality biofeedback training. As Finn (1974) points out,

univariate rather than multivariate procedures are the most appropriate statistics in an exploratory study. Although univariate statistics may not be independent for any one hypothesis, they aid in identifying the variates most and least affected by their antecedents and are a desirable prerequisite to further multivariate analyses. In this study, the identification of which physiological modality or modalities are most influenced by reactive modality biofeedback training is of primary interest. For this reason univariate rather than multivariate statistical procedures were used.

Choice of Level of Significance

To further assist in the identification of salient dependent measures, the .10 level of statistical significance was employed. Results throughout the study are, however, reported at their achieved level of significance, $p < .10$, to facilitate individual interpretations.

Experimental Procedure

During the first week of the study all subjects were administered a psychophysiological stress profile and the State-Trait-Anxiety Inventory (Spieberger, 1968). Based on pre-test scheduling, subjects were alternately assigned to either a biofeedback treatment group or to a delayed treatment progressive relaxation training control group. Treatment subjects were scheduled for two half hour biofeedback sessions per week for four weeks or until learning to criteria had been reached, whichever came first. When learning to criteria occurred before the eighth session, a control subject was called in and both subjects were

post-tested. All post-tests were carried out on the same hour of the day and the same day of the week for which pretesting for that subject had occurred. At the post-test a question was appended to the STAI asking subjects to rate their anxiety before arrival as being more anxious, less anxious, or about the same as their initial visit.

The Sample

Subjects for the study were solicited from an undergraduate course in educational psychology. In return for their participation, subjects were offered training in a procedure previously shown to be effective in dealing with stress. It was suggested that such training would be beneficial in coping with the rigors of examinations, oral presentations, and generally be a worthwhile skill to develop for any future endeavors. Additional incentives were offered to encourage participation were exposure to, and possible though not certain training on, biofeedback equipment, and a hands on experience with experimental procedure. Subjects were urged to volunteer if and only if they could commit themselves to a six week period.

Due to the logistics of scheduling subjects within a six week time frame and of having only one experimenter, the size of the sample was of necessity small. Of the 26 subjects who volunteered, two treatment and one control subjects dropped out due to time pressures of upcoming mid-term examinations and term papers. Data from one other treatment subject and two control subjects were deleted due to questionable validity of EMG recordings and equipment malfunction leaving two

groups of 10 subjects each.

The final demographic composition of the two groups were six females and four males with a mean age of 21.5 years in the treatment group, and eight females and two males with a mean age of 23.5 years in the control group. Initial self-report anxiety measures as measured by the STAI placed treatment subjects in the 45th (male) to 63rd (female) percentile on state anxiety, and in the 61st (male) to 72nd (female) percentile for trait anxiety. Corresponding anxiety levels for the control group fell between the 12th (male) and 71st (female) percentiles on the state construct, and in the 39th (male) to 70th (female) range on trait anxiety. (STAI means by group and sex are presented in Table 1.)

Equipment and Facilities

All sessions were conducted in a quiet, temperature monitored two room laboratory. Subjects were seated on a comfortable recliner chair facing a curtained window. The experimenter sat on a stool adjacent to the subject facing the biofeedback equipment housed in a cabinet on the wall opposite the curtained window. The position enabled the experimenter unobstrusively to monitor subject movement. The STAI was filled out at a desk in an adjoining room.

Frontal EMG, heart rate, peripheral skin temperature and galvanic skin resistance were simultaneously monitored using Colborne Instruments modular biofeedback equipment. Frontal EMG electrodes, following standard clinical procedures, were applied on an imaginary line

Table 1

STAI Means by Group and Sex

Group	Sex	State		Trait	
		Mean	Percentile	Mean	Percentile
Treatment	Male	34.0 (7.528)*	45	37.75 (6.021)	61
	Female	36.0 (4.561)	63	41.5 (11.327)	72
Control	Male	24.5 (2.121)	12	32.5 (.707)	39
	Female	40.25 (10.606)	71	41.0 (10.980)	70

* Standard deviations are presented in brackets in this and all subsequent tables.

an inch and a half above and horizontal to the eyebrow line with the central electrode serving as the reference for recording located equidistant from the two active electrodes. Impedences of 10,000 ohms or less were maintained throughout recording and output channelled through a Colborne Instruments hi-gain bioamplifier. Heart rate was recorded using a Colborne Instruments S71-40 blood flow pulse monitor which measured pulsatile blood flow from an optical densitometer applied to the palmar surface of the left thumb. Peripheral skin temperature was taken from a Yellow Springs thermistor applied to the ventral surface of the middle finger of the nondominant hand and monitored through an S71-30 temperature module. Basal galvanic skin response was recorded by measuring the voltage drop between two lead strips applied to the index and ring fingers, respectively, of the left hand. Output was through a Colborne Instruments S71-20 skin resistance module.

Continuous checks for proper functioning of equipment were possible using visual voltmeters, and all data were processed through a cumulative averaging integrator and printed simultaneously on a Colborne multichannel microprocessing printer.

Assessment Procedure

The Stress Profile

A stress profile employing an A-B-A design with three stressors was used as an assessment and evaluation technique. The procedure emulated that of Budzynski (1977, 1978) in which subjects were asked to relax for the first 12 minutes using whatever procedure they usually used to relax.

A three minute serial sevens subtraction task was then administered followed by a final 12 minute relaxation period. In this study, two additional stressors were included to enhance the reliability of determining most reactive modality and to explore physiological reactivity to stress over time. The two additional tasks were a reading task taken from the Gilmore Oral Reading Test (Gilmore & Gilmore, 1968), and on which subjects were told they would be asked questions parallel to the WAIS arithmetic test. (See Appendix A for a set of instructions employed). The sequence of the stress profile was, therefore, a 12 minute relaxation period followed by a three minute serial sevens task and a subsequent two minute relaxation period; a reading task followed by another two minute relaxation period; and an arithmetic task followed by a final 12 minute relaxation period.

Upon initial greeting, subjects were introduced to the biofeedback equipment and told that it would be used to determine their individually unique response to stress. They were then requested to sit in the recliner chair and the function of each measurement device was explained as it was attached to them. After electrode application, the sequence of relaxation and stressor episodes was outlined. Physiological recordings on frontal muscle tension, skin temperature, skin resistance and heart rate were simultaneously recorded at three-minute intervals during the 12-minute relaxation periods and stressor episodes, and at 15-second intervals during the two three-minute recovery periods. The resultant output yielded 27 data points for each physiological modality.

The post-test stress profile followed the same sequence but used alternate forms of the cognitive tasks (see Appendix B). The State-Trait Anxiety Inventory was administered at the end of each profile.

Assessment of Most Reactive Modality

Intraindividual comparisons between response modalities using the ZR transformation outlined in chapter two were made to determine most reactive modality. The figures were calculated using the mean and standard deviation for the initial 12-minute baseline period and the raw score for each of the subsequent 23 data points. Most reactive modality was determined by comparing the ZR score values at the three stimulus points. Where one response modality was not readily discernable using these figures, recovery period ZR score values were used to make the final decision. The sequence of decision rules employed are given below:

1. Where only one modality had ZR scores above 1 on the three stressors, that modality was determined to be the most reactive modality.

2. Where two or more modalities had ZR values exceeding 1, that modality which had the highest ZR values was determined to be the most reactive physiological modality.

3. Where two or more modalities had ZR values exceeding 1 but the ZR scores were of approximately equal value, the modality which had successively greater values as one went from stressor 1 to stressor 3 was determined to be the most reactive modality.

4. Where most reactive modality was not discriminable using rule 3, the modality with the most recovery period ZR scores above one was determined to be the most reactive modality.

Several notes should be made regarding these rules. The second and third stimuli required vocalization by the subject that would produce data artifact on all of the physiological recordings, but especially on frontal EMG. Stimulus 1, therefore, was the only pure data recording point and was given preferential weighting. Second, a characteristic of a person's physiological response to stress is that successive stimuli have a cumulative effect and lead to progressively increasing physiological recovery times (Malmo, 1975). This feature of the physiological stress response accounted for the stipulation in rule 3, that the ZR scores be in ascending order as succesive stressors were presented, and the use of recovery periods in rule 4, as the final criterion for establishing most reactive modality.

Two more general observations should be made about the ZR statistic employed. First, since the numerator of the transformation consists of the baseline mean minus the raw score at the stimulus points, and since there is a ceiling effect above which physiological responses are not likely to exceed, subjects with a high initial baseline value had a lower probability of being identified as most reactive in that modality. It was possible, therefore, for most-reactive modality to be chosen despite elevated baselines in another modality that may have been clinically significant. Second, the standard deviation in the denominator means that subjects with low response variability over

the initial baseline period would have a greater likelihood of being reactive on that modality than on a modality for which initial response variability was high. Subjects with highly variable resting responses would not, in all probability, have been determined reactive in that modality.

These two points emphasize that the central concern of this study was reactivity. In an attempt to study reactivity prior to the onset of overt symptomatology, subjects were drawn from a normal population where over elevated initial physiological baselines would not be an expected result. The ZR statistic furthered this intent by controlling for initial resting response variability and operationally defining reactivity as the response to a stressor over and above any initial variability at resting physiological levels.

Dependent Measures

Several physiological dependent measures were employed in the study. For the mean level of physiological activity analyses, each modality was separately assessed using the pre- and poststressor baselines, and the values from the data point at stressor 1. The response variability analysis was conducted using the stress profile SD score (to be described below). The state-trait anxiety inventory (STAI) administered at the end of each profile, was used as a self-report index of anxiety. A brief discussion of these measures is described below.

Pre- and Post-Stressor Baselines

As mentioned above, the stress profile included an initial 12 minute relaxation period with four three minute data recording points. The pre-stressor baseline was calculated by taking the mean of these points. The post-stressor mean was calculated as the mean of the final four three minute data points of the stress profile. These baselines were individually used as dependent measures for the nonstressor or resting level episodes.

Stressor 1

The serial sevens subtraction task employed as stressor 1, is normally used as the exclusive stressor in the stress profile procedure (Budzynski, 1977; 1978). Since stressors 2 and 3 were included primarily for enhanced reliability and for exploratory purposes, and since they were tainted by vocalization during their measurement, stressor 1 was used as the physiological dependent measure for the stress condition. The three minute mean score yielded by the Colborne Instruments multi-channel printer was employed.

Stress Profile Standard Deviation (SD) Score

Each subject's response variability over the entire nonstress-stress-nonstress sequence was measured by taking the standard deviation over the initial four three minute relaxation data points, the three three minute stressor data points, and the final four three minute data points for each modality. The result was a profile SD score for each physiological modality which was used as the dependent measure for a

subject's response variability.

Consistent with the ZR transformation to determine most reactive modality, the profile SD provided a measure of a subject's response variability that minimized initial overelevated baselines. It was theoretically possible for a subject to have a low profile SD score on a specific physiological modality despite having elevated initial baseline levels on that modality. In fact, because of ceiling effects to physiological responding, a subject with elevated initial baselines would have been more likely to have a low profile SD score since his/her response to a stressor would be restricted by the decreased range between the already elevated baseline, and the ceiling level.

Stait-Trait Anxiety Inventory

As a psychological self-report measure of anxiety, the STAI was used. Each of the state and trait inventories is a self-evaluation questionnaire consisting of 20 items rated on a four-point scale. Test-retest stability coefficients are high (r = .79) for the trait inventory and low (median r = .32) for the state inventory, the latter representing the influence of situational factors (Spielberger, Gorsuch & Lusheno, 1970). Concurrent validity of the trait scale is demonstrated by its high correlation with the IPAT Anxiety Scale (r = .75), and the Taylor Manifest Anxiety Scale (r = .80) (Spielberger, 1968). Construct validity for the STAI-state scale was determined on 977 undergraduate students. The mean score for the state scale was "significantly higher" prior to simulated exam conditions than under normal conditions (Spielberger et al., 1970).

Treatment Procedures

Group 1: Most Reactive Modality Biofeedback Training

Using the assessment procedure outlined above, treatment subjects were given biofeedback training in their most reactive modality while the other three modalities were simultaneously monitored. At the first session, subjects were given continuous auditory feedback and asked to decrease the pitch of the tone using any strategy they chose. Subjects who had difficulty in altering the pitch were questioned and alternate strategies based on passive volition (letting go on demand; Green & Green, 1977) suggested. In subsequent sessions, subjects were asked to review their successful strategy from the previous session(s) and to continue using it during training sessions. In later sessions, alternate periods of continuous feedback and no feedback were initiated. Subjects monitored their success during no feedback intervals and were given oral and visual (the printer output) feedback of their success in self-monitoring at the end of the session.

All subjects received eight 30-minute training sessions or as many training sessions as were necessary to reach training criteria, whichever came first. Consistent with the procedure for determining most reactive modality, training criteria were established as being able to reduce training session target response baselines to a value two initial baseline profile standard deviations below initial baseline profile mean level, and to maintain this reduction for two consecutive three-minute intervals. For example, a heart rate reactive subject whose initial heart rate baseline mean was 78 bpm with a standard deviation of 2.5,

would be required to maintain a heart rate level of 73 bpm for six consecutive minutes. In order to demonstrate reliability of this self control, it was further necessary for the subject to achieve his criterion over two successive training sessions.

Once training criteria had been achieved, a final session was conducted during which they were requested to maintain their decrements in a no feedback condition while simultaneously listening to questions from a previous educational psychology mid-term examination. Since the last session was conducted one week prior to their educational psychology midterm, this was deemed to provide training at maintaining physiological reductions during a stress induction procedure.

Group 2: No Contact Control

The control group was administered the stress profile and STAI during the same week as the treatment subjects. Rather than immediate treatment, however, control subjects were told that physiological recordings are often influenced by meteorological and time of semester variables, and that in order to assure the reliability of the individually unique response to stress, a readministration of the stress profile would be necessary in approximately five weeks. Control subjects were called back and post-tested on the same week as treatment subjects and were provided with progressive relaxation training sessions after post-testing.

Control subjects, therefore, were provided both with exposure to biofeedback equipment which has been shown to have expectancy parameters

(Wickramaskera, 1981: Miller, 1978; Miller & Dworkin, 1977), and with the promise of an empirically demonstrated effective stress management procedure.

Summary

This experiment employed a 2x2 factorial (treatment, control X T_1, T_2) design with repeated measures on the time factor. Twenty volunteer undergraduate subjects were administered a psychophysiological stress profile and randomly assigned to either biofeedback training on their most reactive physiological modality or to a treatment deferred but biofeedback equipment exposed group. Posttesting was conducted using an alternate form of the stress profile. Dependent measures used were physiological levels of responding under resting and stress conditions, a stress profile SD score, and the State-Trait-Anxiety Inventory.

In the next chapter, the results will be presented.

CHAPTER IV

DATA ANALYSIS

The main purpose of the study was to test the effectiveness of providing biofeedback training in a subject's most reactive modality. Further intentions were to explore some of the implications of using response variability as physiological dependent measure and to provide support for the construct validity for the ZR transformation approach of determining most reactive modality. Analyses were performed comparing the treatment with the control groups as a whole and, where numbers permitted, specific reactive modality subjects with control subjects reactive in that same modality.

Before dealing with the hypotheses, however, it is first necessary to describe the sample in terms of its reactive modalities, and determine whether group differences on initial physiological baseline values existed.

Preliminary Analyses

Reactive Modalities of the Sample

Using the ZR transformation and the decision rules outlined in Chapter III, standardized scores were calculated for the three stressor episodes and each subject's most reactive modality was identified. (The ZR transformations for each subject are presented in Appendix C.) With the exception of subject 2, sufficient information was provided by the three data points to identify reactive modality. Subject 2 had no

Z scores exceeding 1 on either stressor or recovery points, and most reactive physiological modality for this subject was selected by choosing the only modality to show a physiological response in the direction of heightened arousal to a stressor. The final configuration of the treatment group was six heart rate reactive subjects, two peripheral skin temperature reactive subjects, and one each of frontal EMG reactive and GSR reactive subjects. The composition of the control group was seven heart rate reactive, 2 frontal EMG reactive, and one peripheral skin temperature reactive subjects.

The results reveal a disproportionate number of heart rate reactive subjects. Since hypotheses 2 and 3 state, that where numbers permit, groups receiving reactive modality feedback on the same modality would be analyzed individually, the decision was made to proceed with a separate analysis using heart rate reactive treatment versus heart rate reactive control groups. For this purpose, one heart rate reactive subject was randomly deleted from the control group. In the analyses that follow, results will be presented alternatively for the treatment group as a whole, and for heart rate reactive groups.

Results of Biofeedback Training

Of the ten treatment subjects, eight subjects achieved the performance training criteria of reducing physiological responses in their most reactive physiological modality to two standard deviations below initial profile baseline levels for a six minute period over two consecutive training sessions. Two subjects, both of whom received biofeedback training on peripheral skin temperature, failed to reach this criterion

level, but received the full eight sessions of biofeedback training.

Initial Group Baseline Statistics

As Benjamin (1967) points out, the Law of Initial Values (which states that the magnitude of a physiological response to an experimental stimulus is related to the prestimulus level) has important implications for the statistical analyses of physiological data. Normally where initial group physiological baseline levels do not differ significantly from each other, the analysis of variance is the appropriate statistical procedure. Where there are significant physiological baseline differences, however, the analysis of covariance is the preferred procedure. To test for these initial differences, an analysis of variance analogous to a simple t-test was performed on the initial baseline group means for each of the four response systems. No significant differences were found at the $p. < .05$ level for any of the physiological response modalities. Throughout the testing of hypotheses, therefore, the repeated measures analysis of variance was employed. (Initial physiological group means on frontal EMG, PST, GSR and HR are presented in Table 2 for groups as a whole, and Table 3 for heart rate reactive groups).

The Testing of Hypotheses

Hypothesis 1

Subjects receiving reactive modality biofeedback training will, as a group, exhibit significant anxiety decrements on treatment-control postscore comparisons as measured by frontal EMG, peripheral skin temperature, skin resistance, heart rate, and the STAI.

Table 2

Initial Group Means for Groups as a Whole (N=20)

Index of Anxiety	Prestressor Baseline		Stress Condition		Poststressor Baseline	
	Treatment	Control	Treatment	Control	Treatment	Control
EMG (UV)	3.108 (1.811)	3.358 (1.693)	3.226 (1.657)	3.007 (0.853)	3.061 (1.688)	3.360 (1.336)
PST (°C)	31.756 (3.132)	30.691 (2.572)	32.153 (3.267)	31.367 (2.613)	31.290 (3.170)	30.225 (2.498)
GSR (KΩ)	38.532 (1.365)	39.227 (2.699)	39.608 (2.539)	41.426 (2.296)	42.428 (5.962)	42.251 (2.482)
H.R (BPM)	74.503 (11.664)	76.288 (7.518)	81.800 (10.793)	83.650 (5.882)	72.665 (15.076)	71.382 (7.851)

Table 3

Initial Group Means for Heart Rate Reactive Groups (N=12)

Index of Anxiety	Prestressor Baseline		Stress Condition		Poststressor Baseline	
	Treatment	Control	Treatment	Control	Treatment	Control
EMG (UV)	3.342 (2.068)	2.782 (1.566)	3.725 (1.919)	2.598 (0.451)	3.252 (1.923)	2.952 (1.260)
PST (°C)	32.181 (3.515)	30.600 (2.320)	32.613 (3.717)	31.490 (2.420)	32.025 (3.780)	29.906 (2.442)
GSR (KΩ)	38.367 (1.353)	40.005 (2.747)	39.950 (2.817)	41.070 (2.990)	41.529 (3.844)	41.970 (2.990)
H.R (BPM)	71.625 (11.366)	74.333 (8.832)	80.833 (10.872)	83.733 (7.025)	76.479 (17.555)	72.517 (7.614)

Results. The two way analyses of variance used to test this hypothesis on the groups as a whole revealed a significant group x time interaction for frontal EMG under the stress condition, F (1,18) = 4,841, p = .041; and a significant group x time interaction for heart rate also under the stress condition, F (1,18) = 4.478, p = .049. The results indicate a differential treatment effect for frontal EMG and heart rate during stress. The Scheffé Multiple Comparisons procedure was used to determine the critical mean differences both over time and between groups. Scheffé (1959) recommends the adoption of the .10 significance level to overcome the excessively conservative nature of the procedure. Critical differences were calculated here using both the .05 and .10 levels of significance. The lowest level at which significance occurred is reported. The results revealed a significant increase in frontal EMG for the control group, Sch (1,18) $\geq$ .547, p < .10; a significant decrease in heart rate for the treatment group, Sch (1,18) $\geq$ 5.192, p < .05; and a significant difference in heart rate between the two groups during the posttest session, Sch (1,36) $\geq$ 8.446, p < .05. These data suggest that reactive modality biofeedback training is effective in preventing frontal EMG increases under stress conditions and in significantly reducing heart rate reactivity to stress. No other physiological mean levels or the self-report measure showed a significant treatment effect.

Other significant differences noted were a significant decrease in posttest skin resistance at the poststressor baseline, F (1,18) = 6.489, p = .02, indicative of increased arousal for both groups during post-

testing; a significant decrease in trait anxiety, $F(1,18) = 7.512$, $p = .013$, indicating decreased anxiety on the trait construct for both groups during the posttest session; and a significant decreased frontal EMG at the poststressor baseline, $F(1,18) = 4.203$, $p = .055$, indicative of decreased arousal for both groups during posttesting. No group differences were revealed. (The group means for EMG, PST, GSR and HR appear in Table 4. Group means for the self-report measures appear in Table 5, and analyses of variance summaries for significant effects appear in Appendix D.)

The above results show that treatment subjects experienced anxiety decrements only for heart rate under stress conditions. Also, the treatment group as a whole did not experience frontal EMG increases under the stress conditions as did the control group. Both treatment and control groups displayed increased anxiety during the posttesting as evidenced by decreased skin resistance. In contradiction to the skin resistance evidence, there was decreased trait anxiety for both groups during the posttest session and decreased frontal EMG during the poststressor baseline. Support for Hypothesis 1, therefore, was minimal.

Hypothesis 2

Subjects receiving reactive modality biofeedback training on the same modality will exhibit significant anxiety decrements on treatment-control post score comparisons for the physiological modality on which they received training.

Table 4

Physiological Group Means for Groups as a Whole

(N=20)

Group	Anxiety Measure	Baseline 1		Stressor		Baseline 2	
		T_1	T_2	T_1	T_2	T_1	T_2
1*	EMG	3.108 (1.811)	2.578 (1.249)	3.226 (1.657)	2.818** (1.496)	3.061 (1.688)	2.588 (0.977)
2	EMG	3.358 (1.693)	2.960 (1.311)	3.007 (0.853)	3.580 (1.524)	3.360 (1.336)	2.644 (1.008)
1	PST	31.756 (3.132)	32.055 (3.005)	32.153 (3.267)	32.032 (2.989)	31.290 (3.170)	30.725 (3.036)
2	PST	30.690 (2.572)	31.704 (3.001)	31.67 (2.613)	32.148 (2.833)	30.225 (2.498)	31.696 (3.028)
1	GSR	38.534 (1.365)	38.621 (2.699)	39.608 (2.539)	39.544 (3.603)	42.428 (5.962)	38.880 (4.149)
2	GSR	39.227 (2.696)	41.020 (4.684)	41.426 (2.296)	40.129 (4.990)	42.250 (2.482)	41.233 (3.089)
1	H.R	74.502 (11.502)	74.005 (12.472)	81.800 (10.793)	75.310*** (11.117)	72.665 (15.076)	69.512 (11.404)
2	H.R	76.287 (7.518)	76.215 (7.314)	83.650 (5.882)	84.560 (8.460)	71.382 (7.851)	76.527 (5.715)

* Group 1 in this table and subsequent tables refers to the treatment group. Group 2 refers to the control group.

** $T_1 - T_2 \geq .547$ is significant, $\underline{Sch}(1,18) \geq .547$, $\underline{p} < .10$

*** $T_1 - T_2 \geq 5.192$ is significant, $\underline{Sch}(1,18) \geq 5.192$, $\underline{p} < .05$.

$G1 - G2 \geq 8.446$ is significant, $\underline{Sch}(1,36) \geq 8.446$, $\underline{p} < .05$.

Table 5

Self Report Group Means for Groups as a Whole (N=20)

Group	Anxiety Measure	T_1	T_2
1	STAI-T	40.000* (9.333)	37.700 (8.832)
2	STAI-T	39.300 (10.328)	36.500 (9.277)
1	STAI-S	35.200 (5.613)	37.000 (9.357)
2	STAI-S	37.100 (11.493)	36.400 (8.566)

* Increased scores are indicative of increased anxiety.

Results. Results for heart-rate reactive subjects showed significant treatment effects for heart rate during the poststressor baseline period, $F(1,10) = 5.285$, $p = .044$, and for heart rate under the stress condition, $F(1,10) = 4.708$, $p = .055$. Results of the Scheffé Multiple Comparisons procedure revealed a significant decrease in heart rate for the treatment group during the poststressor period, $Sch(1,10) \geq 7.960$, $p < .10$; a significant increase in heart rate under the stress condition, $Sch(1,10) \geq 8.044$, $p < .05$; and a significant difference between the treatment and control groups under the stress condition during posttesting, $Sch(1,20) \geq 10.872$, $p < .05$. The results indicate that heart rate reactive treatment subjects experienced significant reductions in heart rate under both stress and poststress conditions, and that they were significantly different from the control group in this regard under the stress condition.

Significant treatment effects for peripheral skin temperature during the poststressor period, $F(1,10) = 5.998$, $p = .034$, and for frontal EMG under the stress condition, $F(1,10) = 3.615$, $p = .086$, were also noted. The Scheffé Multiple Comparisons procedure revealed that control subjects experienced a significant increase in peripheral skin temperature, $Sch(1,10) \geq 2.293$, $p < .05$, although they did not significantly differ from the treatment group in skin temperature. The Multiple Comparison's procedure for frontal EMG revealed no significant differences either across time within groups, or between groups.

Significant differences between pre- and posttest sessions were also observed. As in the groups as a whole analysis, heart rate reactive

subjects displayed a significant decrease over time in GSR at the post-stressor baseline, $F(1,10) = 4.993$, $p = .049$, indicative of increased arousal. There were, however, increases over time in peripheral skin temperature at the prestressor baseline, $F(1,10) = 4.657$, $p = .056$, and poststressor baseline, $F(1,10) = 4.441$, $p = .061$; and significant decreases in trait anxiety, $F(1,10) = 3.573$, $p = .088$, all indicative of decreased anxiety. (Heart rate reactive group means for EMG, PST, GSR and HR appear in Table 6. Heart rate reactive group means for STAI-S and STAI-T are presented in Table 7. Analyses of variance statistics for significant effects are summarized in Appendix E.)

The data for heart rate reactive groups indicate significant treatment effects for heart rate under the stress and poststressor conditions. There was, however, also a significant treatment effect for frontal EMG under the stress condition. Hypothesis 2, therefore is only partially supported.

Hypothesis 3

Subjects receiving reactive modality biofeedback training on the same modality will not exhibit significant anxiety increments on treatment-control postscore comparisons on modalities in which they did not receive training.

Results. The finding of a group x time interaction in peripheral skin temperature outlined in relation with the results of hypothesis 2, are not in accord with the present hypothesis. Although the poststressor decrease in peripheral skin temperature for the treatment group consideed alone did not reach significance, the finding of a significant increase

Table 6

Physiological Group Means for Heart Rate Reactive Groups

(N=12)

Group	Anxiety Measure	Baseline 1		Stressor		Baseline 2	
		T_1	T_2	T_1	T_2	T_1	T_2
1	EMG	3.342 (3.068)	2.963 (1.523)	3.725 (1.919)	3.092 (1.921)	3.252 (1.923)	2.912 (1.167)
2	EMG	2.782 (1.566)	3.395 (0.966)	2.598 (0.451)	3.150 (1.555)	2.952 (1.260)	2.284 (0.849)
1	PST	32.181 (3.515)	33.114 (2.647)	32.613 (3.717)	33.177 (2.621)	32.025 (3.780)	21.776 (3.322)
2	PST	30.600 (2.320)	33.059 (2.038)	31.490 (2.424)	33.618 (1.095)	29.906 (2.442)	33.224* (1.527)
1	GSR	38.367 (1.353)	37.476 (2.089)	39.950 (2.817)	38.985 (3.205)	41.529 (3.844)	37.637 (4.766)
2	GSR	40.004 (2.747)	41.827 (4.800)	41.070 (2.990)	39.775 (5.261)	41.970 (2.980)	41.630 (3.388)
1	H.R	71.625 (11.366)	69.704 (10.602)	80.833 (10.872)	72.517** (7.025)	76.479 (17.555)	67.879*** (10.801)
2	H.R	74.333 (8.832)	75.637 (3.426)	83.733 (9.968)	86.500 (7.692)	72.517 (7.614)	78.183 (2.829)

* $T_1 - T_2 \geq 2.292$ is significant, $\underline{Sch}(1,10) \geq 2.93$, $\underline{p} < .05$.

** $T_1 - T_2 \geq 8.044$ is significant, $\underline{Sch}(1,10) \geq 8.044$, $\underline{p} < .05$.

G1 - G2 $\geq$ 10.872 is significant, $\underline{Sch}(1,20) \geq 10.872$, $\underline{p} < .05$.

*** $T_1 - T_2 \geq 7.960$ is significant, $\underline{Sch}(1,10) \geq 7.960$, $\underline{p} < .10$.

Table 7

Self Report Group Means for Heart Rate Reactive Groups

(N=12)

Group	Anxiety Measure	T_1	T_2
1	STAI-T	38.667 (4.633)	37.500 (7.259)
2	STAI-T	39.333 (10.013)	36.167 (8.886)
1	STAI-S	35.000 (6.419)	34.333 (9.709)
2	STAI-S	34.000 (10.789)	37.333 (7.659)

in skin temperature for control subjects at that time runs contrary to the hypothesis. The finding of decreased skin resistance indicative of increased arousal, although shared by the control group, also contradicts the hypothesis. Hypothesis 3 must, therefore, be rejected.

Hypothesis 4

Subjects receiving reactive modality biofeedback training will, as a group, experience significant anxiety decrements on a greater number of physiological indices of anxiety when profile SD is used as a dependent measure than when mean levels of physiological activity is used as a dependent measure.

Results. When groups as a whole were analyzed using profile SD as the dependent measure, a significant, $F(1,18) = 3.810$, $p = .067$, treatment effect was found for heart rate variability. Results of the Scheffé Multiple Comparisons procedure showed that both treatment and control groups experienced significant increases in heart rate variability during the posttest session, $Sch(1,18) \geq 3.698$, $p < .05$. A significant difference between the two groups during posttesting, $Sch(1,36) \geq 4.190$, $p < .10$, was also observed indicating a lesser increase in heart rate variability for the treatment group. No other significant treatment effects were detected although there was a significant decrease in frontal EMG variability for both groups during posttesting, $F(1,10) = 3.172$, $p = .092$. Hypothesis 4, therefore, must be rejected. (Table 8 presents the group means for EMG, PST, GSR and HR variabilities for groups as a whole. Analysis of variance summaries are presented in Appendix F.)

Table 8

Group Means for Profile SD (Groups as a Whole)

(N=20)

Group	Anxiety Measure	T_1	T_2
1	EMG	1.509 (1.073)	1.160 (0.725)
2	EMG	1.241 (0.581)	1.183 (0.414)
1	PST	10.267 (0.947)	10.177 (0.924)
2	PST	9.952 (0.815)	10.157 (0.879)
1	GSR	13.032 (1.195)	13.135 (2.120)
2	GSR	13.473 (0.892)	12.752) (1.221)
1	H.R	26.846 (7.684)	36.692 (5.625)
2	H.R	26.457) (3.938)	44.163* (4.075)

* $T_1 - T_2 \geq 3.698$ is significant, Sch(1,18) $\geq$ 3.698, $p < .05$.

G1 - G2 $\geq$ 4.190 is significant, Sch(1,36) $\geq$ 4.190, $p < .10$.

Hypothesis 5

Subjects receiving reactive modality feedback on the same physiological modality will experience significant anxiety decrements on a greater number of physiological indices of anxiety when profile SD is used as a dependent measure than when mean levels of physiological activity is used as a dependent measure.

Results. The reanalysis of the heart rate reactive groups using profile as the dependent measure revealed a significant group x time interaction for heart rate variability, $F(1,10) = 5.870$, $p = .036$; and a significant time difference for heart rate variability, $F(1,10) = 98.710$, $p = .001$. The Scheffé Multiple Comparisons Procedure confirmed that both groups experienced significant increases in heart rate variability $Sch(1,10) \geq 5.299$, $p < .05$, but that the treatment group experienced less of an increase as evidenced by the significant group difference at posttesting, $Sch(1,20) \geq 5.539$, $p < .10$.

A significant group x time interaction was also detected for frontal EMG variability, $F(1,10) = 4.563$, $p = .058$. The Scheffé Multiple Comparisons analysis revealed that the treatment group experienced a significant decrease in frontal EMG variability during posttesting, $Sch(1,10) \geq .485$, $p < .05$. The results are in contradiction to the hypothesis which must be rejected. (Group means on frontal EMG, PST, GSR and HR for heart rate reactive groups are presented in Table 9. Analysis of variance summaries are included in Appendix F.)

Table 9

Group Means for Profile SD (Heart Rate Reactive Groups)

(N=12)

Group	Anxiety Measure	T_1	T_2
1	EMG	1.954 (1.211)	1.412* (0.839)
2	EMG	0.909 (0.370)	1.023 (0.377)
1	PST	10.330 (1.122)	10.523 (0.778)
2	PST	9.937 (0.744)	10.565 (0.404)
1	GSR	12.891 (0.925)	13.496 (2.642)
2	GSR	13.324 (0.995)	12.711 (0.940)
1	H.R	26.558 (8.340)	39.197** (5.542)
2	H.R	24.724 (3.096)	45.518 (3.752)

* $T_1 - T_2 \geq .485$ is significant, Sch(1,10) $\geq .485$, $p < .05$.

** $T_1 - T_2 \geq 5.299$ is significant, Sch(1,10) ≥ 5.299, $p < .05$.

G1 - G2 ≥ 5.539 is significant, Sch(1,20) ≥ 5.529, $p < .05$.

CHAPTER V

DISCUSSION

In this chapter, a discussion of the results is presented. The first section is a more intensive interpretation of the results derived from hypothesis testing. This is followed by a discussion of the validity of the ZR transformation for determining most reactive physiological modality. The final section presents a summary of the major research implications developed from this study.

Hypothesis Results

Due to the interrelatedness of some of the hypotheses, it was decided to group hypotheses 1 and 2, and hypotheses 4 and 5 for the purposes of discussion.

Hypotheses 1 and 2

Hypothesis 1 stated that subjects receiving most reactive biofeedback training would, when treated as a group, experience decrements on all physiological and self-report measures. The findings were that treatment subjects showed decreases in physiological indices of anxiety as a result of training only for heart rate under the stress condition and that treatment subjects did not experience increases in frontal EMG under stress as did control subjects. Additional findings were decreased skin resistance for both groups evidential of increased arousal during the poststressor baseline, and decreased frontal EMG during the postressor baseline and trait anxiety during posttesting indicative of

decreased arousal for both groups.

Hypothesis 2 stated that subjects receiving most reactive biofeedback training on a single modality, in this case heart rate biofeedback training, should experience anxiety decrements as exhibited by physiological indices of arousal only on heart rate. The findings, however, were analogous to the groups as a whole analysis. Treatment subjects experienced significant reductions in heart rate under stress and poststressor conditions, and there was a significant frontal EMG treatment effect under the stress condition although no significant differences were detected across time or between groups. Also, both heart rate reactive treatment and heart rate reactive controls evidenced decreased skin resistance on the poststressor baseline and decreased trait anxiety during posttesting. Additionally, both groups had increased peripheral skin temperature during the prestressor and poststressor periods, and the control group had increased skin temperature during the stressor condition when posttested. The section that follows will deal first with the findings of decreased heart rate and frontal EMG under stress conditions.

Decreased heart rate and frontal EMG. The first main aspect of these findings is that the treatment group experienced physiological reductions only for heart rate and not for all physiological modalities as predicted. The logic behind hypothesis one was, however, that equal proportions of subjects would receive biofeedback training on each modality. Decrements in each group trained on a specific modality would

therefore be sufficiently large to influence the mean on that modality for the entire treatment sample. Given the disproportionate numbers of heart rate reactive subjects constituting 60 percent and 70 percent of the treatment and control groups respectively, it is unlikely that there were sufficient numbers of subjects trained in frontal EMG, PST and for GSR to uniformly influence these means. A plausible explanation for the decreased heart rate under stress conditions is that this decrement was due primarily to the influence of the heart rate reactive subjects in the larger sample.

The finding that treatment subjects as a whole maintained their frontal EMG levels under the stress condition while control groups experienced increased frontal EMG can be explained in two possible ways. One explanation is that the result is attributable to the generalization of heart rate reductions to the frontal EMG physiological response modality by the heart rate biofeedback trained subjects. The other interpretation is that the decreased frontal EMG was due to the decreased frontal EMG in subjects who received biofeedback training in a physiological response system other than heart rate. Although either or both explanations are logically possible, a closer scrutiny of the data supports the first interpretation of physiological response generalization of decreased heart rate to the frontal EMG response modality in heart rate trained subjects. Results from analyzing the heart rate reactive treatment subjects showed significant reductions for both heart rate and frontal EMG under the stress condition. A supplementary 2 x 2 repeated measures analysis of variance for the remaining not heart rate

reactive treatment subjects versus the not heart rate reactive control subjects, on the other hand, revealed no significant reductions on any of the three frontal EMG data points. Since the subjects receiving biofeedback training in response modalities other than heart rate experienced no significant reductions in frontal EMG, it follows that frontal EMG reductions under the stress condition reported on page 55 for the groups as a whole analysis must have been due to the heart rate reactive trained subjects lowering frontal EMG and not due to the subjects trained in other modalities. Although impossible to determine from the data, it is possible to speculate that the increases in frontal EMG experienced by the control group in the groups as a whole analysis and not detected in the heart rate reactive groups analysis was due to the increased statistical sensitivity as a consequence of the larger sample. Additionally, the presence of two frontal EMG reactive subjects in the control group as a whole versus only one in the treatment group would result in proportionately higher frontal EMG stress condition measures for control subjects and contribute to the statistical significance of control subject frontal EMG increases in the groups as a whole analysis.

The disproportionate number of heart rate reactive subjects in the overall study sample can, therefore be used to interpret the results of hypothesis 1. Without adequate representation of frontal EMG, peripheral skin temperature and skin resistance subjects in the sample a complete test of hypothesis 1 was not possible in this study. The hypothesis, however, remains tenable for further testing using an enlarged sample

with equal representation of subjects trained on each physiological modality.

The independent findings that heart rate reactive subjects receiving heart rate biofeedback training experienced heart rate reductions and were able to maintain frontal EMG levels whereas control subjects were not, is of considerable interest. Gatchel et al. (1978) found that subjects given frontal EMG biofeedback training were able to maintain control over the training target response (frontal EMG) under stress conditions, did not include a control group. This study, which did include a deferred contact group, indicates that simply noting increases or lack of increases in physiological responding to a stress induction procedure is not an adequate methodology to evaluate the physiological outcome of biofeedback training. The results here suggest that subjects given biofeedback training in heart rate were able to maintain control of both heart rate and frontal EMG under stress conditions. Caution must be exercised making this conclusion. Statements about the joint occurrence of these two physiological responses would require a multivariate analysis procedure. The use of univariate procedures in this study requires that the nominal alpha level be multiplied by 2 -- the number of independent events in the joint occurrence of decreased heart rate and maintained frontal EMG. Using this criterion, the level of significance becomes $p = .098$ for the groups as a whole analysis and $p = .172$ for the heart rate reactive analysis.

Another caution must be issued since subjects were assigned to a physiological target response on the basis of reactivity rather than randomness. Lott and Gatchel (1978) have shown that subjects who demonstrated high reactivity to a cold pressor test produced significantly larger heart rate changes than low reactor subjects and it is possible that the same mechanism was in operation here. The high reactive subjects employed in this study may have produced greater heart rate changes than a random sample of biofeedback heart rate trainees and, because of the viscerally mediated nature of heart rate (Miller, 1978; Benson, 1975) produced concomitantly greater decrements in frontal EMG. A suggested topic for further research would be, therefore, a multi-response evaluation of heart rate biofeedback training under stress conditions between high heart rate reactive subjects, random assignments to heart rate training subjects, and no-feedback biofeedback equipment exposed subjects to determine the relative physiological response generalization of heart rate to frontal EMG for the three groups. In this study a multivariate analysis of variance would be appropriate to confirm the joint occurrence of self-control of both heart rate and frontal EMG under stress conditions. This finding, if confirmed would indicate that heart rate biofeedback training would be a more efficacious procedure in the treatment of anxiety than frontal EMG training in that it provides self-control of two physiological response systems "for the price of one."

The second main aspect to the finding of maintained frontal EMG and decreased heart rate, is that significant differences between treat-

ment and control groups were found primarily under stress conditions and not on the prestressor and poststressor baselines. The result is in agreement with DeGood and Adam's (1976) finding that groups given biofeedback training in controlling (decreasing) heart rate are indistinguishable from other treatment groups including no feedback control groups except when tested under aversive stimulus conditions. The result is also in contradiction to the cultivated physiological arousal hypothesis that states biofeedback trained groups exhibit lower indices of physiological arousal than simply relaxed control groups under resting conditions. Previous attempts to test the efficacy of biofeedback training for anxiety or stress management may have overlooked the significance of biofeedback training by not including a stress induction procedure. In this study, monitoring physiological response systems solely during resting periods would have lead to the erroneous conclusion that sitting in a comfortable recliner chair located in a quiet sinecure away from the hustle bustle of crowded classrooms, libraries and cafeterias, was equally as effective as biofeedback training in controlling a subject's physiological response to stress. When combined with the fact monitoring of the single training target response even under a stress condition may lead to the equally erroneous conclusion that cultivated physiological low arousal has occurred, it is clear that future research assessing the effectiveness of biofeedback in stress management should employ a multi-response stress induction procedure in order to determine that acquisition of self control of a physiological modality has occurred.

Decreased skin resistance during poststressor baseline. Decreases in skin resistance have previously been noted during biofeedback training (e.g. Gatchel, 1976). Kilpatrick (1971) has found that tonic (mean level) skin resistance versus phasic (responsivity) skin resistance is more responsive to cognitive tasks and to the period of cognitive activity following the task, than to psychological stressors such as the manipulation of threat. Phasic skin resistance, conversely, was found to be more responsive to psychological stressors than cognitive stressors. Since this study employed basal DC coupled skin resistance versus quick change AC coupled skin resistance, variations in skin resistance measured were more of the tonic variety. Increased skin resistance during the poststressor baseline, then, may have been due to the use of the less responsive basal GR and its sensitivity to cognitive activity following mental tasks. Despite this interpretation, however, the major implication of this finding is that even when biofeedback training is provided in a physiological modality which constitutes a major physiological component of anxiety for that individual, learned control of that physiological modality is not necessarily reflected in related physiological response systems.

Decreased trait anxiety. Insofar as this was an analogue study employing subjects with "normal" rather than "high" anxiety levels, the lack of treatment effect on the self-report indices of anxiety is not altogether surprising. This is especially so since little or no training was provided in identifying personal stresses. Nor was there anything but mild encouragement given to practice physiological self-

control outside the laboratory. Nevertheless, a central aim of providing biofeedback training was to decrease anxiety and so a more lengthy discussion of lack of treatment effects on either state or trait anxiety is warranted.

Of the two types of anxiety, state anxiety is the more situationally sensitive construct and one might reasonably have expected a significant treatment effect on this measure. It could be argued that the lack of such an effect may be accounted for by the order of administering the STAI since it was administered after the psychophysiological stress profile when any impending anxiety over the administration of stressors would likely have been relieved. This argument is not wholly convincing. On a logical basis, presession state anxiety could be influenced by any number of factors such as being late for the session or having just completed an examination as was reported by several subjects. Post-session administration of the STAI would be more likely to reflect decrements in self-report anxiety as a result of practicing the developed biofeedback strategy or normal relaxation strategies. Moreoever, there is no evidence to suggest that presession administration of the STAI would have led to a significant treatment effect. Analysis of subject responses to the question of differential anxiety in anticipating the pretest and posttest profiles revealed no significant differences between treatment and control groups. The most likely explanation, therefore, is that since high initial state anxiety was not a feature of the sample, no decrements were found.

The finding of decreased trait anxiety for both groups at the

posttest session is an unexpected outcome and more problemmatical to explain. In fact, the research design of this study does not permit an adequate explanation. The result is, however, consistent with the findings of decreased poststressor frontal EMG for the groups as a whole, and decreased prestressor and poststressor peripheral skin temperature for heart rate reactive groups during posttesting. One might speculate that the therapeutic expectancy/demand characteristics implicit in both the exposure to biofeedback equipment and the promise of treatment led to a placebo effect. An experiment by Gatchel, Hatch, Maynard, Turns, and Taunton-Blackwood (1979) describes the power of such a placebo effect. Comparing a relaxation/biofeedback group, a false feedback group and a systematic desensitization group, Gatchel et al. found that despite lack of physiological arousal reduction in the false biofeedback group, this group demonstrated as much reduction in self-reported speech anxiety as the two treatment groups. This reduction was persistent in that it was maintained for all three groups in a follow up evaluation one month later.

The issue of whether decreased trait anxiety found in both groups was due to a placebo effect associated with exposure to biofeedback equipment during the psychophysiolgoical stress profile or to some other factor such as increased confidence as the academic year progressed might easily have been provided in this study by the inclusion of a second control group. Administering the STAI to a third group during the pretest and posttest weeks and providing no further contact would have permitted comparisons to be made between biofeedback treatment,

biofeedback exposure plus promise of therapeutic benefit, and pure no contact control conditions. Decreased trait anxiety in this second control group would indicate the effects of external factors while no decreases in trait anxiety would confirm the expectancy/demand effect associated with the experimental procedure.

It is suggested that any further research using a psychophysiological profile to assess biofeedback treatment incorporate such an additional control.

Hypothesis 3

Hypothesis 3 stated that subjects receiving reactive modality biofeedback training on a specific modality, in this case heart rate, will not exhibit anxiety increments on frontal EMG, skin resistance or peripheral skin temperature. The findings were a significant treatment effect in favor of increased peripheral skin temperature for the control group at the poststressor baseline, and decreased skin resistance at the post stressor baseline.

Peripheral skin temperature interaction and decreased skin resistance. The hypothesis that learned control of a subject's most reactive physiological modality occurs without concomitant arousal increases in other physiological response modalities must be rejected. Instead, the hypothesis that control of a physiological response system other than the training target response must be entertained regardless of whether the training target response is the subject's most reactive physiological modality or a uniform training target response that does

not constitute a major physiological component of anxiety for the individual. As in the case of discussing the joint occurrence of decreased heart rate and maintained frontal EMG under stress conditions, the caveat concerning actual alpha levels must be made here. Nevertheless, the fact that heart rate biofeedback training is reflected on increased arousal as measured by skin resistance and peripheral skin temperature strongly suggests dissociation of physiological responses. Moreover, the finding of a significant treatment effect in which control subjects experienced significantly increased skin temperature under stress conditions whereas heart rate biofeedback subjects did not is consistent with DeGood and Chisholm's (1976) finding of increased peripheral vasoconstriction accompanying frontal EMG reduction training. In DeGood and Chisholm's study the increased peripheral vasoconstriction occurred despite decreases in frontal EMG, heart rate, and respiration suggesting the possibility that control of visceral or viscerally mediated physiological responses and other autonomic physiological responses such as peripheral skin temperature and skin resistance operate through different mechanisms. It is interesting to speculate whether the converse is true as well -- that acquiring self-control over peripheral skin temperature or skin resistance response would lead to increases in frontal EMG and/or heart rate. Perusal of frontal EMG data for subjects in this study receiving peripheral skin temperature biofeedbcak training strongly suggest there are increases in frontal EMG associated with training in peripheral skin temperature increases. Further systematic investigation of this question is required. If the

resulting evidence supported the dissociation of visceral and autonomic physiological responses, a case could be made that biofeedback training should be provided in two physiological response modalities. Such an approach would be analogous to Schwartz's (1972) patterning of physiological responses. Schwartz (1972) using a cardiovascular response system and pareital alpha has shown that providing training in both physiological modalities but not in one alone leads to subjective self-reports of intense relaxation or what Schwartz describes as "emergent properties." It is possible that it is necessary to provide training in two dissimilar physiological response systems in order to produce the cultivated low physiological effect necessary for Wolpe's (1958) concept of reciprocal inhibition.

Hypotheses 4 and 5

Hypothesis 4 states that the use of profile SD as a dependent measure would yield anxiety decrements on a greater number of physiological response systems than the results of using mean levels of physiological activity. The findings consisted only of a significant treatment and time difference on heart rate variability for both the groups as a whole analysis and the heart rate reactive groups analysis; a significant frontal EMG treatment effect for the heart rate reactive groups analysis; and a significant decrease in frontal EMG variability during posttesting for the groups as a whole analysis. The hypothesis was, therefore, rejected.

In an attempt to "reclaim" this hypothesis, a supplementary analysis

using profile variance (S^2) instead of profile SD was performed. The use of variance as opposed to SD causes high numerical values to be enhanced due to the geometric progression effect of the squaring procedure and should, therefore, logically provide more sensitive measure of response variability. Also, variance has widely been used in the research on heart rate variability (Porges, 1972; Walter & Porges, 1976). The results of the heart rate reactive groups revealed the same number and type of significant effects although, as predicted, the results achieved a greater level of significance than in the profile SD analysis. The results were: a significant group x time interaction for a frontal EMG variability ($F(1,10) = 4.861$, $p = .052$); a significant group x time interaction for heart rate variability ($F(1,10) = 7.034$, $p = .024$); and a significant time effect for heart rate variability ($F(1,10) = 81.375$, $p = .001$). Both groups displayed increased heart rate variability during posttesting with the treatment subjects showing a lesser increase (Group means for the profile variances are presented in Table 10. Analysis of variance summaries are located in Appendix G.)

For the groups as a whole analysis there was an increase in the number of significant effects as well as a greater level of significance when profile S^2 was used over profile SD. There was a significant time effect for frontal EMG variability, $F(1,18) = 4.720$, $p = .043$, with both groups having decreased frontal EMG variability during posttesting; a significant group x time interaction for heart rate variability, $F(1,18) = 5.329$, $p = .0331$; and a significant time effect for heart rate variability at posttesting, $F(1,18) = 130.078$, $p = .001$, with

Table 10

Group Means for Profile S^2 (Heart Rate Reactive Groups)

(N=12)

Group	Anxiety Measure	T_1	T_2
1	EMG	5.040 (4.762)	2.581 (2.724)
2	EMG	0.940 (0.737)	1.166 (0.709)
1	PST	107.763 (22.487)	111.236 (6.191)
2	PST	99.201 (14.619)	11.236 (8.688)
1	GSR	166.900 (23.892)	187.981 (29.833)
2	GSR	178.360 (26.771)	162.306 (24.307)
1	H.R	763.280 (521.054)	1561.987 (431.634)
2	H.R	619.269 (143.971)	2083.228 (346.946)

the treatment group experiencing less of an increase. (Group means for profile variances are presented in Table 11. Analysis of variance summaries are given in Appendix G.)

The differential results of using physiological mean levels of activity, profile SD, and profile S^2 are presented and contrasted in Figure 2. The choice between profile SD and profile S^2 is essentially a choice between a dependent measure that provides a more conservative interpretation of the results (profile SD) and a dependent measure that offers a more generous interpretation (profile S^2). Although the interest here is reactivity and the profile S^2 procedure enhances the finding of reactivity by accentuating the difference between baseline and stressor scores, the profile SD measure seems most appropriate here since it treats both high and low values equally. In so doing, it avoids any argument that reactivity results have been artificially produced by the choice of statistical procedure. It also provides the advantage of giving a conservative estimate of the results of biofeedback training and mitigates against the drawing of over optimistic conclusions, especially in view of the liberal .10 level of significance employed in the study.

A caveat must be made here, however. Due to the skewness of standard deviations between the two groups, the assumptions of normality and homogeneity of variances made in the analysis of variance procedure may have been violated. Although the analysis of variance procedure is robust to such violations, the size of the disparity suggests that caution must be exercised in interpreting the results.

Table 11

Group Means for Profile S^2 (Groups as a Whole)

(N=20)

Group	Anxiety Measure	T_1	T_2
1	EMG	3.314 (4.193)	1.819 (2.270)
2	EMG	1.845 (1.644)	1.437 (1.029)
1	PST	106.213 (18.987)	104.340 (18.620)
2	PST	99.639 (16.021)	103.855 (17.449)
1	GSR	171.120 (32.029)	176.583 (63.392)
2	GSR	182.248 (24.140)	163.954 (32.583)
1	H.R	773.854 (173.392)	1603.951 (447.725)
2	H.R	713.937 (212.763)	1965.353 (360.835)

Figure 2

Significant Effects According to Physiological Dependent Measure

Group	Physiologidal Response	Dependent Measure		
		Mean Levels	Profile SD	Profile S^2
Groups as a Whole	EMG	Treatment Effect (S.C.)* Time Difference	Time Difference	Time Difference
	PST	--	--	--
	GSR	Time Difference (B2.)	--	--
	H.R	Treatment Effect (S.C.)	Treatment Effect Time Difference	Treatment Effect Time Difference
Heart Rate Reactive Groups	EMG	Treatment Effect (S.C.)	Treatment Effect	Treatment Effect
	PST	Treatment Effect (B2.)	--	--
	GSR	Time Difference (B2.)	--	--
	H.R	Treatment Effect (S.C.)	Treatment Effect Time Difference	Treatment Effect Time Difference

*S.C. denotes stress condition and B2 denotes poststressor baseline.

The comparison of results between profile SD and mean levels of physiological activity clearly favours mean levels of physiological activity in terms of the number of significant results. This, however, can be explained by the greater number of analyses carried out using mean levels of physiological activity. Since analyses were conducted at each of the prestressor, stressor, and poststressor baselines for each physiological modality, three times the number of analyses were performed with each measure. On the basis of chance alone, one would expect a greater number of significant results to occur.

Perhaps the issue of a choice between these physiological dependent measures is not their differential sensitivity, but rather the additional information they provide as alternate indices of physiological self control. Considered in this light, the results of the profile variability analyses corroborate the previously noted findings (Shein & Mandel, 1981; Hnatiow & Lang, 1965; Lang, Sroufe & Hastings, 1967; Harrison & Raskin, 1976) that mean level reductions in heart rate can occur despite increased heart rate variability. It is not clear, however how this observation should be interpreted. There is some support for the notions that cognitive tasks produce a decrease in heart rate variability (e.g., Lacey, 1967; Porges, 1972; Porges & Raskin, 1969; Walter & Porges, 1976; Heslegrave, Ogilvie & Furedy, 1981), and that aversive stimuli produce increases in heart rate variability (Heslegrave, Ogilvie & Furedy, 1981). This evidence seems to indicate that the stimuli used in the study behaved more as stressors during the posttest administration of the stress profile than during the pretest administration. Following this reasoning it is possible to interpret the lesser increase in heart rate variability by the treatment

group relative to the control group as an indication of self-control by the treatment group during an aversive stimulus situation.

Another finding of interest from the profile SD results was that a significant increase in heart rate variability was accompanied by a decrease in mean levels of heart rate. Alternatively, however, there was a trend for decreased frontal EMG variability coincident with a decrease in mean levels of frontal EMG. This raises the possibility that physiological response variability behaves differently from mean levels of physiological activity, at least under stress conditions. Logically, one might expect physiological variability to increase in each physiological modality during stress conditions. The evidence from this study, however, suggests that this is not so. Further research using mean levels of physiological activity might simultaneously monitor response variability in order to uncover the relationship between variability and mean levels of physiological activity.

The Method of Determining Most Reactive Modality

The interpretation of results up to this point has been in the context of most reactive modality biofeedback training. It has, therefore, assumed that the ZR transformation used to determine most reactive modality is a tried and tested instrument. In point of fact, the opposite is true. The ZR transformation is a unique instrument derived for this thesis and without previously determined validity or reliability. This section will provide a preliminary discussion of these issues.

Support for the Construct Validity for the ZR Transformation

The most logical approach to establish the construct validity of the ZR transformation would be to compare the most reactive modality as determined by the ZR transformation to some established normative scores. Unfortunately such normative data do not exist. An alternate method is to compare the results of using the ZR transformation with the results of other more established approaches for determining reactivity. As mentioned in Chapter two, the most prevalent method of identifying reactivity has been the median-split method. Comparing the results of these two methods is not, however, a straightforward procedure. There are two reasons for this. First, the median-split method assigns reactivity to only one physiological modality at a time. Even though a subject may be a high reactive on one physiological modality, he may also be high reactive on one or more other modalities. Determining high reactivity simultaneously on four physiological modalities would therefore reveal a response topography of one to four modalities without being able to discriminate which was the most reactive. Although it would be possible to determine most reactive modality by performing the median-split in four dimensional space, such an approach leads to interpretive difficulties and results in assigning the other three modalities equal weights. The ZR statistic, by contrast, easily assigns the subject a most reactive physiological modality even though other modalities may be high reactive as well. A comparison of the two approaches, therefore, must take the form of asking whether a subject's most reactive physiological modality as determined by the ZR statistic would also be considered as a high reactive physiological modality using the median-split method.

A second difficulty in making this comparison is that the split-half method by definition assigns half of the subjects to a high reactive group and the other half of the subjects to a nonreactive (low reactive) group. This occurs regardless of whether the individual scores within the sample justify such a designation on that physiological modality. For example, the entire sample with the exception of one or two subjects may show little or no reactivity on heart rate in response to a stressor. Nevertheless, using the median-split approach, one half of the sample would be designated as high reactors on heart rate. Conversely, the entire population may show marked heart rate reactivity in response to a stressor and yet half the sample would be identified as low reactors. In point of fact, this study revealed such a contradiction. When subjects were assigned to either high reactive or low reactive groups on the basis of their skin resistance responses to stress, ten subjects were identified as skin resistance high reactive despite the fact only five subjects even displayed increased skin resistance in response to the stressor. A difficulty with the median-split approach then, is the problem of defined proportionality. In contrast, the ZR transformation does not require that any specified proportion of subjects be reactive on any one physiological modality. It simply prescribes the one physiological modality of the total number of modalities monitored must be defined as most reactive. The issue of proportionality here is an empirical question. This difference in the proportion of subjects designated as high reactive as a result of using the split-half or ZR transformation methods means that the ZR transformation may assign more (or less) subjects as being most reactive than the split-half method assigns as high reactive. Comparisons

between the ZR transformation and split-half method cannot be made on a straight one for one basis.

Within the context of these limitations, however, a comparison of the two methods was still deemed desirable and carried out. Following the median-split method employed by Light (1981), the difference score between initial baseline and the subject's response to a stressor, in this case the first stressor, was calculated for each subject. The top ten subjects were then assigned to a high reactive group and the bottom ten subjects assigned to a low reactive group for each modality. Table 12 shows that the most reactive modality as assigned by the ZR transformation fell within the high reactive group as determined by the median-split method 65 percent of the time. This result, however, is confounded by the problem of the median-split's defined proportionality. In order to get a clearer view of this problem the difference scores for each physiological modality were listed in sequence and a clinical judgement approach used to discriminate high and low reactive subjects. The results for heart rate reactivity which constituted the major proportion of subjects in this thesis are presented in Table 13. (Median-split difference scores for frontal EMG, PST and GSR are presented in Appendix H.)

Table 13 shows that if the subjects are split into the top 15 subjects and bottom five subjects there is a logical separation of baseline-stressor difference scores between 4.275 beats per minute and 1.875 beats per minute. Given this split, all but two of the top 15 subjects are most reactive according to the ZR transformation, and all of the bottom five subjects are most reactive in a physiological

Table 12

Mean-Split High and Low Reactive Subjects

Subject Number	ZR Transform	Median-Split			
	Most Reactive Modality	EMG Reactivity	PST Reactivity	GSR Reactivity	H.R Reactivity
1	PST	Lo	Hi	Lo	Lo
2	PST	Lo	Lo	Hi	Lo
3	EMG	Hi	Hi	Lo	Hi
4	H.R	Hi	Hi	Hi	Hi
5	GSR	Hi	Lo	Hi	Hi
6	H.R	Lo	Lo	Lo	Lo
7	H.R	Hi	Lo	Hi	Hi
8	H.R	Lo	Lo	Lo	Hi
9	H.R	Lo	Hi	Hi	Lo
10	H.R	Lo	Hi	Hi	Lo
11	H.R	Lo	Lo	Hi	Hi
12	H.R	Hi	Hi	Hi	Lo
13	EMG	Hi	Lo	Lo	Lo
14	H.R	Hi	Hi	Lo	Hi
15	H.R	Hi	Lo	Lo	Hi
16	H.R	Lo	Hi	Lo	Lo
17	H.R	Lo	Hi	Hi	Lo
18	EMG	Lo	Lo	Hi	Lo
19	PST	Hi	Hi	Lo	Lo
20	H.R	Hi	Lo	Hi	Hi

Table 13

Median-Split Difference Scores for Heart Rate Reactivity

Subject Number	Median-Split Difference Score	ZR Assigned Reactive Modality
20	20.75	H.R
14	16.40	H.R.
7	12.85	H.R
15	11.275	H.R
4	10.60	H.R
5	9.975	GSR
9	9.875	H.R
8	8.975	H.R
3	8.50	EMG
11	7.25	H.R
	SPLIT	
6	6.90	H.R
16	6.625	H.R
17	6.225	H.R
10	6.05	H.R
12	4.275	H.R
18	1.875	EMG
1	.90	PST
13	.125	EMG
2	.15	PST
19	-1.40	PST

modality other than heart rate. Looking at the comparable ordering of baseline-stressor difference scores for GSR and frontal EMG (see Appendix I), we see that the GSR subject (subject 5) was also high reactive on GSR (the most reactive in fact), and that the frontal EMG subject (subject 3) was also in the high frontal EMG reactive group. Turning to Appendix C, we also note that the ZR transformation also designated these two subjects as highly reactive on GSR and frontal EMG respectively, but when these reactivities were compared with heart rate they were more reactive on heart rate.

Although the above approach is not a conclusive proof of the construct validity of most reactive modality as determined by the ZR transformation, it is highly suggestive. The ZR transformation approach is consistent with the clinical judgement approach applied to the baseline-stressor difference scores, and justifiable within the limitations imposed by the median-split method.

Reliability of the ZR Transformation

As a preliminary test of the reliability of the ZR transformation for determining most reactive modality, a Spearman's rank order correlation coefficient was calculated between the most reactive physiological modalities as identified by the ZR statistic at the pretest and post-test administrations of the stress profile. Because of the treatment intervention for experimental subjects, the correlation was made only for control subjects. The small (N = 10) sample as a result, means that this index of reliability must be tentative. A correlation of .5222, $p = .061$ was obtained. Using the tau b procedure recommended when the

number of tied ranks relative to the number of observations is large revealed a correlation of .4472, p = .059. As a matter of interest, the equivalent correlations for the treatment group were r = -.0348, p = .462 and r = -.0298, p = .458 indicating less agreement between reactive modalities when biofeedback training in most reactive physiological modality was provided.

Although the rank order correlation for most reactive physiological modality as determined by the ZR statistic is modest, this can be explained, at least in part, by the small numbers employed in the calculation. Future research employing this statistic should, as a matter of course, test further its reliability.

Stressor Analysis

A secondary purpose of this study was to explore the physiological responses to the stressors used in the stress profile. Using the initial stress profile baseline and stressor 1 data points, a 2x2 (Group x time) repeated measures analysis of variance was performed for each physiological modality. The purpose of this procedure was to determine that the stimuli used served equally as stressors for each group. The results for the groups as a whole analysis revealed a significant, $F(1,18) = 30.986$, p = .001 increase in heart rate indicative of arousal for both groups in response to stressor 1. Peripheral skin temperature, however, demonstrated a significant, $F(1,18) = 5.876$, p = .026, increase for both groups, and skin resistance evidenced a significant, $F(1,18) = 7.915$, p = .012, decrease for both groups in response to stressor 1, indicative of decreased arousal. There were no significant

interaction effects representative of differential rates of responding between the two groups. The analyses for heart rate reactive groups revealed only a significant, $F(1,10) = 49.058$, $p = .001$, increase in heart rate in response to stressor 1 for both groups. Again there were no significant interaction effects indicative of differential rates of responding for the two groups. (Group means for the initial baseline and stressor 1 values appear in Table 14 for the groups as a whole, and Table 15 for heart rate reactive groups. Analyses of variance summaries appear in Appendix I.)

The results were clearly not in keeping with the view that the serial sevens task served adequately as a stressor. In order to explore this discrepancy further, a one way repeated measures analysis of variance was performed using the initial baseline and three stressor data points as the time factors. Since there were no differences in the way treatment and control subjects responded to stressor 1, the one way analysis of variance was conducted on the entire sample. Significant effects were found for peripheral skin temperature, $F(3,57) = 8.490$, $p = .001$, and for heart rate, $F(3,57) = 11.708$, $p = .001$. (Group means for the four physiological modalities are presented in Table 16. Analyses of variance summaries for significant effects are located in Appendix J.) The Scheffé Multiple Comparisons procedure was used to determine the critical mean differences. A significant increase in peripheral skin temperature in stressor 1, and a significance decrease in skin temperature at stressor 3 were revealed, $Sch(1,76) \geq .308$, $p \leq .05$. Heart rate demonstrated a significant, $Sch(1,76) \geq 2.074$, $p \leq .05$,

Table 14

Group Means for Initial Baseline and Stressor 1

(Groups as a Whole)

Group	Anxiety Measure	Initial Baseline	Stressor 1
1	EMG	3.108 (1.811)	3.226 (1.657)
2	EMG	3.358 (1.693)	3.007 (0.853)
1	PST	31.756 (3.132)	32.153 (3.267)
2	PST	30.690 (2.572)	31.367 (2.613)
1	GSR	38.534 (1.365)	39.608 (2.539)
2	GSR	39.227 (2.696)	41.426 (2.296)
1	H.R	74.502 (11.502)	81.800 (10.793)
2	H.R	76.287 (7.518)	83.650 (5.882)

Table 15

Group Means for Initial Baseline and Stressor 1

(Heart Rate Reactive)

Group	Anxiety Measure	Initial Baseline	Stressor 1
1	EMG	3.342 (2.068)	3.725 (1.919)
2	EMG	2.782 (1.566)	2.598 (0.451)
1	PST	32.181 (3.515)	32.613 (3.717)
2	PST	30.600 (2.320)	31.490 (2.424)
1	GSR	38.367 (1.353)	39.950 (2.817)
2	GSR	40.005 (2.747)	41.070 (2.990)
1	H.R	71.625 (11.366)	80.833 (10.872)
2	H.R	74.333 (8.832)	83.733 (9.968)

Table 16

Baseline and Stressor Means for the Sample by Modality

(N=20)

Physiological Modality	Initial Baseline	Stressor 1	Stressor 2	Stressor 3
Frontal EMG	3.233 (1.711)	3.116 (1.287)	3.323 (1.953)	3.272 (1.607)
PST	31.222 (2.842)	31.760* (2.907)	31.229 (2.807)	30.663* (2.903)
GSR	38.881 (2.110)	40.517 (2.534)	40.657 (2.624)	40.224 (4.257)
H.R	75.395 (9.595)	82.725** (8.513)	82.180** (10.478)	82.525** (8.281)

* Significant difference, $\underline{Sch}(1,76) \geq .308$, $p \leq .05$.

** Significant difference, $\underline{Sch}(1.76) \geq 2.074$, $p \leq .05$.

increase from initial baseline at each of stressors 1, 2, and 3.

The results of these analyses indicate that a three minute serial sevens subtraction task is not adequate to serve as a stressor. Although heart rate was immediately responsive to the first stimulus, peripheral skin temperature did not demonstrate arousal until stimulus 3. Also, as demonstrated before, basal GSR did not decrease (show increased arousal) until the poststressor baseline. These results can be explained by the lower responsiviity of peripheral skin temperature and basal GSR, and the more instantaneous responsivity of heart rate. Future research should use longer periods of stress or more intense stressors in order to detect these changes. The use of quick change AC coupled skin resistance would also provide greater responsivity.

The lack of increase in frontal EMG in response to the stressors is more problemmatical and may require the use of cognitive stressors with greater difficulty levels. Caciappo and Petty (1981) have demonstrated that electromyographic activity occurs maximally in the lip area in response to covert information processing tasks and that even in this area EMG activity was indetectable when level of meaning and difficulty of encoding were low. In this study, task difficulty was at the grade 12 level. Future studies should alter the task difficulty to reflect the population sample used.

Summary of Research Implications

The research implications of this study can now be summarized. To facilitate this presentation, the implications will be grouped under

three headings -- implications of providing heart rate biofeedback training to heart rate reactive subjects; methodological considerations for further research in most reactive modality biofeedback training; and the current status of most reactive modality biofeedback training.

Most Reactive Biofeedback Training for Heart Rate Reactive Subjects

A main finding for this portion of the study was that heart rate biofeedback training in subjects for whom heart rate constitutes the major physiological component of anxiety lead to a reduction of heart rate under stress conditions. While this reduction in heart rate was accompanied by concomitant decreases in frontal EMG under stress conditions. it did not lead to a state of general cultivated low physiological arousal indicative of physiological relaxation; or to subjective self-report expressions of anxiety. The conclusion to be drawn from these results is that heart rate biofeedback training does not, when used alone, provide a satisfactory treatment for anxiety in heart rate reactive subjects. It does not, in Wolpe's (1958) formulation, provide an adequate competing response to anxiety. This conclusion does not, however, preclude the use of heart rate biofeedback as an innoculation procedure against the development of somatic complaints as a result of anxiety. The theoretical formulation behind this thesis was that somatic complaints occur in a subject's most reactive physiological modality as a result of persistent exposure to stress and consequent over arousal of that physiological modality (Malmo, 1975). Although an adequate test of this hypothesis requires a longitudinal study with heart rate reactive subjects assigned to either heart rate biofeedback

training or control groups, the results of this thesis demonstrate that heart rate reactive subjects are able to acquire voluntary control over their heart rate under stress conditions thus establishing a necessary prerequisite to conducting such a longitudinal study.

A second implication of these findings is that heart rate biofeedback training may be a more efficacious single target response system than the frontal EMG physiological modality. Gatchel et al.'s (1978) finding that frontal EMG biofeedback training was not accompanied by heart rate reductions under stress conditions, and this study's finding that heart rate biofeedback training was reflected by frontal EMG decreases under stress conditions gave credence to this claim. The two studies are not, however, directly comparable. Whereas Gatchel et al. employed subjects without regard to their most reactive modality, this study provided heart rate training only to heart rate reactive subjects. The issue of whether these conflicting physiological generalization effects are a function of single target training modality in different physiological response systems, or are specific to providing biofeedback training in a subject's most reactive modality, remains answered. A full scale version of this study with equal and sufficiently large numbers of frontal EMG and heart rate reactive subjects would be able to resolve this issue. Given the results of this thesis and the importance of the issue, such a study seems certainly warranted.

The second main finding for heart rate reactive subjects was that heart rate and frontal EMG reductions occurred despite arousal increments in other physiological response modalities. These increments in

arousal were due to both situational factors as indicated by decreases in skin resistance for both groups, and due to training as evidenced by decreased peripheral skin temperature for the treatment group versus the control group. In addition to corroborating the previous conclusion that heart rate reactive biofeedback training did not lead to cultivated low physiological arousal, this finding led to the suggestion that biofeedback training may have to be provided in two physiological response systems. These results suggest that for heart rate reactive subjects this secondary training target response should be peripheral skin temperature. Whether this is also the optimal secondary training site when reactive modality training is provided in other response systems, must again await a full scale version of this study.

Methdological Implications for Further Biofeedback Research

There are several methodological considerations that have emerged from this study. Consistent with other research (Gatchel et al., 1978; DeGood & Adams, 1976; Burish et al., 1981), this study found that it was impossible to discriminate between treatment and control subjects during the relaxation periods of the psychophysiological stress profile. This means that in order to demonstrate that acquisition of self-control over a physiological response has occurred, future research should include a stress innoculation procedure.

The second implication concerns the nature of the stressor(s) used in the stress innoculation procedure. The level of difficulty of the stressors used should be matched to the abilities of the sample, and be of sufficient duration to detect changes in response systems with

longer response latencies such as peripheral skin temperature and basal skin resistance. Moreoever, future research should demonstrate, prior to analysis, that the stimuli employed did serve as stressors in all of the physiological response modalities monitored.

A third implication concerns the use of control groups. As shown in this study, the use of a subject as his own control and the reliance on increases or lack of increases in physiological indices of arousal may not be sufficient to detect treatment effects in biofeedback training. A no contact control with physiological measurements taken during pretest and posttest sessions should be a mandatory feature of future biofeedback research. Also, in order to overcome the possible placebo effect associated with the simple administration of the psychophysiological stress profile, future research ought to include the precaution of including a second self-report dependent measure group as a control for biofeedback exposure placebo effects.

Finally, the use of the ZR transformation to determine most reactive modality had promising results in the study. With the advantage of overcoming the problem of disproportionality which is inherent to the more prevalent median-split method for choosing high reactive subjects, future research to establish the validity and reliability of this assessment instrument would appear to be worthwhile.

Current Status of Most Reactive Modality Biofeedback Training

Statements about most reactive modality biofeedback training as a generic treatment cannot be made based on the results of this study. This is due to the underrepresentation of subjects receiving biofeedback

training in reactive modalities other than heart rate. Moreoever, attempts to broaden the generalizability of this study by integrating other research which employed frontal EMG as the target training response are thwarted by this study's inclusion of most reactive modality as a presage variable. As a preliminary investigate into most reactive modality biofeedback training, however, this study generated some interesting findings, namely that

a) Differential physiological generalization effects may be associated with choice of physiological training modality.

b) Biofeedback training in a specific physiological target response does not occur in isolation from other physiological response systems. Other physiological response systems may increase in arousal concomitant with training.

c) Therapeutic placebo effects may occur as a result of administration of the physiological stress profile and/or expectation of treatment benefit.

These three findings have important implications for the choice of physiological biofeedback training target response, for the choice of secondary physiological target response, and for future research concerning the influence of expectancy variables in psychophysiological assessment procedures. The results of this thesis, therefore, warrant a fuller and multivariate investigation of most reactive physiological modality biofeedback training.

Appendix A

Instructions and Tasks for the Psychophysiological Stress Profile

Initial Instructions

In this study we are investigating what happens to people's bodies when they relax and when they engage in different kinds of mental tasks. Throughout the entire procedure we will be monitoring muscle tension, skin temperature, sweatgland activity (GSR), and heart rate. We will first of all prepare and connect the recording sensors. We will then ask you to relax for 15 minutes, using whatever strategy you usually use to relax. During this time the equipment will be monitoring your body functioning while you are relaxing. Periodically you will hear some clicking sounds. This is the printer printing out the information. After your relaxation session we will be asking you to do several mental tasks. We will be monitoring your body's functioning during these tasks.

Between each task there will be a 3 minute relaxation period. After the last task there will be a 15 minute relaxation period. The first session will be mainly a recording session.

Terminate Initial Relaxation

O.K. That's fine for now. Slowly let your attention drift back to this room. I'm going to ask you to do several tasks now. In each case I will tell you what I want you to do, then give you some time to do the task. After each task I will give you 3 minutes to just relax before I present the next task. After the last task there will be a 15 minute relaxing period when you can relax again as much as possible.

Serial Sevens

In a few moments I am going to tell you a number. I want you to subtract 7 from that number, and then subtract 7 from that answer, and then subtract 7 again, and keep on subtracting 7 until I tell you to stop. Do not say your answers out loud. Do all the subtracting silently. After 3 minutes I will tell you to stop and give me the number you have reached. Are you ready? O.K. The number is 1000, go ahead and start subtracting.

Terminating Serial Sevens

Stop... Tell me your answer (write down the answer). You did very well. Just relax now for 3 minutes. Again use whatever strategy you usually use to relax... Just go ahead and relax.

Reading Task

O.K. That's fine for now. The next task is a reading task. I am going to give you two passages to read. After you finish each passage, I will ask you some questions to see how well you remember what you read.

Here is the first passage. Read it silently and then turn the card over.

.....

(After the last question of the last passage, say).

That's all for the reading task. Now I want you to relax again for 3 minutes. Use whatever strategy you usually use to relax... Just go ahead and relax.

Arithmetic Task

O.K. That's fine for now. The last task is an arithmetic task. I am going to ask you questions. I want you to work the question out in your head and tell me your answer when you're through. Do you understand?

.....

(After the last answer, say)

Those are all the tasks. Now I am going to give you a chance to relax completely. Again use whatever strategy you usually use to relax. Try to relax as completely as possible. Just go ahead and relax.

Arithmetic Task

1. How much is 6 dollars and 3 dollars?

2. If a woman buys four cents worth of stamps and she gives the clerk ten cents, how much change should she get back?

3. A newsboy collected 25 cents from each of seven customers. What is the total amount he collected?

4. How many inches are there in three and one half feet?

5. How many candies can you buy for 49 cents if one candy costs seven cents?

6. How many hours will it take for a man to walk 28 miles at the rate of four miles an hour?

7. If a man buys eight four cent stamps and gives the clerk a half dollar, how much change will he get back?

8. A woman with $19 spent $8.50. How much does she have left?

9. The price of canned beans is two cans for 61 cents. What is the price of one dozen cans?

10. A woman bought some second hand furniture for two thirds of what it cost new. She paid $600 for it. How much did it cost new?

11. A part-time worker's salary is $90 per week. If 15% of her pay is withheld for taxes, how much does she receive each week?

12. Ten men can finish a job in four days. How many men will be needed to finish it in a half day?

Appendix B

Alternate Tasks for the Posttest Psychophysiological Stress Profile

Serial Sevens Task - B

The number given to begin the serial sevens task was 950.

Reading Task - B

Two passages from an alternate form of the Gilmore Oral Reading Test were presented.

Arithmetic Task - B

An alternate arithmetic task of equivalent difficulty to the WAIS grade 12 level was developed and employed. The questions are presented on the next page.

Arithmetic Task-B

1. How much is 7 dollars and 2 dollars?

2. If a women buys six cents worth of stamps and she gives the clerk ten cents, how much change should she get back?

3. A newsboy collected 25 cents from each of nine customers. What is the total amount he collected?

4. How many inches are there in four and one half feet?

5. How many candies can you buy for 56 cents if one candy costs seven cents?

6. How many hours will it take a man to walk 35 miles at the rate of five miles an hour?

7. If a man buys seven four cent stemps and gives the clerk a half dollar, how much change will he get back?

8. A women with $19 spent $7.50. How much does she have left?

9. The price of canned beans is two cans for 71 cents. What is the prize of one dozen cans?

10. A woman bought some second hand furniture for three wuarters of what it cost new. She paid $600 for it. How much did it cost new?

11. A part-time worker's salary is $80 per week. If 15% of her pay is withheld for taxes, how much does she receive each week?

12. Twelve men can finish a job in four days. How many men will be needed to finish it in a half day?

Appendix C

ZR Transformations for Determining Most Reactive Physiological Modality

Z-Score Type Transformations and Most Reactive Modality*

Subject No.	Physiological Modality	Stressor 1	Stressor 2	Stressor 3
1	EMG	-.598	-.630	-.291
	GSR	.303	.776	2.198
	PST*	-1.165	-1.981	-6.147
	H.R	-1.236	-2.610	-1.786
2	EMG	-.051	-1.382	-.952
	GSR	.202	.032	.044
	PST*	.549	.429	-.143
	H.R	.047	-1.929	-.674
3	EMG*	10.843	7.241	5.367
	GSR	1.209	1.335	1.288
	PST	-.282	-.345	-.470
	H.R	4.515	7.011	4.515
4	EMG	3.589	7.285	4.515
	GSR	-7.226	-9.603	-3.336
	PST	1.289	-12.170	.535
	H.R*	7.825	5.832	8.785
5	EMG	-2.117	.284	-1.570
	GSR*	-1.185	-1.202	-1.263
	PST	.869	.391	-.780
	H.R	6.553	4.385	-2.841
6	EMG	.004	-.669	-.703
	GSR	1.955	1.498	.892
	PST	.947	.656	-.032
	H.R*	1.137	1.203	1.631
7	EMG	5.443	5.682	3.002
	GSR	.538	.874	.864
	PST	.284	.589	-.533
	H.R*	14.068	13.740	22.060

* Since heightened arousal is in the direction of increased EMG, decreased GSR, decreased PST and increased H.R, the Z score sign must be positive for EMG and H.R. and negative for GSR and PST to be determined the most reactive modality.

Z-Score Type Transformations and Most Reactive Modality (Continued)

Subject No.	Physiological Modality	Stressor 1	Stressor 2	Stressor 3
8	EMG	-.129	.360	1.022
	GSR	1.166	1.638	2.288
	PST	2.127	.561	.666
	H.R*	.504	1.049	1.158
9	EMG	-1.070	.032	-1.005
	GSR	.531	.584	.561
	PST	-.595	-.751	-.647
	H.R*	1.735	1.383	1.383
10	EMG	-3.751	-15.960	-6.174
	GSR	-.497	-.856	-.761
	PST	-3.666	-3.462	-2.342
	H.R*	14.781	9.895	12.338
11	EMG	-.501	5.179	-.931
	GSR	.255	.251	.205
	PST	.858	.671	.257
	H.R*	5.024	-1.213	1.074
12	EMG*	4.249	2.630	9.465
	GSR	-.310	-.385	-.546
	PST	8.295	.204	6.083
	H.R	1.320	2.740	3.357
13	EMG*	1.052	.077	9.427
	GSR	1.939	2.862	2.242
	PST	.879	-.142	-1.013
	H.R	.064	-1.978	-.140
14	EMG	-3.057	-2.967	-1.735
	GSR	1.364	1.377	.753
	PST	-.690	-.259	-.517
	H.R*	14.194	23.455	15.666
15	EMG	5.333	.611	8.959
	GSR	.510	.844	1.052
	PST	.786	.749	-.289
	H.R*	8.319	9.057	11.492

Z-Score Type Transformations and Most Reactive Modality (continued)

Subject No.	Physiological Modality	Stressor 1	Stressor 2	Stressor 3
16	EMG	-.008	.098	-1.351
	GSR	.985	.834	.766
	PST	-.991	-1.123	-1.189
	H.R*	4.357	5.212	7.646
17	EMG	-2.130	2.497	.128
	GSR	.633	.067	-5.174
	PST	-1.995	-2.410	-1.840
	H.R*	25.035	16.988	25.030
18	EMG	-1.215	1.606	11.510
	GSR	.214	-.304	-.610
	PST	.765	-.481	-1.333
	H.R*	6.762	10.368	13.974
19	EMG	-.716	.006	-.243
	GSR	1.447	1.625	1.018
	PST*	-.620	.620	-4.433
	H.R	-1.235	-2.999	-.706
20	EMG	7.059	2.927	3.478
	GSR	-.633	-.791	-.795
	PST	.924	-.563	-1.085
	H.R*	27.188	14.478	27.581

Appendix D

Analyses of Significant Physiological Mean Levels and Self Report Measures for Groups as a Whole

Analysis of Variance for H.R. Mean Levels (Stress Condition)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	2874.297	19			
'A' Main Effects	308.047	1	308.047	2.161	0.159
Subjects within Groups	2566.250	18	142.569		
Within Subjects	764.915	20			
'B' Main Effects	77.852	1	77.852	2.547	0.128
'AxB' Interaction	136.875	1	136.875	4.478	0.049
'B' x Subjects Within Groups	550.188	18	30.566		

Analysis of Variance for EMG Mean Levels (Stress Condition)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	64.114	19			
'A' Main Effects	0.737	1	0.737	0.209	0.653
Subjects within Groups	63.377	18	3.521		
Within Subjects	11.421	20			
'B' Main Effects	0.068	1	0.068	0.137	0.716
'AxB' Interaction	2.406	1	2.406	4.841	0.041
'B' x Subjects Within Groups	8.947	18	0.497		

Analysis of Variance for EMG Mean Levels (Poststressor)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	44.611	19			
'A' Main Effects	0.314	1	0.314	0.127	0.725
Subjects within Groups	44.297	18	2.461		
Within Subjects	18.812	20			
'B' Main Effects	3.533	1	3.533	4.203	0.055
'AxB' Interaction	0.148	1	0.148	0.176	0.680
'B' x Subjects Within Groups	15.131	18	0.841		

Analysis of Variance for GSR Mean Levels (Poststressor)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	483.462	19			
'A' Main Effects	11.837	1	11.837	0.452	0.510
Subjects within Groups	471.625	18	26.201		
Within Subjects	212.524	20			
'B' Main Effects	52.070	1	52.070	6.489	0.020
'AxB' Interaction	16.016	1	16.016	1.996	0.175
'B' x Subjects Within Groups	144.438	18	8.024		

Analysis of Variance for STAI-T

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	3073.915	19			
'A' Main Effects	9.063	1	9.063	0.053	0.820
Subjects within Groups	3064.852	18	170.270		
Within Subjects	221.477	20			
'B' Main Effects	65.039	1	65.039	7.512	0.013
'AxB' Interaction	0.586	1	0.586	0.068	0.798
'B' x Subjects Within Groups	155.852	18	8.658		

Appendix E

Analysis of Significant Physiological Mean Levels and Self Report Measures for Heart Rate Reactive Groups

Analysis of Variance for Heart Rate Mean Levels (Poststressor)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	1936.453	11			
'A' Main Effects	60.328	1	60.328	0.322	0.583
Subjects within Groups	1876.125	10	187.612		
Within Subjects	895.899	12			
'B' Main Effects	12.914	1	12.914	0.224	0.647
'AxB' Interaction	305.297	1	305.297	5.285	0.044
'B' x Subjects Within Groups	577.688	10	57.769		

Analysis of Variance for Heart Rate Mean Levels (Stress Condition)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	1666.553	11			
'A' Main Effects	427.570	1	427.570	3.451	0.093
Subjects within Groups	1238.983	10	123.894		
Within Subjects	621.836	12			
'B' Main Effects	46.195	1	46.195	1.180	0.303
'AxB' Interaction	184.266	1	184.266	4.708	0.055
'B' x Subjects Within Groups	391.375	10	39.137		

Analysis of Variance for Frontal EMG Mean Levels (Stress Condition)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	45.869	11			
'A' Main Effects	1.712	1	1.712	0.388	0.547
Subjects within Groups	44.157	10	4.416		
Within Subjects	7.942	12			
'B' Main Effects	0.010	1	0.010	0.017	0.898
'AxB' Interaction	2.106	1	2.106	3.615	0.086
'B' x Subjects Within Groups	5.826	10	0.583		

Analysis of Variance for Skin Temperature Mean Levels (Prestressor)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	111.196	11			
'A' Main Effects	4.008	1	4.008	0.374	0.555
Subjects within Groups	107.188	10	10.719		
Within Subjects	57.855	12			
'B' Main Effects	17.273	1	17.273	4.657	0.056
'AxB' Interaction	3.492	1	3.492	0.942	0.355
'B' x Subjects Within Groups	37.090	10	3.709		

Analysis of Variance for PST Mean Levels (Poststressor)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	136.940	11			
'A' Main Effects	0.674	1	0.674	0.049	0.828
Subjects within Groups	136.366	10	13.627		
Within Subjects	65.040	12			
'B' Main Effects	14.133	1	14.133	4.441	0.061
'AxB' Interaction	19.087	1	19.087	5.998	0.034
'B' x Subjects Within Groups	31.820	10	3.187		

Analysis of Variance for GSR Mean Levels (Poststressor)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	266.496	11			
'A' Main Effects	29.953	1	29.953	1.266	0.287
Subjects within Groups	236.543	10	23.654		
Within Subjects	98.68	12			
'B' Main Effects	26.438	1	26.438	4.993	0.049
'AxB' Interaction	19.289	1	19.289	3.643	0.085
'B' x Subjects Within Groups	53.953	10	5.295		

Analysis of Variance for STAI-T

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	1188.848	11			
'A' Main Effects	0.680	1	0.680	0.006	0.941
Subjects within Groups	1188.168	10	118.817		
Within Subjects	112.985	12			
'B' Main Effects	28.172	1	28.172	3.573	0.088
'AxB' Interaction	5.977	1	5.977	0.758	0.404
'B' x Subjects Within Groups	78.836	10	7.884		

Appendix F

Analyses of Significant Profile SD Measures for Groups as a Whole and Heart Rate Reactive Groups

Analysis of Variance for Heart Rate Profile SD (Groups as a Whole)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	867.754	19			
'A' Main Effects	41.680	1	41.680	0.908	0.353
Subjects within Groups	826.074	18	45.893		
Within Subjects	2671.677	20			
'B' Main Effects	2333.594	1	2333.594	150.544	0.001
'AxB' Interaction	59.063	1	59.063	3.810	0.067
'B' x Subjects Within Groups	279.020	18	15.501		

Analysis of Variance for Frontal EMG Profile SD (Groups as a Whole)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	17.462	19			
'A' Main Effects	0.150	1	0.150	0.156	0.698
Subjects within Groups	17.312	18	0.962		
Within Subjects	2.981	20			
'B' Main Effects	0.415	1	0.415	3.172	0.092
'AxB' Interaction	0.212	1	0.212	1.625	0.219
'B' x Subjects Within Groups	2.354	18	0.131		

Analysis of Variance for Heart Rate Profile SD (Heart Rate Reactive Groups)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Groups	480.032	11			
'A' Main Effects	30.141	1	30.141	0.670	0.432
Subjects within Groups	449.891	10	44.989		
Within Subjects	1945.669	12			
'B' Main Effects	1676.180	1	1676.180	98.710	0.001
'AxB' Interaction	99.680	1	99.680	5.870	0.036
'B' x Subjects Within Groups	169.809	10	16.981		

Analysis of Variance for Frontal EMG Profile SD (Heart Rate Reactive Groups)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	13.913	11			
'A' Main Effects	3.082	1	3.082	2.846	0.122
Subjects within Groups	10.831	10	1.083		
Within Subjects	2.338	12			
'B' Main Effects	0.275	1	0.275	1.938	0.194
'AxB' Interaction	0.646	1	0.646	4.563	0.058
'B' x Subjects Within Groups	1.417	10	0.142		

Appendix G

Analyses of Significant Profile Variance Analysis

Analysis of Variance for Frontal EMG Profile S^2 (Heart Rate Reactive Groups)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	F	p
Between Subjects	179.096	11		
'A' Main Effects	45.615	1	3.417	0.094
Subjects within Groups	133.481	10		
Within Subjects	40.530	12		
'B' Main Effects	7.483	1	3.365	0.096
'AxB' Interaction	10.810	1	4.861	0.052
'B' x Subjects Within Groups	22.237	10		

Analysis of Variance for Heart Rate Profile S^2 (Heart Rate Reactive Groups)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	F	p
Between Subjects	2271136.000	11		
'A' Main Effects	213456.000	1	1.037	0.332
Subjects within Groups	2057680.000	10		
Within Subjects	9287037.063	12		
'B' Main Effects	7679491.000	1	81.375	0.001
'AxB' Interaction	663834.063	1	7.034	0.024
'B' x Subjects Within Groups	943712.000	10		

Analysis of Variance for Frontal EMG Profile S^2 (Groups as a Whole)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	F	p
Between Subjects	212.514	19		
'A' Main Effects	8.567	1	0.756	0.396
Subjects within Groups	203.947	18		
Within Subjects	46.532	20		
'B' Main Effects	9.052	1	4.720	0.043
'AxB' Interaction	2.961	1	1.544	0.243
'B' x Subjects Within Groups	34.519	18		

Analysis of Variance for Heart Rate Profile S^2 (Groups as a Whole)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	F	p
Between Subjects	4128510.063	19		
'A' Main Effects	227230.063	1	1.048	0.319
Subjects within Groups	3901280.000	18		
Within Subjects	12774393.125	20		
'B' Main Effects	10831743.000	1	130.078	0.001
'AxB' Interaction	443770.125	1	5.329	0.033
'B' x Subjects Within Groups	1498880.000	18		

Appendix H

Median Split Baseline-Stressor Difference Scores

Median-Split Difference Scores for Frontal EMG Reactivity

Subject Number	Median-Split Difference Score	ZR Assigned Reactive Modality
19	2.0825	PST
4	1.2525	H.R.
7	1.1375	H.R.
12	1.01825	H.R.
3	.7525	EMG
15	.6325	H.R.
14	.5425	H.R.
20	.5125	H.R.
5	.3875	GSR
13	.2425	EMG
	SPLIT	
6	.0125	H.R.
16	-.0075	H.R.
2	-.025	PST
18	-.2025	EMG
11	-.21	H.R.
8	-.262	H.R.
9	-.33	H.R.
1	-.37	PST
10	-.4025	H.R.
17	-2.50	H.R.

Median-Split Difference Scores for PST

Subject Number	Median-Split Difference Score	ZR Assigned Reactive Modality
17	-.77	H.R.
1	-.685	PST
16	-.4525	H.R.
10	-.36	H.R.
9	-.3425	H.R.
12	-.235	H.R.
19	-.13	PST
14	-.08	H.R.
3	-.045	EMG
4	.0525	H.R.
	SPLIT	
7	.205	H.R.
18	.565	EMG
8	.58	H.R.
2	.595	PST
15	.855	H.R.
13	.99	EMG
5	1.455	GSR
20	1.945	H.R.
11	2.525	H.R.

Median-Split Difference Scores for GSR

Subject Number	Median-Split Difference Score	ZR Assigned Reactive Modality
5	-2.725	GSR
20	-1.565	H.R.
12	-1.5225	H.R.
4	-1.3375	H.R.
10	- .525	H.R.
11	.5525	H.R.
18	.715	EMG
2	.845	PST
7	1.12	H.R.
17	1.3075	H.R.
	SPLIT	
15	1.985	H.R.
9	2.4925	H.R.
8	3.1925	H.R.
3	3.8475	EMG
19	4.1474	PST
6	4.5425	H.R.
14	5.0475	H.R.
16	5.6475	H.R.
13	5.6875	EMG
1	8.395	PST

Appendix I

Analysis of Significant Stressor Effects for Groups as a Whole and Heart Rate Reactive Groups

Analysis of Variance for H.R. Stressor 1 (Groups as a Whole)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	2813.547	19			
'A' Main Effects	33.047	1	33.047	0.214	0.646
Subjects within Groups	2780.500	18	154.472		
Within Subjects	849.43	20			
'B' Main Effects	537.305	1	537.305	30.986	0.001
'AxB' Interaction	0.0	1	0.0	0.0	0.999
'B' x Subjects Within Groups	312.125	18	17.340		

Analysis of Variance for PST Stressor 1 (Groups as a Whole)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	305.042	19			
'A' Main Effects	8.569	1	8.569	0.520	0.480
Subjects within Groups	296.473	18	16.471		
Within Subjects	11.908	20			
'B' Main Effects	2.883	1	2.883	5.876	0.026
'AxB' Interaction	0.193	1	0.193	0.393	0.539
'B' x Subjects Within Groups	8.832	18	0.491		

Analysis of Variance for GSR Stressor 1 (Groups as a Whole)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	142.422	19			
'A' Main Effects	15.781	1	15.781	2.243	0.152
Subjects within Groups	126.641	18	7.036		
Within Subjects	90.863	20			
'B' Main Effects	26.797	1	26.797	7.915	0.012
'AxB' Interaction	3.125	1	3.125	0.923	0.349
'B' x Subjects Within Groups	60.941	18	3.386		

Analysis of Variance for H.R. Stressor 1 (Heart Rate Reactive)

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	1814.680	11			
'A' Main Effects	47.180	1	47.180	0.267	0.617
Subjects within Groups	1767.50	10	176.750		
Within Subjects	625.320	12			
'B' Main Effects	519.398	1	519.398	49.058	0.001
'AxB' Interaction	0.047	1	0.047	0.004	0.948
'B' x Subjects Within Groups	105.875	10	10.587		

Appendix J

One way Analyses of Variance for Significant Stressors

One Way Analysis of Variance for H.R. Stressors

Time = Initial Baseline, Stressor 1, Stressor 2, Stressor 3

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Groups	755.313	3	251.771	11.708	0.001
Within Groups	1225.688	57	21.503		
Total	1981.001	60			
Between Subjects					
Subjects Within Groups	5288.375	19	278.335		

One Way Analyses of Variance for Frontal EMG Stressors

Time = Initial Baseline, Stressor 1, Stressor 2, Stressor 3

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Groups	39.609	3	13.203	2.486	0.070
Within Groups	302.688	57	5.310		
Total	342.297	60			
Between Subjects					
Subjects Within Groups	378.688	19	19.931		

One Way Analysis of Variance for PST Stressors

Time = Initial Baseline, Stressor 1, Stressor 2, Stressor 3

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Groups	12.036	3	4.012	8.490	0.001
Within Groups	26.938	57	0.473		
Total	38.974	60			
Between Subjects					
Subjects Within Groups	596.813	19	31.411		

BIBLIOGRAPHY

Alexander, A. B. An experimental test of assumptions relating to the use of electromyographic biofeedback as a general relaxation technique. Psychophysiology, 1975, 12, 656-662.

Alexander, A. B., & White, D. D. Clinical applications of EMG biofeedback. In Gatchel, R. J. & Price, K. P. (Eds.), Clinical applications of biofeedback: Appraisal and status. New York: Permagon, 1979, 112-133.

Almy, T. P. The stress interview: Unfinished business. Journal of Human Stress, 1978, 4, 3-8.

Asterita, M. F., Smolnicky, J. E., & Iatridis, I. P. Effect of caffeinated versus decaffeinated coffee on the voluntary control of muscle tension levels. Proceedings of the Biofeedback Society of America Twelfth Annual Meeting. White Ridge Colorado: 1981, 1-3.

Averill, J. R. Autonomic response patterns during sadness and mirth. Psychophysiology, 1969, 5, 399-414.

Ax, A. F. The physiological differentiation between fear and anger in humans. Psychosomatic Medicine, 1953, 15, 433-442.

Benson, H. The relaxation response. New York: Avon, 1975.

Benjamin, L. S. Facts and artifacts in using analysis of covariance to "undo" the law of initial values. Psychophysiology, 1967, 4, 187-202.

Black, A. H., Cott, A., & Pavloski, R. The operant learning theory approach to biofeedback. In Schwartz, G. E., & Beatty, J. (Eds.). Biofeedback: Theory and practice. New York: Academic, 1977, 89-128.

Blanchard, E. B., Haynes, M. R., Kallman, M. D., & Harkey, L. A comparison of direct blood pressure feedback and electromyographic feedback on the blood pressure of normotensives. Biofeedback and Self-Regulation, 1976, I, 445-451.

Budzynski, T. H. Anxiety and psychological stress: A biofeedback approach. New Jersey: Hoffman-LaRoche, Roche Scientific Series, 1977.

Budzynski, T. H. Biofeedback applications to stress related disorders. The International Review of Applied Psychology, Liverpool: Liverpool University Press, Oct., 1978.

Burish, T. G., Hendrix, E. M., & Frost, R. O. Comparison of frontal EMG biofeedback and several types of relaxation instructions in reducing multiple indices of arousal. Psychophysiology, 1981, 18, 594-602.

Cacioppi, J. T., & Petty, R. E. Electromyographic specificity during covert information processing. Psychophysiology, 1981, 18, 518-523.

Cannon, W. B. Bodily changes in pain, hunger, fear and rage. (2nd Ed.) New York: D. Appleton, 1929.

Carney, P. Psychophysiological stress, biofeedback and migraine therapy. Unpublished doctoral dissertation, University of Alberta, 1981.

Corson, J. A., Schneider, M. J., Biondi, C. G. & Meyers, H. K. Psychophysiological assessment: Toward a general strategy. American Journal of Clinical Feedback, 1980, 3, 52-67.

DeGood, D. E., & Adams, A. S. Control of cardiac response under aversive stimulation. Biofeedback and Self-Regulation, 1976, 1, 373-385.

DeGood, D. E., & Chisholm, R. C. Multiple response comparison of pareital EEG and frontal EMG feedback. Psychophysiology, 1977, 14, 258-265.

Engel, B. T. Stimulus-response and individual response specificity. Archives of General Psychiatry, 1960, 2, 305-313.

Fair, P. F. Biofeedback strategies in psychotherapy. In Basmajian, J. V. (ed.). Biofeedback principles and practice for clinicians. Baltimore: Williams & Williams, 1979, 112-131.

Finn, J. D. A general model for multivariate analysis. New York: Holt, Rinehart & Winston, 1974.

Fisher, L. E., & Winkel, M. H. Time of quarter effect: An uncontrolled variable in electrodermal research. Psychophysiology, 1979, 16, 158-163.

Fridlund, A. J., Fowler, S. C., & Pritchard, D. A. Striate muscle tensional patterning in frontalis EMG biofeedback. Psychophysiology, 1980, 17, 47-55.

Friedman, M. & Rosenman, R. H. Type A behavior and your heart. Greenwich, Conn.: Fawcett, 1979.

Gatchel, R. J. The effect of voluntary control of heart rate decleration on skin conductance level: An example of response fractionalization. Biological Psychology, 1976, 4, 241-248.

Gatchel, R. J. Biofeedback and the treatment of fear and anxiety. In Gatchel, R. J., & Price, K. P. (Eds.). Clinical applications of biofeedback: Appraisal and status. New York: Pergamon, 1979, 148-172.

Gatchel, R. J., Korman, M., Weis, C. B., Smith, D. & Clark, L. A multi-response evaluation of EMG biofeedback performance during training and stress induction conditions. Psychophysiology, 1978, 15, 253-258.

Gatchel, R. J., Hatch, J. P., Maynard, A., Turns, R., & Taunton-Blackwood, A. Comparative effectiveness of heart-rate biofeedback, and systematic desensitization in reducing speech anxiety. Journal of Consulting and Clinical Psychology, 1979, 47, 620-622.

Gilmore, J. V., & Gilmore, E. C. Gilmore oral reading test. New York: Harcourt Brace Jovanovich Inc., 1968. Forms C9, C10, D9, D10.

Glaus, K. D., & Kostes, H. Generalization of conditioned frontalis tension: A closer look. Psychophysiology, 1979, 16, 513-519.

Goldstein, I. B., Grinker, R. R., Heath, H. A., Oken, D., & Shipman, W. G. Study in psychophysiology of muscle tension. I: Response specificity. Archives of General Psychiatry, 1964, 11, 322-330.

Green, E., & Green, A. Beyond biofeedback. New York: Delta, 1977.

Harrison, R. S., & Raskin, D. C. The role of feedback in the control of heart rate variability. Psychophysiology, 1976, 13, 135-139.

Heslegrave, R. J., Ogilvie, J. C., & Furedy, J. J. Measuring baseline-treatment differences in heart rate variability: Variance versus successive difference mean square and beats per minute versus interbeat intervals. Psychophysiology, 1981, 16, 151-157.

Hiebert, B. A. Physiological response patterning: A review. Paper presented to the fourth annual general meeting of the Biofeedback Association of Alberta, 1980, Edmonton, Alta.

Hnatiow, M. & Lang, P. J. Learned stabilization of heart rate. Psychophysiology, 1965, 1, 330-336.

Kilpatrick, D. G. Differential responsiveness of two electrodermal indices of psychological stress and performance on a complex cognitive task. Psychophysiology, 1971, 9, 218-226.

Knight, M. L., & Borden, R. J. Autonomic and affective reactions of high and low socially anxious individuals awaiting public performance. Psychophysiology, 1979, 16, 209-213.

Lacey, J. I. Psychophysiological approaches to the evaluation of psychotherapeutic process and outcome. In Rubenstein, E. A., & Parloff, M. B. (Eds.). Research in Psychotherapy. Washington, D. C.: American Psychological Association, 1959.

Lacey, J. I. Somatic response patterning and stress: some revisions of activation theory. In Appley, M. H., & Trumball, R. (Eds.). Psychological stress: Issues and research. New York: Appleton-Century-Crofts, 1967, 14-37.

Lacey, J. I., & Lacey, B. C. Verification and extension of the principle of autonomic response sterotypy. American Journal of Psychology, 1958, 71, 50-73.

Lacey, J. I., & Lacey, B. C. The law of initial value in the longitudinal study of autonomic constitution: Reproducibility of autonomic responses and response patterns over a four year period. Annals of the New York Academy of Sciences, 1962, 98, 1257-1290.

Lacey, J. I., Bateman, D. E., & Van Lehn, R. Autonomic response specificity: An experimental study. Psychosomatic Medicine, 1953, 15, 8-21.

Lacey, J. I., Kagan, J., Lacey, B. C., & Moss, H. A. The visceral level: Situational determinates and correlates of autonomic response patterns. In Knapp, P. H. (Ed.). Expression of emotions in man. New York: International Univ. Press, 1963.

Lang, P. J., Sroufe, A., & Hastings, J. Effects of feedback and instructional set on the control of cardiac-rate variability. Journal of Experimental Psychology, 1967, 75, 425-31.

Lawler, K. A. Cardiovascular and electrodermal response patterns in heart rate reactive individuals during psychological stress. Psychophysiology, 1980, 17, 464-470.

Lazarus, R. S. A cognitively oriented psychologist looks at biofeedback. American Psychologist, 1975, 553-561.

Light, K. C. Cardiovascular responses to effortful active coping: Implications for the role of stress in hypertension development. Psychophysiology, 1981, 18, 216-225.

Light, K. C., & Obrist, P. A. Cardiovascular reactivity to behavioral stress in young males with and without marginally elevated casual systolic pressures: A comparison of clinic, home and laboratory measures. Hypertension, 1980, 2, 802-808.

Lott, G. G., & Gatchel, R. J. A multi-response analysis of learned heart control. Psychophysiology, 1978, 15, 576-581.

Malmo, R. B. On emotions, needs and our archaic brain. New York: Holt, Rinehart & Winston, 1975.

Malmo, R. B., Shagass, C., & Davis, F. H. Symptom specificity and bodily reactions during psychiatric interview. Psychosomatic Medicine, 1950, 12, 362-376.

Malmo, R. B., & Wallerstein, H., & Shagass, C. Headache proneness and mechanisms of motor conflict in psychiatric patients. Journal of Personality, 1953, 22, 163-187.

Manuck, S. B., & Schaefer, D. C. Stability of individual differences in cardiovascular reactivity. Physiology and Behavior, 1978, 21, 675-678.

Manuck, S. B., Corse, C. D., & Winkelman, P. A. Behavioral correlates of individual differences in blood pressure reactivity. Journal of Psychosomatic Research, 1979, 23, 281-288.

Meichenbaum, D. Cognitive factors in biofeedback therapy. Biofeedback and Self-Regulation, 1976, 2, 201-216.

Miller, N. E. Biofeedback and visceral learning. Annual Review of Psychology, 1978, 29, 373-404.

Miller, N. E., & Dworkin, B. R. Critical issues in therapeutic applications of biofeedback. In Schwartz, G. E. & Beatty, J. (Eds.). Biofeedback: Theory and practice. New York: Academic, 1977, 129-161.

Obrist, P. A., & Light, K. C. Heart rate reactivity to stress as a predictor of systolic blood pressure lability. Psychophysiology, 1979, 6, 336-351.

Oken, D., Grinker, R. R., Heath, H. A., Hertz, M., Korchin, S. J., Sabshin, M., & Schwartz, N. B. Relation of physiologic response to affect expression. Archives of General Psychiatry, 1962, 6, 336-351.

Patton, G. W. R. Instressor consistency of subject differences in autonomic activity. Journal of Psychology, 1969, 72, 190-209.

Pelletier, K. R., Gladman, A., & Mikuriya, T. Handbook of Physiological Feedback. Vol. I. Berkeley, Calif.: Autogenic Systems Inc., 1976.

Porges, D. W. Heart rate variability and decleration as indices of reaction time. Journal of Experimental Psychology, 1972, 92, 103-110.

Porges, S. W., & Raskin, D. C. Respiratory and heart rate components of attention. Journal of Experimental Psychology, 1969, 81, 497-503.

Roessler, R., & Engel, B. T. The current status of the concepts of physiological response specificity and activation. In Lipowski, Z. J., Lipsitt, D. R., & Whybrow, P. C. (Eds.). Psychosomatic medicine: Current trends and applications, New York: Oxford, 1977, 50-57.

Roessler, R., Greenfield, N. S., & Alexander, A. A. Ego strength and response stereotypy. Psychophysiology, 1964, I, 142-150.

Scheffe, H. The analysis of variance. New York: John Wiley & Sons, 1959.

Schwartz, G. E. Voluntary control of human cardiovascular integration and differentiation through feedback and reward. Science, 1972, 175, 90-93.

Schwartz, G. E. The current status of biofeedback. Paper presented to the Vancouver Institute, Vancouver, Feb., 1981.

Sersen, E. A., Clausen, J., & Lidsky, A. Autonomic specificity and stereotypy revisited. Psychophysiology, 1978, 15, 60-67.

Shein, G. F., & Mandel, A. R. Standard deviation as an indicator of self-regulation in biofeedback. Proceedings of the Biofeedback Society of America Twelfth Annual Meeting. Wheat Ridge, Colorado, 1981, 218-221.

Spielberger, C. D. The State-trait-anxiety inventory. Palo Alto: Consulting Psychologists, 1968.

Spielberger, C. D., Gorsuch, R. L., & Lushene, R. F. The State-trait-anxiety inventory. Palo Alto: Consulting Psychologists, 1970. (Manual)

Sroufe, L. A. Learned stabilization of cardiac rate with respiration experimentally controlled. Journal of Experimental Psychology, 1969, 81, 391-393.

Sternbach, R. A. Principles of psychophysiology. New York: Academic, 1966.

Stoyva, J. M., & Budzynski, T. H. Cultivated low arousal-An antistress response? In DiCara, L. V. (Ed.), Limbic and autonomic nervous systems research. Chicago: Plenum, 1974, 369-394.

Townsend, R. E., House, J. F., & Addario, D. A comparison of biofeedback-mediated relaxation and group therapy in the treatment of chronic anxiety. American Journal of Psychiatry, 1975, 132, 598-601.

Walker, B. B., & Sandman, C. A. Physiological response patterns in ulcer patients: Phasic and tonic components of the electrogastrogram. Psychophysiology, 1977, 14, 393-400.

Walter, G. F., & Porges, S. W. Heart rate and respiratory responses as a function of task difficulty: The use of discriminant analysis in the selection of psychologically sensitive physiological responses. Psychophysiology, 1976, 13, 563-571.

Waters, W. F., Koresko, R. L., Rossie, G. V., & Hackley, S. A. Short-, medium-, and long-term relationships among metorological and electrodermal variables. Psychophysiology, 1979, 16, 445-451.

Wenger, M. A., & Cullen, T. D. ANS response patterns to fourteen stimuli. American Psychologist, 1958, 13, 423. (abstract)

Wickramaskera, I. A conditioned response model of the placebo effect: Predictions from the model. Biofeedback and Self-Regulation, 1981, 5, 1-10.

Wolpe, J. Psychotherapy by reciprocal inhibition. Stanford: Stanford Univ. Press, 1958.

Yates, A. J. Biofeedback and the modification of behavior. New York: Plenum, 1980.