

McMASTER  
UNIVERSITY  
LIBRARY



## INSTRUCTED CONTROL OF HEART RATE AND SKIN CONDUCTANCE

PROPERTIES AND MECHANISMS  
OF  
INSTRUCTED HEART RATE AND SKIN CONDUCTANCE CONTROL

by  
TERRANCE DALE WILTON, B.SC.

A Thesis  
Submitted to the School of Graduate Studies  
in Partial Fulfilment of the Requirements  
for the Degree  
Master of Arts

McMaster University

May 1979

MASTER OF ARTS (1979)  
(Psychology)

McMASTER UNIVERSITY  
Hamilton, Ontario

TITLE: Properties and Mechanisms of Instructed Heart Rate and Skin  
Conductance Control.

AUTHOR: Terrance Dale Wilton, B.Sc. (McMaster University)

SUPERVISOR: Dr. L.E. Roberts

NUMBER OF PAGES: viii, 114

## Abstract

Instructed control of skin conductance and heart rate was compared using experimental groups trained to produce changes in one or the other of these responses. Subjects were required to produce increases and decreases in the visceral target with the aid of visual analogue feedback for five training sessions. Differences arising between the groups were necessarily attributable to differences in the neural organization of the target responses, since the groups differed only with respect to the response that was identified by the feedback display.

Instructed control of both targets was demonstrated. Subjects trained on the heart rate target produced both increases and decreases in response level, whereas only control in the increase direction was demonstrated by the skin conductance group. Instructed changes in skin conductance were approximately three times larger than those reported in previous studies of this response.

Performance mechanisms underlying control also differed between the two groups. Increases in heart rate were associated with increases in somatomotor and respiratory activation, but control of skin conductance was manifested in varying physiological contexts.

The bearing of the results on several issues in visceral learning was discussed.

### Acknowledgements

The author wishes to express his appreciation to Dr. L.E. Roberts for his guidance and support in the preparation of this thesis. Without his aid and instruction this thesis could not have attained its present form. Any errors or omissions are totally the responsibility of the author and do not reflect upon the supervision and assistance of Dr. Roberts.

Gratitude is also extended to Mr. Richard Marlin and Mr. John Lyons who provided computer and invaluable electrophysiological expertise in the establishment and maintenance of the experimental environment. Appreciation is also extended to Beverly Pitt for the final typing of this manuscript.

Special gratitude is reserved for my wife, Mary, for her patient support throughout the experimentation, data analysis and writing of this thesis. Without this support the completion of this thesis would have been beyond the realm of possibility.

## Table of Contents

	<u>Page</u>
CHAPTER ONE INTRODUCTION.....	1
CHAPTER TWO METHOD.....	19
CHAPTER THREE RESULTS.....	33
CHAPTER FOUR DISCUSSION.....	76
APPENDICES.....	88
REFERENCES.....	108



### List of Figures

	<u>Page</u>
Figure 1. Control of skin conductance and heart rate in four groups of subjects (N=8 each). (From Lacroix and Roberts, 1978).	9
Figure 2. Response patterns observed in groups instructed to control either heart rate or sudomotor activity. (From Lacroix and Roberts, 1978).	13
Figure 3. Feedback Display.	21
Figure 4. Control of heart rate within trials on the last day of the experiment.	34
Figure 5. Acquisition of instructed control of heart rate across the five experimental sessions.	37
Figure 6. Changes in heart rate on Training, Test and Blank Trials within each session.	39
Figure 7. Response patterns associated with control of heart rate on Training and Blank Trials of the last day of the experiment.	41
Figure 8. The response patterns produced by heart-rate subjects on Increase Training trials are given for the five experimental sessions.	44
Figure 9. The response patterns produced by heart-rate subjects on Decrease Training Trials are given for the five experimental sessions.	46

Figure 10. Spearman rank-order correlations relating	49
in heart rate and skin conductance with changes in	
muscle tension, movement, temperature, and respiratory	
frequency are given for the three subjects trained to	
control heart rate.	
Figure 11. Control of skin conductance within-trials on	52
the last day of the experiment.	
Figure 12. Acquisition of instructed control of skin	54
conductance across the five experimental sessions.	
Figure 13. Changes in skin conductance on Training, Test	57
and Blank Trials within each experimental session.	
Figure 14. Response patterns associated with control of	59
skin conductance on Training and Blank Trials on the	
last day of the experiment.	
Figure 15. Response patterns are given for the three	61
subjects trained on the skin-conductance target for	
each of the five experimental sessions.	
Figure 16. Spearman rank-order correlations relating	64
changes in skin conductance and heart rate with each of	
the other variables (excluding respiratory amplitude) are	
given for the three subjects trained to control skin	
conductance.	
Figure 17. Changes in heart rate for both target groups	67
across five days of training.	
Figure 18. Changes in skin conductance for both target	70
groups across five days of training.	

Figure 19. Change scores in the concomitant measures  
over five days of training.

72

### List of Tables

	<u>Page</u>
Table 1. Summary of Trials Deleted from Data Analysis	31
Table 2. Verbatim Verbal Reports	75
Table 3. Disposition of the Nineteen Subjects	93

## CHAPTER ONE

### INTRODUCTION

Considerable attention has been directed over the last ten years to the study of the ability of human subjects to comply successfully with verbal instructions to control their visceral responding. While a variety of responses has been studied in this respect, greatest effort has focused upon activities of the cardiovascular and sudomotor response systems (Brener, 1977; Kimmel, 1974; Katkin and Murray, 1968). The dependent variable in studies of sudomotor control has typically been skin conductance or skin potential responding. Cardiovascular studies, on the other hand, have been concerned principally with heart rate, although other measures such as blood pressure (Surwit and Shapiro, 1977), pulse wave velocity (Steptoe, 1977), pulse amplitude (Johnston, 1977), and skin temperature (Roberts, Schuler, Bacon, Zimmerman, and Patterson, 1975) have been investigated as well.

A variety of terms has been used to describe the control that has been evidenced in these experiments. Included among these terms are voluntary control (Brener, 1974a), self-regulation (Blanchard and Young, 1973), instructed control (Lacroix and Roberts, 1978), and discriminative operant conditioning (Black, Cott, and Pavloski, 1977; Kimmel, 1974). The main purpose of these terms is to indicate that subjects have complied successfully with a verbal instruction to change a visceral response. However, a further and more debatable implication

of terms such as voluntary control or self-regulation is that compliance is a volitional act. This view of compliance is incompatible with particular ideas regarding the origin and properties of visceral control. For example, a volitional interpretation of compliance would not be proposed if the response were shown to be an unconditioned reaction to the verbal instructions the subjects received. Nor would a volitional interpretation be suggested if the response were found to depend upon association of the instructions with some reinforcing event (classical conditioning). Although such limitations are clearly implied by a volitional characterization of instructional compliance, the requirements for such characterizations have rarely been specified in the visceral learning literature (Black, 1974; Black et al., 1977; Brener, 1974b).

It is useful to have available a term that designates successful compliance with verbal instructions to change a response without prejudging the origin of visceral control, since in many instances in the literature the origin of compliance cannot be ascertained (for example, see Bell and Schwartz, 1975; Brener, 1974a; Johnston, 1976; Levenson, 1976; Lacroix and Roberts, 1978). However, some minimal specification of the response seems necessary to define the phenomenon under investigation in this area of research. The terminology to be adopted in this thesis is adapted from Lacroix and Roberts (1978) and is as follows. Briefly, an instance of instructed visceral control will be said to have been observed when a subject complies successfully with a verbal instruction to differentially manipulate a visceral response. For example, a subject may be asked to

increase and decrease the response, or to produce a change in responding in one part of the body and then another. The criterion that is intended here is not that responding change precisely as specified by the experimenter but that it differ as a function of the instructional manipulation. This concept of instructed control is based upon the notion that a subject who can be said to control a response ought at least to be able to exert a systematic influence over the course of responding when he is instructed to do so. Thus, according to this concept of instructed control, target responding that occurs as a part of an orienting reaction that is elicited by the receipt of a verbal command does not qualify as an instance of instructed control, unless target responding is shown further to be sensitive to a component of the verbal instructions that requires manipulation of the response. It should also be noted that instances of instructed control that are shown further to be a product of experience with a feedback contingency could also be described as instances of discriminative operant conditioning. Demonstration of operant conditioning was not the central purpose of the experiment to be reported in this thesis, but evidence bearing upon operant conditioning as a source of instructed control is provided.

The experiment to be reported in this thesis was undertaken to compare the properties and mechanisms of instructed changes in heart rate and sudomotor responding. A brief summary of what is currently known about this problem is followed by a description of the present research.

### Instructed Control of Heart Rate

Considerable evidence has accumulated concerning the properties of instructed changes in heart rate. It is known, for example, that subjects can comply successfully with a verbal instruction to increase heart rate in the absence of explicit prior training for this skill (Blanchard and Young, 1972; Brener, 1974a). Performance has been reported to improve as a result of exposure to exteroceptive feedback conditional upon cardiac activity (Blanchard, Scott, Young and Edmundson, 1974; Brener, Kleinman, and Goesling, 1969), although this effect may not be forthcoming in the presence of instructions that bias the subject's choice of response strategy (Lacroix and Roberts, 1978). Increases in heart rate obtained under conditions of exteroceptive feedback have further been reported to vary as a function of (a) whether subjects are told that heart rate is the response to be produced (Blanchard et al., 1974); (b) the compatibility of the required response state with the directional set induced by the feedback display (Shapiro, in press); (c) properties of the feedback display (Gatchel, 1974; Lang and Twentyman, 1974); and (d) whether monetary incentive for successful performance is provided (Lang and Twentyman, 1976).

It has also been demonstrated that subjects are capable of producing decreases in heart rate, when feedback training is given for this response (Engel and Hansen, 1966; Lang and Twentyman, 1974). However, these decreases are of a smaller magnitude than is the case for heart-rate increases (-2 beats per minute, compared to approximately +8 beats per minute for increases). Another difference



between instructed increases and decreases in heart rate is that increases have been shown to transfer to non-feedback conditions whereas decreases have been reported not to do so (Lang and Twentyman, 1974).

The mechanisms underlying the performance of instructed increases in heart rate have been studied by several investigators. The results have indicated that heart-rate increases are typically associated with increases in somatomotor and respiratory function (Brener, 1974c; Blanchard et al., 1974; Lacroix and Roberts, 1978). Furthermore, the magnitude of heart-rate change is sharply reduced when these concomitant activities are constrained by verbal instructions or respiratory pacing techniques (Obrist, Galosy, Lawler, Gaebelein, Howard, and Shanks, 1975). This evidence is consistent with the view that changes in somatomotor and respiratory activity are sufficient and perhaps necessary for the production of substantial increases in heart rate (see Roberts, 1978, for a review).

#### Instructed Control of Sudomotor Activity

Less evidence is available concerning the properties and mechanisms of instructed sudomotor control. Unlike the situation that pertains with respect to heart rate, subjects do not appear able to comply successfully with a verbal instruction to increase or decrease volar sweating in the absence of prior feedback training for this response (Lacroix, 1976). It has been reported, however, that sudomotor activation is increased when subjects are instructed to perform various cognitive and emotional manoeuvres with which sudomotor

responding has typically been associated (Klinge, 1972). The ability to produce increases in sudomotor activation is also established when feedback training is given for electrodermal responding (Crider, Shapiro, and Tursky, 1966; Kimmel, 1967). The increases obtained following feedback training are larger than those observed when subjects are instructed to think emotional thoughts (Klinge, 1972) and have been shown to transfer to test trials on which subjects are instructed to increase the response with exteroceptive feedback removed (Lacroix, 1976). In most instances, the sudomotor control produced by experience with feedback has been found to develop slowly over the course of training (Crider et al., 1966; Johnson and Schwartz, 1967).

The ability of subjects to produce decreases in sudomotor activation has not been widely investigated. Lacroix and Roberts (1978) observed small decrements in skin conductance when subjects were instructed to decrease finger sweating in the presence of exteroceptive feedback. However, they acknowledged the possibility that these decrements may have reflected a continuation of the pre-trial trend and therefore may not have been learned responses. The instructed increases in skin conductance produced by feedback training in this experiment were substantially larger than the observed decreases in this response (Lacroix and Roberts, 1978).

A small literature has addressed the question of how instructed changes in sudomotor activation are achieved. Crider et al. (1966) monitored heart rate, respiration rate, and gross skeletal activity while contingent reinforcement was given for the production or suppression of phasic increases in skin conductance (galvanic skin

responses or GSRs). The effect of reinforcement on electrodermal activity was independent of changes in the other measured variables. The relationship of operantly-conditioned changes in galvanic skin response to changes in muscle activity of the fingers was examined by Rice (1966). There was no indication that the differences between experimental and control groups in the ability to control GSRs could be explained by differences in muscle activity. However, a subsequent attempt to establish operant control by reinforcing only GSRs elicited in the absence of muscle tension responses was unsuccessful. Rice (1966) attributed his failure to insufficient occurrence of the response pattern that was to have been shaped under the conditions of his test.

The aforementioned studies by Crider et al. (1966) and Rice (1966) examined the relationship of sudomotor function to somatomotor and respiratory activity when subjects were discouraged from employing skeletal-muscular or breathing strategies to control the target response. A different approach was used by Gavalas (1968), who reinforced subjects for deep inspirations that were associated with a GSR. The frequency of deep inspirations increased over the course of training, but the GSR associated with the respiratory response habituated as training progressed. Still another approach was followed by Lacroix and Roberts (1978), who measured somatomotor and respiratory concomitants of sudomotor control when response strategies were unconstrained. Bidirectional differences were observed in the concomitant measures, but these were not augmented by feedback training as was the target response. These findings, taken together with

earlier studies by Crider et al. (1966) and Rice (1966), suggest that instructed control of sudomotor activation may not be dependent upon somatomotor and respiratory action (see Kimmel, 1974, for a review).

#### Comparison of Instructed Cardiac and Sudomotor Control

Comparison of the available data pertaining to cardiac and sudomotor control suggests that there are substantial differences between these two response systems. These differences appear to involve properties of the responses as well as the mechanisms by which instructed control of the viscera is achieved.

The possibility of differences in the properties and mechanisms of cardiac and sudomotor control was examined explicitly in the aforementioned study by Lacroix and Roberts (1978). The findings of this experiment are relevant to the current thesis and are depicted in Figure 1. Subjects in Group I/HR were instructed to produce increases and decrease in cardiac activity on discrete 30-sec trials, in the absence of exteroceptive feedback for this response. Target responding was defined to the subjects as "heart rate", but no mention was made of possible response strategies or of explicit performance constraints. Inspection of the right-hand portion of Figure 1 shows that verbal instructions alone (Group I/HR) were sufficient to produce substantial control of heart rate from the outset of training. Addition of binary feedback for successful performance (Group IF/HR) did not improve heart-rate control significantly at any stage of experimentation. These findings are consistent with earlier evidence indicating that experience with an explicit feedback contingency is not necessary for

Figure 1. Control of skin conductance and heart rate in four groups of subjects (N=8 each). Experimental conditions are as described in the text. Bidirectional performance on test trials during which feedback was absent is shown.

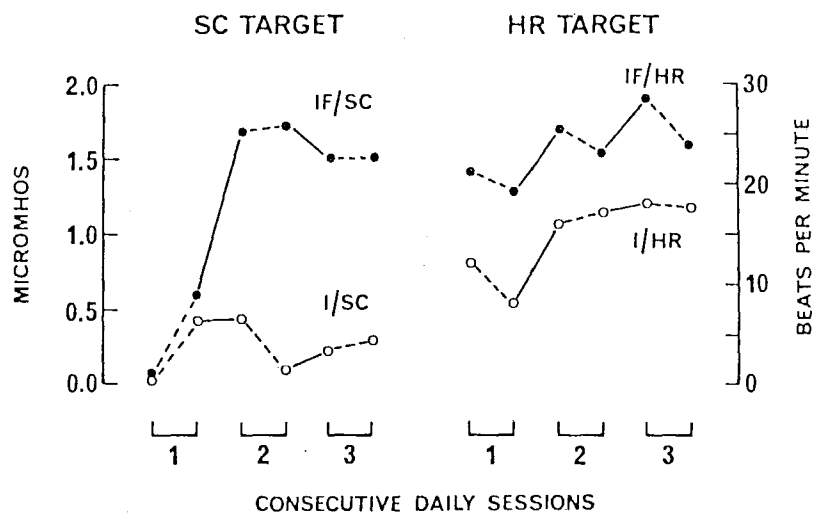


Figure 1.

cardiac control, when a change in heart rate is designated as the behavioural goal (Brener, 1974a).

The properties of instructed control of sudomotor responding, however, appeared to be different. The data are presented in the left panel of Figure 1. Subjects in Group I/SC were instructed to increase and decrease volar sweating, in the absence of exteroceptive feedback for this response. Volar sweating was defined to the subjects as "sweating of the fingers" and was assessed by measuring skin conductance. As before, no mention was made of possible response strategies or performance constraints in the instructions the subjects received. Inspection of the left portion of Figure 1 shows that subjects without prior feedback experience (Group I/SC) were unable to control the target response over three days of testing. Subjects in Group IF/SC, on the other hand, received identical instructions but were given binary feedback when they performed successfully. Comparison of these groups shows that control of skin conductance was apparent in the feedback condition after the second day of training. It appears, therefore, that feedback training contributed more to instructed control of sudomotor activation than to instructed control of heart rate under the circumstances of this study. Subjects were unable to produce bidirectional differences in sudomotor activation without prior feedback training, but such training was not required for, and contributed little to, the performance of instructed changes in heart rate.

Lacroix and Roberts (1978) also examined the mechanisms associated with the performance of instructed sudomotor and cardiac

control. The main findings are given in Figure 2, which depicts the response patterns observed in each training condition on the last day of testing. Inspection of the heart-rate groups in the right hand panels of Figure 2 (Groups I/HR and IF/HR) reveals that substantial changes in somatomotor activity and respiratory function were observed when subjects were instructed to produce increases and decreases in this target response. Lacroix and Roberts also found that two variables that influenced the magnitude of instructed cardiac change - the designation of either sweating or heart rate as the target response and the provision of explicit strategy suggestions - affected the magnitude of respiratory and somatomotor concomitants as well (Lacroix and Roberts, 1978, p. 122-123). These findings are consistent with earlier evidence concerning the mechanisms of operant heart-rate conditioning and suggest that changes in somatomotor and respiratory action may be necessary for substantial control of this response (Obrist et al., 1975; Roberts, 1978).

The response patterns associated with sudomotor control are shown in the left hand panels of Figure 2 (Groups I/SC and IF/SC). Statistical analyses of these data revealed that bidirectional differences in gross body movement and respiration amplitude were present when skin conductance was designated as the target response. However, changes in these concomitant activities were of small magnitude and were not augmented by feedback training, as was the sudomotor target. These findings indicate that augmentation of concomitant changes in somatomotor and respiratory function may not be necessary for augmentation of skin-conductance control by feedback training, when



Figure 2. Response patterns observed in groups instructed to control either heart rate or sudomotor activity. Experimental conditions are described in text. Bidirectional performance on test trials of the last day of training is shown. SC = skin conductance in micromhos, HR = heart rate in beats per minute; GBM = gross body movement in arbitrary units; RF = respiration frequency measured as respiratory period in milliseconds (negative up); RA = respiratory amplitude in arbitrary units; EM = eye movement in arbitrary units.

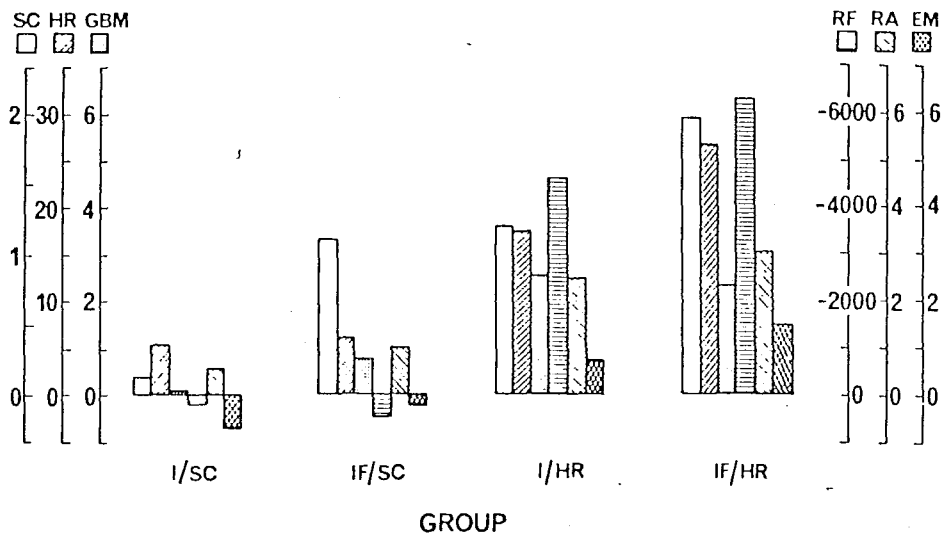


Figure 2.

a change in volar sweating is designated as the behavioural goal.

It should be noted that the differences that were evident in the properties and mechanisms of cardiac and sudomotor control in the Lacroix and Roberts study, and in earlier research relevant to this topic (cf. Obrist, 1975; Crider et al., 1966), may have derived from one of two sources. The first possibility is that these differences may have been attributable to differences in the neural organization of the two response systems. The response patterns associated with the sudomotor and cardiac targets in the Lacroix and Roberts (1978) and earlier studies are consistent with this view, since prior evidence has suggested that these two visceral responses differ in the extent of their coupling with movement control mechanisms (Roberts, 1974). However, a second possibility is that differences between the responses may have derived from differences in the procedures that were used to assess sudomotor and cardiac control. Such procedural differences, which pertain mainly to instructional conditions and feedback procedure, are numerous when comparisons are made across studies that deal with only one target response (for example, compare Crider et al., 1966, with Obrist et al., 1975 or Levenson, 1976). The problem exists in the Lacroix and Roberts experiment as well, in which subjects were instructed to produce increases and decreases in either "sweating of the fingers" or "heart rate". Comparison of Groups I/SC and I/HR in Figure 2 reveals that the effect of referring to one of these two targets was persistent and surprisingly large. Consequently, it is possible that somatomotor correlates failed to materialize in the sudomotor condition, not because these activities do not contribute to

sudomotor control, but because reference to volar sweating as the visceral target may have led subjects to employ response strategies that did not involve generalized somatomotor or respiratory change.

Exploration of the extent and basis of differences in the properties and mechanisms of visceral control is relevant to several issues in visceral learning. Study of these differences provides information concerning the generality of principles of visceral self-regulation. It also provides information concerning the nature of differences in the neural organization of visceral control systems. Finally, there is reason to suggest that an exploration of differences in the properties and mechanisms of visceral control may provide insight into the question of how subjects learn to control a visceral effector as a consequence of experience with a feedback contingency (Roberts and Marlin, in press). The experiment to be reported in this thesis was therefore undertaken to explore the basis and extent of differences in sudomotor and cardiac control, and to provide information on these more general issues.

### The Present Research

There were four objectives of the present experiment. The first was to provide further information regarding several properties of instructed changes in sudomotor and cardiac responding. Comparison of the present results with earlier findings by Lacroix and Roberts (1978) was expected to provide information concerning (a) the effects of extended training on the control of heart-rate and sudomotor responding; (b) the possibility of differentiated visceral responding

on decrease trials; (c) an effect of feedback procedure, or of reference to a particular response, on instructed visceral control; and (d) some appreciation of the stability of individual performance with continued training and the extent of individual differences in response pattern under each target condition.

The second goal was to explore further the mechanisms by which instructed changes in these visceral effectors is achieved. This objective was attempted by examining the response patterns associated with instructed changes in sudomotor and cardiac activity. Continuous analogue feedback was employed in an effort to accentuate effects deriving from the organization of the sudomotor and cardiovascular systems. The basis of this expectation was that analogue feedback would provide greater information concerning changes in the target response and correlated activities than would the discontinuous binary feedback that has been used in much of the previous work on these response systems (Klinge, 1972; Lacroix and Roberts, 1978; Obrist et al., 1975).

The third goal of the present research was to provide information on the basis of differences between the target systems in the properties and mechanisms of instructed visceral control. Consequently, comparison across the target conditions was carried out when all aspects of training were identical except for the response that was identified by the feedback contingency. Differences that materialized under these conditions were necessarily attributable to differences in the organization of the sudomotor and cardiac control

systems, since there was no other means by which to explain differences between the target groups.

A final goal of the present work was to develop a computer-directed environment that could subsequently be used to investigate a range of problems relating to instructed and operant visceral control. Priority was given to (a) the automation of verbal instructions and training procedure, including trial sequencing and feedback display; (b) standardization of subject preparation including pre- and post-experimental interviews and electrode configuration; and (c) computerization of data collection and analysis. The purpose of this effort was not only to expedite the collection and analysis of data in subsequent studies, but also to build into the research program a degree of control that would facilitate replication of basic findings.

The procedure that was devised during the course of this work is described in Chapter 2. Experimental findings are presented in Chapter 3 and discussed in Chapter 4. Chapter 4 also considers some problems for future research.

## CHAPTER TWO

### METHOD

#### Subjects

Twenty-one male volunteers between the ages of 18 to 37 years were screened in a brief medical interview to establish their suitability for participation. The details of this procedure and the criteria for exclusion are reported in Appendix A. Two volunteers were excluded on the basis of the interview, one due to a heart murmur and the other because of high blood pressure.

Five sessions of training were completed for each of the remaining 19 volunteers. Of these, ten who were tested primarily in the early stages of experimentation were dropped from the data analysis because of artifact on the target channel that seriously distorted the feedback display. This problem was overcome for the remaining subjects by (1) improvements in electrode placement and preparation; (2) development of electronic circuitry that discriminated the R-wave of the electrocardiogram from attending electromyographic artifact; and (3) alteration of system software to detect and reject artifactual signals (a change in successively sampled signals of greater than 1 volt) on both the skin-conductance and heart-rate channels.

Three additional subjects were rejected as well. In one instance, the subject (whose native tongue was not English) failed to understand the tape-recorded instructions. In the second case, intervention by the experimenter was required to keep heart rate below

the acceptable maximum for the task (150 beats per minute). In the last instance, the subject fell asleep during the fourth session of training. A summary of the technical problems encountered in each of the total sample of 19 subjects is given in Appendix B.

These exclusions reduced the sample to six subjects, each of which was included in the analyses reported herein. Three of these subjects were trained to control heart rate and three were trained to control skin conductance. Subjects were paid \$2 an hour for their participation. In addition, they received bonus money conditional upon their performance, to a maximum of \$1 per day.

#### Apparatus

During the experimental session the subject was seated in an electrically shielded, sound deadened room. Ambient light level was dimmed and the walls were curtained and the floor carpeted to provide an undistracting environment. A padded chair, fixed in a non-reclining position, was provided for the subject.

Visual feedback was provided by a Sony Model 110 videomonitor with a screen size of 18 by 23 cm. This monitor was situated approximately 1.2 meters in front of the subject at eye level. The feedback display, illustrated in Figure 3, consisted of a vertical line which varied in length proportional to the difference between the current value of the target response (skin conductance or heart rate) and the last recorded value during the pretrial period. A fixed horizontal line represented the starting point at the beginning of the trial. The sensitivity of the feedback display was set before the onset of the first training trial and approximated 1 micromho/cm for



Figure 3. Feedback Display. The vertical line denoted the direction and magnitude of the change in response level from its value at the end of the pretrial period. Movement of the vertical line toward the top of the screen denoted increases in response level; movements toward the bottom denoted decreases. The magnitude of the change was represented by the length of the vertical line. The horizontal line was fixed and represented the response value at the end of the pretrial. The feedback display was updated every 250 milliseconds.

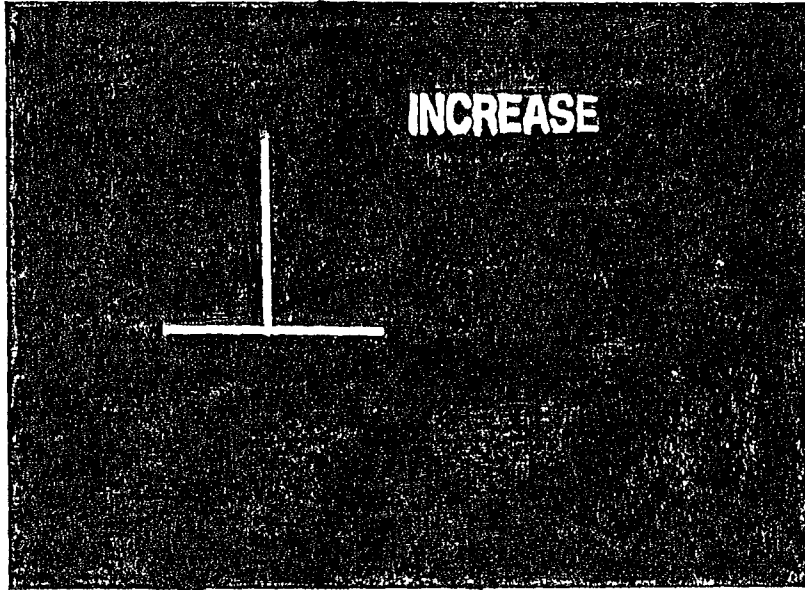


Figure 3.

skin-conductance subjects and 20 bpm/cm for heart-rate subjects. This sensitivity remained constant throughout the five sessions. The words "INCREASE" and "DECREASE" were also presented on the video display.

Trial sequences and the feedback display were administered by a PDP-8/L computer. Additionally, five channels of electrophysiological data were fed into the computer through an analog-to-digital converter and stored on Cartrifile tape for future analysis. The feedback display was generated by a computer C118 Display Interface and a Textronix 4501 Scan Converter.

Electrophysiological data were also monitored throughout the experimental session by a Beckman Type R polygraph operating at a paper speed of 1mm/second.

#### Electrophysiological Recording

Bilateral skin conductance was recorded from the hypothenar eminence using Beckman bipotential Ag/AgCl skin electrodes 15 mm in diameter. The active and reference electrodes were prepared with a paste consisting of 1-part Parke and Davis Unibase mixed with 3-parts .1M NaCl (by volume). Surface contact was through an adhesive collar with a 3/8 inch diameter opening. Prior to the application of the electrodes, active and reference sites were cleaned with alcohol. The reference site, located on the ventral surface of the wrist, was also abraded with a medium grade sandpaper and rubbed with Beckman Electrolytic paste to lower epidermal resistance. Skin conductance was measured as the current generated by a 500mv DC source applied between the active and reference sites through a series resistance of 2 K-ohms.

A Beckman AC/DC coupler (9806A) set on the DC mode was used to make the recordings. A calibrated zero-suppression circuit was used to suppress and retain the tonic level.

Beckman Ag/AgCl electrodes placed over the upper sternum and the lower left rib cage were used to measure the electrocardiogram. These electrode sites were prepared in the same manner as the skin-conductance reference site except that the skin was not abraded each day if flushing from the previous preparations was evident. Beckman Electrode paste was used as the electrode medium. A beat-by-beat measure of heart rate was provided by a cardiometer (Beckman 9857B). The analog output of this device was amplified (X 5) and fed to the computer to provide a continuous measure of this response. The raw electrocardiogram was also recorded through a Beckman AC/DC coupler (9806A) set to an RC constant of 0.03 seconds.

Respiration was recorded by means of a mercury filled strain gauge (Parks Electronics Laboratory) encircling the subject's upper torso. A Beckman mercury gauge coupler (9875B) measured the expansion of the gauge on each respiratory cycle.

Forearm electromyographic activity was recorded through Beckman Ag/AgCl skin electrodes placed over the ventral surface of the forearm as described by Lippold (1967). The preparation of the electrode sites was the same as that for the heart-rate sites. The signal from the forearm electrodes was fed through a Beckman AC/DC coupler (9806A) set to an RC constant of 0.03 seconds with an amplifier gain of 40mv/cm. Preamplifier output was amplified (X 50) and rectified and integrated by a Beckman 9873B integrator coupler (2 mv/cm; IC = 1; TMW = 3.0).

Gross body movement was measured using an inflatable cushion fixed in the seat of the subject's chair. The air valve of the cushion was connected to a Beckman pressure coupler (9853A). The cushion was inflated to 25 mmHg and the coupler calibrated to 1 mmHg/mv. Preamplifier output was amplified (X 5) and rectified for integration by a Beckman 9873B integrator coupler (50 mv/cm; IC = 1; TMW = 3.0).

Skin temperature was measured through two thermistor probes (YSI 429) attached to the middle of the palm of each hand with medical adhesive tape. The signal from the probe was recorded through a Beckman thermistor coupler (9858).

For the final five subjects Beckman Ag/AgCl mini-electrodes were attached to the head as dummy electrodes. Both sites were cleaned with alcohol before the adhesive collar and electrode were applied. One site was located behind the right ear, the other on the extreme upper left forehead. The electrodes were kept shorted during the experiment and were not used for recording purposes.

The polygraph recorded the following data during the experimental session: bilateral skin conductance, heart rate, respiration, electrocardiogram, gross body movement, and forearm electromyographic activity and skin temperature from the target limb. The target limb, which was chosen on the first day and retained subsequently throughout all five sessions, was the side which provided the best skin-conductance signal on the first day of training. Measurements of skin conductance, heart rate, respiration and skin temperature were subject to post-amplification (X 5) prior to being fed through the analog-to-digital converter into the computer. Integrator

resets for electromyogram and gross body movement were recorded by digital buffers on the PDP-8/L.

### Procedure

The subject was assigned to one of the target groups prior to his arrival at the laboratory. This assignment was carried out randomly by an experimenter (designated herein as E1) who had no direct contact with the subject. A second experimenter (the author, designated herein as E2) prepared the subject for testing. E2 was blind with respect to target condition, as was the subject himself.

After a brief intake interview (Appendix A), the subject was told:

- We are going to teach you to control a physiological response that is not normally thought of as being controlled voluntarily.
- We are not going to tell you what the response is because we have reason to believe this might interfere with your performance.
- On the final day of the experiment we will explain the experiment to you and answer any questions concerning it.
- Each day we will attach a number of recording devices to the surface of your body. There will be no needles, no risk.
- It is best if we keep our conversation to a minimum during the course of the experiment.
- You will be paid bonus money in addition to your daily earnings.

-- It is important that you attend each session. You will be paid your entire earnings on the final day.

After the subject had been prepared with electrodes and seated in the experimental chamber, the following procedure was carried out to test for electrophysiological artifact. These statements were read by E1 over an intercom from an adjoining room while E2 demonstrated for the subject.

-- For the first fourteen subjects:

"I am going to check to make sure the recordings are OK. Would you move about in the chair? (pause) Make a fist with both your hands. (pause) Close your eyes and then open them again. (pause) Take a couple of deep breaths."

-- For the final five subjects:

"I want to check to make sure the electrodes are firmly attached. Terry will show you what I want you to do. First make a fist with both hands, raise your arms off the chair and shake them a little (pause). Fine. Now, close your eyes and shake your head. (Pause) Fine. The experiment will begin in a minute."

Tape recorded instructions were then played over the intercom.

For the first day, these were as follows:

In this experiment we are going to try to teach you to control a physiological response that is not usually thought of as being controlled voluntarily. We are not going to tell you what the response is, because we have reason to believe this will interfere with your performance. At designated times during the course of the session we will ask you to increase this response by displaying the word "INCREASE" on the videomonitor in front of you. Similarly, when the word "DECREASE" comes on, you are to decrease the response. During those times when there is no instruction word on the monitor you should sit quietly and wait for the next trial.

To help you perform this task, we will give you feedback on some trials to show you how well you are doing. Here is an example of what the feedback display looks like. The horizontal line represents your starting point at the beginning of the trial. The vertical line, on the other hand, shows changes in the response. Movements of the vertical line toward the top of the screen correspond to increases in responding. Movements of the vertical line toward the bottom of the screen correspond to decreases in responding. Your task is to keep the vertical line above the horizontal line on increase trials, and to keep it below the horizontal line on decrease trials.

There will be some trials on which feedback will be given but you will still be instructed by the videomonitor to increase or decrease the response. On these trials we want you to control the response as best you can without feedback.

Feel free to use any method you wish to control this response but please do not get out of the chair or touch the electrodes. If you need to talk to us during the experiment you can do so simply by speaking out loud. We will hear you over the intercom and will reply if we think a reply is necessary.

To provide extra incentive, we are going to pay you bonus money for responding correctly. You could earn as much as an extra dollar for every session of the experiment, were you to respond correctly all of the time. We will tell you how much bonus money you have earned at the end of each session.

If you would like to have these instructions repeated would you please tell us now. Otherwise, we will begin the experiment in two or three minutes. Good luck.

Instructions for subsequent days were as follows:

The procedure for this session will be the same as for the last session. You may use any method you wish to perform the task but do not get out of the chair or touch the electrodes. If you need to, you can speak to us through the intercom. Remember, the amount of bonus money you earn depends upon how well you perform. Good luck.

Each experimental session consisted of 32 trials lasting 30 sec each. Twenty of these trials were "Training Trials" during which both visual analogue feedback and an instruction word (INCREASE or DECREASE) were presented. Training Trials were administered in a random sequence of ten INCREASES and ten DECREASES. Preceding and following this block of 20 Training Trials was a block of four "Test Trials" (2 INCREASE and 2 DECREASE) on which the instruction to increase or decrease was presented but no feedback was available. Also included in each test



block were two "Blank Trials" (extended inter-trial intervals) during which neither an instruction word nor feedback was presented to the subject. The interval between trials was variable and averaged 1 minute. Each trial was preceded by a 30 sec pretrial period during which neither feedback nor instruction words were presented. All physiological data were monitored and recorded throughout the pretrial and trial periods. A summary of trial sequences is given in Appendix C.

Immediately following the fifth session of training, the subject was asked to fill out a questionnaire. This questionnaire, given in Appendix D, asked subjects to describe what they did to control the feedback display on Increase and Decrease Trials. Answers were examined by E2 to determine whether clarification of handwriting was necessary, but were not otherwise reviewed. The subject was then paid and debriefed. He was also reminded not to discuss details of the study with others in case they should be recruited as subjects.

#### Data Analysis

The polygraph records of each subject were inspected on a trial-by-trial basis for artifact by E2. If on a given trial there was artifact on either of the skin-conductance or heart-rate measures that trial was completely deleted from analysis; 6.1% of the trials were deleted for this reason. If only respiratory measures were obscured by artifact the other measures were retained and the respiratory variables were deleted from analysis for that trial; 7.6% of the trials included in the data analysis were without respiratory measures for

this reason. Similarly, if gross body movement or electrogram were affected by equipment artifact those measures were deleted from analysis for that trial but the autonomic and respiratory variables were retained; 3.4% of the trials were without the somatomotor measures for this reason. Deletions for each of the six subjects are summarized in Table 1.

Bilateral skin conductance and skin temperature from the target limb were measured every 250 milliseconds. Heart rate was measured every 125 milliseconds. These measurements were made throughout the trial and a 30-sec pretrial period. These data were used to compute 5-sec averages spanning the pretrial and trial periods. Change scores were also calculated by subtracting pre-trial measures from trial measures.

Change scores for electromyographic activity and gross body movement were calculated directly from the polygraph records. The number of integrator resets during the pretrial period was subtracted from the number of resets during the trial period to obtain these scores.

Respiratory amplitude and respiratory frequency were also scored by inspection of the polygraph records. Change scores for respiratory frequency were calculated by subtracting the number of  $360^\circ$  oscillations of the polygraph pen during the pretrial period from the number of oscillations during the trial period. In addition, increases and decreases in respiratory amplitude were determined by comparing the amplitude of the respiratory cycles in the pre-trial period with those in the trial period. Each trial was scored as a "+1", "0" or "-1" to

Table 1

Summary of Trials Deleted from Data Analysis.

Autonomic Variables (SC, HR, Temp)		Respiratory Variables (Freq. and Ampl.)	Somatomotor Variables (EMG, Movement)
<hr/>			
Skin Conductance Subjects			
KM	3.1%	2.6%	0.0%
RP	3.1%	2.6%	3.2%
DH	1.9%	3.2%	2.5%*
<hr/>			
MEAN	2.7%	2.8%	1.9%
<hr/>			
Heart Rate Subjects			
RS	11.3%	16.9%	4.2%
PT	15.0%	11.8%	5.1%
KK	1.9%	8.3%	5.1%
<hr/>			
MEAN	9.4%	12.2%	4.8%
<hr/>			

\* This excludes loss of integrated EMG throughout the final two sessions for subject DH. Inspection of the raw EMG indicated that there was very little activity on this channel on these two days. This was true for sessions 1 - 3 for this subject as well when the EMG integrator was functional.

designate an increase, absence of change, or a decrease in amplitude respectively. These numeric values were summed across trials to provide a single measure of change in respiration amplitude on Increase and Decrease Trials in each session.

Because of the small number of subjects which could be included in the data analysis no inferential statistical analysis was undertaken. The results presented in the next chapter are based on a visual inspection of the data.

## CHAPTER THREE

### RESULTS

The experimental results are presented in three sections. An analysis of the performance of the group trained on the heart-rate target appears first and is followed by a parallel analysis of the skin-conductance group. After this the performance of the two groups is compared.

#### Heart-Rate Group

Changes in heart rate within trials on the last (fifth) day of training to control this response are shown in Figure 4. The mean heart rate during the pretrial period has been subtracted from all trial and pretrial values for each trial type so as to depict heart-rate performance as a change from a common baseline. Comparison of heart-rate performance on Training and Test Trials with performance on Blank Trials shows that both increases and decreases in heart rate were produced. Furthermore, transfer from Training to Test Trials was evident under both directional conditions, although it was not as complete in the case of increases as it was in the case of decreases. Heart-rate increases tended to be larger than heart-rate decreases. By the end of the 30 sec trial period, increases averaged approximately 15 beats per minute, whereas decreases averaged about -8 beats per minute. Both responses appeared to have reached asymptote by the end of the trial stimulus.

Figure 4. Control of heart rate within trials on the last day of the experiment. Performance with feedback (Training Trials) and without feedback (Test Trials) is compared to the Blank Trial baseline. Also reported is the mean heart rate in beats per minute during the pre-trial period.

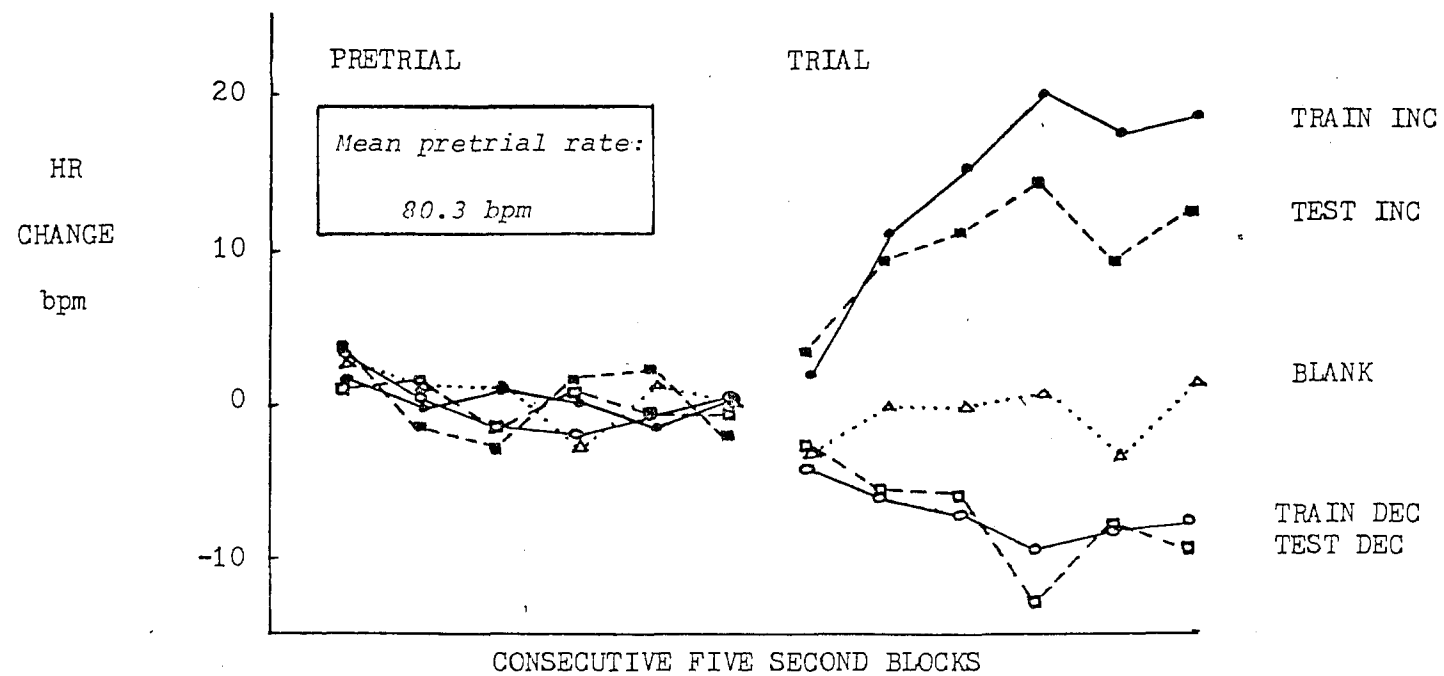


Figure 4.

Figure 5 depicts the acquisition of heart-rate control over the course of five days of training. The data at the extreme left of this figure are taken from the first block of Test Trials on the initial day of training and show that some ability to increase heart rate existed before training began. However, decreases in heart rate were not evident at this point. Introduction of exteroceptive feedback improved increase control in the first session, but subsequent improvements for increase Training and Test Trials were not observed over the course of training. Comparison of Decrease and Blank Trials shows that control in this direction developed more slowly and was present on Training Trials of the third day. Transfer of decrease control was not evident until the last two days of testing.

The course of performance on the heart-rate target within each training session is depicted in Figure 6. Sizeable increases were evident on the first Training Trial of the first day with little systematic improvement thereafter. As suggested above, decrease control was not apparent on the first day of training but was present by the final two days on both Training and Test Trials.

The response patterns accompanying heart-rate control on Training Trials of the final day, averaged across the three heart-rate subjects, are given in Figure 7. Data for Increase Training Trials are given in the right panel of this figure with Blank Trial data in the middle panel and performance on Decrease Training Trials at the left. Sizeable changes in skin conductance, skin temperature, electromyographic activity, and movement accompanied the heart-rate increases. Changes in respiratory function (frequency and amplitude)



Figure 5. Acquisition of instructed control of heart rate across the five experimental sessions. Pretest data were taken from Test Trials given before feedback training on Day 1.

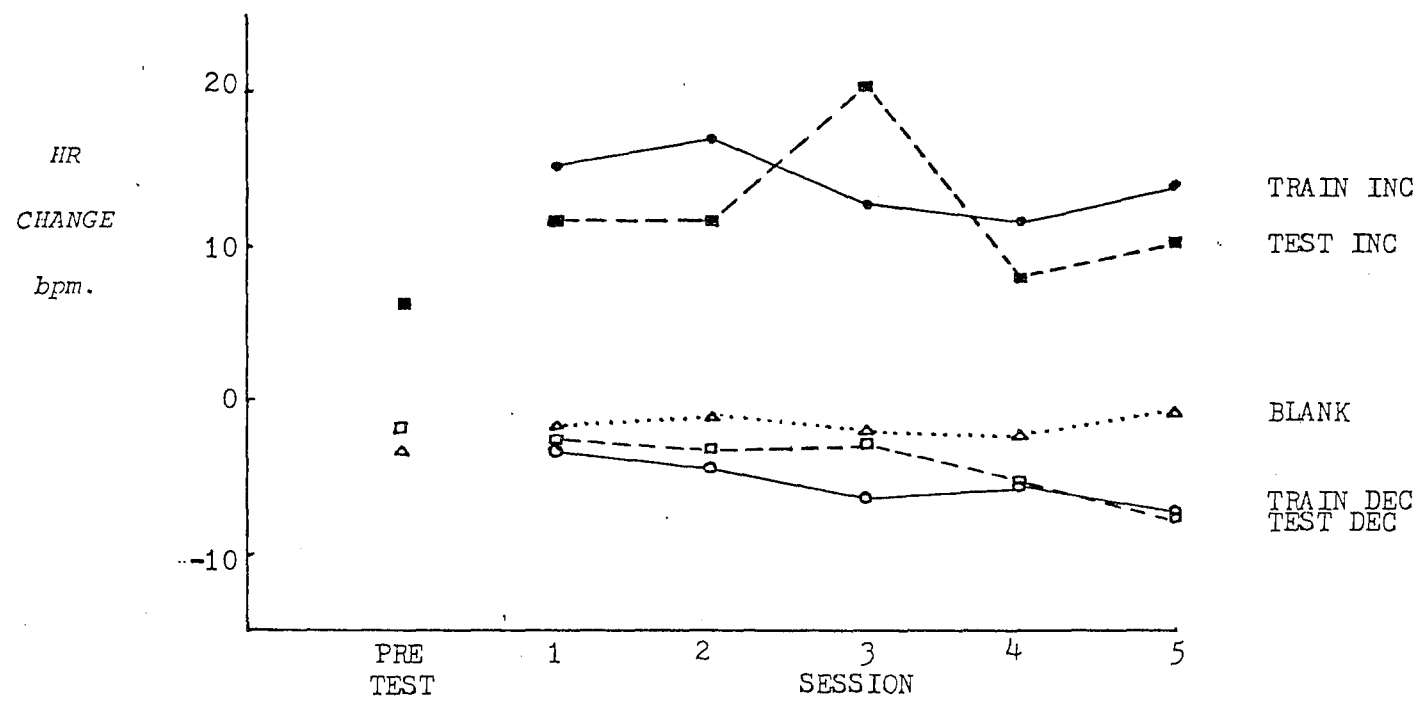


Figure 5

Figure 6. Changes in heart rate on Training, Test and Blank Trials within each experimental session.

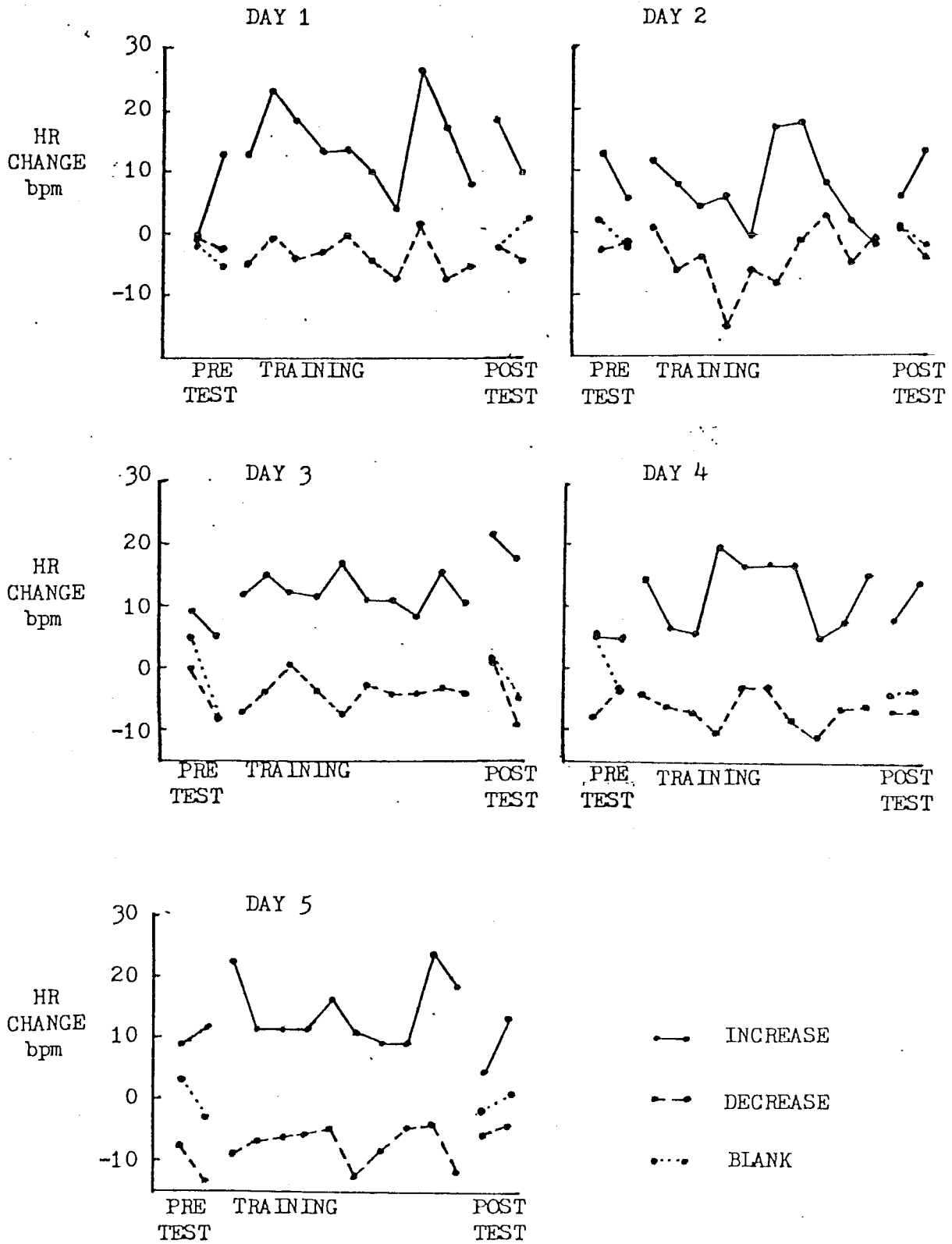


Figure 6.

Figure 7. Response patterns associated with control of heart rate on Training and Blank Trials of the last day of the experiment. SC = skin conductance in micromhos HR= heart rate in beats per minute; TEMP = skin temperature in degrees celsius; EMG = electromyographic activity in arbitrary units; MVT = gross body movement in arbitrary units; RF = respiratory frequency in cycles per 30 sec; RA = respiratory amplitude (proportion of trials on which the respiratory amplitude increased during the trial minus the proportion of trials on which respiratory amplitude decreased during the trials).

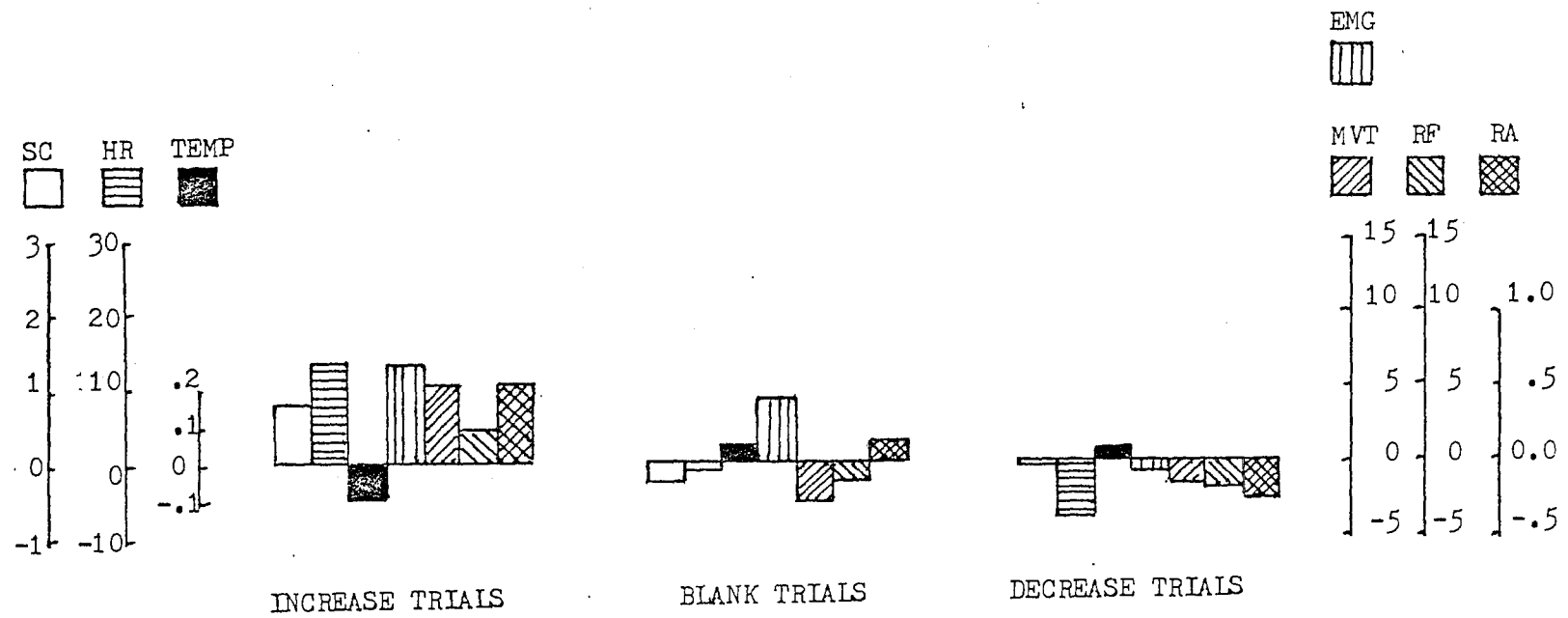


Figure 7 .

were also present on Increase Trials. Decrease heart-rate control was accompanied by decreases in all variables except for skin temperature and skin conductance. Heart-rate decreases were larger on Decrease Training Trials than on Blank Trials, and the direction of changes and the concomitant activities was also more consistent on Decrease Training Trials than on Blank Trials.

The response patterns generated by individual subjects on Increase Training Trials are given in Figure 8 for each of the five experimental sessions. Inspection of these data show that one subject, KK, evidenced little control of heart rate or changes in the concomitant variables during the first three days of training. On the final day of training this subject showed a larger heart-rate increase, although this increase was still modest compared to the control produced by other subjects; the changes in gross body movement and electromyographic activity seen on this day were also the largest produced by subject KK. The other two subjects demonstrated more substantial heart-rate control and also produced larger changes in the concomitant variables. The most consistent finding across all three subjects is that large changes in heart rate were not observed unless concomitant changes in somatomotor activity and respiratory function were also present.

The individual response patterns associated with decreases in heart rate are given in Figure 9. No particular response pattern appears to have been associated with this control, although decreases in respiratory amplitude were of the largest magnitude in the case of KK, the subject who also tended to show the largest heart-rate

Figure 8. The response patterns produced by heart-rate subjects on Increase Training Trials are given for the five experimental sessions. Response measures are as defined in Figure 7.



Figure 8.

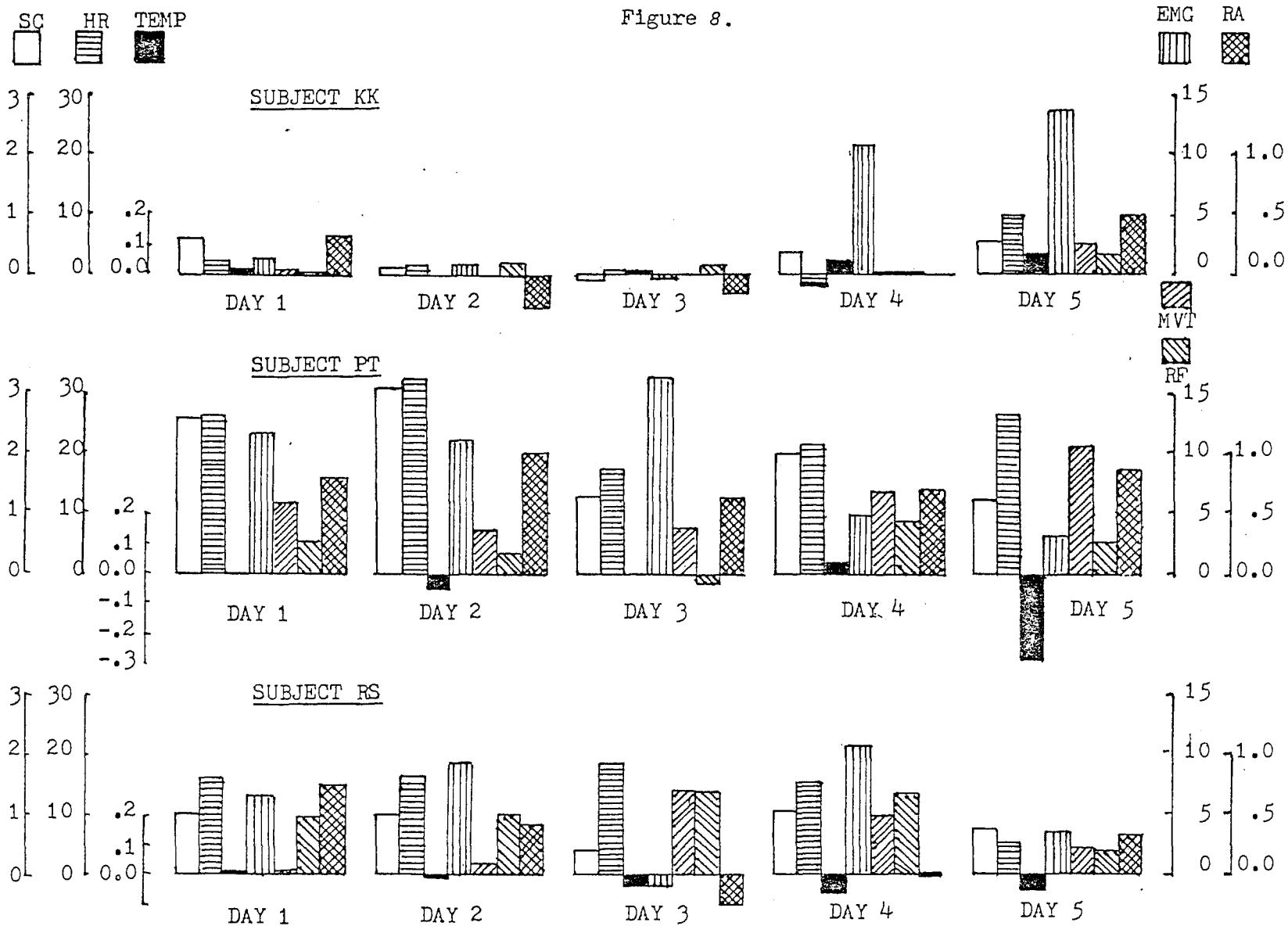
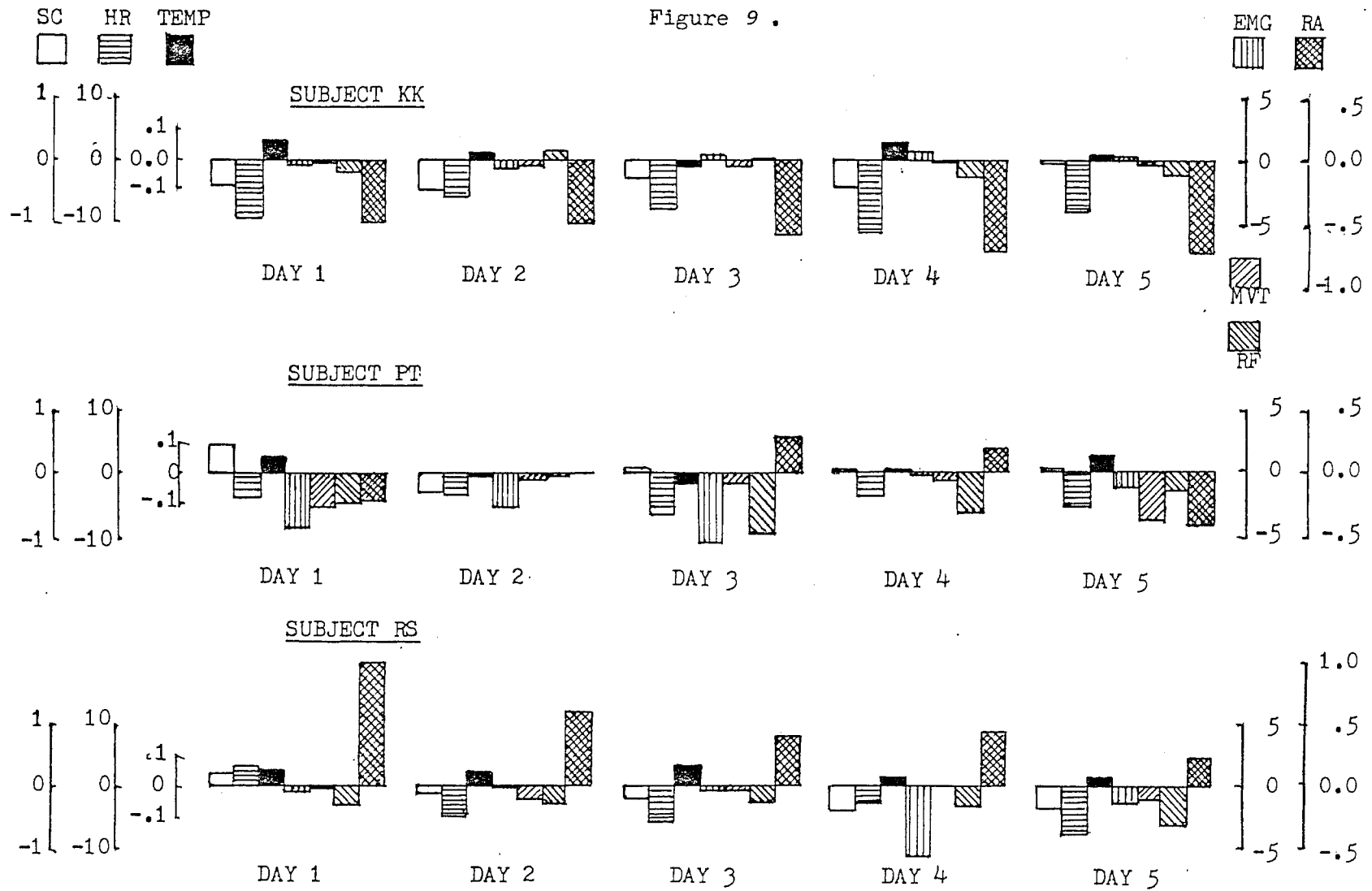


Figure 9. The response patterns produced by heart-rate subjects on Decrease Training Trials are given for the five experimental sessions. Response measures are as defined in Figure 7.

Figure 9 .



decreases. It is also possible to discern a relationship between heart-rate decreases and the disappearance of a paradoxical increase in respiratory amplitude, in subject RS. Further findings of possible note include a decrease in respiratory frequency over days (subject RS) and the presence of decreases in movement in two of the three subjects tested (PT and RS).

The relationship of changes in heart rate to the other responses was examined further by computing within-subject correlations (Spearman rank-order) among the responses observed on Training Trials of the last day of the experiment. These correlations, which were calculated for Increase Trials only, are reported in Figure 10. PT was the only subject who showed significant correlations between heart rate and the other variables. In his case, heart-rate change was positively correlated with changes in skin conductance, electromyographic activity, and gross body movement, and negatively correlated with changes in skin temperature. It is noteworthy that PT, the subject who demonstrated the greatest heart-rate control, is the only subject that showed significant correlations between the target and other responses. The heart-rate changes seen in this subject appear to have been more closely related to somatomotor variables (gross body movement and electromyographic activity) than to respiratory function. It should be noted that the negative correlation with skin temperature may also have a somatomotor basis, since constriction of the peripheral vasculature is a predictable concomitant of increased somatomotor responding (Forsyth, 1974).

Figure 10. Spearman rank-order correlations relating changes in heart rate and skin conductance with changes in muscle tension, movement, temperature and respiratory frequency are given for the three subjects trained to control heart rate. Point-biserial correlations relating respiratory amplitude to skin conductance and heart rate are also reported. These correlations are based on change scores observed on Increase Training Trials of the final experimental session.

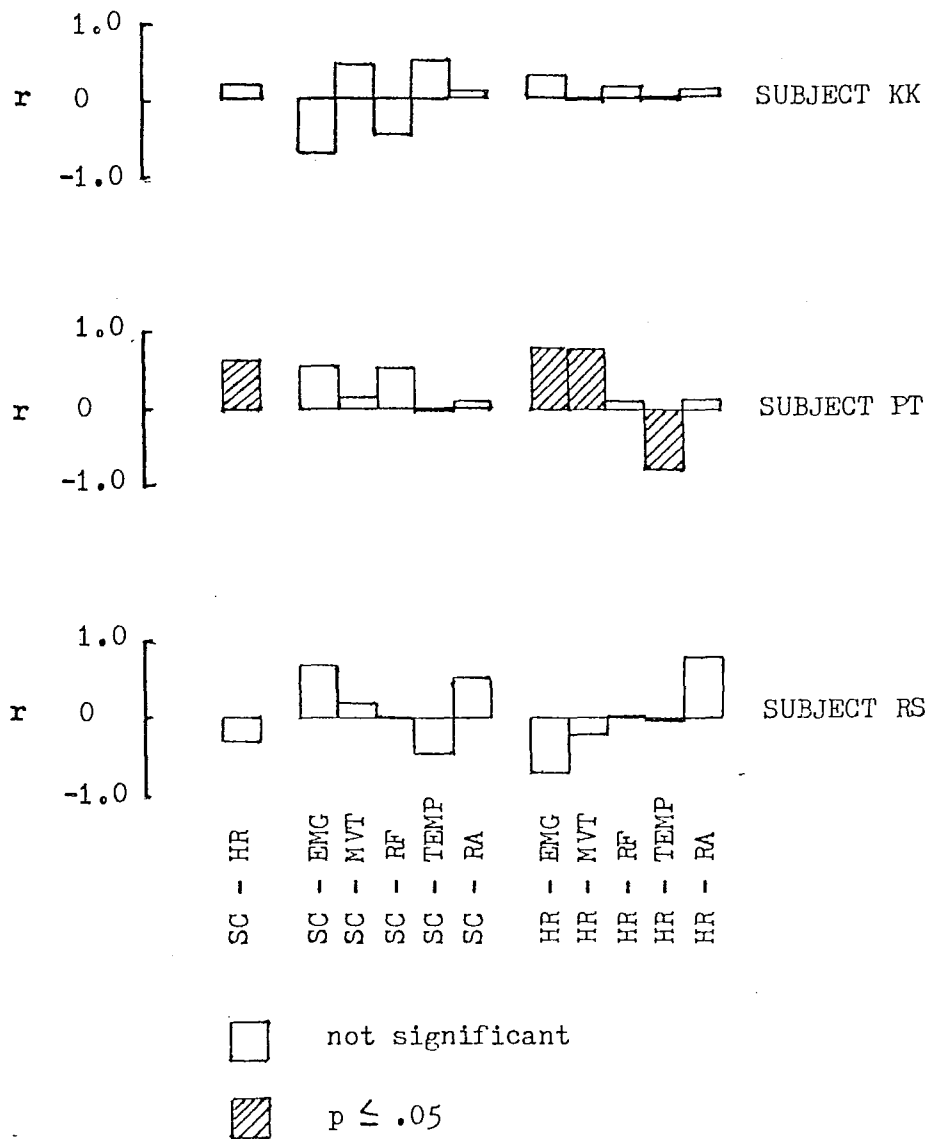


Figure 10.

Skin-Conductance Group

Control of the skin-conductance target within trials on the last day of the experiment is shown in Figure 11. As before, the mean skin conductance during the pretrial period has been subtracted from all trial and pretrial values so as to depict changes in responding from a common baseline. Inspection of these data shows that subjects were able to produce increases in skin conductance on Training Trials where feedback was present, and that transfer to Test Trials on which feedback was removed was complete. However, decreases in skin conductance were not evident on either type of trial after five days of training. Instead, skin conductance tended to decrease steadily as a function of time throughout both Blank and Decrease Training Trials, with little difference between them. Further inspection of increase performance shows that the magnitude of instructed control was quite large (approximately 2.8 micromhos in the last five seconds of the trial). It appears that an even larger increment in conductivity would have been observed had a longer trial duration been used.

The acquisition of skin-conductance control over the five days of training is shown in Figure 12. Inspection of performance during the first block of Test Trials on the initial day of training shows that little control of skin conductance was evident before exposure to the feedback contingency. However, control of skin conductance in the increase direction was manifested subsequently on Training Trials of this day, although the magnitude of this control did not improve substantially over the remaining sessions. Transfer, on the other hand, was not apparent at the end of Day One, but had developed by the

Figure 11. Control of skin conductance within-trials on the last day of the experiment. Performance with feedback (Training Trials) and without feedback (Test Trials) is compared to the Blank Trial baseline. Also reported is the mean skin conductance in micromhos during the pre-trial period.



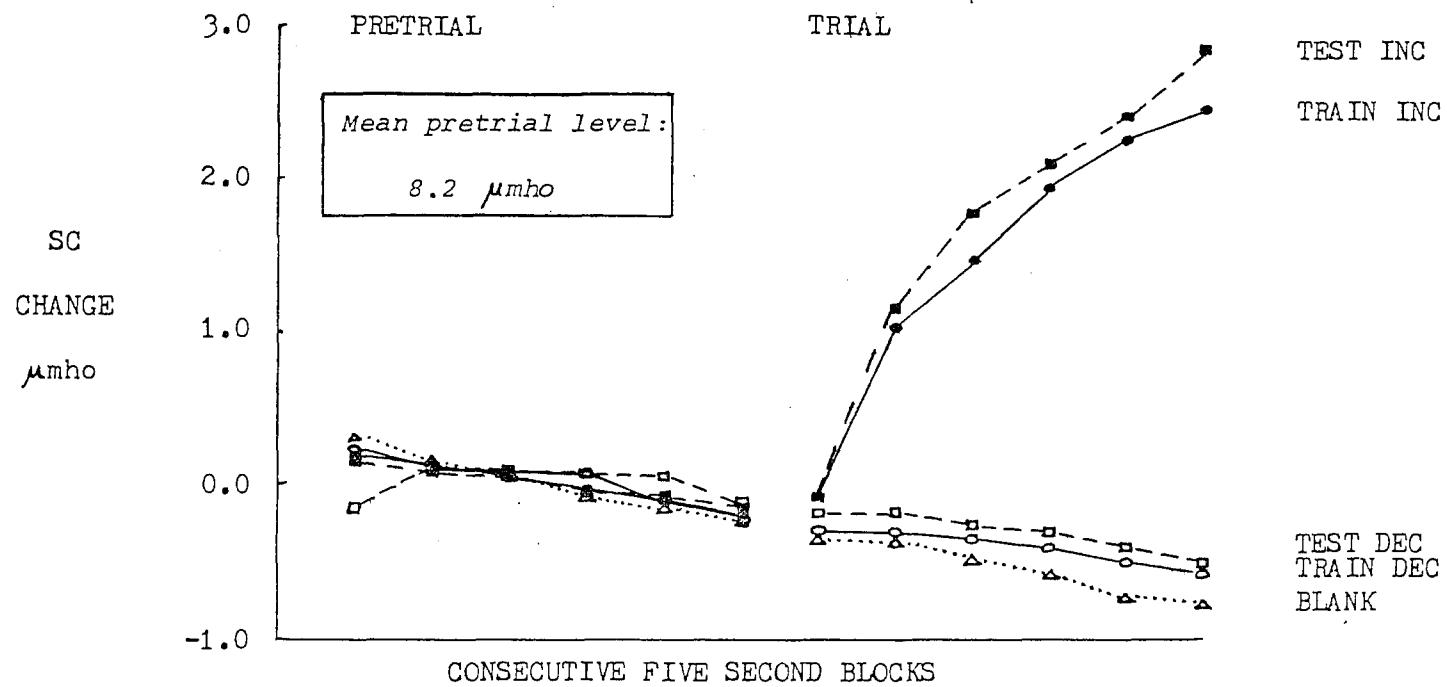


Figure 11.

Figure 12. Acquisition of instructed control of skin conductance across the five experimental sessions. Pretest data were taken from test trials given before feedback training on Day 1.

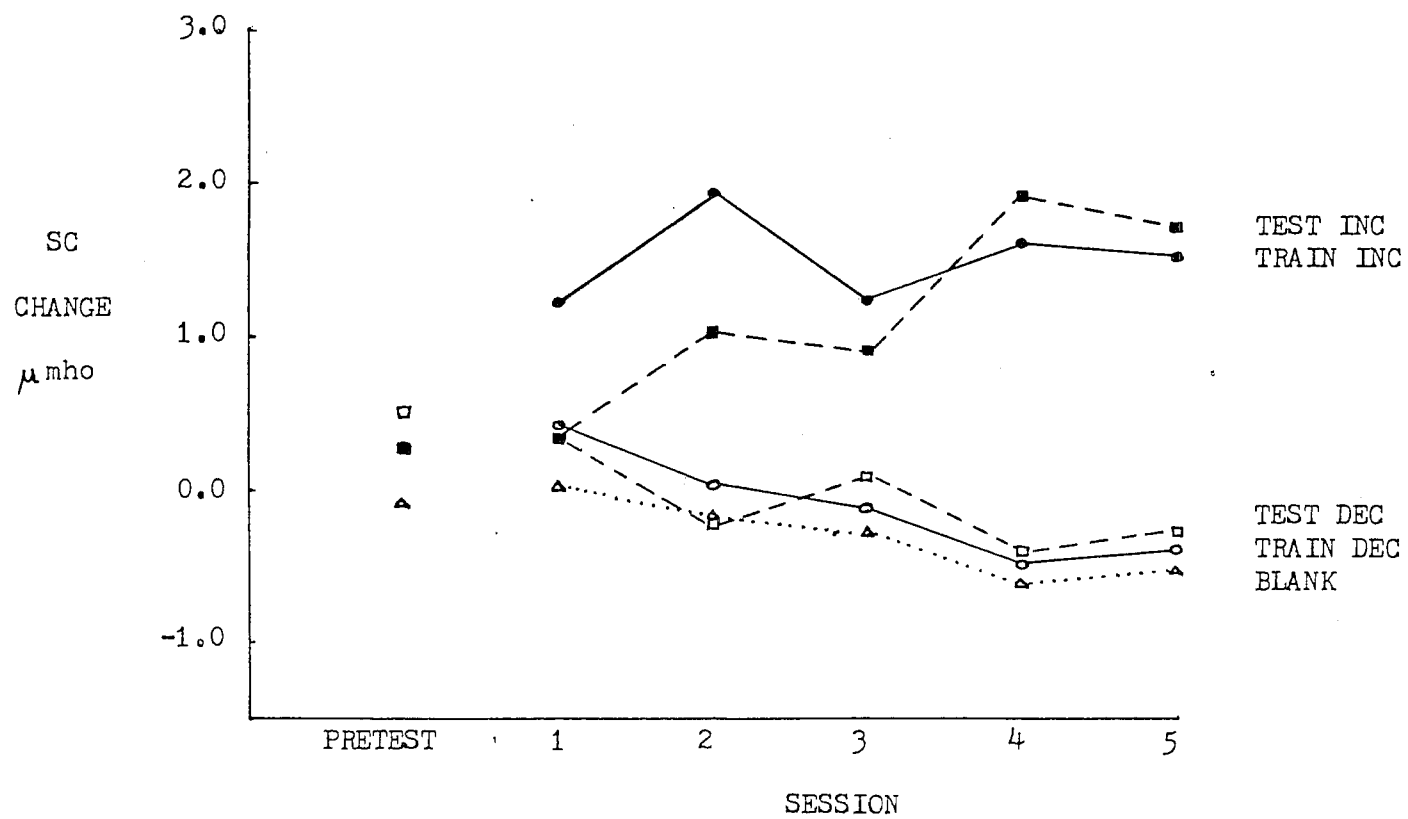


Figure 12.

fourth day of training. No control in the decrease direction was demonstrated during any session of the experiment.

Within-session trends in the performance of the skin-conductance target can be examined in Figure 13. A slight improvement in bidirectional control of this response was apparent over the course of the first day of training. Transfer appeared to have developed on Test Trials at the end of Day Two, but performance diminished on Test Trials (and to some extent on Training Trials as well) on Day Three. Bidirectional performance on both types of trials was substantial and stable on Days Four and Five, however.

The response pattern accompanying skin-conductance control, averaged over the three subjects for the final day of training, is given in Figure 14. Data for Increase Training Trials are shown in the left panel with the decrease response pattern on the right and Blank Trial performance in the center. Substantial changes in respiratory amplitude and frequency, forearm electromyographic activity, gross body movement, and heart rate appeared to accompany skin-conductance increases, but no change in skin temperature was observed. Changes in responding on Blank and Decrease Trials, on the other hand, were small and not substantially different from one another.

The response patterns of individual subjects on each day of training are given in Figure 15. Bidirectional performance is shown, but since responding in the decrease direction differed little from responding on blank trials, the bidirectional measure is almost totally comprised of changes occurring in the increase direction. An examination of these response patterns shows that all subjects

Figure 13. Changes in skin conductance on Training, Test, and Blank Trials within each experimental session.

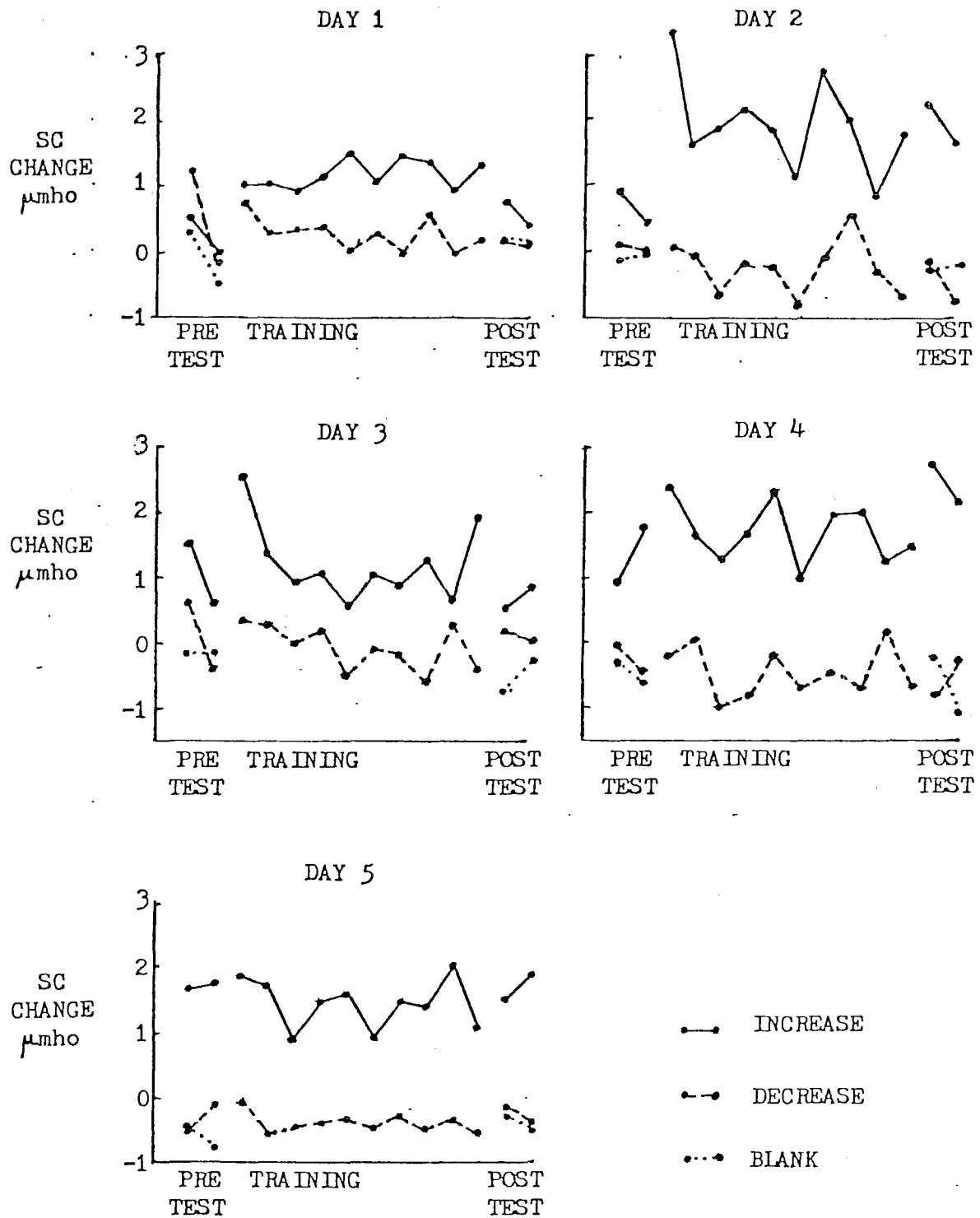
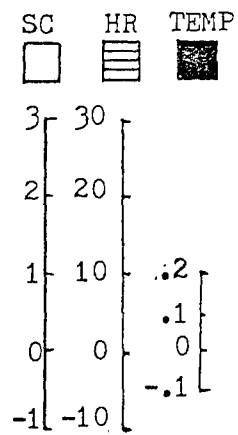


Figure 13.

Figure 14. Response patterns associated with control of skin conductance on Training and Blank Trials on the last day of the experiment. Response measures are as defined in Figure 7.



INCREASE TRIALS



BLANK TRIALS



DECREASE TRIALS

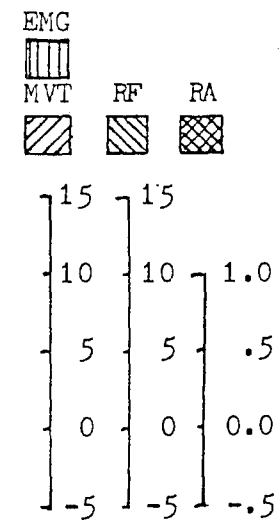
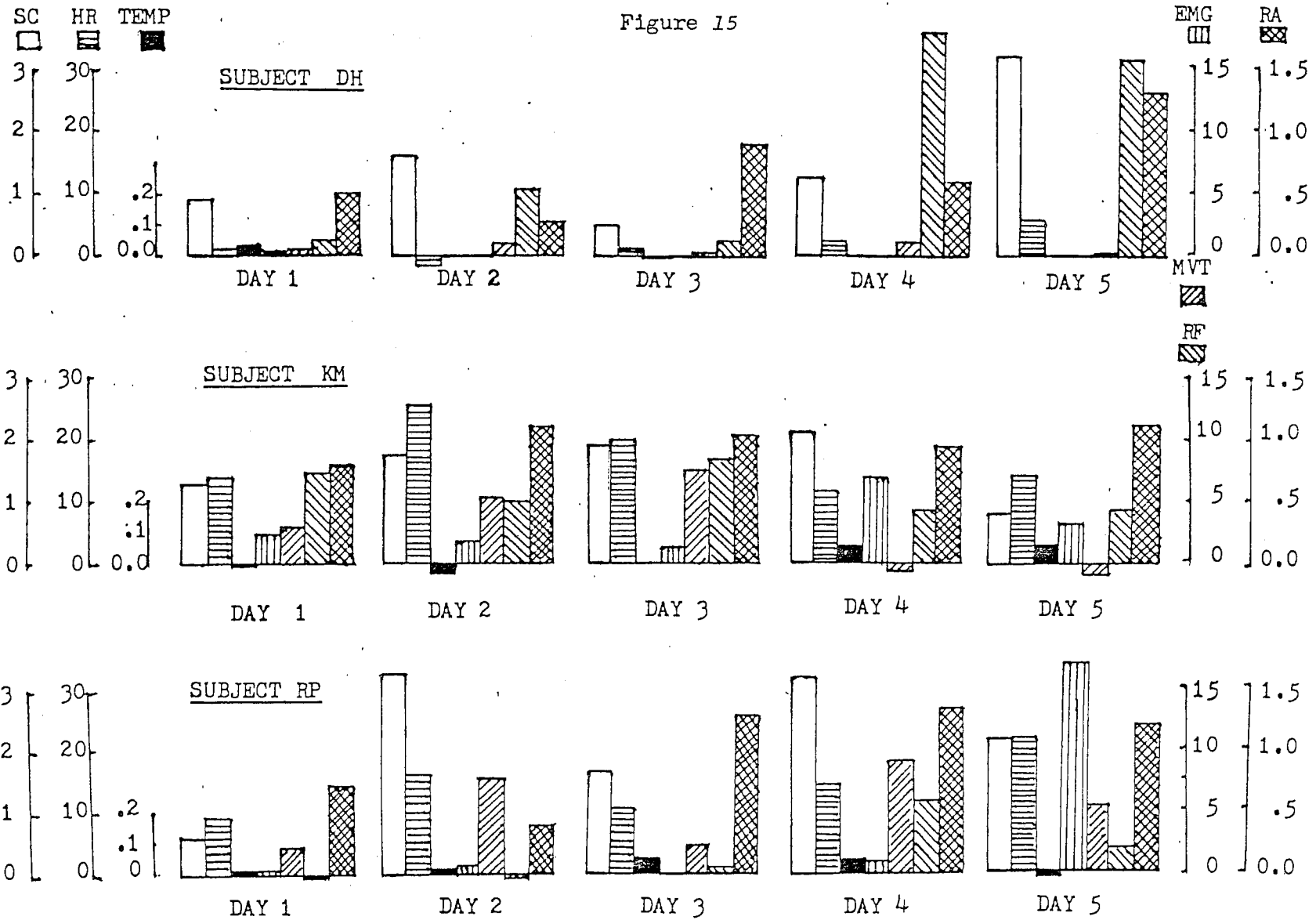


Figure 14.



Figure 15. Response patterns are given for the three subjects trained on the skin-conductance target for each of the five experimental sessions. Bidirectional performance on Training Trials is depicted. Response measures are as defined in Figure 7.

Figure 15



demonstrated control of the target response accompanied by changes in the concomitant measures. Subject DH demonstrated large changes in respiratory amplitude and frequency with little change in the other variables. Both KM and RP demonstrated similar changes in respiratory amplitude; however, they also showed large changes in heart rate. Both of these latter two subjects demonstrated sizeable changes in movement and electromyographic activity but these somatomotor changes were not stable over days. When the movement or electromyographic changes were large, heart-rate changes also tended to be large for these subject.

Further examination of Figure 15 indicates that changes in movement, electromyographic activity, and respiratory frequency may not have been necessary for the production of increases in skin conductance. DH demonstrated large conductance changes in the absence of movement and muscle tension responses throughout his training, whereas RP showed substantial control of skin conductance in the absence of a change in respiratory frequency in Sessions Two and Three. However, increases in skin conductance were not observed in the absence of an increase in respiration amplitude. This suggests that this concomitant may have been necessary for a substantial increase in skin conductance, although it should be noted that a change in respiration amplitude does not appear sufficient to produce a substantial increase in skin conductance (see DH, Session Three).

Spearman rank-order correlations between the target and concomitant measures are given for skin-conductance subjects in Figure 16. None of the correlations was significant, suggesting that changes in the concomitant responses were not directly related to performance

Figure 16. Spearman rank-order correlations relating changes in skin conductance and heart rate with each of the other variables (excluding respiratory amplitude) are given for the three subjects trained to control skin conductance. These correlations are based on change scores for Increase Training Trials during the final day. None of the correlations was significant.

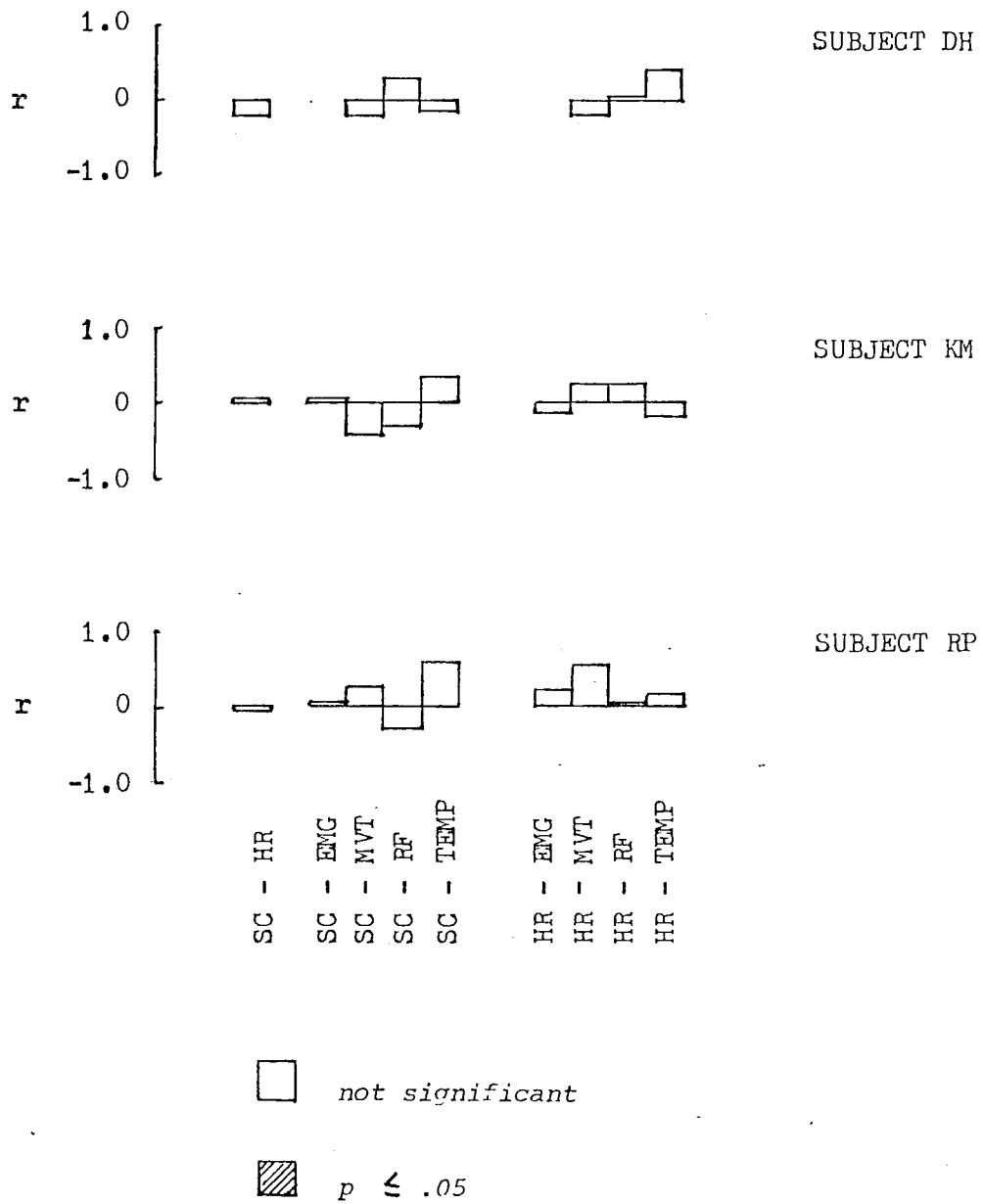


Figure 16.

of the target. It should be noted, however, that the relationship between skin-conductance responses and respiratory amplitude could not be assessed in this analysis, because there was no variability in this concomitant on Increase Trials of the fifth day of training (all Increase Trials received a "+1" on the amplitude measure on this day). However, although respiration amplitude increased on all these trials, increases in skin conductance were not always present. This provides further evidence that even though respiration amplitude may be necessary for skin-conductance control, it may not be sufficient for sudomotor activation.

#### Comparison of Heart-Rate and Skin-Conductance Groups

A comparison of the heart-rate and skin-conductance groups suggests that differences in both target responding and concomitant activities were present.

Changes in heart rate are compared across the two target groups in Figure 17. Increases in heart rate were larger in subjects that were given heart-rate feedback, on each day of training. However, the difference that was evident between the two target groups was not augmented by continued feedback training. The results concerning decreases in heart rate, on the other hand were different. Once again superior performance was observed in the heart-rate group, in this case in the decrease direction, but this superiority appears to have materialized only on the last three days of training. This result is consistent with prior evidence suggesting that subjects given feedback

Figure 17. Changes in heart rate for both target groups across five days of training. Performance on Training and Blank Trials is shown.

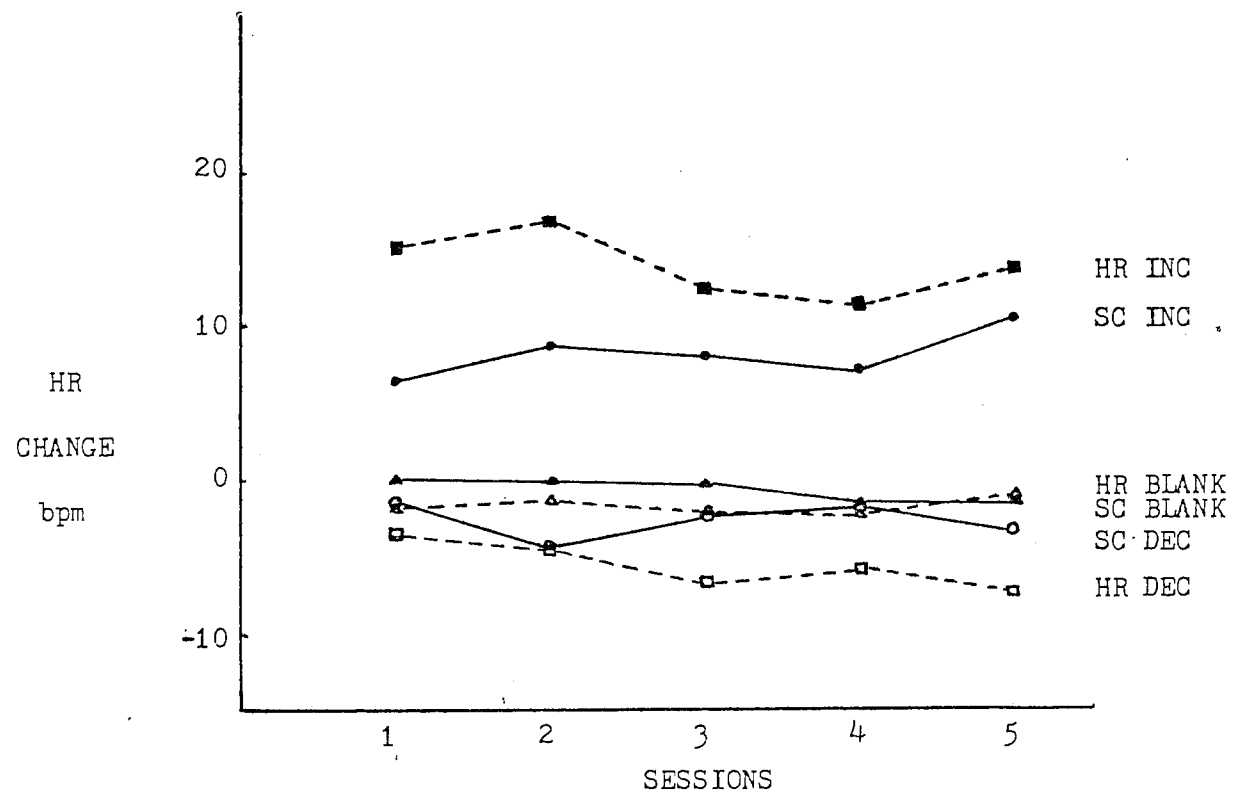


Figure 17.



training for heart rate successfully decreased this response, and that such decreases developed gradually over training.

Figure 18 depicts changes in skin conductance in the two target groups. Both groups demonstrated substantial increases in this response on Increase Trials. However, the groups tended to diverge over the course of training, owing largely to a decrement in the magnitude of skin-conductance responses evidenced by subjects trained to control heart rate. No differences were evident between the target groups on either Blank or Decrease trials.

Comparisons between the target groups for each of the remaining response measures are given in Figure 19. Inspection of the data presented for Increase Trials suggests that somatomotor changes tended to be more prominent in the heart-rate group. Larger changes in electromyographic activity were observed in this group during the first four experimental sessions, and larger changes in gross body movement in the last experimental session. Changes in respiratory function, on the other hand, tended to be larger in the skin-conductance group. This was particularly true of respiration amplitude, which was substantially elevated in skin-conductance subjects on each day of training. Inspection of the data provided for Decrease Trials shows that heart-rate subjects tended to display less somatomotor activity (electromyographic responding and gross body movement) and slower rates of breathing (respiration frequency) than did skin-conductance subjects. Although responses on Decrease Trials were small, it should perhaps be noted that the decrement in respiration frequency seen in heart-rate subjects developed on Day 3, which was the same day on which

Figure 18. Changes in skin conductance for both target groups across five days of training. Performance on Training and Blank Trials is shown.

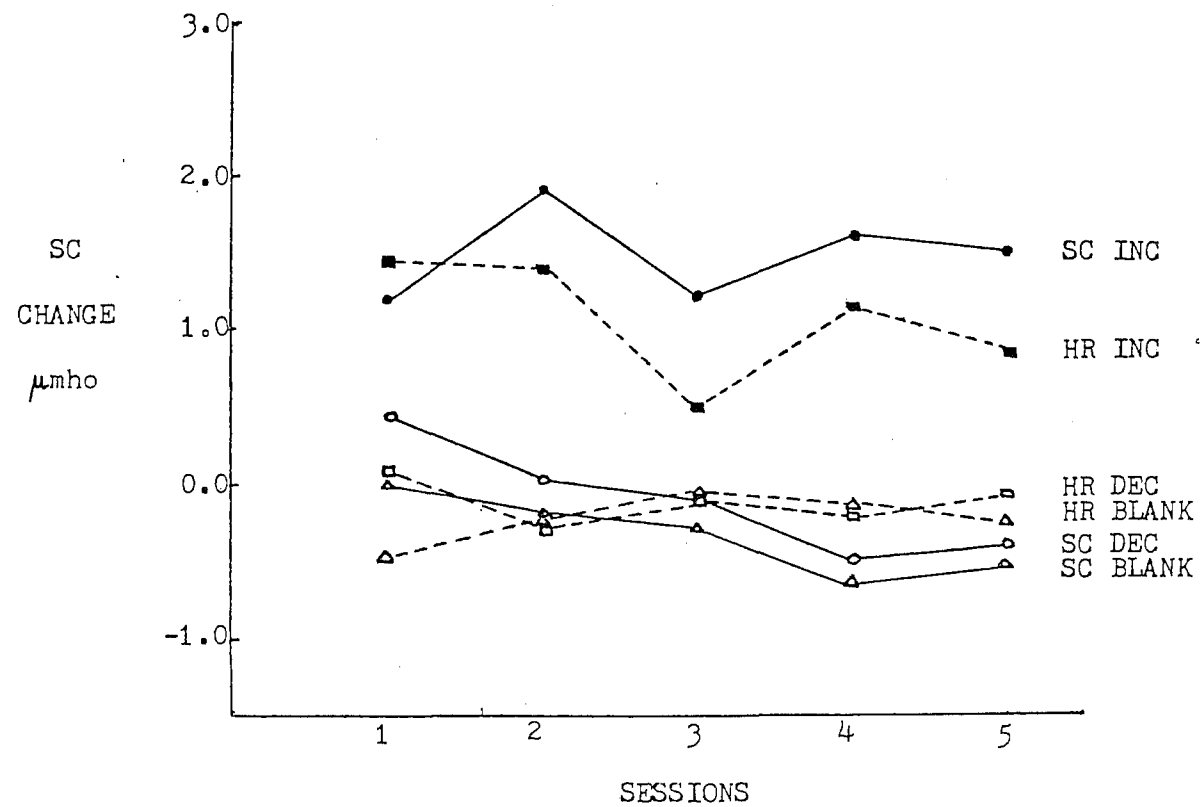


Figure 18

Figure 19. Change scores in the concomitant measures over five days of training. Responding is shown on Increase and Decrease Training Trials, with Blank Trials omitted for clarity. Response measures are as described in Figure 7.

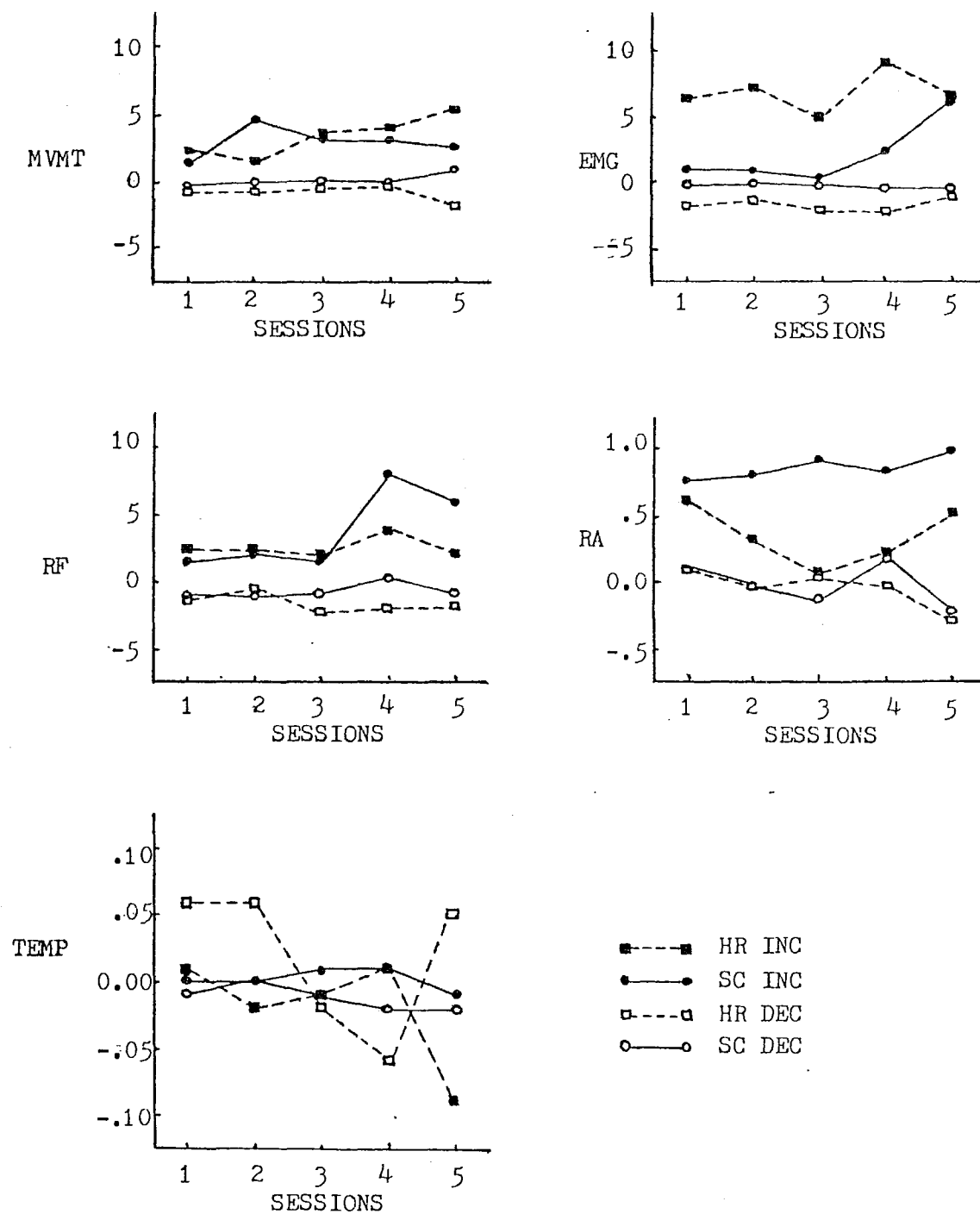


Figure 19.

control of heart-rate decreases appears to have materialized in this group.

A further comparison across the two training conditions examined the differences in the response strategies that were reported on the post-experimental questionnaire. Table 2 presents the strategies that were mentioned by subjects during debriefing, separately for Increase and Decrease Trials and for each training condition. The protocols reported here are verbatim replies to items two and three on the questionnaire, which asked the subject to specify activities that worked best on Increase and Decrease trials. Inspection of these reports shows that increases in somatomotor activity and excitement were reported when subjects were asked to describe how they controlled the feedback display on Increase Trials. In addition, every subject in the skin-conductance group reported taking deep breaths on this type of trial, whereas subjects in the heart-rate group did not make reference to respiratory action when an increase in visceral responding was the behavioural goal. Verbal reports given on Decrease Trials, on the other hand, typically alluded to general relaxation and a slowing of movement. In addition, "rhythmic breathing" and "exhaling" were mentioned as response strategies, but this time by subjects in the heart-rate rather than in the skin-conductance group.

Table 2

## Verbatim Verbal Reports\*

(Q2) What worked best on increase trials?

- HR    KK   getting myself excited
- RS   increasing bodily functions by increasing physical activity
- PT   tensing my muscles. I found that if I pushed down on the arms of the chair and back against the back of the chair it would provide maximum increase.
- SC    DH   more excited feeling - associated with mild perspiration in palmar (GSR) - expansion of the chest and rapid breathing.
- RP   Heavy chest breathing and muscle contraction in general
- KM   deep breaths with simultaneous wiggling of fingers.

(Q3) What worked best on decrease trials?

- HR    KK   tryed (sic) to depress myself
- RS   relaxing and rhythmic breathing
- PT   Relaxing totally; exhaling.
- SC    DH   warm relaxed feeling in the pit of stomach and chest
- RP   Relaxing as much as possible
- KM   Leaning forward and looking downward with eyes closed.

---

\* Replies to remaining items on the questionnaire are transcribed in

## CHAPTER FOUR

### DISCUSSION

Two purposes of the present research were to (a) provide further information regarding several properties of instructed changes in sudomotor and cardiac responding, and to (b) explore further the mechanisms by which instructed changes in these visceral effectors is achieved. A third goal was to (c) provide information concerning the origin of differences between the responses in properties and mechanisms of instructed control. In the latter respect it will be recalled that differences that materialized between the training conditions were necessarily attributable to differences in the organization of the response systems, since the only difference between the training conditions concerned the response that was identified by the feedback display.

#### Properties of Instructed Control

Subjects given feedback training for changes in heart rate successfully produced increases and decreases in this response. Increase performance approximated 15 beats per minute at the end of the trial and reached asymptote during the first session of training. Decreases in heart rate, on the other hand, were smaller (approximately -8 beats per minute at the end of the trial) and did not develop until the third session of the experiment. The magnitude of the changes seen in this small sample of subjects deviated only slightly from that



reported earlier by Lacroix and Roberts (1978), in which increases of approximately 20 beats per minute and decreases of -4 beats per minute were observed on test trials of the third day of training.

It will be recalled that the present procedure differed from that of Lacroix and Roberts (1978) in two major respects. First, subjects in the present study were not informed of the response they were being trained to control. Second, subjects in the present experiment received visual analogue rather than auditory binary feedback. The similar magnitude of heart-rate control observed in the two studies suggests that these changes did not affect cardiac performance, although the possibility of an interaction between the variables cannot be discounted.

A larger discrepancy in response magnitude is found when the present research and the preceding study by Lacroix and Roberts (1978) are contrasted with the results of earlier investigations of instructed cardiac control (Blanchard et al., 1974; Brener, 1974; Engel and Chisholm, 1967; Engel and Hansen, 1967; Lang and Twentyman, 1974; Levenson, 1976; Schwartz, 1977). These latter experiments have typically reported increases in heart rate of approximately 4 to 8 beats per minute, compared to the 15 to 20 beats per minute reported here. Decreases in heart rate, on the other hand, have typically averaged about -2 beats per minute in the earlier research, compared to -4 to -8 beats per minute in the current procedure.

The origin of these discrepant findings is difficult to ascertain, since there are many differences between the procedure used by Lacroix and Roberts (1978) and the present research and those

employed in previous investigations. Most conspicuous among the possibilities, however, is that constraints were not placed upon somatomotor and respiratory action in the present research or in earlier experimentation by Lacroix and Roberts (1978). Such constraints have been shown to be an important determinant of instructed cardiac change (Obrist et al., 1975) and have been employed with few exceptions in the visceral learning literature. Such constraints could be important to the expression of decrease as well as increase control, insofar as somatomotor activity is likely to elevate the heart-rate baseline from which decreases are differentiated. In this respect it should be noted that the baseline heart rates observed in the current experiment and earlier research by Lacroix and Roberts (1978) approximated 78 to 80 beats per minute, which is about 6 beats per minute higher than those reported in other studies in which decreases in heart rate have been sought (Lang and Twentyman, 1974; Engel and Chisholm, 1967; Blanchard et al., 1974).

There is an indication in the present findings that both increases and decreases in heart rate derived in part from operant conditioning. This was clearest in the case of decreases, which developed gradually over the course of training only in subjects that were given feedback for heart rate. The failure of subjects given skin-conductance feedback to show substantial decreases in heart rate indicates that the decrements seen in heart-rate subjects were not caused merely by an attempt to decrease a response or by visual attention to the feedback display. In contrast, increases in heart rate were demonstrable on test trials given prior to experience with

exteroceptive feedback. However, heart-rate subjects subsequently tended to show larger increases in heart rate than did subjects given feedback for skin conductance, again suggesting the possibility of a feedback effect.

A further variable of importance concerned individual differences in cardiac performance. Although all subjects appeared to have produced modest decrements in heart rate, only two of the three subjects showed substantial increases in this response. The fact that the unsuccessful subject succeeded in producing decrements in responding and performed consistently over five days of training suggests that an effort was made to comply with the requirements of the task. The explanation for this deviant performance in the increase direction is not apparent, although claims of substantial individual variability have been made previously (McCanne and Sandman, 1975).

The results concerning instructed control of skin conductance differed from those concerning heart rate in certain respects. As was the case with heart rate, subjects given feedback training for skin conductance successfully produced increases in this response. Furthermore, the fact that these increments tended to be larger than those observed in subjects that were given feedback for heart rate suggests that this control originated in part from operant conditioning. However, unlike the findings with regard to heart rate, increases in skin conductance were not evident at the outset of training but developed gradually over the course of the first session of the experiment.

Another finding of interest concerned the magnitude of the sudomotor response observed in the present study. This response, which approached 2.8 micromhos at the end of the trial stimulus in the fifth session, was approximately twice as great as that observed earlier by Lacroix and Roberts (1978). Superior performance in the current work does not appear to have been attributable to one or two exceptional subjects, since all subjects demonstrated substantial increments in skin conductance over the course of training. Nor does the effect appear to have derived from differences in electrode preparation in the two studies, since the magnitude of skin-conductance changes observed in subjects given heart-rate training were similar in the two experiments. The training procedure that was applied to sudomotor subjects in the present research appears to have produced a degree of control that exceeds that of every other published report in which a comparison can be made (Klinge, 1972; Lacroix and Roberts, 1978; see Kimmel, 1974 for a review).

The superiority of skin-conductance control that was observed in the present work may have arisen from one of two sources. The first possibility is that utilization of continuous analogue feedback in place of the binary feedback procedures that have typified earlier experimentation (Klinge, 1972; Lacroix and Roberts, 1978) may have provided more information about the response and led to the development of more effective response strategies. Continuous analogue feedback has been shown to be superior to binary procedures when heart rate is the target response (Lang and Twentyman, 1974) although heart-rate performance does not appear to have been augmented in the present work

by the use of such a procedure (cf. Lacroix and Roberts, 1978). The effect of different feedback procedures on the control of skin conductance, and the interaction of this effect with instructional conditions, has not been investigated in previous research.

The second possibility is that superior sudomotor performance may have been attributable to the verbal instructions that were employed in the present work. Important in this respect may have been (a) the omission of a reference to sweating of the fingers as a behavioural goal (cf. Lacroix and Roberts, 1978) and (b) the absence of constraints on somatomotor and respiratory response strategies (cf. Klinge, 1972). The effect of respiratory and somatomotor constraints on instructed sudomotor control has not been explored in previous studies, but prior research has shown that verbal description of the behavioural goal as a change in volar sweating diminishes the magnitude of instructed changes in both skin conductance (Lacroix and Roberts, 1978; Figure 1, this thesis) and heart rate (Blanchard et al., 1974).

Another conspicuous difference between the sudomotor and heart-rate groups in the present study concerned the absence of instructed decrements in the target response. In contrast to the heart-rate group, skin-conductance subjects were unable to reduce target responding below Blank Trial levels or below the level observed in subjects that were given feedback training for heart-rate decreases. There was, however, a tendency for skin conductance to decrease throughout Blank Trials. This suggests that the small decrements observed by Lacroix and Roberts (1978) may have been attributable to a continuation of the pretrial trend.

The failure to demonstrate instructed decrements in skin conductance in the present work could have arisen from several sources. One possibility is that subjects simply cannot produce decreases in skin conductance, owing to properties intrinsic to this biological system. However, while this must be considered a possibility, psychophysiological (Edelberg, 1973) and neurophysiological (Wang, 1964) evidence indicates that reabsorption of sweat from the epidermal layer is an active process that is under neurogenic control.

A second possibility is that the procedure used in the present research may have been ineffective for the production of sudomotor decreases. For example, longer trial durations might be more favourable to the demonstration of such decrements, since reabsorption appears to be a much slower process than is secretion in the eccrine sweat gland (Lloyd, 1961).

A third explanation of the present failure to observe sudomotor decrements is as follows. Although skin conductance on Decrease Trials did not differ substantially from skin conductance on Blank trials, subjects may nevertheless have learned to suppress phasic responses that would otherwise have been elicited by the feedback display. Lack of sudomotor differentiation between subjects trained on skin conductance and subjects trained on heart rate is not favourable to this interpretation, although it may be that the effect of feedback training for heart rate on decreases in skin conductance was the same as the effect of feedback training for sudomotor decreases.

It is not possible at the present time to establish which of these interpretations is correct. However, continued efforts to

produce sudomotor decrements may have practical utility in view of recent reports by Patel (1973, 1975; Patel and North, 1975) that hypertensives given feedback training for decrements in skin conductance displayed decreases in blood pressure that were substantially larger than those reported in studies employing other feedback procedures (see Blanchard and Miller, 1977 for a review). Subjects with labile sudomotor function have also been reported to be at risk for familial cardiovascular disease. (Hastrup, Swaney, Beeler and Chaska, 1978).

#### Performance Mechanisms

As in previous studies of learned cardiac control, instructed increases in heart rate in the present experiment were accompanied by increases in respiratory and somatomotor activation. Within-subject correlations revealed further that target responding was correlated with somatomotor concomitants (gross body movement, electromyographic activity, and skin temperature) in the subject that showed the largest heart-rate control. These findings are to be expected on the basis of prior evidence indicating a coupling between cardiovascular and movement control mechanisms (see Roberts, 1974, for a review). On the other hand, increases in heart rate were not correlated with concomitant changes in respiratory function, although such relationships have been reported previously (see Brener, 1974c). It is possible that correlations with respiratory function were diminished by the present procedure of allowing subjects to utilize a more effective somatomotor response strategy.

The basis for decreases in heart rate is less apparent. Small decrements in gross body movement were seen in two of the three subjects tested, but changes in this response and in electromyographic activity did not appear to parallel changes in heart rate as well as did the decrements in respiration frequency (Figure 19). Respiratory strategies such as "rhythmic breathing" and "exhaling" were also cited in the verbal reports; furthermore, such manoeuvres were not mentioned by subjects given feedback for sudomotor decreases. Consequently it is possible that respiratory changes contributed more to decreases in heart rate than did other concomitants of this response.

The origin of increases in skin conductance appears to have differed from that of heart rate. Although increases in movement and electromyographic activity were observed in this training condition, inspection of individual performances revealed that changes in these responses were neither necessary nor sufficient for control of sudomotor activation. Within-subject analysis also failed to reveal a single correlated concomitant even though, as mentioned previously, control was exceedingly large and observed in each subject. While it is important to remember that respiratory amplitude was not included in this analysis, inspection of the response patterns across training sessions nevertheless suggested that changes in this response were not sufficient for the control of skin conductance. These data are congruent with earlier evidence suggesting the dissociability of sudomotor and movement control processes (Roberts, 1974). They are also consistent with the report by Gavalas (1967) that deep



inspirations do not inevitably result in the production of GSRs (but also see Stern and Anchel, 1968).

The absence of demonstrable correlations between sudomotor responding and concomitant activities leads one to ask why concomitant changes were present at all in this training condition. Although there are several possible answers to this question, three appear most plausible. First, changes in the concomitant activities may have been neither necessary nor sufficient for sudomotor control. Changes in these activities may have materialized as a consequence of instructions to increase and decrease a response, rather than as a consequence of coupling with the target effector. This explanation holds that concomitants made no contribution whatsoever to the observed sudomotor control.

A second possibility, on the other hand, proposes that concomitant activities were related to sudomotor activation. However, changes in any one of a subset of these activities may have been sufficient for sudomotor control, although no particular concomitant was necessary so long as one of the subset was present. Failure to demonstrate significant within-subject correlations is not unavoidably fatal to this view, since it is conceivable that the composition of performance could have changed both within and between training sessions. In this respect it should be noted that the variability in individual response patterns across training that was evident in the present work perhaps lends some credence to this latter possibility.

Finally, a third explanation proposes that changes in respiratory action contributed to sudomotor control in the present

work, but more sensitive assessments of this variable are needed to demonstrate the dependency between these variables. The first steps in this direction would appear to include (a) development of an improved method of measuring respiratory activity and (b) application of dissociative training procedures.

Comparison of concomitant activities across the target conditions is relevant to an assessment of these three explanations. It will be recalled that differences that were demonstrable between the target conditions in the present research were necessarily attributable to differences in the organization of the response systems, since there was no other basis on which to explain differential patterning as a function of target group. Although the differences that materialized in response patterns were not pronounced, they were confirmed by an interesting intersection with the verbal report. On Increase Trials, all three skin-conductance subjects reported deep breathing as an effective response strategy. On the other hand, no mention was made of respiratory action when an increase in heart rate was the target response. These reports were corroborated by the measured response patterns, which suggested that respiration amplitude was much greater in the skin-conductance condition. Unfortunately it is impossible to say whether this difference developed because respiratory action contributed uniquely to sudomotor control, or because movement strategies were more effective at controlling heart rate, or some combination of these possibilities. Nevertheless, these observations implicate respiratory action in sudomotor control and point to the need

for a more detailed analysis of the relationship between sudomotor and respiratory function.

Although the present data are not extensive enough to establish that subjects reported activities differentially related to the visceral targets, the question of whether they are capable of doing so under any circumstance is of interest to accounts of the process by which control of responding is established through feedback training. One possibility recently put forth by Roberts and Marlin (in press) suggests that visceral learning is a process in which subjects seek information about the behavioural goal. According to this view, concepts of pertaining to this goal are based initially upon procedural details of training (instructional conditions, feedback method, and so forth) and are modified subsequently as feedback identifies instances of the behaviour that is to be produced. The attractiveness of this descriptive account of learning derives in part from its ability to explain a variety of instructional effects that have been reported in the visceral learning literature (see Lacroix and Roberts, 1978). It also gives reason to expect that subjects that have learned to produce a visceral response on a transfer test are capable of reporting activities that are related to the performance of visceral change. The study of performance mechanisms, which seeks to determine which aspects of response state enter into necessary and sufficient relationships with a visceral target, offers one point of departure for the study of veridical self-report and its relationship to performance on visceral learning tasks.

List of Appendices

	<u>Page</u>
Appendix A Initial Interview Form	89
Appendix B Summary of 19 Subjects	92
Appendix C Trial Sequences of the Experimental Sessions	95
Appendix D Debriefing Questionnaire	96
Appendix E Verbal Reports of Acceptable Subjects	100

APPENDIX AInitial Interview FormCONFIDENTIALSTANDARD PSYCHOPHYSIOLOGICAL LABORATORY INTERVIEW FORM

<u>NAME:</u>	<u>SEX:</u>	M	F
<u>TELEPHONE NUMBER:</u>	<u>AGE:</u>		
<u>OCCUPATION:</u>	<u>WEIGHT:</u>		
<u>WITH WHICH HAND DO YOU WRITE?</u>	<u>HEIGHT</u>		

Have you ever taken part in any other experiment in which physiological recordings were made? (If biofeedback, reject. Were you trained to control the response? Were you given feedback? If so, reject).

Are you taking any medications?

antibiotics (o.k. but note)

mood altering or psychiatric drugs (reject)

antihistamines (o.k. but note)

other (if psychoactive, reject. If drug name is not recognized by interviewer subject is asked reason he is taking the medication).

Have you had any respiratory disorders? (If current, reject).

Have you had any skin conditions? (If current and on target, reject).

Are you epileptic? (If yes, reject).

Have you every had any heart or cardiovascular problems?

high blood pressure (If yes, reject)

angina (If any cardiac problem is current and physician has  
restricted subject's physical activites/sports, then  
reject).

heart attack

Blood pressure: \_\_\_\_\_ (Taken by experimenter with standard  
blood pressure cuff, sphygomanometer and stethoscope) (if  $\geq 130/90$ ,  
reject).

Neurological examination:

balance

finger to nose

finger to tongue

do you experience any fainting spells or spells of dizziness?

(If so reject).

Do you smoke?

- 
1. If a subject is rejected, experimenter must state basis on this questionnaire.
  2. If pressure up on first measure, take another at end of introducing experiment to subject. If still high, ask subject to return next day on a pretext and if it is still high, casually mention he might

consult his family doctor and reject. Also make sure this report gets to L.E.R.; we may follow up if problem looks serious enough.

APPENDIX BSummary of 19 Subjects

Table 3 summarizes the disposition of all subjects exposed to the training procedure. Although the potential loss of blindness was not a principal reason for the rejection of any subject it was a contributing factor for three skin conductance subjects (CC, NH and VT). Not only were the same number of subjects acceptable for data analysis for each target condition, but also the same number of subjects in each group were rejected from the analysis for reasons which specifically included movement artifact on the target channel. Other than the fact that movement artifact alone as a reason for rejection was more prevalent for the heart rate target condition, the two targets of training were remarkably similar with respect to the disposition of individual subjects.

It is likely the case that the rejection of subjects on the basis of excessive movement artifact selectively deleted the subjects who produced the greatest amount of movement from data analysis. Furthermore, it has been established that the magnitude of the heart-rate change is directly related to the amount of movement present (see introductory discussion). On this basis it is possible that the sample selected for data analysis on the heart-rate target is biased in a conservative manner such that the results reported in this study underestimate the true magnitude of control possible.

In future experimentation it will be necessary to resolve the problem of movement artifact on the target channels to reduce the



Table 3.

## Disposition of the Nineteen Subjects

	<u>Target of Training</u>	
	<u>SC</u>	<u>HR</u>
<u>Subjects Accepted</u>	3 DH, RP, KM.	3 KK, RS, PT.
<u>Subjects Rejected (reasons given)</u>		
1. Movement artifact on target.	0	4 GK, AT, PC, RM.
2. Movement artifact on both skin conductance and heart rate.	2 ZM, AB.	1 NR
3. Movement artifact on target and repeated need to replace electrodes	2 NH, BD.	0
4. Movement artifact on target and need to change target limb.	1 VT	0
5. Failure to understand instructions	0	1 MU
6. Additional instructions interfering with performance	1 CC	0
7. Asleep during session	1 RPh	0
<u>Total</u>	3 Accepted 10 Rejected	3 Accepted 9 Rejected.

number of subjects whose data is lost to comprehensive analysis. Instructions toward quiescence may serve only to limit the amount of heart rate change and dissociate it from its usual context. Technical improvements, perhaps in the attaching of electrodes, choosing electrode sites or in the filtering of the incoming signals, would be the preferred solution.

APPENDIX CTrial Sequences of the Experimental Sessions.

<u>TRIAL #</u>	<u>TYPE OF TRIAL</u>	<u>INSTRUCTION WORD</u>	<u>FEEDBACK</u>
1	Blank Trial	absent	absent
2 - 5	Test Trial	Two INCREASE (I) and two DECREASE (D) presented in one of the following sequences: IDID, DIDI, DIID, IDDI, IIDD, DDII	absent
6	Blank Trial	absent	absent
7 - 26	Training Trial	Ten INCREASE (I) and ten DECREASE (D) presented in one of the following sequences: IIDIDDIIDIIDDIIDDIDD, IDIIDIDIIDDIDDIIDIDD, DDIDIIDDIDDIIDDIIDII, DIDDIDIDDIIDIIDDIDII.	present
27	Blank Trial	absent	absent
28 - 31	Test Trial	as in trials 2 - 5	absent
32	Blank Trial	absent	absent

---

The combination of six test trial sequences and four training trial sequences yields a total of 24 combinations of trials.

APPENDIX D

Debriefing Questionnaire

Name of Subject \_\_\_\_\_

Name of Experimenter \_\_\_\_\_

Date

1. How did you go about controlling the feedback display?

2. What worked best on increases trials?

3. What worked best on decrease trials?

4. Are there things that did not work well on increase or decrease trials?

5. Did you do as well when the feedback was removed as when it was present?

6. Did you go about attempting to control the feedback display in the same way every day?

7. What physiological response do you suppose we were trying to teach you to control?

APPENDIX E

Verbal Reports of Acceptable Subjects

KK(HR)

1. HOW DID YOU GO ABOUT CONTROLLING THE FEEDBACK DISPLAY?

Tryed (sic) to control my breathing and used my mind.

(Upon further questioning by the experimenter:

For "INCREASE" tried to get excited;

For "DECREASE" tried the other way to depress it.)\*

2. WHAT WORKED BEST ON INCREASE TRIALS?

Getting myself excited.

3. WHAT WORKED BEST ON DECREASE TRIALS?

Tryed (sic) to depress myself.

(Upon further questioning by the experimenter: Tried to slow down a bit.)

4. ARE THERE THINGS THAT DID NOT WORK WELL ON INCREASE OR DECREASE TRIALS?

Using my arm muscles.

5. DID YOU DO AS WELL WHEN THE FEEDBACK WAS REMOVED AS WHEN IT WAS PRESENT?

I don't think so.

---

\* In some instances the subject was asked to repeat a question to clarify an answer that was incomplete or vague. These additional replies were recorded separately and are included here in parentheses prefaced by the phrase "Upon further questioning by the experimenter."



6. DID YOU GO ABOUT ATTEMPTING TO CONTROL THE FEEDBACK DISPLAY IN THE SAME WAY EVERY DAY?

I tryed (sic) different things each time to improve.

(Upon further questioning by the experimenter:

Getting excited and arm muscles on the first day. Then tried to loosen up a bit. Used different breathing.)

7. WHAT PHYSIOLOGICAL RESPONSE DO YOU SUPPOSE WE WERE TRYING TO TEACH YOU TO CONTROL?

Self control.

(Upon further questioning by the experimenter: my mind, or my muscles or something.)

RS (HR)

1. HOW DID YOU GO ABOUT CONTROLLING THE FEEDBACK DISPLAY?

With some mental instruction for my body to increase/decrease its bodily functions and with increased or decreased physical activity. i.e., relaxing or tensing up. I tried holding my breath but didn't notice much results. I also tried recalling stress situations or passive situations in my personal history.

2. WHAT WORKED BEST ON INCREASE TRIALS?

Increasing bodily functions by increasing physical activity.

3. WHAT WORKED BEST ON DECREASE TRIALS?

Relaxing and rhythmic breathing.

4. ARE THERE THINGS THAT DID NOT WORK WELL ON INCREASE OR DECREASE TRIALS?

Holding my breath did not help on either increase or decrease. Or

mentally positioning myself in a stressful or passive situation did not seem to help.

5. DID YOU DO AS WELL WHEN THE FEEDBACK WAS REMOVED AS WHEN IT WAS PRESENT?

I was more relaxed, therefore I think the results were more stable then. I think I did do as well.

6. DID YOU GO ABOUT ATTEMPTING TO CONTROL THE FEEDBACK DISPLAY IN THE SAME WAY EVERY DAY?

Yes, however, the first and second days I did not have much of an idea about the procedures that would work so I tried most of the things then. The last three days I did just tense up muscles and relax.

7. WHAT PHYSIOLOGICAL RESPONSE DO YOU SUPPOSE WE WERE TRYING TO TEACH YOU TO CONTROL?

Heart beat?

PT (HR)

1. HOW DID YOU GO ABOUT CONTROLLING THE FEEDBACK DISPLAY?

To increase the feedback display I would tense my body up and tell myself lies. I would also inhale for a long time and exhale for a short time. Exactly the opposite made the feedback display decrease: relaxing and telling myself trues (sic.).

2. WHAT WORKED BEST ON INCREASE TRIALS?

Tensing my muscles. I found that if I pushed down on the arms of the chair and back against the back of the chair it would provide maximum increase.

3. WHAT WORKED BEST ON DECREASE TRIALS?

Relaxing totally. Exhaling.

4. ARE THERE THINGS THAT DID NOT WORK WELL ON INCREASE OR DECREASE TRIALS?

Trues (sic) did not have as great an effect as lies did.

5. DID YOU DO AS WELL WHEN THE FEEDBACK WAS REMOVED AS WHEN IT WAS PRESENT?

I don't know.

6. DID YOU GO ABOUT ATTEMPTING TO CONTROL THE FEEDBACK DISPLAY IN THE SAME WAY EVERY DAY?

Yes, although amounts varied slightly I used the same methods.

7. WHAT PHYSIOLOGICAL RESPONSE DO YOU SUPPOSE WE WERE TRYING TO TEACH YOU TO CONTROL?

Blood pressure or maybe body temperature.

DH (SC)

1. HOW DID YOU GO ABOUT CONTROLLING THE FEEDBACK DISPLAY?

Decrease - warm relaxed feeling in the pit of stomach and chest.

Increase - more excited feeling

- associated with mild perspiration in palmar (GSR)

- expansion of chest and rapid breathing.

2. WHAT WORKED BEST ON INCREASE TRIALS?

see number 1.

3. WHAT WORKED BEST ON DECREASE TRIALS?

see number 1.

4. ARE THERE THINGS THAT DID NOT WORK WELL ON INCREASE OR DECREASE TRIALS?

I tried a general slowdown of physical processes for decreases by beginning a meditation. It didn't work, nor did eyes closed. Tensing up didn't work for increase. Breathing out and attempting to control chest didn't work for decrease which puzzled me because the opposite seemed to work for increase.

5. DID YOU DO AS WELL WHEN THE FEEDBACK WAS REMOVED AS WHEN IT WAS PRESENT?

I felt I did much better with the feedback present because there were some things I could do to influence the results which were too vague and intangible to replicate without feedback.

6. DID YOU GO ABOUT ATTEMPTING TO CONTROL THE FEEDBACK DISPLAY IN THE SAME WAY EVERY DAY?

The past three days I used essentially the same procedure outlined in #1. Before that trial and error were used.

7. WHAT PHYSIOLOGICAL RESPOSNE DO YOU SUPPOSE WE WERE TRYING TO TEACH YOU TO CONTROL?

It could be GSR or chest expansion-contraction or may even be blood pressure. I am not sure.

RP (SC)

1. HOW DID YOU GO ABOUT CONTROLLING THE FEEDBACK DISPLAY?

On increase trials I tensed up muscles and whole body in general by the third day, before, I did deep breathing in my chest and moved my legs.

On decrease trials I tried to relax as much as possible, light breathing, mostly in stomach.

2. WHAT WORKED BEST ON INCREASE TRIALS?

Heavy chest breathing and muscles contraction in general.

3. WHAT WORKED BEST ON DECREASE TRIALS?

Relaxing as much as possible.

4. ARE THERE THINGS THAT DID NOT WORK WELL ON INCREASE OR DECREASE TRIALS?

On decrease trials, sometimes the harder I tried to relax, seemed to have the opposite effect, i.e., line would go up.

On increase trials moving my legs or arms.

5. DID YOU DO AS WELL WHEN THE FEEDBACK WAS REMOVED AS WHEN IT WAS PRESENT?

I don't think I did as well when feedback was removed for the odd trial.

6. DID YOU GO ABOUT ATTEMPTING TO CONTROL THE FEEDBACK DISPLAY IN THE SAME WAY EVERY DAY?

Yes on decrease.

No on increase.

(Upon further questioning by the experimenter:

Decrease: relax right from the beginning.

Increase: Monday - general tensing up

Tuesday - moving legs and arms

Wednesday - heavy breathing

Friday - tensing up muscles in whole body

7. WHAT PHYSIOLOGICAL RESPONSE DO YOU SUPPOSE WE WERE TRYING TO TEACH YOU TO CONTROL?

Something that causes blood pressure to rise.

KM (SC)

1. HOW DID YOU GO ABOUT CONTROLLING THE FEEDBACK DISPLAY?

a) to increase: Take deep beraths and wiggle your fingers, sometimes shaking of head vigorously increases the response.

b) to decrease: Keep quite (quiet?) with no movements at all. Of late, I found that leaning forward with eyes closed decreases the response. At times turning head to right or left slowly helped to decrease the response.

2. WHAT WORKED BEST ON INCREASE TRIALS?

Deep breaths with simultaneous wiggling of fingers.

3. WHAT WORKED BEST ON DECREASE TRIALS?

Leaning forward and looking downward with eyes closed.

4. ARE THERE THINGS THAT DID NOT WORK WELL ON INCREASE OR DECREASE TRIALS?

Yes, turning your body, stretching your legs, stiffening of the facial muscles.

5. DID YOU DO AS WELL WHEN THE FEEDBACK WAS REMOVED AS WHEN IT WAS PRESENT?

As I don't know my performance when feedback was removed, I would not be in a position to make the comparison.

(Upon further questioning by the experimenter:

I think that it should be the same because I do the same movements, but I am not sure.)

6. DID YOU GO ABOUT ATTEMPTING TO CONTROL THE FEEDBACK DISPLAY IN THE SAME WAY EVERY DAY?

No. I started to change the movements each day to the maximum possible.

(Upon further questioning by the experimenter:

First day - deep breaths

Second day - wiggle fingers                      Designation of days approximate.

Third day - both together

7. WHAT PHYSIOLOGICAL RESPONSE DO YOU SUPPOSE WE WERE TRYING TO TEACH YOU TO CONTROL?

I guess that you are trying to control the movements of various organs of the body. I don't know any better answer than this.

REFERENCES

- Bell, I. R., Schwartz, G. E. Voluntary control and reactivity of human heart rate. Psychophysiology, 1975, 12, 339-348.
- Black, A. H. Summary: The nature of operant and voluntary control. In P. A. Obrist, A. H. Black, J. Brener, & L. V. DiCara (Eds). Cardiovascular Psychophysiology: current issues in response mechanisms, biofeedback and methodology. Chicago: Aldine. 1974.
- Black, A. H., Cott, A., & Pavloski, R. The operant learning theory approach to biofeedback training. In G. E. Schwartz and J. Beatty (Eds), Biofeedback Theory and Research. San Francisco: Academic Press, 1977, 89-127.
- Blanchard, E. B. & Miller, N. E. Psychological Treatment of Cardiovascular Disease, Archives of General Psychiatry, 1977, 34, 1402-1413.
- Blanchard, E. B. & Young, L. D. Relative efficacy of visual and auditory feedback for self-control of heart rate. Journal of General Psychology, 1972, 87, 195-202.
- Blanchard, E. B. & Young, L. D., Self-control of cardiac functioning: A promise as yet unfulfilled. Psychological Bulletin, 1973, 79, 143-162.



- Brener, J. A general model of voluntary control applied to the phenomena of learned cardiovascular change. In P. A. Obrist, A. H. Black, J. Brener, L. V. DiCara (Eds) Cardiovascular Psychophysiology: current issues in response mechanisms, biofeedback and methodology. Chicago: Aldine, 1974(a).
- Brener, J. Summary: The nature of operant and voluntary control. In P. A. Obrist, A. H. Black, J. Brener & L. V. DiCara (Eds) Cardiovascular Psychophysiology: current issues in response mechanisms, biofeedback, and methodology. Chicago: Aldine, 1974(b).
- Brener, J. Factors influencing the specificity of voluntary cardiovascular control. In L. V. DiCara (Ed.), The limbic and autonomic nervous systems: Advances in Research. New York: Plenum Press 1974(c).
- Brener, J. Visceral perception. In J. Beatty and H. Legewie (Eds.) Biofeedback and Behaviour. New York: Plenum Press, 1977, 235-260.
- Brener, J., Kleinman, R. A., & Goesling, W. J. The effects of different exposures to augmented sensory feedback on the control of heart rate. Psychophysiology, 1969, 5, 510-516.
- Crider, A., Shapiro, D., & Tursky, B. Reinforcement of spontaneous electrodermal activity. Journal of Comparative and Physiological Psychology, 1966, 61, 20-27.

- Edelberg, R. Mechanisms of electrodermal adaptations for locomotion, manipulation, or defense. In E. Stellar & J. M. Sprague (Eds.), Progress in physiological psychology, Vol. 5. New York: Academic Press, 1973.
- Engel, B. T., Chism, R. A. Operant conditioning of heart rate speeding. Psychophysiology, 1967, 3, 418-426.
- Engel, B. T., Hansen, S. P. Operant conditioning of heart rate slowing. Psychophysiology, 1966, 3, 176-187.
- Forsyth, R. P. Mechanisms of the cardiovascular responses to environmental stressors. In P. A. Obrist, A. H. Black, J. Brener, L. V. DiCara (Eds) Cardiovascular Psychophysiology: current issues in response mechanisms, biofeedback and methodology. Chicago: Aldine, 1974.
- Gatchel, R. J. Frequency of feedback and learned heart rate control. Journal of Experimental Psychology, 1974, 103, 274-283.
- Gavalas, R. J. Operant reinforcement of a skeletally mediated autonomic response: Uncoupling of the two responses. Psychonomic Science, 1968, 11, 195-196.
- Hastrup, J. L., Swaney, K., Beeler, C., Chaska, L. Electrodermal lability and family history of cardiovascular disease. Paper presented at 18th Annual Meeting of the Society for Psychophysiological Research, Wisconsin, 1978.
- Johnson, H. J., Schwartz, G. E. Suppression of GSR activity through operant reinforcement. Journal of Experimental Psychology, 1967, 75, 307-312.

- Johnston, D. Criterion level and instructional effects in the voluntary control of heart rate. Biological Psychology, 1976, 4, 1-17.
- Johnston, D. Biofeedback, verbal instructions and the motor skills analogy. In J. Beatty and H. Legewie (Eds) Biofeedback and Behavior. New York: Plenum, 1977.
- Katkin, E. S. & Murray, E. N. Instrumental conditioning of autonomically mediated behavior: Theoretical and methodological issues. Psychological Bulletin, 1968, 70, 52-68.
- Kimmel, H. D. Instrumental conditioning of autonomically mediated behavior. Psychological Bulletin, 1967, 67, 337-345.
- Kimmel, H. D. Instrumental conditioning of autonomically mediated responses in human beings. American Psychologist, 1974, 29, 325-335.
- Klinge, V. Effects of exteroceptive feedback and instructions on control of spontaneous galvanic skin responses. Psychophysiology, 1972, 9, 305-317.
- Lacroix, J. M. Determinants of voluntary electrodermal and cardiac control. Doctoral dissertation: McMaster University, 1976.
- Lacroix, J. M. & Roberts, L. E. A comparison of the mechanisms and some properties of instructed sudomotor and cardiac control. Biofeedback and Self-Regulation, 1978, 3, 105-132.
- Lang, P. J., & Twentyman, C. T. Learning to control heart rate: Binary vs analogue feedback. Psychophysiology, 1974, 11, 616-629.

- Lang, P. J., & Twentyman, C. T. Learning to control heart rate: Effects of varying incentive and criterion of success on task performance. Psychophysiology, 1976, 13, 378-385.
- Levenson, R. W. Feedback effects and respiratory involvement in voluntary control of heart rate. Psychophysiology, 1976, 13, 108-114.
- Lippold, O. C. J., Electromyography. In P. H. Venables and I. Martin (Eds) Manual of psycho-phsyiological methods. New York: Wiley, 1967.
- Lloyd, D. P. Action potential and secretory potentials of sweat glands. Proceedings of the National Academy of Sciences, U. S., 1961, 47, 351-358.
- McCanne, T. R., Sandman, C. A., Determinants of Human Operant Heart Rate Conditioning: A systematic investigation of several methodological issues. Journal of Comparative and Physiological Psychology, 1975, 88, 609-618.
- Obrist, P. A., Galosy, R. A., Lawler, J. E., Gaebelein, C. J., Howard, J. L., & Shanks, E. M. Operant conditioning of heart rate: somatic correlates. Psychophysiology, 1975, 12, 445-455.
- Patel, C. H., Yoga and biofeedback in the management of hypertension. Lancet, 1973, 2, 1053-1055.
- Patel, C. H. Twelve month follow-up of yoga and biofeedback in the management of hypertension. Lancet, 1975, 2, 62-67.
- Patel, C. H., & North, W. R. S., Randomized controlled trial of yoga and biofeedback in management of hypertension. Lancet, 1975, 2, 93-99.

- Rice, D. G., Operant conditioning and associated electromyogram responses. Journal of Experimental Psychology, 1966, 71, 908-912.
- Roberts, A. H., Schuler, J., Bacon, J. G., Zimmerman, R. C., & Patterson, R. Individual differences and autonomic control: Absorption, hypnotic susceptibility and unilateral control of skin temperature. Journal of Abnormal Psychology, 1975, 84, 272-279.
- Roberts, L. E. Comparative psychophysiology of the electrodermal and cardiac control systems. In P. A. Obrist, A. H. Black, J. Brener, & L. V. DiCara (Eds) Cardiovascular Psychophysiology: current issues in response mechanisms biofeedback and methodology. Chicago: Aldine, 1974.
- Roberts, L. E. The role of exteroceptive feedback in learned electrodermal and cardiac control: Some attractions of and problems with discrimination theory. In J. Beatty and H. Legewie (Eds) Biofeedback and Behavior, New York: Plenum Press, 1977.
- Roberts, L. E. Operant conditioning of autonomic responses: One perspective on the curare experiments. In G. E. Schwartz and D. Shapiro (Eds) Consciousness and self-regulation: Advances in research. Vol. 2. New York: Plenum Press, 1978, 241-320.

- Roberts, L. E., & Marlin, R. G. Some comments on the self-description and discrimination of visceral responses states. To appear in N. Birbaumer and H. D. Kimmel (Eds) Biofeedback and Self-Regulation. Hillsdale, New Jersey: Lawrence Erlbaum Associates, in press.
- Schwartz, G. E. Biofeedback and patterning of autonomic and central processes: CNS-cardiovascular interactions. In G. E. Schwartz and J. Beatty (Eds) Biofeedback: Theory and Research. New York: Academic Press, 1977.
- Shapiro, D. Paper to appear in N. Birbaumer and H. D. Kimmel (Eds) Biofeedback and Self-Regulation, Hillsdale, New Jersey: Lawrence Erlbaum Associates, (in press).
- Stephens, A. Blood pressure control with pulse wave velocity feedback: Methods of analysis and training. In J. Beatty and H. Lewegie (Eds) Biofeedback and Behavior. New York: Plenum Press, 1977.
- Stern, R. M., Ansel, C. Deep inspirations as stimuli for responses of the autonomic nervous system. Psychophysiology, 1968, 5, 132-141.
- Surwit, R. S., & Shapiro, D. Biofeedback and meditation in the treatment of borderline hypertension. In J. Beatty & H. Legewie (Eds) Biofeedback and Behavior. New York: Plenum Press, 1977.
- Wang, G. H. The neural control of sweating. University of Wisconsin Press, Madison, Wisconsin, 1964.