DETERMINANTS OF VOLUNTARY ELECTRODERMAL AND CARDIAC CONTROL

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Abstract

The thesis was concerned with the general question of how voluntary control, defined as compliance with verbal instructions to change the response, is achieved over skin conductance and heart rate. This question encompasses three separate but related issues. First, what features of the experimental procedure contribute to the establishment of voluntary electrodermal and cardiac control? Second, what other response systems are affected when control is achieved over electrodermal and cardiac functions, and how are changes in these systems related to the occurrence of the target response? Finally, what is the process by which subjects learn to comply with an instruction to control skin conductance and heart rate, and is this process different for the two responses? The thesis bears on all three of these issues.

Experiment 1 was designed to develop and test a procedure for producing voluntary control of skin conductance and heart rate. Ten subjects received either two days of training to control skin conductance followed by two days of training to control heart rate, or the converse. Subjects attempted to produce increases and decreases in the target response during discrete 30-second trials. They were given instructions to control sweating or heart rate, were provided with strategy suggestions likely to facilitate control, and also with auditory feedback whenever they produced changes exceeding a predetermined criterion. The results provided evidence of control over both skin conductance and heart rate.

This control could have resulted from the instructions to control sweating and heart rate which were given to the subjects, from the strategy suggestions which were also provided, and/or from the exteroceptive feedback which was also provided. Experiment 2 examined the role of these variables in electrodermal and cardiac control. Sixty-four subjects were divided into eight groups. Four of these received three days of electrodermal training; the other four received three days of heart rate training. Two groups (one electrodermal and one cardiac) received only instructions to produce increases and decreases in the target response. Two others received strategy suggestions in addition to these instructions. Two more received instructions plus response-contingent feedback. Finally, the last two received instructions, strategy suggestions, and feedback.

Instructions to control palmar sweating were insufficient to generate reliable bi-directional control of skin conductance. However, electrodermal control was established when feedback for skin conductance changes was also provided. On the other hand, providing subjects with strategy suggestions appeared to interfere with electrodermal control, when such control was evident in the first place. The results with respect to heart rate were somewhat different. Instructions to control heart rate were sufficient to generate reliable, bi-directional heart rate differences. Adding feedback to these instructions had little effect on performance. However, adding strategy suggestions clearly interfered with performance.

Bi-directional differences in skin conductance were accompanied by differences in heart rate, body movement, respiration amplitude, and in a number of affective scales. However, these correlates did not appear to be intrinsically related to the skin conductance changes, and were probably due to the increase-decrease component of the instructions. Bi-directional differences in heart rate when heart rate was controlled were accompanied by differences in skin conductance, body movement, respiration frequency, respiration amplitude, and in several affective scales. Contrary to what was obtained with respect to skin conductance, however, there was evidence that the autonomic, somatomotor, and respiratory correlates of heart rate change were intrinsically involved in the performance of the heart rate response. It was suggested that the production of skin conductance changes was embedded in either the neural systems controlling specific hand movements or in those controlling a nonmotor arousal process. On the other hand, the production of heart rate changes was viewed as embedded in the neural systems controlling somatomotor and respiration functions.

While the production of skin conductance and heart rate changes appeared to involve different processes, the acquisition of control over the two responses was viewed as involving the same basic process. That process was depicted as one in which feedback related to the occurrence of the target response was utilized to identify response strategies which led to appropriate changes in target behaviour. It was suggested that changes in cardiovascular activity produced discriminable interoceptive afferentation that allowed subjects to identify

effective response strategies without having to undergo training with exteroceptive feedback. On the other hand, it was suggested that afferentation arising as a consequence of electrodermal activity was less discriminable than that associated with cardiovascular function. Consequently, training with exteroceptive feedback was necessary in order to identify response strategies which led to appropriate changes in electrodermal responding.

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Chapter 1: General Introduction

The autonomic nervous system is that branch of the nervous system which controls smooth muscle, cardiac muscle, and glands. It governs what are generally referred to as vegetative functions, and plays a vital role in the maintenance of homeostasis. The system comprises two branches, the sympathetic and the parasympathetic, which may be distinguished on anatomical, physiological, and functional grounds. Functionally, the two branches tend to have antagonistic effects, activation of the sympathetic branch exerting a generally mobilizing influence on the organism, activation of the parasympathetic branch, a generally relaxing one.

Historically, the autonomic nervous system has been of interest to psychologists primarily because of its involvement in emotional experiences (Cannon, 1929), and as a tool for studying the mechanisms of classical conditioning (Pavlov, 1927). Much of our knowledge about how the autonomic nervous system is organized stems from work concerned with these two general areas of inquiry. Among the issues which were raised by this work, one is of particular relevance to this thesis. This is the question of whether autonomic responses can be operantly conditioned or brought under "voluntary" control.

Operant and Voluntary Control

In this thesis, the terms "operant control" and "voluntary control" will be used to designate behavioural phenomena that are observed during the application of two, slightly different, training procedures.

The responses that are produced by application of these procedures will be referred to as "operant responses" and "voluntary responses", respectively. These terms are preferred to alternative ways of designating the phenomena of interest, because they are widely used in the literature pertaining to self-regulation of autonomic functions (Blanchard and Young, 1973; Brener, 1974a; Schwartz, 1974). The relationship between operant and voluntary control, and the procedures used to establish these phenomena,are discussed briefly below.

The phenomenon of operant control has been studied extensively by experimental psychologists and a consensus has developed as to its meaning. A response may be considered an instance of operant control if it can be demonstrated that its production stems from the existence of a contingency between that response and a reinforcer. The procedure that arranges such a contingency is called operant conditioning, and the responses that result from such conditioning are usually called operant responses, or simply operants. The procedures of operant conditioning have been defined with considerable precision and have been subjected to conceptual analysis by several investigators (Keller and Schoenfeld, 1950; Platt, 1974; Skinner, 1938). In addition, these procedures have been applied extensively to the study of skeletal-motor-behaviour for many decades, and several principles of conditioning have been established. Application of the methods of operant conditioning to the study of autonomic responding is of more recent origin, and has been carried out intensively only within approximately the last ten years.

The study of "voluntary" control of behaviour is almost as old as the discipline of Experimental Psychology itself (see Irwin, 1969,

and Kimble and Perlmutter,1970, for reviews). However, experimental attempts to establish voluntary control of autonomic responses, like efforts to operantly condition these responses, are of relatively recent origin. At the present time there is less consensus with respect to the meaning of the term "voluntary control", than with respect to the meaning of the term "operant control". This lack of consensus is due largely to a failure to agree, despite much prior discussion (Black, 1974a; Brener, 1974a; Irwin, 1969; Kimble and Perlmutter, 1970) on what constitutes a voluntary response. One definition that has guided much of the current research on voluntary control of autonomic responses is that a voluntary response is one that can be operantly conditioned (Miller, 1969). This view equates the concepts of operant and voluntary and reduces the study of voluntary processes to the study of operant conditioning.

An alternative view, promoted recently be Brener (1974a, b), defines a voluntary response as one that can be influenced systematically by verbal instructions to control the behaviour in question. One advantage of this definition is that it captures, perhaps better than an operant specification of voluntary behaviour, the meaning of the term voluntary as it is used in the English language. Another advantage may be that it simplifies the requirements that must be met before a given behaviour may be relegated to a voluntary response class. On the other hand, this definition may be too simple. One can question whether a response produced simply in answer to instructions to produce the response can always be properly characterized as "voluntary" (Black, 1974a). Additional criteria may be imposed before concluding that a

response is "voluntary". These criteria might consist of additional performance requirements. For instance, one might require that subjects be able to withhold the response as well as produce it. Or, one could require that subjects be able to produce bi-directional changes in the response. Or, one could require that there be no evidence of gross somatic mediation in the performance of the response. Alternatively, whether instructional compliance is considered evidence of voluntary control might be made to depend upon how this control developed. For example, one might want to rule out classical conditioning as an account of how the response was acquired before calling the response voluntary. There is as yet no consensus on what constitutes a voluntary act.

Brener's definition of a voluntary response is accepted in this thesis as a starting point in the analysis of voluntary control. A response will be considered "voluntary" if it can be produced following an instruction to do so. The thesis experiments examine the role of two variables, feedback contingency and information about the response, in establishing voluntary control. Other possible sources of control, such as Pavlovian or response-eliciting properties of the instructional stimulus, are not explicitly evaluated. It should be noted, however, that compliance with both increase and decrease instructions make these unlikely determinants of performance in both the increase and the decrease conditions.

The use of the terms operant control and voluntary control to designate separate phenomena in this thesis could be interpreted to mean that these phenomena, and the procedures used to demonstrate them,

differ fundamentally from one another. However, this need not be the case. The relationship between operant control and voluntary control defined as compliance with verbal instructions, is unclear and controversial (Black, 1974a; Black et al., in press; Brener, 1974a).

The procedures typically used to establish operant and voluntary control do appear to differ in certain respects, however. The method of Brener and his colleagues may be taken to illustrate a typical voluntary control study (Brener, 1974a; see also Brener, Kleinman, and Goesling, 1969; Lang and Twentyman, 1974; Levenson, 1974). Since voluntary control is evidenced by compliance with a verbal instruction to change the response, human subjects are used in these experiments. They are usually informed of the response they should strive to bring under control, and are told whether they are to increase the response, decrease the response, or both. In addition, some experimenters (for example, Klinge, 1972) have given their subjects information about various response strategies that might be expected to facilitate performance. Another feature of voluntary control studies is that the extent to which the subjects are able to control the target response in the absence of any special training is usually ascertained, either by presentation of a few test trials before training begins (for example, Lang and Twentyman, 1974), or through the use of a separate control group (Brener, 1974a; Levenson, 1974). Special training designed to establish voluntary control is then carried out. During training, the subjects are provided with exteroceptive feedback for their performance. This feedback typically consists of a tone or a visual display, some feature of which varies in proportion to changes in the subject's target

response. Periodically, the subjects may be asked to produce appropriate changes in their response without the benefit of the exteroceptive feedback, on "transfer" trials. Voluntary control is evidenced by the ability of the subjects to comply with the instruction to change the response. Comparison of responding before and after feedback training provides information on the role of the training procedure in establishing voluntary control of the response.

The typical operant conditioning procedure, on the other hand, may be described as follows. Although not a requirement of the procedure, consideration here is restricted to those experiments in which human subjects have been employed (for example, Crider, Shapiro and Tursky, 1966; Schwartz, Shapiro and Tursky, 1971; Schwartz, 1972). Typically, subjects in operant conditioning experiments are not informed of the response they are to bring under control. Nor are they told whether the response is to be increased or decreased. Instead, the subjects are typically instructed to try to produce changes in "certain physiological responses that are considered involuntary" (Schwartz, Shapiro and Tursky, 1971). After the instructions have been given, operant conditioning begins. Typically, this training consists of the presentation of discrete trials, indicated by appropriate exteroceptive stimulus, on which the subjects are rewarded by presentation of a pleasant slide or a light indicating monetary reward, whenever responses of the appropriate direction and magnitude are produced. Operant control of the response is evidenced by the development of a bi-directional difference that is significantly greater than that observed in control groups receiving non-contingent feedback presentations. These

comparisons provide information on whether experience with the operant contingency was sufficient to establish control of autonomic responding.

Comparison of these two procedures for establishing control of a response suggests that they differ in at least two possibly important respects. One difference between the two procedures pertains to the instructions which are given to the subjects prior to feedback training. The instructions given to subjects in voluntary control training refer both to a particular target system, and to the direction of the changes which the subjects are to effect in that target response. Successful compliance with these instructions provides information on the extent to which they are able to achieve the behavioural goal specified by the instruction, before feedback training is carried out. No such prior assessment is taken in operant conditioning, where the behavioural goal is specified by the exteroceptive feedback alone. Prior assessment of the subject's ability to comply with the instruction is relevant to the question of whether experience with a feedback contingency in operant conditioning acts to define a new and previously unattainable goal for the subject, as some researchers have suggested (Brown, 1974), or whether it is used by the subject merely to identify a previously learned performance (Johnston, in press).

One can question whether this difference between the procedures of operant and voluntary control is necessary or important. One objection is that there is no a priori basis for assuming that the processes of selecting a previously learned goal and of establishing a new one are different. On the contrary, these processes and the role played

by exteroceptive feedback in them may be the same. Another problem is that the prior assessments taken during voluntary control training do not by themselves provide full information about the subject's ability to achieve the specified behavioural goal. For example, successful compliance with an instruction to "increase heart rate" indicates that the subject possesses within his current repertoire a response strategy that achieves this goal, but it does not indicate whether the subject knows that this strategy is effective specifically for heart rate. Moreover, failure to comply with instructions to increase heart rate does not necessarily indicate that the subject is unable to comply with these instructions. Failure to comply may be due to a misunderstanding of the instructions, or to a lack of motivation, or the subject may be able to comply with the instructions, but not within the constraints of the experiment. Additional data are required to describe the subject's knowledge and abilities more precisely. Despite these interpretative problems, it is true that the instructions included in the voluntary training procedure provide information about the subject's ability to achieve the behavioural goal that is not provided by operant conditioning experiments. Successful compliance with a verbal instruction to alter heart rate prior to feedback training indicates that the subject is able to change his heart rate in the prescribed direction, without explicit exteroceptive feedback training for this skill. This information seems worth having for the light it sheds on the subject's initial skills, and on the necessity of explicit feedback training for establishing control of an autonomic response.

A second difference between the procedures pertains to the criteria which are employed to determine whether operant or voluntary

control has been demonstrated. In order to demonstrate voluntary control, all that is necessary is that the subject be able to produce the required response when asked to do so. There is no requirement that the ability to comply with the instruction be acquired within the course of the experiment, or that provision of exteroceptive feedback be essential for development of this ability. However, more is required before one can conclude that operant control has been demonstrated. Here it is incumbent upon the experimenter to show not only that the response occurs, but also that production of the response is a result of experience with the contingency between the response and the reinforcer that was employed during training. Often, this is accomplished through comparisons with appropriate control conditions in which the reinforcer is made contingent on changes in the target response in the opposite directions (the "bi-directional" control procedure). These comparisons can be made either between different types of conditioning trials within the same subject, or between different experimental groups, when all other variables including instructional manipulations are held constant. The difference in the criteria used to determine whether voluntary or operant control have been established can be illustrated by comparing how changes in the response on transfer trials would be interpreted in the two cases. It will be recalled that on these trials subjects are instructed to produce increases or decreases in the target response in the absence of exteroceptive feedback. Evidence that subjects can control the target response when asked to do so on transfer trials would constitute prima facie evidence of voluntary control, insofar as the subject has complied with the

instruction given to him. On the other hand, appropriate changes in the target response on transfer trials would not necessarily constitute evidence for operant control, since these data do not by themselves show that the contingency between response and reinforcer was essential for the development of such control.

It should be stressed that these differences in the procedures for demonstrating operant and voluntary control do not necessarily imply that the two types of control are fundamentally different. A feature central to both training procedures is the presence of a response-feedback contingency. There is no a priori basis for assuming that the mechanisms by which experience with such a contingency establishes control is different in the two procedures. Furthermore, it may be noted that voluntary control training can be viewed as a special case of operant conditioning, in which the operant contingency is correlated with an exteroceptive cue consisting of a verbal instruction to produce the response. According to this view, voluntary control training is merely a form of discriminative operant conditioning in which the discriminative stimulus is a verbal label rather than a more conventional exteroceptive cue such as a tone or a light. This view is accepted by many researchers engaged in the study of operant and voluntary control, including Brener (1974a).

Nevertheless, it would be premature to conclude on the basis of these similarities that the phenomena of operant and voluntary control are the same. Clearly, the ability of a subject to comply with a verbal instruction to alter an autonomic response cannot be taken as evidence for operant control of the response, since there is in this

demonstration no evidence that experience with an operant contingency was necessary to establish the effect. Nor can a demonstration of successful operant conditioning necessarily be taken to imply voluntary control. Operant conditioning may not produce an ability to comply with a verbal instruction to change a response, even if the subject is informed of the response he successfully produced during prior operant conditioning. Moreover, the way in which a human subject learns to control a response may differ during voluntary and operant training, depending upon whether he is told of the response to be trained. The neural systems involved in the performance of the response may depend on information contained in the instructions given in the two procedures. In view of these uncertainties, the relationship between the phenomena of operant and voluntary control, which seems in many respects to be similar, may best be left unjudged at present.

The Acquisition of Voluntary Control

The goal of the research to be reported in this thesis was to provide information on the question of how voluntary control, defined as compliance with verbal instructions, is established over two autonomic responses, electrodermal activity and heart rate. Although the thesis is concerned specifically with voluntary control, reference is made to the literature on operant conditioning of autonomic responses when this appears to be instructive.

The question of how voluntary control is established encompasses three separate but related issues. First, what features of the experimental procedure contribute to the development and maintenance of voluntary control over a given autonomic response? An assumption which

is prevalent in much of the current literature on voluntary control is that successful compliance with an instruction to control a response depends heavily upon prior experience with exteroceptive feedback for autonomic changes of the appropriate direction and magnitude (Brener, 1974a; Brown, 1974; Lang, 1970). Alternatively, it is possible that mere instructions to increase or decrease responding are sufficient to produce changes in the appropriate direction, or that compliance is heavily dependent upon behavioural strategies that are sometimes made explicit in the instructions which are given to the subjects prior to training. One goal of the experiments to be reported in this thesis was to examine the extent to which these three variables (instructions to produce changes in the response, behavioural strategies that may be included in these instructions, and provision of feedback for changes in the appropriate direction) contribute to voluntary control of electrodermal and heart rate responding.

A second major issue related to the question how voluntary control is achieved deals with the changes that are produced in various response systems when voluntary control is established. Are other autonomic and somatomotor functions affected when voluntary control is established over a given autonomic response? If so, which systems are affected, and how are changes in these systems related to the occurrence of the target response? The answers to these questions provide information on the neural systems that are involved in the performance of the target response. In the present thesis, this problem was approached by evaluating concomitant changes in a variety of autonomic, somatomotor, and affective variables when voluntary control was established over heart rate and electrodermal responding.

A third issue related to the problem of how voluntary control is established has to do with how subjects learn to comply with an instruction to alter an autonomic response. This question has received little attention so far, except in Brener's writings (1974a,b, in press). Brener argues that the process of gaining control over a response which the subject cannot control at the outset is essentially a process of learned discrimination. Subjects gradually learn to identify interoceptive cues that are related to changes in the target response, on the basis of temporal associations between these cues and exteroceptive feedback. The ability to control the target response follows when afferentation arising from performance of the response is discriminated and associated with the verbal labels used to denote the response in the instructions. Brener's interpretation appears to place a particularly heavy emphasis on the discrimination of effector activity as a prerequisite for voluntary control over an autonomic response. In this thesis, a similar hypothesis will be offered to account for differences in the voluntary control of electrodermal and cardiac responding.

This thesis, then, is concerned with the problem of how voluntary control is achieved over two autonomic functions, electrodermal activity and heart rate. The experiments attempt to provide information on the importance of different features of the training procedure typically employed to establish voluntary control of autonomic responding, and on the changes which take place in a variety of response systems when voluntary control of electrodermal activity and heart rate is established. The thesis is also concerned with the process underlying the acquisition of voluntary control over internal responding. Comparison

of the characteristics of voluntary electrodermal and cardiac control was undertaken as a first step toward developing an understanding of this process.

The Electrodermal and Cardiac Control Systems

The two autonomic responses studied in this thesis are heart rate and electrodermal activity. A brief summary of the organization of these two response systems is given here.

Heart rate is simply the frequency with which the heart contracts, measured in beats per minute. Heart rate is heavily influenced by neurogenic and to a lesser extent by hormonal influences, and is the major determinant of cardiac output in most mammalian species (Rushmer, 1965). Neurogenic influences converge on the sino-atrial pacemaker and consist of parasympathetic slowing and sympathetic speeding effects, with parasympathetic control predominating. Control of the heart by centrally-organized processes, and peripherally by baroreceptor and chemoreceptor activities, are mediated over these pathways. It should be noted that fluctuations in heart rate in human subjects are closely related to somatomotor activity (Obrist et al., 1970). Cardio-somatic coupling is maintained primarily by modulation of parasympathetic outflow (Freyschuss, 1970a) and is normally evident except under conditions of extreme stress, where sympathetic effects of beta-adrenergic origin are potentiated and produce heart rate changes that may be temporarily inappropriate for concurrent somatomotor arousal (Obrist et al., 1974).

Electrodermal activity is recorded from palmar surfaces, and is generated primarily by sweat glands situated in these surfaces. Unlike cardiac muscle, the sweat glands are innervated only by the sympathetic branch

of the autonomic nervous system. The neuro-transmitter in the post-ganglionic sudomotor fibre is acetylcholine. The activity of the sweat glands may be measured by recording the transcutaneous potential (skin potential), or by measuring the conductivity of the volar surface (skin conductance). Regardless of the method of measurement, electrodermal activity manifests both a tonic and a phasic component. Phasic activity in turn may be assessed by a variety of discrete measures, such as rise time, recovery time, amplitude, and frequency, which reflect (preferentially to some extent) active secretion and reabsorption of sweat in palmar and plantar skin (see Edelberg, 1972a for a review). In this thesis, electrodermal activity was recorded as skin conductance. Conductance was sampled every five seconds. This measure was sensitive to changes in both tonic and phasic activity, although it did not distinguish between the two, and can be assumed to have reflected peripheral events that increased the hydration of the epidermal surface, including an increase in secretion, an inhibition of reabsorption, or both (Edelberg, 1972a).

Palmar and plantar sweating and reabsorption, and thus electrodermal activity, are influenced to some extent by changes in ambient temperature. Exposure to extreme temperatures provokes volar sweating, but provokes considerably more sweating in non-volar areas (Wilcott, 1963; 1967). In the absence of intense thermal stimulation, volar glands do not seem to be influenced by thermoregulatory processes to any great extent. Moreover, correlations between electrodermal changes and room temperature have generally been found to be low, as have correlations between electrodermal changes and humidity (Edelberg,

1972a; Wenger and Cullen, 1962). Thus, thermoregulatory functions, although they may exert considerable influence on non-volar sweating, do not appear to be a major determinant of electrodermal behaviour, at least under typical psychophysiological conditions. Rather, electrodermal activity appears embedded within two other neural systems. First, there is evidence that electrodermal changes are related to changes in somatomotor activity (Culp and Edelberg, 1966; Freeman, 1938; Pinneo, 1961; Roberts, 1974). And second, there is also some evidence that electrodermal changes are subject to the influence of a non-motor arousal process (Roberts, 1974; Roberts and Young, 1971). While this process appears to play an important role in electrodermal functioning, its nature is as yet poorly understood. However, there is some suggestive evidence that this non-motor process may be manifest in terms of increased secretion and inhibition of reabsorption, and that the recovery limbs of electrodermal phasic responses may be particularly sensitive to the latter process (Edelberg, 1970; 1972b).

There are at least two important differences in the functional organization of the electrodermal and cardiac control systems. First, although both somatomotor and non-motor arousal processes have been shown to influence the activity of both systems, non-motor influences appear to be more important determinants of electrodermal responding than of heart rate responding under most circumstances (Roberts, 1974). Second, the two response systems appear to differ with respect to the afferentation arising as a direct consequence of effector activity. Interoceptive feedback consequent on the occurrence of discrete heart beats may arise from a variety of possible sources, including

mechano-receptors in muscle and the overlying skin, baroreceptors in the carotid sinus and the aortic arch, and possibly also from the myocardium and arteries themselves. There is, on the other hand, no afferentation arising directly from myoepithelial contraction in the sweat glands (Wang, 1964). There is reason to suspect that the activity of other sense organs in skin may be influenced by extreme variations in epidermal hydration that may be consequent to sudomotor activation (Edelberg, 1972a), but it is unlikely that these effects are substantial over the range of electrodermal responding typically observed in psychophysiological experiments.

Plan of the Thesis

The organization of this thesis is as follows. The next chapter reviews what is presently known about the contribution of instructions, strategy information, and feedback to voluntary control of skin conductance and heart rate. This chapter also reviews the available data pertaining to correlates of voluntary control over these two autonomic responses. The chapter which follows presents the first experiment, which was designed primarily to develop and test a procedure for bringing skin conductance and heart rate under voluntary control.

The major empirical findings of the thesis lie in the second experiment, which is presented in Chapter 4. This experiment was designed specifically to evaluate the role of instructions, strategy information, and feedback in voluntary cardiac and electrodermal control. It also examined the response profiles generated when skin conductance and heart rate were controlled voluntarily, and examined whether these profiles were dependent on how training was carried out. Finally, the

experiment examined whether voluntary control of skin conductance and heart rate was affected differently by the different training variables, and considered the extent to which the response profiles associated with the two target responses overlapped.

The last chapter of the thesis examines the bearing of the results on the central issue of how voluntary control is established over electrodermal and heart rate responding. This discussion summarizes the role of instructions, explicit strategy suggestions, and feedback in the development of voluntary control over electrodermal and heart rate responding, considers data pertaining to the neural systems that are involved in performance of the target responses, and develops an account of how subjects learn to control an internal response as a result of feedback training.

Chapter 2: Review of the Literature

The previous chapter distinguished three features of a typical voluntary control procedure. First, subjects are instructed to increase or decrease a particular autonomic response. These instructions contain both a directional requirement and a reference to a particular response system. Second, subjects may also be told about response strategies that may facilitate performance. Although not a central feature of the voluntary control paradigm, such information has been provided in experiments on voluntary control of electrodermal responding, in which subjects have typically been asked to think emotional thoughts on increase trials, and to refrain from such activity on decrease trials (for example, Klinge, 1972). Third, subjects are given exteroceptive feedback when they comply successfully with the instructions that have been given.

This chapter reviews what is presently known about the contribution of these three features to voluntary control of electrodermal and heart rate responding. Also reviewed in the chapter are data pertaining to the behavioural correlates that occur when voluntary control is established over these response systems. The problems of determinants and correlates of voluntary control are considered separately in the sections that follow. The review begins with the electrodermal system and proceeds to heart rate.

Determinants of Voluntary Electrodermal Control

Instructions

It is probably fair to say that most subjects in psychological experiments are only dimly aware, if at all, that they generate electrodermal activity. Hence, there may not be much point in looking at the effects of instructing subjects to try to exert control over a system of which

most of them are ignorant. However, given that electrodermal behaviour reflects primarily palmar sweat gland activity, and that most subjects are probably aware of what sweating is, it seems reasonable to examine the effects of instructions to produce increases and decreases in palmar sweating activity. Unfortunately, there does not appear to be a single published report in which electrodermal activity was examined while subjects attempted to comply with instructions to try to produce increases or decreases in either their electrodermal behaviour or palmar sweating. There is one experiment in which the degree of control over electrodermal behaviour was compared between a group which received instructions to decrease electrodermal activity (not "sweating") and a group which did not receive these instructions (Quy and Kubiak, 1974). However, the effect of instructions was confounded by the presentation of random electric shocks (these groups served as yoked controls for groups provided with shock-feedback for electrodermal changes). The electrodermal performance of subjects provided with instructions was not reliably different from that of subjects not so provided.

Strategies

There is evidence which suggests that electrodermal behaviour can be modified through the suggestion of appropriate strategies. In fact, a whole body of literature has concerned itself with the effects on electrodermal activity of varying instructions to subjects in conditioning experiments (see Grings, 1973, for a review). However, it is very difficult to pinpoint exactly the nature of the relevant strategies. The problem is complicated by the fact that different investigators have used different features of electrodermal activity as dependent variables. Hence, there

is the possibility that the strategies which a given investigator deduces to be important determinants of electrodermal behaviour might be feature-specific, e.g. might have an effect on recovery rate, but not on rise time, amplitude, frequency, or tonic level. Despite these difficulties, there has been no dearth of attempts to tie electrodermal activity with various psychological processes. Both co-discoverers of electrodermal behaviour, Féré and Tarchanoff, believed electrodermal behaviour to have a basis in emotions (Neuman and Blanton, 1970). Although this view enjoyed a good deal of popularity, it was by no means universally accepted:

> In an attempt to classify statements which various investigators have made concerning the psychological significance or the psychological concomitants of this reflex, I have formulated the following résumé: forty investigators hold that it is specific to or a measure of emotions or the affective qualities; ten others state that it is not necessarily of an emotional or affective nature; twelve men hold that it is somehow to be identified with conation, volition, or attention, while five hold very definitely that it is non-voluntary; twenty-one authorities state that it goes with one or the other of the higher mental processes; eight state that it is the concomitant of all sensation and perception; five have called it an indicator of conflict and suppression; while still four others have used it as an index of character, personality or temperament. (Landis, 1930, p. 391).

Nevertheless, the notion that electrodermal activity is related to emotional arousal, and that changes in the level of emotional arousal should produce changes in the level of electrodermal behaviour, is supported by the literature on voluntary control. Most investigators of voluntary control over electrodermal activity have told their subjects to "think emotional thoughts" when trying to produce increases, and to "relax" when trying to produce decreases in electrodermal behaviour. Shean (1970) reported one of three studies in which such emotional strategies were explicitly manipulated while establishing control over electro-

dermal behaviour. A discrimination procedure was utilized, in which subjects could avoid delivery of an electric shock during presentation of one stimulus by producing a phasic increase in electrodermal activity, and during presentation of another stimulus by inhibiting phasic electrodermal activity. Subjects in the experimental group were told that they could avoid delivery of the shock by, respectively, having frightening thoughts and remaining calm. Subjects in the control group were given the same avoidance contingency, but without the instruction containing strategy information. While experimental subjects were found to produce reliable differences between respond and inhibit conditions, control subjects were not.

Another study (Stern and Kaplan, 1967) included a group of subjects who were told that they should try to control their electrodermal activity, and that they could do this by thinking of emotional events on Raise trials, and by relaxing on Lower trials. No feedback was provided. Unfortunately, the investigators did not provide statistical comparisons between the behaviour of these subjects on Raise trials, and their behaviour on Lower trials. However, visual examination of the published figures indicates that substantial differences in frequency of phasic electrodermal activity were obtained between Raise and Lower trials in these subjects.

Finally, Klinge (1972) instructed her subjects to "think arousing thoughts" and to "relax" during presentations of appropriate stimuli. Subjects were observed to produce a greater frequency of electrodermal responses during presentation of the "think" stimulus than during presentation of the "relax" stimulus. The difference between "think" and "relax" trials was statistically reliable.

In sum, it appears that simply telling subjects to think emotional thoughts and to relax is sufficient to bring about differences in electrodermal activity. Whether these differences are of greater magnitude than differences which could be produced by simply instructing subjects to "sweat" and "not sweat" is not known. Moreover, whether these differences can be maintained for significant durations and over a large number of trials also remains to be answered. Klinge did not report on these questions. In the Stern and Kaplan study, the magnitude of the difference in electrodermal activity between Raise and Lower trials diminished over time, while in Shean's experiment the magnitude of the difference between Raise and Lower trials increased over trials.

In addition to the emphasis on strategies based on "emotional arousal", interest has also been shown in strategies based on somatomotor activity. While somatic strategies have not been specifically manipulated in studies of voluntary control over electrodermal behaviour, there is evidence which suggests that these might play an important role in controlling electrodermal behaviour. For example, Pinneo (1961) observed that level of palmar electrodermal activity changed as a function of the level of muscular activity, as measured by recorded pressure on a hand dynamometer, and confirmed this relationship by electromyographic measurements. Nor does palmar electrodermal behaviour appear related only to the activity of the muscle fibers in close proximity to the sweat glands generating palmar electrodermal activity. Freeman (1938) monitored palmar electrodermal activity in subjects engaging in various tasks while varying the amount of weight on their lower limbs (from 0 to 80 pounds). Level of electrodermal arousal was observed to increase as a function of the level of tension in the legs, regardless

of the task in which the subjects were engaged. These and similar data suggest that electrodermal activity is related to general somatomotor activity (or its central nervous system components) in Man. However, Culp and Edelberg (1966) have reported evidence that there is at least some degree of specificity to muscle groups in electrodermal behaviour. Muscular activity of either the foot or the hand produced greater relative electrodermal arousal in the ipsilateral limb than in the contralateral limb.

These data suggest a close relationship between electrodermal and motor functions. In addition, there are two experiments which investigated the relationship between respiratory variables and electrodermal behaviour. In the first, Stern and Anschel (1968) noted that isolated deep breaths produced consistent increases in skin conductance, and that skin conductance typically took longer than 20 seconds to return to baseline. In the other, Gavalas (1968) noted that changes in respiratory variables did not necessarily lead to electrodermal changes. She rewarded phasic electrodermal responses, but only when these were associated with deep inspirations. While an acquisition function was observed with respect to deep inspirations, no such function was observed with respect to electrodermal responses. The frequency with which deep inspirations elicited electrodermal responses diminished over time.

Feedback

There appears to be three reports in which the degree of control over electrodermal behaviour when feedback was presented was compared with the degree of electrodermal control in the absence of feedback. In all cases, explicit strategy information was provided in the control condition. Feedback effects were examined by contrasting performance on training trials,

on which exteroceptive feedback was present, with performance on control trials, where subjects attempted to comply with the strategy suggestions alone.

Of the three published reports, the one by Klinge (1972) was the most extensive and the one which was the most methodologically sound. In two very similar experiments Klinge reported reliable effects of feedback training. A between-subjects design was employed, in which all subjects were told to think arousing thoughts during discrete, one minute, Raise trials, and to relax during discrete, one minute, Lower trials. Raise and Lower trials alternated. Experimental subjects were provided with feedback on their electrodermal behaviour via a meter, whereas control subjects received no such feedback. While both groups were able to generate differences in the frequency of electrodermal responding between Raise and Lower trials, these were reliably greater for experimental subjects than for control subjects. This result was found in both of Klinge's experiments.

On the other hand, there are two reports in which facilitation of electrodermal control through feedback was not observed. In a study by Stern and Kaplan (1967), all subjects were told to think of emotional thoughts during 10-minute Raise trials, and to relax during 10-minute Lower trials. Experimental subjects received feedback for their performance, while control subjects did not. Both groups generated electrodermal differences between Raise and Lower trials. However, although experimental subjects produced more electrodermal responses than control subjects during the Raise condition, they also produced more electrodermal responses during the Lower condition, and it is quite evident that the magnitude of the difference in electrodermal activity between Raise and Lower trials was approximately the same in both groups. Hence, provision of feedback did not appear to facilitate control (this observation is based on visual analysis

of the published figures, since the investigators did not report the appropriate statistical comparisons).

Finally, Chalmers (1970) employed a within-subjects, unidirectional design in which subjects were asked to try to increase either the amplitude or the frequency of their electrodermal activity, and were told that they could do this by thinking of emotional events. Feedback was provided by a meter. The subjects' performance during feedback presentation was compared to their performance during a preceding adaptation period. No difference in electrodermal activity was observed between feedback and non-feedback periods. However, feedback and temporal variables were confounded in this experiment, so as to weigh against obtaining feedback effects, since any effect of feedback would not only have to counter, but also to surpass any habituation effects (Crider, Schwartz, and Shnidman, 1969).

In sum, of three investigations on the contribution of feedback to voluntary control over electrodermal activity, one (Klinge, 1972) reported that feedback facilitated control, while two others (Chalmers, 1970; Stern and Kaplan, 1967) reported that it did not. It should be noted that there is a methodological difference between the positive report and the negative ones. Klinge employed discrete one-minute trials, while in both the Stern and Kaplan and the Chalmers studies feedback was presented continuously for ten minutes. It is possible that feedback effects may be more evident in experiments utilizing discrete trials of relatively short duration.

The foregoing review has examined feedback effects primarily within the context of voluntary control paradigms. Studies of operant conditioning, on the other hand, have shown that experience with a feedback contingency can result in operant control over electrodermal functions (see Kimmel, 1973, for a review). However, these studies did not inform the subjects

of the response to be brought under control to begin with, nor did they evaluate the ability of subjects to control the target behaviour prior to conditioning. Thus these studies cannot comment on whether feedback is necessary for voluntary electrodermal control, or whether its provision enhances the degree of control that can be produced in response to instructions prior to training. Experiments on voluntary control have been concerned primarily with the last two problems.

Summary

Present data on the role of instructions, strategies, and feedback, in the voluntary control of electrodermal activity may be summarized as follows. The effect of instructions simply to increase or decrease palmar sweating does not appear to have been assessed. However, instructions that include explicit strategy suggestions of an emotional nature have been reported to produce reliable electrodermal changes. The extent to which these changes are attributable to the instructions to increase or decrease a response or to the strategies that were suggested cannot be determined from the data presently available. The role of feedback in voluntary control is also unclear. Only three investigations appear to have examined feedback effects. The more extensive and adequate of these studies (Klinge, 1972) reported better control with the provision of feedback than without it, whereas the other two did not. Thus, there is reason to expect that provision of feedback facilitates control of electrodermal responding, although the available data are limited and somewhat contradictory.

Correlates of Voluntary Electrodermal Control

This section examines the literature pertaining to the correlates of voluntary control over electrodermal activity. The studies which are reviewed here have met two criteria. First, they have reported reliable

evidence that at least one feature of electrodermal behaviour was brought under control. Second, they have provided data on the relationship of electrodermal responding to at least one other variable.

Unfortunately, there is a dearth of evidence on the correlates of electrodermal control. There appears to be only one experiment specifically concerned with voluntary control over electrodermal activity in which concomitant measures were recorded (Klinge, 1972). However, there is a number of experiments concerned with operant control over electrodermal behaviour in which variables other than electrodermal activity were measured. Results from both types of study will be presented here. It should be noted that the correlates of electrodermal control reported in operant studies may not be the same as those found in studies of voluntary control, in which subjects are informed of the response that is to be controlled prior to feedback training and are asked to produce changes in a specified direction.

Autonomic correlates

Klinge (1972) measured heart rate while subjects were attempting to produce different levels of electrodermal behaviour on discrete Raise and Lower trials. On Raise trials, subjects were told to think emotional thoughts, and watched a meter analog of their electrodermal activity; on Lower trials they were told to relax, and were also provided with meter feedback. Klinge found heart rate to be reliably higher on Raise than on Lower trials.

There are also several reports in which heart rate was monitored while attempts were made to bring electrodermal behaviour under operant control. Martin, Dean, and Shean (1968), in a shock avoidance study, noted

that heart rate was more elevated on trials when increases in electrodermal activity were required than on trials when decreases were required. However, this observation was based on only four subjects. Other investigators have failed to find a correspondence between electrodermal activity and heart rate in similar experiments employing positive rather than negative reinforcement (Crider, Shapiro, and Tursky, 1966; Gavalas, 1967; Schwartz and Johnson, 1969; and Shapiro and Crider, 1967).

Somatomotor activity

Rice (1966), and VanTwyver and Kimmel (1966), in experiments concerned with operant control of electrodermal behaviour in noninstructed subjects, observed no relationship between electromyographic activity and electrodermal behaviour. Crider, Shapiro, and Tursky (1966) recorded gross body movement in similar experiments, and also failed to detect a relationship between electrodermal activity and movement.

Respiration

It appears that all studies reporting on respiratory correlates of electrodermal control have focussed on frequency rather than amplitude of respiration. Klinge (1972), in a study of voluntary control, observed that subjects breathed faster on Raise trials, where they were told to think emotional thoughts and provided with electrodermal feedback, than on Lower trials, where they were told to relax and also provided with feedback. However, correlations computed between electrodermal changes and changes in respiration frequency were statistically unreliable.

Other investigators have reported on the relationship between electrodermal behaviour and respiration frequency in electrodermal operant conditioning situations involving noninstructed subjects, and have found that the two did not covary (Crider, Shapiro, and Tursky, 1966; Schwartz and Johnson, 1969; Shapiro and Crider, 1967; and VanTwyver and Kimmel, 1966). Finally,

Gavalas (1968), in an experiment reported earlier in another context (p.24), rewarded phasic electrodermal responses which followed deep inspirations. Contrary to expectations, she observed an increase in the frequency of deep inspirations generally, but a decrease in the frequency of deep inspirations which elicited electrodermal responses, over the course of training.

Summary

In sum, correlated changes in somatomotor and autonomic variables have been observed when electrodermal responding was brought under operant and voluntary control. Although the data are sparse, there appears to be a tendency for these correlates to be more prominent in voluntary control studies (Klinge, 1972) than in studies of operant conditioning (for example, Crider et al., 1966). However, data on the nature of the response correlates and on the conditions that are conducive to them are generally lacking. Nor are there sufficient grounds to decide whether response correlates are merely concomitants of voluntary electrodermal changes, as might be the case were they attributable to the up-down component of verbal instructions, or whether they are necessary for performance of the voluntary electrodermal change.

Determinants of Voluntary Cardiac Control

While the literature dealing with the experimental determinants and with the correlates of voluntary electrodermal control was sparse, and permitted only tentative conclusions, the literature pertaining to the determinants and correlates of voluntary heart rate control is more substantial. As in the first half of this review, attention will focus first on the experimental determinants of voluntary control over heart rate, and will then turn to an examination of the correlates of cardiac control.

Instructions

Several experiments have examined the effects of simply instructing subjects to raise and lower their heart rate. Before examining the results of these studies, however, a methodological point should be made. It appears that only one experiment has actually investigated the effects of instructions to control heart rate in the absence of constraints on movement, or respiration, or both, and the report of that experiment was informal (Brener, 1974a). Brener noted that heart rate differences of seven beats per minute were generated between trials when subjects were attempting to increase their heart rate and trials when they were attempting to decrease it. All other studies which have examined the effects of instructions to raise and lower heart rate appear to have done so in situations in which the subject's behaviour was constrained considerably, usually by further instructions not to engage in motor or respiratory maneuvers. It should be noted that Brener (1973) and Obrist et al. (1975) have reported that the degree of control evidenced over heart rate is inversely related to the extent to which somatomotor and respiratory maneuvers are discouraged by verbal instructions. Thus, the extent to which heart rate can be changed following verbal instructions without prior feedback training appears to have been underestimated in most studies.

Despite the attenuating effect of somatomotor constraints, it has generally been found that subjects are capable of controlling their heart rate when asked to do so. Brener, Kleinman, and Goesling (1969) were the first to report on the behaviour of subjects asked merely to attempt to produce heart rate changes. They observed that their subjects were able to produce mean heart rate differences of two to three beats per minute between trials when they were to slow their heart rate (Lower trials)

and trials when they were to speed their heart rate (Raise trials). Brener (1974a) subsequently corroborated these data, reporting heart rate differences of four to five beats per minute between Raise and Lower trials. Neither of these reports specified whether the difference in heart rate between Raise and Lower trials was due to a speeding of heart rate on Raise trials, a slowing on Lower trials, or both.

Bergman and Johnson (1971) examined the issue with a between-subjects design in which subjects in one group were asked to try to raise their heart rate, and subjects in another group were asked to try to lower their heart rate during discrete trial periods. Differences of two to three beats per minute were observed between Raise and Lower trials, and these were found to result mostly from raising of heart rate on Raise trials. In a partial replication of this study, Bergman and Johnson (1972) obtained mean increases of one beat per minute in a group of subjects told to try to speed their heart rate, but no decrease group was included. While these differences in heart rate between Raise and Lower trials are lower than those obtained in Brener's studies, the procedures used by the two groups of investigators were different. Brener's results were obtained with a within-subjects design, whereas Bergman and Johnson utilized a between-subjects design. Furthermore, Bergman and Johnson had subjects pace their respiration, while Brener merely asked his subjects not to move, and not to engage in respiratory maneuvers. Finally, Brener's trials lasted 50 inter-beat intervals, while in the first Bergman and Johnson experiment trials lasted 6 inter-beat intervals, and in the second, they were 20 seconds in duration.

Blanchard and Young (1972) investigated the behaviour of subjects asked to attempt to raise and lower their heart rate during randomly

presented 60-second trials, and observed mean heart rate differences of two to five beats per minute between Raise and Lower trials. Moreover, these appeared to result mostly from lowering on Lower trials. These findings were confirmed in two subsequent investigations (Blanchard et al.,1974b; Young and Blanchard, 1974), in which no evidence of raising was obtained, while mean decreases of two beats per minute were produced in response to instructions.

Levenson (1974), also using a within-subjects design, reported mean heart rate increases of three beats per minute, and decreases of two beats per minute on Raise and Lower trials, respectively. Similarly, Ray (1974) observed both raising and lowering of heart rate of approximately three beats per minute. Finally, Schwartz (1974) reported informally that his subjects could raise their heart rate by a mean of 7.5 beats per minute, and lower it by a mean of 0.5 beat per minute, but no procedural details were given.

Thus, it is clear that instructions to increase and decrease heart rate generally result in appropriate heart rate changes. It was noted earlier that the instructions typically given to subjects in voluntary control studies specify both the direction of desired changes, and the response system to be brought under control. Brener and Goesling (1968, cited in Brener, in press) examined whether the directional requirement alone is sufficient to produce appropriate heart rate changes. Subjects were provided with heart rate feedback consisting of high-pitched tones for short inter beat intervals (high heart rates), and low-pitched tones for long inter beat intervals (low heart rates). Some subjects were required to attempt to increase their heart rate, while others were required to attempt to decrease their heart rate. The subjects

were not informed that heart rate was the response to be controlled. They either were told that their task was to produce high- (or low-) pitched tones, or that their task was to inhibit high- (or low-) pitched tones. Subjects for whom the directional requirement was consistent with the require response (producing high-pitched tones and increasing heart rate, and inhibiting high-pitched tones and decreasing heart rate) generated larger heart rate changes in the desired direction than did subjects for whom the directional requirement was inconsistent with the required response (inhibiting low-pitched tones and increasing heart rate, and producing low-pitched tones and decreasing heart rate). Since the groups did not differ reliably with respect to the number of success and failure signals which were delivered, it was concluded that differences between the groups were due to the directional component of the instructions. Similar results have been reported by Bouchard and Corson (1975; cited in Brener, in press). Unfortunately, the magnitude of the heart rate changes produced through directional manipulations in these experiments was not specified.

In addition to these studies on directional specifications, some attention has also been paid to the other component of the instructions, the reference to a given response system. Blanchard et al. (1974a) instructed one group of subjects to try to control heart rate, another to try to control skin conductance, and a third to try to control some unspecified physiological response. All groups were provided with heart rate feedback. The best heart rate performance was observed in the group given heart rate instructions. Thus, both the directional and the reference components of the instructions appear capable of influencing heart rate control.

If subjects are capable of controlling their heart rate in response to instructions alone, does this control increase or diminish over time, in the absence of feedback training? The results on this point are conflicting. Levenson (1974) reported a statistically reliable improvement over trials, within a single session, in the degree of heart rate control, on both Raise and Lower trials. Brener, Kleinman, and Goesling (1969) also reported a reliable improvement in heart rate control over trials, this time over two training sessions. Brener (1974a) subsequently replicated this effect. However, in none of the studies originating in Blanchard's laboratory was a reliable improvement in heart rate control observed between sessions, in subjects given instructions alone (Blanchard and Young, 1974b; Young and Blanchard, 1974).

In sum, it is evident that subjects can voluntarily control their heart rate in response to instructions alone. Bi-directional heart rate differences averaging two to five beats per minute are typically produced when subjects are asked to refrain from engaging in motor and respiratory maneuvers. Moreover, it appears that larger heart rate differences can be produced when no constraints are placed on movement and respiration. Part of these changes can be ascribed separately to the directional component of the instructions and to the reference to heart rate, but the relative importance of these components is as yet undetermined. Also remaining at issue is the question of whether the degree of heart rate control changes over trials during the course of a session, or over sessions. Finally, the question of why some investigators obtain evidence of both increases and decreases in heart rate while others find evidence only of increases or only of decreases, also remains unsettled.

Strategies

As in the case of electrodermal behaviour, extensive data indicate that heart rate can be modified by instructions to execute specific

response strategies. Two kinds of strategies which have been shown to be of particular relevance are alterations in respiratory parameters and in gross somatic activity. The importance of these strategies can be inferred from studies in which subjects were asked to engage in respiratory or somatic maneuvers while their heart rate was measured.

Westcott and Huttenlocher (1961) examined the heart rates of subjects who were systematically manipulating depth and frequency of respiration. It was observed that variability in the heart rate record was related to changes in the respiratory parameters. The largest changes in heart rate were produced when respiration was at its deepest and slowest, and heart rate variability was found to be an inverse function of the amplitude of respiratory cycles, and of respiratory rate. The effects of the respiratory maneuvers on mean heart rate were not assessed. Westcott and Huttenlocher also observed that some specific irregular respiratory activities (e.g. gasps) were often accompanied by consistent changes in heart rate.

Engel and Chism (1967a) extended these observations by measuring both mean heart rate and heart rate variability as a function of changes in respiratory rate. Westcott and Huttenlocher's results were replicated. Heart rate variability was found to be inversely related to rate of respiration. However, changes in respiration rate did not affect mean heart rate.

Sroufe (1971) further extended these findings by examining mean heart rate and heart rate variability when both depth and rate of respiration were manipulated. He confirmed that heart rate variability was inversely related to respiratory rate, and that changes in respiration rate did not affect mean heart rate. On the other hand, both

heart rate variability and mean heart rate were affected by changes in depth of respiration: the deeper the breath, the faster and the more variable was the heart rate. By appropriately altering respiratory parameters, mean heart rate differences of up to 12 beats per minute could be generated between different conditions. These observations, which were based on adults, were subsequently replicated in children (Sroufe and Morris, 1973). Finally, Brener (1974a) observed heart rate differences of 8 beats per minute between a condition in which subjects were asked to hyperventilate and a condition in which they were asked to hypoventilate.

All these studies examined the effects of tonic as opposed to phasic respiratory maneuvers on heart rate. Stern and Anschel (1968), on the other hand, examined the effects of isolated deep breaths of varying rates on heart rate. They noted that the cardiac response to an abnormal breath consists of an increase in heart rate followed by a decrease. The acceleratory component of this response was not affected by whether these deep breaths were fast or slow. However, slow breaths produced larger and more sustained decelerations than did fast breaths.

In addition to these experiments in which heart rate was observed while subjects were simply asked to produce changes in respiratory functions, there is one study in which respiration was manipulated while subjects were attempting to control their heart rate (Sroufe, 1971). In this case, the focus was on heart rate variability. The subjects' task was to maintain their heart rate within a specified range. Subjects in the experimental group were told that their task was to control their heart rate through the use of respiration, and they were instructed to practise various respiratory maneuvers while watching a display

of their cardiac activity during a 2-minute "demonstration" period. Subjects in the control group received no such "demonstration" period, and were simply told to try to control their heart rate, but to breathe normally. Both groups then received five minutes of training during which they tried to maintain their heart rate within the designated range, and were provided with feedback on their performance. This training period was followed by a 5-minute transfer period during which feedback was discontinued. Subjects who received practice with respiratory strategies were observed to exert better control over their heart rate than subjects in the control group, during both training and transfer periods. Although these results suggest that provision and practice with respiratory strategies facilitated heart rate control, it is also possible that the difference between the experimental and control groups was due simply to the extra two minutes of practice in the experimental group (training lasted only five minutes), or to some interaction between strategies and feedback. Moreover, even if the experimental-control difference did indeed stem from the strategies, it is not clear whether this came about through mere provision of strategies, or through practice with these strategies.

In summary, evidence indicates that changes in respiratory activity can produce reliable changes in heart rate and heart rate variability. The magnitude of the changes in mean heart rate which can be produced by manipulating respiratory amplitude and frequency (8 to 12 beats per minute between increase and decrease conditions) appears to exceed somewhat the magnitude of the changes in heart rate which are produced when subjects are told to increase and decrease their heart rate, and

are not allowed to engage in respiratory maneuvers.

Another factor which has been found to affect heart rate reliably is muscular activity. Investigations of the relationship between heart rate and somatic activity fall in one of two categories. First, an extensive literature has developed, since the turn of the century, concerned with the effects of physical exercise on a number of cardiovascular parameters, including heart rate. There is no question that increases in motor activity produce marked increases in heart rate. As an example, Freyschuss (1970a,b) investigated the effects of elbow flexion and handgrips on heart rate. Onset of muscular activity generated an immediate increase in heart rate, and heart rate increased by an average maximum of 18 beats per minute in one study and 19 beats per minute in the other, during muscle contraction. Moreover, it was shown that these changes in heart rate were not secondary to respiratory maneuvers.

A second stream of investigations has examined the relationship between heart rate and motor activity in circumstances in which large changes in motor activity were not produced by instructions, as in exercise, but rather were a by-product of other experimental manipulations. Obrist and his colleagues (see Obrist et al., 1970) have performed an extensive series of experiments in which the cardio-somatic relationship was examined while events of a motivational, arousal, and attentional nature were manipulated by means of reaction time and classical conditioning procedures. Nonsomatic influences on heart rate were found to be minimal: changes in heart rate were related primarily to changes in motor activity. Other investigators have been led to essentially the same conclusions, both in human (Cohen and Johnson, 1971)

and animal preparations (Black and deToledo, 1972; Roberts and Young, 1971). The only clear exception to these results is that heart rate may be dissociated from motor activity when subjects are exposed to severe electric shock. Under these conditions, control of the heart by sympathetic non-motor influences is potentiated (Obrist et al., 1974).

Finally, there is one published study in which gross somatomotor activity (or, rather, the prevention of such activity) was manipulated as an independent variable while an attempt was made to bring heart rate under operant control (Obrist et al., 1975). Although the subjects in this study were not told specifically to try to control their heart rate, the results are relevant to the question of the importance of strategies, and therefore will be reported here. An avoidance procedure was employed, in which subjects could avoid delivery of a mild electric shock (and receive a monetary bonus) by producing heart rate changes of desired direction and magnitude during a one-minute trial period. For three groups of subjects, increases in heart rate were reinforced, whereas decreases in heart rate were reinforced for subjects in two other groups. For two of the groups (one increase and one decrease) no attempt was made to maintain control over somatic activity. For two more groups (also one increase and one decrease) somatic activity was controlled through both instructions not to move and the use of paced respiration. Finally, for the fifth group, which was rewarded for heart rate increases, somatic activity was controlled through instructions but not paced respiration. It was observed that the magnitude of heart rate control was inversely related to the extent to which somatic activity was restrained when increases in heart rate were reinforced. With no restrictions on somatic activity, mean heart rate

increases of up to 20 beats per minute were obtained; with maximal restrictions, these increases ranged between 4 and 8 beats per minute. For the decrease groups, mean decreases of up to 4 beats per minute were observed, and these were somewhat greater in the subjects whose somatic activity was restricted than in those who were free to move.

In sum, it is clear that changes in respiratory function and gross motor activity are related to changes in heart rate. Moreover, there are indications that the degree of control which subjects can exert over their heart rates is diminished if respiratory and motor strategies are prevented. The question of whether voluntary control of heart rate is augmented by provision of information relating to somatomotor strategies has not been investigated.

Feedback

One training variable which has received considerable attention, however, is exteroceptive feedback. The effect of feedback on heart rate control has typically been investigated by comparing a condition in which instructions and feedback were provided with a condition in which instructions alone were provided. If subjects can control their heart rate in response to instructions, does adding feedback to these instructions improve performance?

There appear to be twelve studies that have included a comparison between a condition in which subjects are simply told to try to increase or decrease their heart rate, and a condition in which feedback was provided for such increases and decreases. In all of these, movement and respiratory maneuvers were specifically prohibited. Seven of the twelve studies reported better heart rate control with feedback than without it. Brener, Kleinman, and Goesling (1969) instructed

subjects to try to raise their heart rate during presentation of one visual stimulus, and to try to lower their heart rate during presentation of another stimulus. One group of subjects received auditory feedback on their heart rate during training, while another group received no feedback. Subjects in the feedback group performed consistently better than subjects in the no-feedback group, showing greater heart rate differences between Raise and Lower conditions (6 beats per minute versus 3 beats per minute). Similar results were obtained in a subsequent experiment by Brener (1974a). Subjects told simply to try to produce heart rate differences generated mean heart rate differences of five beats per minute between Raise and Lower trials. Subjects given the same instructions, and also given feedback on their heart rate, generated mean heart rate differences of 15 beats per minute between Raise and Lower trials. However, neither Brener (1974a) nor Brener et al. (1969) specified whether this control was generated primarily on Raise trials, on Lower trials, or whether the magnitude of the control was similar on Raise and Lower trials.

Blanchard et al. examined this question of whether feedback was differentially effective on Raise and Lower trials, and in two different experiments (1974a and b) observed that feedback had an effect on both increases and decreases in heart rate. They compared the behaviour of subjects receiving instructions to increase and decrease heart rate with that of subjects who received the same instructions, and who were also provided with feedback. Subjects receiving feedback produced reliably larger heart rate changes on both Raise and Lower trials than did subjects who did not receive feedback (on Raise trials: 5 beats per minute versus 2 beats per minute; Lower trials: 4 beats per minute versus 1 beat per minute).

Blanchard and Young (1972), using a similar design, also obtained evidence for a feedback effect. However, in this case, the effect was manifest only on Raise trials. Subjects receiving both instructions and feedback generated mean increases of 6 beats per minute on Raise trials, while subjects receiving instructions but no feedback did not generate any heart rate change on Raise trials. Heart rate changes produced on Lower trials were of similar magnitude (3 beats per minute). Very similar findings were obtained in a subsequent replication (Young and Blanchard, 1974).

Finally, Ray (1974) also reported a facilitation of heart rate control by feedback. In this case, a within-subjects design was employed in which subjects first received four Raise and four Lower trials during which they were asked to try to alter their heart rate in the appropriate direction, without benefit of feedback. This was followed by four more Raise and Lower trials, during which feedback was presented. The results indicated that feedback improved performance, but that this effect was statistically reliable only on Raise trials.

In each of the foregoing studies, feedback was observed to have an effect beyond instructions on voluntary control of heart rate. Of the seven experiments reviewed, three reported that feedback improved heart rate control only on Raise trials, two others noted that feedback affected performance on both Raise and Lower trials, and the last two reported reliable effects of feedback presentations, but did not specify whether these occurred on Raise trials, on Lower trials, or on both types of trial. However, there appears to be a total of five studies in which feedback was not observed to facilitate heart rate control. One of these (Levenson and Strupp, 1972) used a design and parameters

almost identical to those utilized by Ray (1974). Nevertheless, Levenson and Strupp found that adding feedback to instructions to try to control heart rate did not improve performance.

Other failures to obtain feedback effects were reported by Bergman and Johnson (1972), Johns (1970), Levenson (1974), and Manuch et al. (in preparation), all of whom employed between-subjects designs. In Levenson's study, subjects were required to breathe through a mouthpiece, and this might have interfered with the development of feedback-mediated heart rate control (although use of the mouthpiece did not interfere with the voluntary production of changes in heart rate in the absence of feedback). Otherwise, there do not appear to be systematic or methodological differences between studies reporting positive results and studies reporting negative results. Neither the number of training trials, nor trial duration, duration of habituation before training, type of feedback, or instructions regarding respiratory and motor nameuvers systematically differentiated between experiments in which feedback effects were observed and experiments in which feedback effects were not observed. Why feedback has an effect on heart rate control beyond instructions under some circumstances but not under others is unclear.

The studies just reviewed provide evidence of feedback effects, although the reasons for discrepant findings are not apparent. Further evidence for feedback effects comes from studies which have compared the effects of different types of feedback on performance. Much of this work has originated in Lang's laboratory. In separate but very similar experiments, Gatchel (1973, 1974), and Lang (1974) examined

voluntary control of heart rate increases and decreases in four different groups of subjects. Three of these groups were provided with feedback for heart rate changes. In one group, feedback was provided after every heart beat, in another after every five heart beats, and in the third after every ten heart beats. A control group received exposure to the feedback stimulus contingent upon performance in a tracking task. The best performance on increase trials was found in the group given feedback following every heart beat. The groups given feedback following every five and every ten heart beats also produced reliable increases in heart rate on Raise trials, but these were of comparable magnitude and smaller than those produced when feedback followed every heart beat. On Lower trials, however, while heart rate was lower in the feedback groups than in the control group, the feedback groups did not differ from one another. These experiments by Lang and Gatchel are among the few studies in which performance was observed not only during training, but also during transfer trials, when feedback was no longer provided. On Raise trials, similar results were obtained on training and transfer trials. However, on Lower trials, there was no evidence of any lowering of heart rate on transfer trials, although such evidence was obtained on training trials.

Lang and Twentyman (1974) published a similar experiment in which they compared the merits of analog and binary feedback. Subjects receiving analog feedback received feedback following every heart beat. Subjects receiving binary feedback received feedback only when their heart rate exceeded a certain criterion. Analog procedures were more efficient than binary procedures in bringing about heart rate control on Raise trials, but both types of feedback were equally efficient in

bringing about heart rate control on Lower trials. The degree of heart rate control exhibited on Raise trials was maintained on transfer trials in the analog group, but not in the binary group. On Lower trials, neither group showed evidence of heart rate control on transfer trials. Evidence of superior heart rate control under conditions of analog rather than binary feedback on Raise trials, but not on Lower trials, was also reported by Blanchard et al. (1974b)

In sum, it appears that provision of feedback for heart rate changes has an effect on voluntary heart rate control, and that feedback typically results in a greater degree of heart rate control than is obtained when only instructions to control heart rate are given. Moreover, feedback appears to have a more consistent effect on the production of increases in heart rate than on the production of decreases in heart rate. Also, there is evidence that when feedback affects the production of heart rate changes in both directions, increases and decreases in heart rate are affected differently (Lang, 1974). Nevertheless, it should be noted that there are a number of experiments in which feedback effects have failed to materialize. Why feedback effects are not obtained more consistently is presently unclear.

Summary

This section has reviewed what is known about the contribution of three training variables to voluntary control of heart rate. It is clear that subjects can generate reliable changes in heart rate following instructions to increase or decrease this response. Part of these changes can likely be ascribed to the directional component of the instructions, and part of them also to the reference to heart rate in the instructions. As yet unsettled, however, is the extent to which the directional component of the instructions alone, and the information that

heart rate is the target response,contribute to the total effect. Depriving the subjects of somatomotor strategies diminishes performance, but the question of whether provision of explicit strategy information has the opposite effect has not been investigated. Data pertaining to the role of exteroceptive feedback in voluntary control are contradictory. Several studies have reported that provision of feedback facilitates voluntary control of heart rate, and this effect is usually more substantial with respect to the production of increases in heart rate than with respect to the production of decreases. However, the effect of feedback is not always sustained on transfer trials when the feedback is discontinued, particularly on decrease trials. There are, on the other hand, several studies in which feedback failed to generate a heart rate change that was superior to that seen in subjects given verbal instructions alone. The question of why feedback appears to have an effect under some conditions but not others remains to be resolved.

Correlates of Voluntary Cardiac Control

This section examines the literature relevant to the question of whether changes occur in other systems when heart rate is voluntarily controlled. As was the case with the comparable review with respect to electrodermal behaviour, only studies which have reported reliable evidence of cardiac control, and which have provided empirical evidence of the presence or absence of changes in at least one other response system, will be examined. Moreover, as was also the case with the review of the correlates of electrodermal control, pertinent results from studies of operant conditioning will also be presented.

Autonomic correlates

A number of investigators have recorded the activity of other autonomic systems while heart rate was voluntarily controlled. Bergman and Johnson (1971, 1972) and Chalmers (1970) focussed on electrodermal behaviour. In no case was a relationship observed between heart rate and electrodermal tonic level, frequency of electrodermal phasic responses, or amplitude of electrodermal phasic responses. However, it should be noted that the heart rate changes which were obtained in these studies were of small magnitude (1 to 5 beats per minute).

Brener (1974a), and Shapiro, Tursky and Schwartz (1970) measured blood pressure (diastolic and systolic, respectively) while heart rate was brought under voluntary control. Brener observed that heart rate and diastolic blood pressure exhibited changes in the same direction when subjects were simply instructed to try to control their heart rate. However, the provision of feedback for heart rate changes accentuated the magnitude of the heart rate changes, but diminished the magnitude of the changes in diastolic blood pressure. In the Shapiro et al. study systolic blood pressure was examined while heart rate was brought under operant control. Subjects were not informed that heart rate was the target response. No relationship was observed between heart rate and systolic pressure.

Somatomotor activity

Brener (1973, 1974a) investigated the effects of exerting voluntary control over heart rate on electromyographic activity. Merely instructing subjects to raise and lower their heart rate generated reliable concomitant changes in chin electromyographic activity. Moreover, the provision of feedback for changes in heart rate resulted in substantially

larger changes in both heart rate and electromyographic activity.

Cohen (1973) and Obrist et al.(1975) have also examined chin electromyographic activity during the establishment of operant control of heart rate. In both experiments a shock avoidance procedure was employed, and in neither study were the subjects explicitly told that heart rate was the target response. In Cohen's experiment electromyographic activity and heart rate did not covary, whereas in the study by Obrist et al. the responses did covary. The latter study also incorporated two other measures of somatic activity, gross body movement and eye movement, and a relationship between both these activities and heart rate was found in that experiment.

Respiration rate

There appear to be eight experiments on voluntary control of heart rate in which some measure of respiration rate was recorded (Blanchard et al., 1974a and b; Brener et al., 1969; Gatchel, 1973; Lang, 1974; Lang and Twentyman, 1974; Levenson, 1974; and Levenson and Strupp,1972)[1]. The experiments differ widely in the type and duration of training, and in the magnitude of the heart rate changes which were observed. Nevertheless, in each of these studies, reliable differences in respiration rate were observed between conditions in which subjects were to raise their heart rate and conditions in which they were to lower their heart rate. Moreover, in the two studies in which the relationship between heart rate and respiration rate was scrutinized particularly carefully (Gatchel, 1973, and Levenson, 1974), the relationship was found to be a close one.

Levenson (1974) instructed his subjects to try to increase and decrease their heart rates on Raise and Lower trials, respectively, but

to refrain from using respiratory maneuvers. One group of subjects was provided with heart rate feedback, a second was provided with both heart rate and respiration rate feedback, and a third was not provided with feedback of any kind. Subjects breathed into a respirometer while attempting to change their heart rate. No group differences were observed with respect to either the degree of heart rate control or the relationship between heart rate and respiration rate. Mean heart rate increases of 2.9 beats per minute were obtained on Raise trials, and mean decreases of 1.6 beats per minute were manifest on Lower trials. These were accompanied by changes in the duration of respiratory cycles: a mean decrease of 402 msec was observed on Raise trials, and a mean increase of 569 msec was obtained on Lower trials. It should be noted that these changes in the rate of respiration, though highly reliable, are rather slight: a 500 msec change in respiratory cycle duration would result in a difference of only 2 cycles per minute in a subject breathing normally. However, the heart rate changes observed by Levenson were also of small magnitude.

Gatchel (1973) also examined the heart rate - respiration rate relationship in some detail. Although, like Levenson's, his subjects were instructed to breathe normally, the changes in heart rate obtained in Gatchel's study were approximately two to three times the magnitude of those obtained in Levenson's experiment, on both Raise and Lower trials. Moreover, on Lower trials, respiratory cycle duration lengthened by several times the magnitude observed in Levenson's study. Changes in respiration rate were also present on Raise trials, although the magnitude of these was not reported. On Raise trials, a significant

negative correlation was found between the magnitude of heart rate changes, and that of changes in respiration rate; the corresponding correlation on Lower trials was positive, and approached significance.

In addition to the studies on voluntary control of heart rate, there have been a number of experiments concerned with operant control of heart rate in which respiration rate was monitored. In two of these experiments (Obrist et al., 1975, and Shearn,1962), changes in respiration rate were concomitant with changes in heart rate, whereas in four other reports,there was no difference in respiration rate between Raise and Lower conditions (Engel and Chism, 1967b; Engel and Hansen, 1966; Finley, 1971; and Headrick, Feather, and Wells, 1971). The relevance of these observations to the relationship between heart rate and rate of respiration in voluntary control studies is unclear. First, both the Obrist et al. and the Shearn studies, in which differences in respiration rate were observed, differ from traditional voluntary control experiments not only in that the subjects were not told what they should try to control, but also in the use of shock avoidance as reinforcement. As for those studies in which respiration rate differences were not observed, in two of these (Engel and Chism, 1967b, and Engel and Hansen, 1966) there are convincing reasons to believe that heart rate control was not achieved either (see Murray and Katkin, 1968), whereas in the other two, the subjects were deliberately misinformed about the nature of the response which they should try to control.

In sum, considerable data are available on the relationship between heart rate and respiration rate in voluntary control training and operant conditioning studies. The evidence indicates that heart rate and respiration rate covary, and this even when subjects are specifically

told not to utilize respiratory maneuvers.

Respiration amplitude

Four experiments appear to have incorporated measures of respiration amplitude while establishing voluntary control over heart rate. In two of these studies (Bergman and Johnson, 1972, and Wells, 1973) differences in respiration amplitude were observed to parallel the differences in heart rate between Raise and Lower trials, whereas in the other two (Bergman and Johnson, 1971, and Levenson, 1974) changes in respiration amplitude did not accompany the heart rate changes. It might be noted that of these four studies, the only one to report substantial heart rate differences between Raise and Lower trials was Wells' (mean heart rate increase of 18.5 beats per minute; mean heart rate decrease of 1.5 beats per minute). Highly reliable correlations were found between heart rate and respiration amplitude in that study (rho = .82 and .78 for Raise and Lower trials, respectively, between the percent change in respiration amplitude and the percent change in heart rate; these are based on the data presented in Table 1 of Wells' paper.)

In addition to these studies, there appear to be four experiments concerned with operant control of heart rate in which the relationship between heart rate and respiration amplitude was assessed. In three of these, differences in respiration amplitude were observed to parallel the differences in heart rate between Raise and Lower conditions (Cohen, 1973; Obrist et al., 1975; and Shearn, 1962), while the last one failed to uncover evidence for such a relationship (Headrick, Feather, and Wells, 1971).

Thus, while there is little doubt that changes in respiration rate accompany changes in heart rate when heart rate is brought under voluntary control, the evidence with respect to whether changes in respiration amplitude also parallel changes in heart rate is less consistent. This is perhaps a bit surprising in view of the results of experiments discussed earlier,(Sroufe, 1971, and Sroufe and Morris, 1973), in which it was observed that mean heart rate was susceptible to changes in respiration amplitude but not to changes in respiration frequency. However, it should be noted that in Sroufe's investigations, in which respiration amplitude was explicitly manipulated, large differences in respiration amplitude were produced, and large differences in heart rate resulted. In the Bergman and Johnson (1971) and in the Levenson (1974) studies the heart rate changes which were observed were quite small, and it is possible that accompanying changes in respiration amplitude were too small to be detected. As noted above, the only experiment reporting large heart rate differences in which respiration amplitude was monitored also reported large differences in respiration amplitude (Wells, 1973).

Summary

In sum, there are concomitant changes in other systems when heart rate is voluntarily controlled. Consistent changes in respiration frequency were observed to parallel heart rate changes. Moreover, it is probably safe to say that when large magnitude changes in heart rate are produced, these can be expected also to be accompanied by changes in respiration amplitude, body movement, and diastolic blood pressure. Surprisingly, perhaps, changes in electrodermal behaviour were not

found to parallel voluntary changes in heart rate, whereas it was reported in an earlier section of this chapter that heart rate changes parallelled changes in electrodermal activity when electrodermal activity was voluntarily controlled. However, those studies in which electrodermal behaviour was examined while heart rate was brought under voluntary control reported only minimal changes in heart rate, and it is possible that more substantial heart rate changes would be accompanied by electrodermal changes.

Conclusion

This chapter has examined the literature pertaining to the role of instructions, explicit strategy suggestions, and feedback, in the voluntary control of electrodermal activity and heart rate. Also reviewed were data pertaining to the correlates of electrodermal and cardiac control.

The contribution of instructions, strategies and feedback to voluntary electrodermal control has received little attention in the literature. The effect of simple instructions to increase or decrease palmar sweating does not appear to have been investigated. However, instructions to increase and decrease emotional arousal have been observed to produce reliable changes in electrodermal responding, although the extent to which these are attributable to the reference to an emotional response strategy as opposed to the instructions to change a response is unclear. Data pertaining to the role of feedback in voluntary electrodermal control are very sparse. Only three experiments appear to have examined whether provision of exteroceptive feedback facilitates voluntary control of electrodermal responding. The most satisfactory of these studies (Klinge, 1972) provided evidence of feedback effects,

but the remaining two experiments did not. There is some indication that voluntary changes in electrodermal responding are associated with respiratory and heart rate changes, but once again the data pertaining to this question are sparse. The basis for these correlates, and the nature of their relationship to changes in the target response, are not well understood.

The role of instructions, strategies, and feedback in voluntary cardiac control has received more attention. Instructions to increase and decrease heart rate have resulted in heart rate differences of up to eight beats per minute between increase and decrease conditions. Part of these changes can be attributed to the directional component of the instructions and part of them to the reference specifically to heart rate. Moreover, while it appears that the largest heart rate changes are observed when no somatomotor and respiratory restrictions are placed on the subject's behaviour, whether cardiac control is augmented further by provision of explicit strategy suggestions has yet to be determined. Adding feedback to instructions often results in better heart rate control, ranging up to 20 beats per minute between increase and decrease trials, but several experiments have failed to confirm this observation. The basis for these discrepancies is not apparent. Finally, examination of correlated responses indicates that changes in somatomotor and respiratory functions typically parallel voluntary changes in heart rate. The fact that the relationship of heart rate changes to these correlates is frequently substantial, and that voluntary control of heart rate is diminished when somatomotor and respiratory correlates are constrained, suggests that these correlates may be necessary for performance of voluntary cardiac change.

It is apparent from this review that present knowledge of the characteristics of voluntary electrodermal and heart rate control is incomplete. It is further evident that data are particularly lacking for the electrodermal system. The research to be described in this thesis was undertaken in an effort to fill some of the gaps in our knowledge of this response, and to contrast the properties of voluntary electrodermal control with those of voluntary heart rate control. The experiments also provided data bearing on some of the currently unresolved issues pertaining to variables that determine voluntary heart rate change. The specific goals of the experiments were (1) to evaluate the contribution of instructions, explicit strategy suggestions, and exteroceptive feedback to voluntary control of electrodermal responding and heart rate, (2) to determine how other response systems are affected when voluntary control of these responses is achieved, and (3) to compare voluntary electrodermal and cardiac control with respect to response correlates and the role of the three experimental variables in establishing voluntary control of each autonomic system.

The first experiment is presented in the next chapter. In this experiment, instructions, strategy suggestions, and exteroceptive feedback were combined in an attempt to devise a procedure that would demonstrate voluntary control over the electrodermal and cardiac systems. A subsequent experiment, presented in chapter 4, examined the contribution of these variables separately. The properties of voluntary electrodermal and heart rate control were compared in both studies.

Chapter 3: Experiment 1

The first and main goal of this experiment was to develop and test a procedure for bringing skin conductance and heart rate under voluntary control. Since the concern here was simply with demonstrating control, instructions, explicit information about response strategies, and feedback were combined in an attempt to bring about a high degree of control. Subjects were instructed to try to control their electrodermal behaviour or heart rate, were provided with potentially useful behavioural strategies, and were given feedback for producing increases and decreases of appropriate magnitude in skin conductance and heart rate during designated Raise and Lower trials. Performance was examined both during training trials, when the feedback stimulus was presented, and during transfer trials, when the feedback stimulus was removed.

A second goal of the experiment was to examine the correlates of voluntary control of skin conductance and heart rate. Behaviours which were monitored under both Target conditions included skin conductance, heart rate, gross body movement, respiration frequency, respiration amplitude, and eye movements. Subjects were also requested to rate the Raise and Lower trials on a battery of affective scales. In addition to examining correlates of control, the experiment compared the correlates of electrodermal control with those of cardiac control.

A third goal of the experiment was to evaluate whether transfer of training resulted from gaining control over one response to gaining control over the other. Such an effect would suggest some overlap in the neural mechanisms controlling the performance of the electrodermal and cardiac changes.

Method

Subjects

The study was carried out on ten male volunteers who were naive to the experimental situation. The subjects were recruited by means of advertisements placed around the McMaster University campus, and ranged in age from 18 to 27 years, with a mean of 20.9 years.

Apparatus

The subjects were tested in an electrically shielded room, and were left alone during each experimental session. All recording and programming activities took place in an adjoining room. Inside the experimental room the subject and the various pieces of equipment used in the experiment were encircled by dark curtains to minimize distracting influences by other objects in the room. A rectangular hole was cut in the curtain facing the subject to accommodate a television camera, placed behind the curtain, for monitoring the subject's behaviour. A small 60-watt lamp was left on in the room during experimental sessions, and this provided enough light for the camera. This lamp was placed slightly to the left of and in front of the subject, behind the enclosure.

Each subject sat on an air cushion in a custom-made black wooden chair, provided with arm-, foot-, and head-rests. The subject faced a table on which he could lean if he so wished. The table supported a trapezoid-like enclosure (120 cm at the front, 60 cm at the rear, 60 cm in depth, and 60 cm in height), open at the top and at the front. At the base of this enclosure,facing the subject, was the stimulus display. This consisted of a rectangular white lucite plaque (25 cm in length and 10 cm in height). Two lucite stimuli (4 cm^2) were embedded side by side within this rectangle, one marked with a black "R", the other

with a black "L". Behind each of these squares was a 15-watt bulb. One of these stimuli was illuminated during each trial period, the "R" stimulus indicating a Raise trial, the "L" a Lower trial. Above the stimulus display, but behind the enclosure was an intercom, which was set so that vocalizations and noises made by the subject were audible to the experimenter. The experimenter could also communicate with the subject over the intercom if the need arose.

The feedback stimulus consisted of an 800 hz, 75 db tone (intensity measured at the speaker) delivered via a 10 cm speaker located on the subject's chair, directly behind his head. All programming was accomplished by means of BRS logic modules and Foringer electronic equipment.

Electrophysiological recordings

All electrophysiological variables were recorded on an 8-channel Beckman Type R polygraph, operating at a chart speed of 2.5 mm/second.

Skin conductance was recorded through Beckman biopotential Ag/AgCl skin electrodes, 7 mm in diameter. The face of each electrode was filled with Beckman electrode paste and then topped with Unibase (0.1 molar with respect to NaCl). Beckman electrode collars (10 mm in diameter) were used to attach the electrodes to the skin. Contact area was thus approximately circular and 10 mm in diameter. Electrodes were kept shorted together in a weak salt solution when not in use, and were pre-selected from several electrodes to ensure standing potentials of less than 1 mv between recording surfaces. Active electrodes were placed on the tip of the middle finger of both hands, but records from only one of these (the one subject to less electrical noise and fewer movement artifacts, if there was a difference) were kept throughout the session. A reference electrode was placed on the left forearm, approxi-

mately over the fascia of the palmaris longus muscle. All sites were cleaned with 70% alcohol prior to application of the electrodes. In addition, the reference site was abraded daily with a pumice stone. Skin conductance was measured as the current generated by 500 mv applied between active and reference electrodes, through a total series resistance of 1.5 KΩ. Current flow through a typical subject was 12.5 μa (32.5 μa/cm^2). Recordings were accomplished through a Beckman Type 9806 A AC/DC coupler, set in the DC mode.

Heart rate was recorded by means of electrodes identical to those used to monitor skin conductance. Heart rate was recorded with a standard Lead II configuration, between the left leg (over the gastrocnemius muscle) and the right forearm (over the palmaris longus muscle). An expanded-range cardiotachometer (Beckman Type 9857B) provided a beat-by-beat measure of heart rate throughout each experimental session. The raw EKG was also recorded through a Beckman AC/DC coupler (Type 9806A) set at an RC constant of 0.03 second.

In addition to being recorded as noted above, whichever response was trained on a given day (either skin conductance or heart rate) was also recorded at a higher gain on two other channels. These channels formed the initial stage of the feedback circuit. One of these was used to detect when skin conductance or heart rate was below a certain criterion; the other was used to detect when skin conductance or heart rate was above a certain criterion.

Respiration was recorded via a mercury-filled strain gauge (Parks Electronics Laboratory) which encircled the subject's upper torso, and was connected to a Beckman Type 9853H Voltage/Pulse/Pressure coupler.

Gross body movement was recorded from the air-filled cushion on which the subject sat. The air valve was connected to a Beckman Physiological Pressure transducer, which in turn was connected to a Beckman Voltage/Pulse/Pressure coupler (Type 9853H). This method proved to be sensitive to slight movements of the arms, legs or torso. Even respiratory changes could always be observed in the body movement record. The only types of movement to which this measure did not prove sensitive were head movements. However, most of those appeared in the eye movement records.

Eye movements were recorded by means of Beckman miniature Ag/AgCl skin electrodes, 3 mm in diameter, which were treated like the skin conductance and heart rate electrodes, except that Unibase was not used with these. The electrodes used to record eye movements were placed over the zygomatic process above the left eye, and over the zygomaticofacial foramen, below the right eye. These sites were cleaned daily with alcohol and abraded slightly with a special cleansing pad (Ferris Medical Corporation). Eye movements were recorded through a Beckman AC/DC coupler (Type 9806A), set at an RC constant of 1.0 second.

Procedure

Subjects were divided into two groups. Subjects in the first group received two days of feedback training with skin conductance followed by two days of feedback training with heart rate. Subjects in the other group were treated similarly, except that they were given heart rate training first and skin conductance training second. The subjects were run for four consecutive days, and received $3.00 per day for their participation in the study, plus whatever bonus they succeeded in earning.

At the beginning of each session, after application of the electrodes, and after verification of the quality of the electrophysiological recordings, the subject was read a standard set of instructions (see Appendix A.) These informed him of the general purpose of the experiment, and of the response system which would be subjected to feedback training on that day. He was also instructed about the contingencies which would be in effect during the session, and some sample strategies were suggested. However, the subject was strongly encouraged to develop his own ways of meeting the response requirements. The experimenter then left the room and the experiment began two to three minutes later.

Each session comprised 41 trials, including 20 Raise and 21 Lower trials, or the reverse. Six Raise and six Lower trials were test trials, and the feedback stimulus was not delivered on these trials. The purpose of these trials was to enable examination of behaviour without any confounding effects of the feedback stimulus.

Raise and Lower trials were indicated by means of lights placed in front of the subject. Each trial lasted 30 seconds. The inter-trial intervals lasted 30, 35, 40, 45, 50 or 60 seconds, with a mean of 40 seconds. Trials were presented in a mixed order and were randomized with respect to the inter-trial intervals. The first and last Raise and Lower trials were always test trials. Three different sequences of trial presentations were used. These sequences were counter-balanced across days and between subjects, and are presented in Appendix B.

The procedure used to determine the response criterion for feedback presentation was selected because it enabled identical treatment of the

cardiac and electrodermal systems. On Raise trials subjects were rewarded with the feedback tone for raising their skin conductance (or their heart rate if heart rate was the rewarded response) above a predetermined criterion. This criterion was based on performance on the previous Raise trial, and consisted of the skin conductance level which was equal to 75% of the difference between the maximal and minimal level on that trial. For example, if on trial $Raise_{n-1}$ skin conductance oscillated between 20 and 28 micromhos, criterion on trial $Raise_n$ would be set at 26 micromhos, and feedback would be presented whenever skin conductance exceeded 26 micromhos. On Lower trials subjects were rewarded for keeping their skin conductance level below a given criterion. Criterion was equal to 25% of the difference between maximal and minimal skin conductance on the previous Lower trial. If, then, skin conductance varied between 20 and 24 micromhos on trial $Lower_{n-1}$, criterion would be set at 21 micromhos on trial $Lower_n$, and feedback would be given whenever skin conductance fell below 21 micromhos. These parameters were chosen because pilot work had indicated that they resulted in feedback frequency and duration which seemed optimal. It was thought that too much or too little feedback would impair performance, too much because it would hinder identification and control of appropriate behaviours, and too little because it would lead to a decrement in the subject's motivation.

Whenever the subject met the criterion requirement on a Raise or a Lower trial, the feedback tone was turned on, and remained on as long as the criterion was surpassed. In addition, subjects earned 10¢/minute when they achieved criterion. On test trials the feedback tone was not provided, but subjects still earned bonus money when they met the

response requirements, and they were so informed in the instructions.

At the end of days 2 and 4 subjects were asked to complete a questionnaire which attempted to find out what cognitive or affective strategies, if any, they had used in trying to meet the response requirements. Among other things, they were asked to rate the Raise and Lower stimuli along various dimensions, which were presented in Semantic Differential fashion. In addition, questions were included to evaluate perceived success at controlling skin conductance and heart rate, relative difficulty of controlling the target response on Raise and Lower trials, and general attitudes toward the experiment. The questionnaire is presented in Appendix C.

Data analysis

Skin conductance and heart rate were sampled at the midpoints of consecutive 5-second intervals during the 30 seconds immediately preceding the start of a trial, during the trial period, and during the 15 seconds immediately following the end of the trial. This provided 6 baseline measures, 6 trial measures, and 3 post-trial measures of skin conductance and heart rate. Analyses of response forms, to be presented later, indicated that analyses of performance would be most appropriate if they were based on the last 10 seconds of the trial period. Change scores were computed by subtracting measurements taken from the last 10 seconds of the pre-trial period from measurements taken during the last 10 seconds of the trial period, thus yielding a measure of responding that took baseline behaviour into account.

Analyses of variables other than skin conductance and heart rate also focussed on the late portion of the trial. Evaluations of body movement were based on the number of 3 mm deflections on either side

of the midline during the last 10 seconds of the baseline and trial periods. The number of 3 mm deflections in the two intervals was compared. An increase in movement from baseline to trial period was scored as +1, a decrease as -1, and no change was scored as a 0. These scores were summed across test trials in each session, to yield a single measure of the movement response for each subject on each day of training.

Respiration frequency was evaluated by recording the time interval between the peaks of the last two inspirations during the trial and subtracting from this value the time interval between the peaks of the last two inspirations preceding trial onset. These scores were averaged across test trials in each session, separately for each subject on each day of training. Changes in respiration amplitude were evaluated by noting the amplitude of the largest deflection (inspiration to expiration) in the respiratory record, both in the last 10 seconds of the baseline period and in the last 10 seconds of the trial period. If no activity was in evidence in the respiratory record during either time interval, the amplitude of the deflection most closely preceding the appropriate time interval was noted. The magnitudes of the two deflections were then compared. An increase in maximum respiratory amplitude from baseline to trial period was scored as +1, a decrease as -1, and no change was scored as a 0. These scores were summed across test trials for each subject on each day of training.

Finally, eye movement was evaluated by counting the number of 2 mm deflections of the pen on either side of the midline during the last 10 seconds of the baseline and trial periods. The number of such deflections

between the two intervals was compared. The response was scored as an increase (+1), a decrease (-1), or no change (0), as was done previously with respiration amplitude and gross body movement. These scores were summed across test trials in each daily session to yield an eye movement measure for each subject in the experiment.

Results

Control over skin conductance

Figure 1 presents test trial performance on the last day of skin

Figure 1. Mean skin conductance on Raise and Lower test trials on the second day of training to control skin conductance. Subjects receiving skin conductance training first (SC-1) and second (HR-1) are shown separately. Skin conductance was measured at the midpoint of consecutive 5-second intervals, beginning 30 seconds before trial onset, and ending at the offset of the trial.

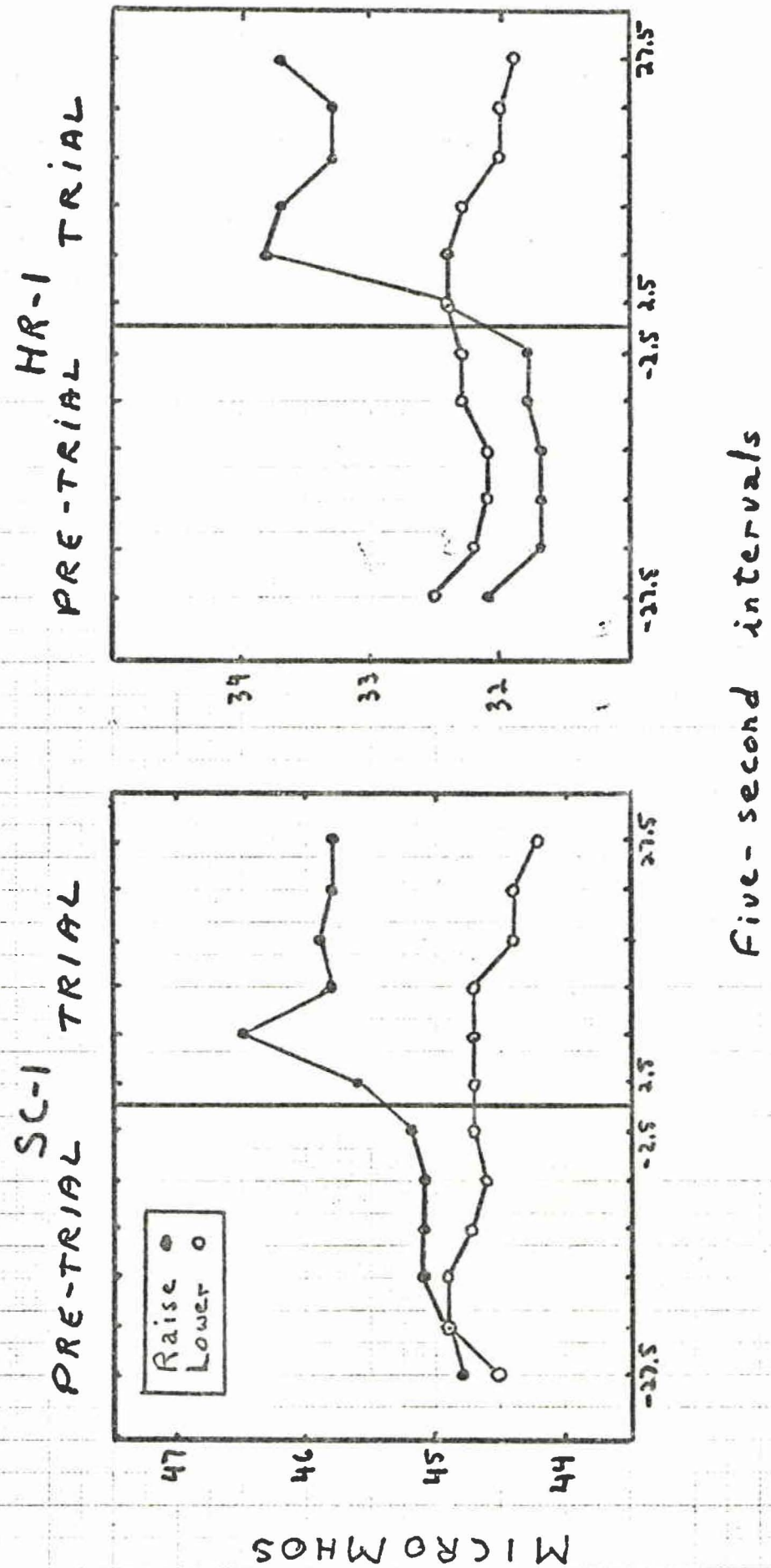
SC-1
PRE-TRIAL TRIAL
Raise ●
Lower ○
47
46
45
44
-22.5
-2.5
2.5
22.5
HR-1
PRE-TRIAL TRIAL
34
33
32
-22.5
-2.5
2.5
22.5
MICROMHOS
Five-second intervals

Figure 1

conductance training for subjects who received skin conductance training first (the SC-1 group, in the left panel), and for subjects who received skin conductance training second (the HR-1 group, in the right panel). Skin conductance is plotted every five seconds during the 30-second periods preceding Raise and Lower trials (left-hand side of each panel) and during the trial periods (right-hand side of each panel).

Two preliminary analyses were carried out on these data. First, baseline differences were examined through analyses of variance employing Types of Trial (Raise vs Lower) and Seconds as variates, which were applied to baseline skin conductance in each group. Neither the Types of Trial main effect nor the Types of Trial-by-Seconds interaction approached reliability in either group. Second, since inspection of Figure 1 suggests that responding took rather different forms on Raise and Lower trials, this difference in response forms was evaluated by noting, for each subject, which of the six trial measurements represented the maximum change in skin conductance in the targeted direction, and then comparing where this point occurred on Raise and Lower trials. The maximum change from baseline in the targeted direction occurred reliably earlier on Raise trials than on Lower trials (sign test, 8 of 9 subjects, $p < 0.05$). This suggests that conclusions regarding the magnitude of control on Raise and Lower trials will vary depending on the portion of the trial which is examined. It was decided to focus attention on the last ten seconds of the trial period, where there was the least likelihood of confusing any orienting response which might occur to trial onset with a voluntarily-produced change in electrodermal behaviour. Subsequent analyses focussed on the difference in performance between the last ten seconds of the trial period and the last ten

seconds of the preceding baseline period. Except where indicated, statistical tests were based on these change scores.

The right-hand portion of each panel in Figure 1 depicts skin conductance activity during Raise and Lower trials on the second day of skin conductance training. The performance of the two groups was compared through an analysis of variance which took Groups and Types of trial as variates. This analysis yielded a main effect of Types of trial which approached reliability ($F_{1,8} = 3.68$, $p < .10$), with no evidence for a Groups-by-Types of trial interaction ($F < 1$). Further, nonparametric, analyses of the data provided stronger evidence for learned control of electrodermal responding. Nine out of the ten subjects tested were found to perform in the direction of training when the difference between Raise and Lower trials was examined for each subject separately ($p = .02$, sign test). Evaluation of performance by Wilcoxon T's also proved reliable when applied to each training group (for SC-1 group, Wilcoxon T ($N=5$) = 0, $p < .025$; for the HR-1 group, T (5) = 1, $p < .05$, one-tail tests). These analyses suggest that voluntary control of skin conductance was achieved, and that it was evidenced under both training conditions.

Further analyses evaluated whether differences between Raise and Lower trials reflected primarily a raising of skin conductance on Raise trials, a lowering on Lower trials, or both. The raising effect approached reliability only in the HR-1 group (T (5) = 2, $p < .07$, one-tail test). However, the lowering effect proved statistically significant in both groups (in both cases, T (5) = 0, $p < .025$, one-tail tests). Thus, there was evidence of electrodermal control in both directions, although this evidence was statistically more prominent on Lower trials.

Subsequent analyses determined whether Lower trial performance reflected a genuine voluntary decrease in skin conductance, or whether it merely reflected a long-term trend in the skin conductance baseline. Several observations suggest the former alternative. First, there was no apparent trend in skin conductance during the baseline periods. This was evidenced by the lack of reliable Seconds main effects or Types of Trial-by-Seconds interactions in analyses of variance

employing Types of Trial and Seconds as variates, which were applied separately to baseline skin conductance in the two groups. Second, analyses of variance confirmed that the amplitude of responding on Lower trials was the same regardless of the duration of the preceding inter-trial interval (intertrial interval $F < 1$, SC-1 and HR-1 groups combined). Similar results were obtained with respect to performance on Raise trials. Third, an examination of the recovery functions following trial offset also suggested that the lowering effect was genuine. These functions are presented in Figure 2. There was a tendency for skin conductance to decrease following Raise trials, and to increase following Lower trials, in both groups. The reliability of these changes was evaluated by means of an analysis of variance employing Groups, Types of Trial, and Time (last trial point versus third post-trial point) as variates. While this analysis failed to confirm the reliability of the differences in recovery functions following the two types of trial, the results were in the expected direction (Types of Trial-by-Time interaction, $F_{1, 8} = 3.412$, $p < .105$).

Figure 3 examines acquisition effects in skin conductance. The figure depicts the mean change in skin conductance from baseline on the first trial and, subsequently, over the four blocks of three test trials. An analysis of variance employing Groups and Types of Trial as variates was applied to performance on the first trial. Neither the main effects nor the interaction of these variables approached statistical significance, indicating that the performance of the two groups was comparable on the first trial, with neither group exhibiting much control over skin conductance. Acquisition effects were evaluated statistically through an analysis of variance which employed Groups, Types of Trial,

Figure 2. Skin conductance before and after trial offset on the second day of training to control palmar sweating. Groups given skin conductance training first (SC-1) or second (HR-1) are plotted separately. Skin conductance is shown as a change from the pre-trial baseline.

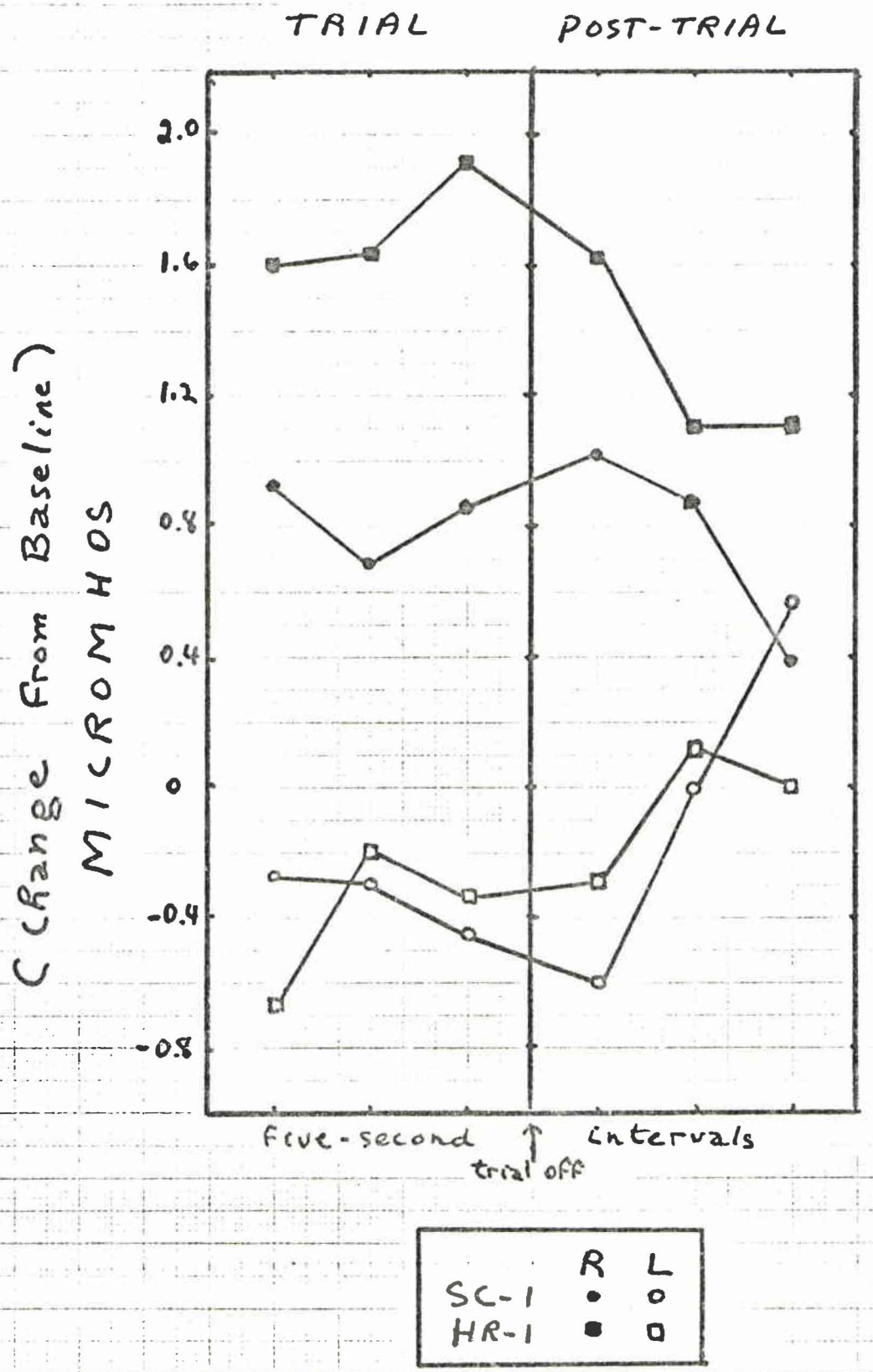
TRIAL
POST-TRIAL
2.0
1.6
1.2
0.8
0.4
0
-0.4
-0.8
(Change From Baseline)
MICROMHOS
Five-second intervals
trial off
R L
SC-1 • o
HR-1 ■ □

Figure 2

Figure 3. Acquisition of skin conductance control. The difference between skin conductance during the last 10 seconds of the trial and the preceding baseline period is plotted for the very first trial (a test trial), and for successive blocks of test trials over two training days. Groups receiving skin conductance training first (SC-1) and second (HR-1) are shown separately.

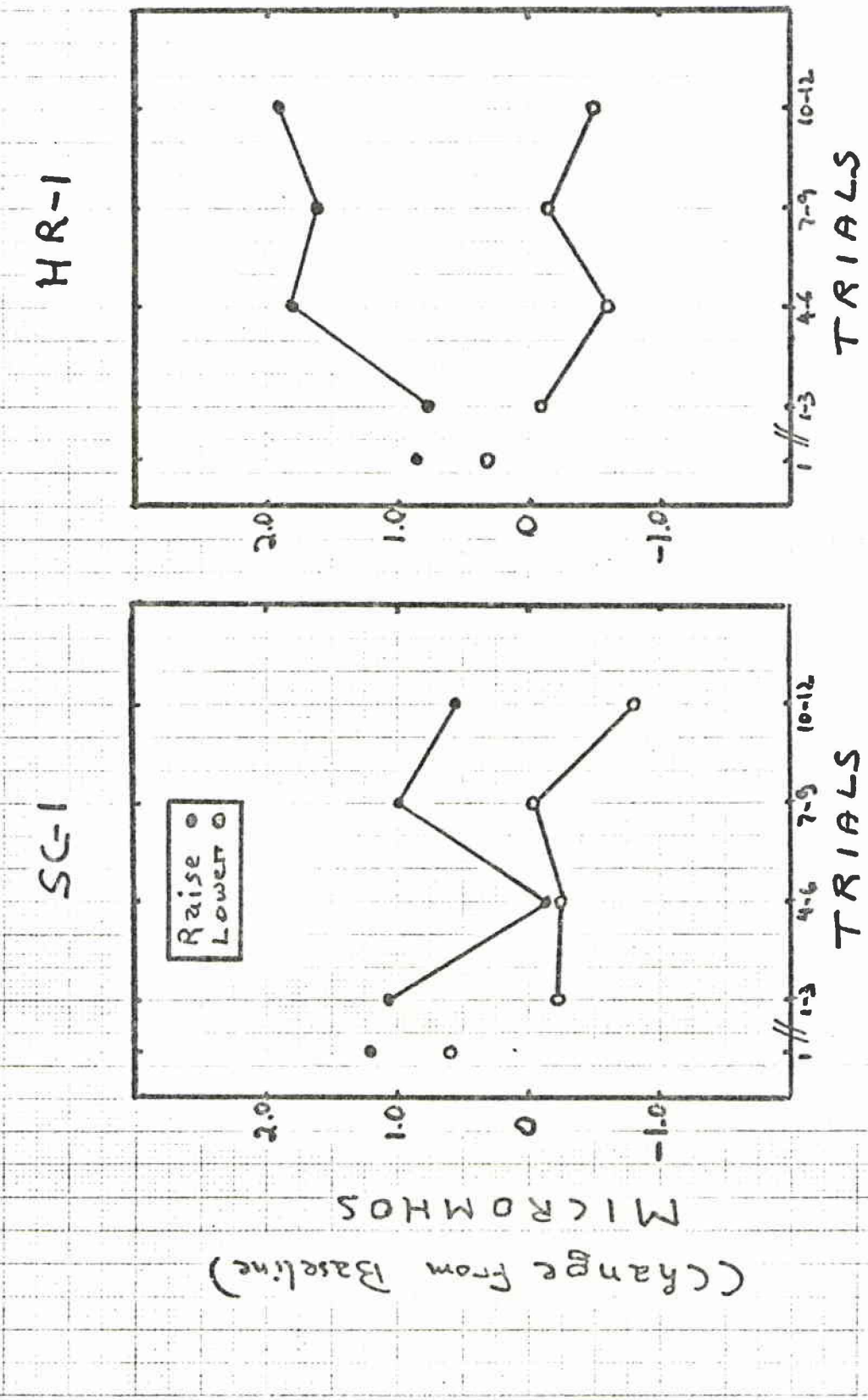
HR-1
SC-1
Raise •
Lower ○
2.0
1.0
0
-1.0
1-3
4-6
7-9
10-12
TRIALS
(Change From Baseline)
MICROMHOS

Figure 3

and Trial Blocks as variates. This analysis yielded a reliable interaction between Groups, Types of Trial, and Trial Blocks ($F_{3,24} = 3.48$, $p < .05$). Inspection of Figure 3 suggests that this interaction reflected a tendency for acquisition on Raise trials to occur only in subjects who received prior training to control heart rate. Unlike previous analyses, which compared performance on the second day of training alone, this analysis implies that voluntary control of skin conductance was more substantial in subjects who were pre-trained on heart rate.

The data presented thus far were all obtained on test trials. The analysis which follows compared performance on test and training trials to determine the extent to which transfer took place. The pertinent data are presented in Figure 4, which depicts the mean change in skin conductance observed in the two groups on Raise and Lower trials over the four blocks of training, on test trials and training trials. Analyses of variance using Groups, Feedback (present/absent), and Trial Blocks as variates were applied to performance on Raise and Lower trials separately. The Feedback main effect approached reliability on Raise trials ($F_{1,8} = 3.66$, $p < .10$), and interacted reliably with other variables only in the 3-way Groups-by-Feedback-by-Types of Trial Interaction ($F_{3,24} = 6.03$, $p < .01$). The interaction appears to reflect the generally larger increases in skin conductance on training trials than on test trials in the HR-1 group which is apparent in Figure 4 except on the third trial block. None of the main effects or interactions involving Feedback (present/absent) were reliable on Lower trials.

Figure 4. A comparison of skin conductance control on test trials (when the feedback stimulus was never presented) and on training trials (when the feedback stimulus was always presented). Groups receiving skin conductance training first (SC-1) and second (HR-1) are plotted separately.

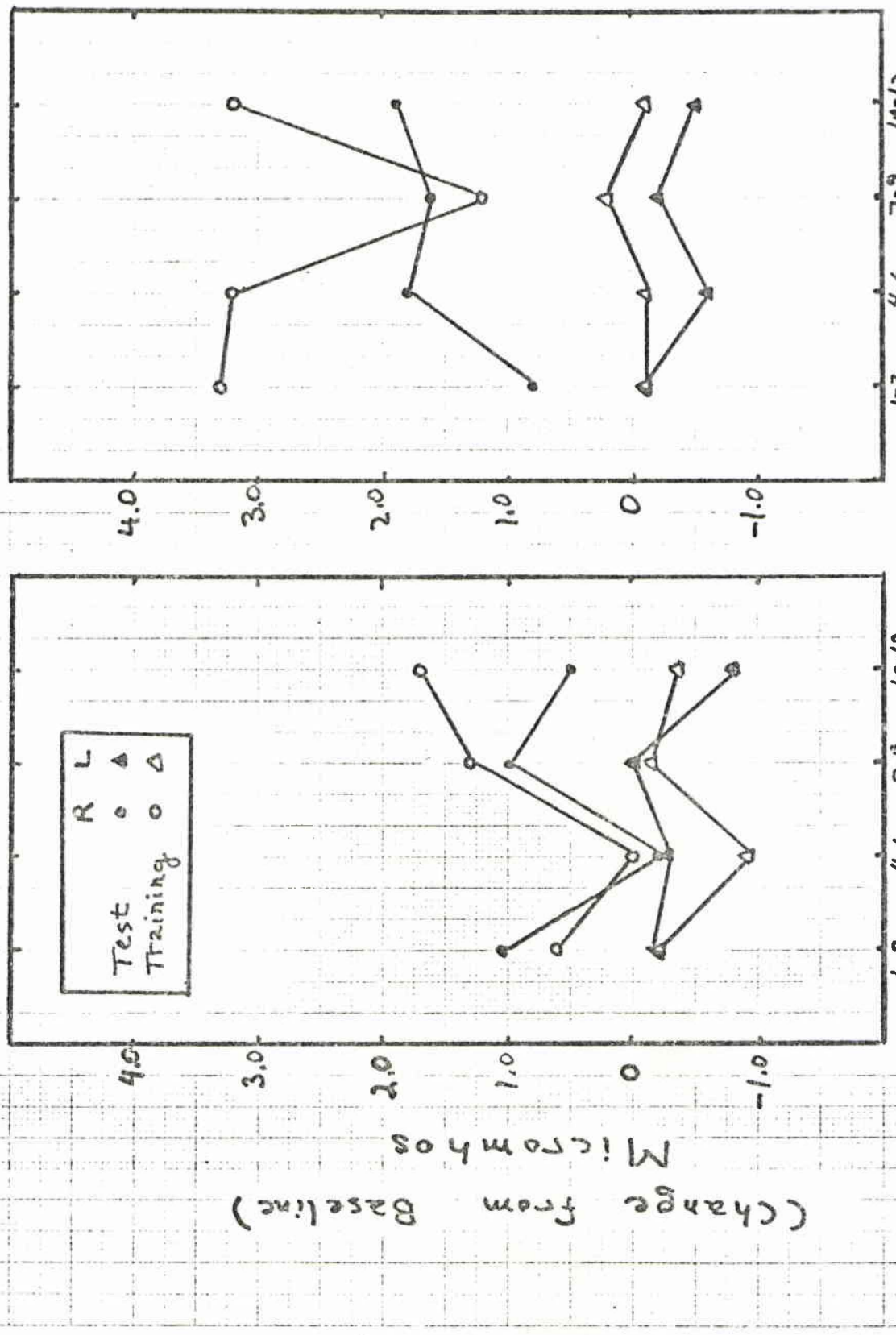
HR-1
SC-1
R L
Test ● ▲
Training ○ △
4.0
3.0
2.0
1.0
0
-1.0
1-3
4-6
7-9
10-12
TRIALS
(Change from Baseline)
Micromhos

Figure 4

Both the number of presentations of the feedback stimulus on each day, and the total duration of feedback presentations per day, were recorded for all subjects. Analyses of variance employing Groups, Types of Trial, and Days as variates were applied to both the duration and the frequency data. No main effect or interaction approached statistical significance with respect to frequency. However, a reliable Days effect was obtained with respect to duration, as well as reliable Groups-by-Days, and Groups-by-Types of Trial-by-Days interactions (for main effect, $F_{1,8} = 6.18$, $p < .05$; for Groups-by-Days interaction, $F_{1,8} = 5.55$, $p < .05$; for Groups-by-Types of Trial-by-Days interaction, $F_{1,8} = 11.31$, $p < .01$). The Types of Trial main effect also approached reliability with respect to duration ($F_{1,8} = 4.77$, $p < .10$). Altogether, these effects reflected tendencies for the feedback stimulus to be presented longer on day 1 than on day 2 on Raise trials in the SC-1 group, and on Lower trials in the HR-1 group, and opposite tendencies, i.e. for it to be presented longer on day 2 than on day 1, on Lower trials in the SC-1 group, and on Raise trials in the HR-1 group.

The two left-hand panels of Figure 5 examine the response profiles generated on Raise and Lower trials when voluntary control was exerted over skin conductance. The SC-1 and HR-1 groups have been combined for purposes of this graph, since they did not differ reliably with respect to any of the variables which were recorded. Perusal of Figure 5 suggests that Raise and Lower trials are differentiable with respect to skin conductance and heart rate when skin conductance was the target response. This was confirmed by statistical analyses, which indicated that only with respect to the two autonomic variables could Raise and Lower trials be differentiated at a level approaching reliability (for

Figure 5. Median changes in skin conductance (SC), heart rate (HR), gross body movement (GBM), respiration frequency (RF), respiration amplitude (RA), and eye movement (EM) on Raise and Lower test trials, on the last day of training to control skin conductance (skin conductance target), and on the last day of training to control heart rate (heart rate target). All measures are based on the difference between performance during the last ten seconds of the trial and pre-trial periods. Groups SC-1 and HR-1 have been combined. Skin conductance is plotted in micromhos, heart rate in beats per minute. Respiration frequency is plotted as respiratory period in milliseconds (negative-up). Body movement, respiration amplitude, and eye movement are in arbitrary units.

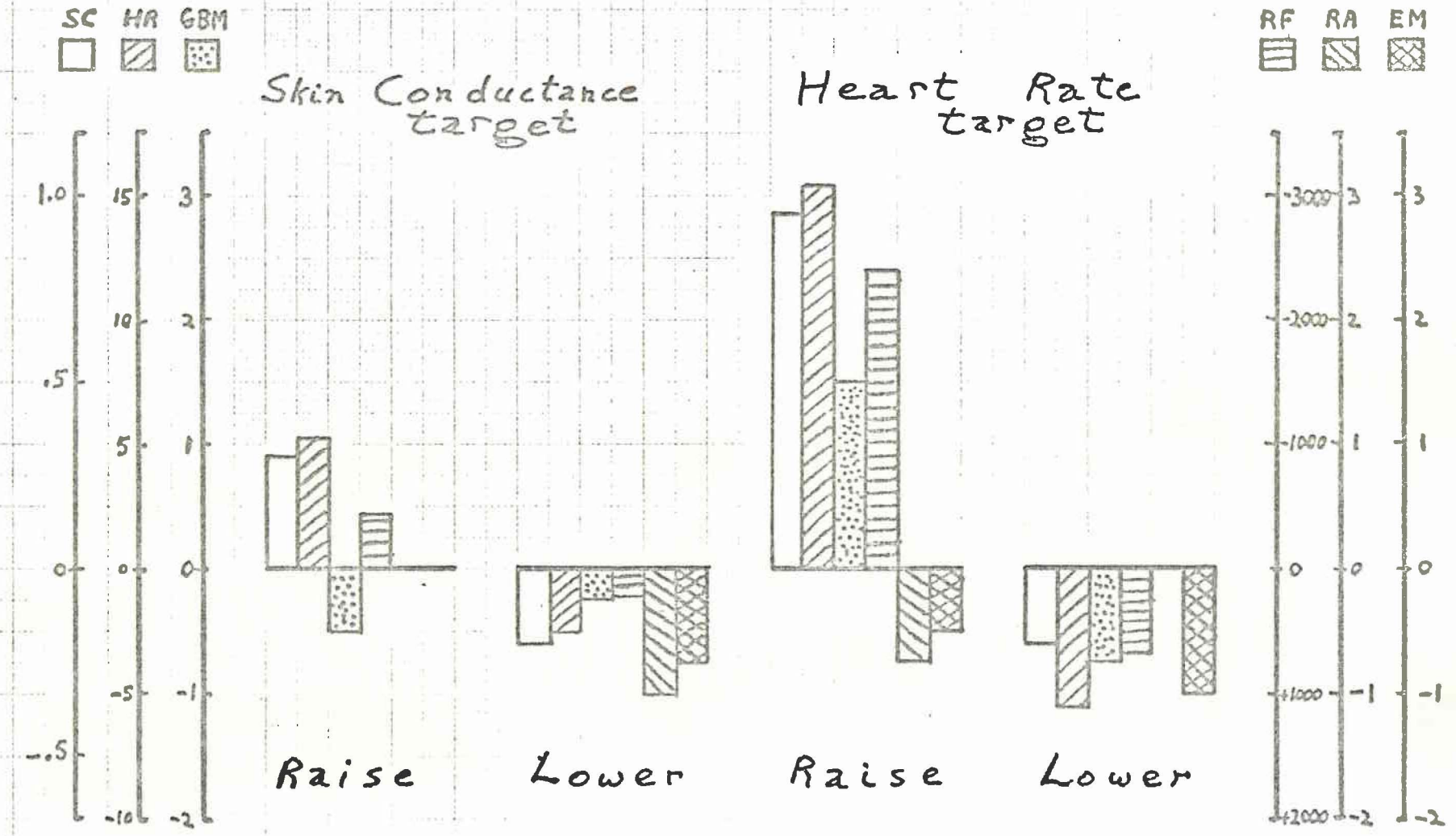
SC
HR
GBM
Skin Conductance target
Heart Rate target
RF
RA
EM
1.0
.5
0
-.5
15
10
5
0
-5
-10
3
2
1
0
-1
-2
-3000
-2000
-1000
0
+1000
+2000
Raise
Lower
Raise
Lower

Figure 5

skin conductance, $\underline{T}$ (10) = 2.5, $p < .005$, one-tail test; for heart rate, $\underline{T}$ (10) = 4, $p < .02$).

Inspection of the questionnaire results failed to reveal any difference between the two groups with respect to age or educational level. The overall mean perceived success at controlling skin conductance was rated at 3.3 on a 7-point scale (1 = not at all successful; 7 = very successful). Subjects perceived Raise trials to be reliably easier than Lower trials, the former rating a mean of 4.6 and the latter a mean of 2.1 on a 7-point scale of difficulty -- 1 = impossible; 7 = could control the activity at will -- ($\underline{T}$ (10) = 1, $p < .01$). Differences between the SC-1 and HR-1 groups did not approach significance with respect to either the success or the difficulty scales.

Finally, Raise and Lower trials could be differentiated reliably with respect to seven of the eleven affective scales. These were scales A (tense-relax), B (uncomfortable-comfortable), C (work-rest), E (sexual-asexual), G (unpleasant-pleasant), J (strained-flaccid), and K (anxious-calm), in all cases Raise trials being rated closer to the first pole of the dyad than Lower trials (for scale A, $\underline{T}$ (10) = 0, $p < .01$; for scale B, $\underline{T}$ (9) = 0, $p < .01$; for scale C, $\underline{T}$ (9) = 1, $p < .01$; for scale E, $\underline{T}$ (10) = 5, $p < .02$; for scale G, $\underline{T}$ (10) = 4, $p < .02$; for scale J, $\underline{T}$ (9) = 1, $p < .01$; for scale K, $\underline{T}$ (10) = 1.5, $p < .01$). The two training groups responded similarly on the scales, and did not differ reliably with respect to any of them.

Control over heart rate

Figure 6 presents test trial performance on the last day of heart rate training for subjects who received heart rate training first

Figure 6. Mean heart rate on Raise and Lower test trials on the second day of training to control heart rate. Subjects receiving heart rate training first (HR-1) and second (SC-1) are shown separately. Heart rate was measured at the mid-point of consecutive five-second intervals, beginning 30 seconds before trial onset and ending at the offset of the trial.

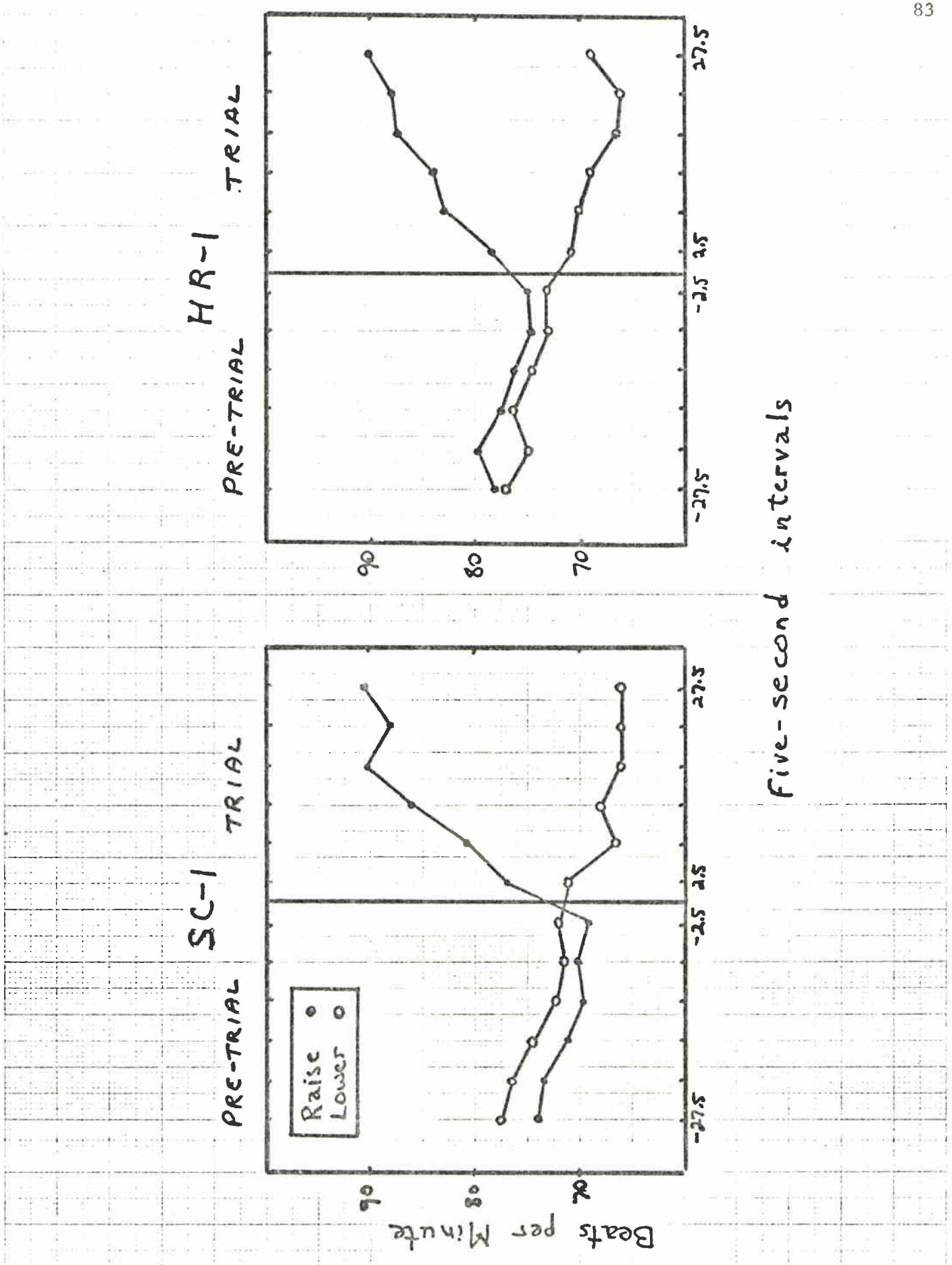
HR-1
PRE-TRIAL
TRIAL
SC-1
PRE-TRIAL
TRIAL
Raise
Lower
90
80
70
-27.5
-2.5
2.5
27.5
Beats per Minute
Five-second intervals

Figure 6

(HR-1, right-hand panel) and for subjects who received heart rate training second (SC-1, left-hand panel). Heart rate is plotted every five seconds during the 30-second periods preceding Raise and Lower trials (left-hand side of each panel), as well as during the trial periods (right-hand side of each panel).

As was the case when control over skin conductance was examined, two preliminary analyses were carried out. First, baseline differences were examined through analyses of variance employing Types of Trial and Seconds as variates, which were applied to baseline heart rate in each group. Only one resulting F statistic met or approached statistical significance, and this was a reliable main effect of Types of Trial in the SC-1 group ($F_{1,4} = 11.56$; $p < .05$). Heart rate was lower, by a mean of 2.5 beats per minute, during the baseline periods preceding Raise trials than during those preceding Lower trials, in the SC-1 group.[3] Second, differences in response forms were evaluated, as they were with respect to skin conductance, by noting, for each subject, which of the six trial measurements represented the maximum heart rate change in the targeted direction, and then comparing where this point occurred on Raise and Lower trials. The maximum change from baseline tended to occur earlier on Lower trials than on Raise trials (sign test, 8 of 10 subjects, $p = .11$). As was the case when skin conductance was the target response, subsequent analyses were based on the difference in performance between the last ten seconds of the trial period and the last ten seconds of the preceding baseline period, unless otherwise indicated.

The right-hand portion of each panel in Figure 6 depicts heart rate during Raise and Lower trials on the second day of heart rate training.

The performance of the two groups was compared through an analysis of variance employing Groups and Types of Trials as variates. This analysis yielded a reliable Types of Trial main effect ($F_{1,8} = 21.73$, $p < .01$), and an unreliable Groups-by-Types of Trial interaction ($F < 1$), indicating that voluntary control was present and comparable in both training conditions. Nonparametric analyses confirmed that the difference in performance between Raise and Lower trials was statistically significant in both groups (for the SC-1 group, T (5) = 1, $p < .05$; for the HR-1 group, T (5) = 0, $p < .025$, one-tail tests).

Subsequent analyses examined whether differences between Raise and Lower trials reflected primarily a raising of heart rate on Raise trials, a lowering on Lower trials, or both. The raising effect proved statistically significant in both groups (for the SC-1 group, T (5) = 1, $p < .05$; for the HR-1 group, T (5) = 0, $p < .025$, one-tail tests). However, the lowering effect proved reliable only in the HR-1 group (T (5) = 0, $p < .025$; for the SC-1 group, T (5) = 3, $p < .10$). Thus, there was evidence of cardiac control in both directions, although this evidence was statistically more prominent on Raise trials.

Subsequent analyses determined whether Lower trial performance reflected a genuine decrease in heart rate, or whether it merely reflected a long-term trend in the heart rate baseline. On the one hand, statistical analyses failed to confirm the reliability of the trend in heart rate apparent during the baseline periods of Figure 6. Analyses of variance employing Types of Trial and Seconds as variates, which were applied separately to baseline heart rate in the two groups, yielded neither reliable Seconds main effects nor Types of Trial-by-Seconds interactions. Moreover, examination of the recovery functions following trial offset also suggested that the lowering effect was genuine. These functions

are presented in Figure 7. There was a tendency for heart rate to decrease following Raise trials, and to increase following Lower

Figure 7. Heart rate before and after trial offset on the second day of training to control cardiac responding. Groups given heart rate training first (HR-1) or second (SC-1) are plotted separately. Heart rate is shown as a change from the pre-trial baseline.

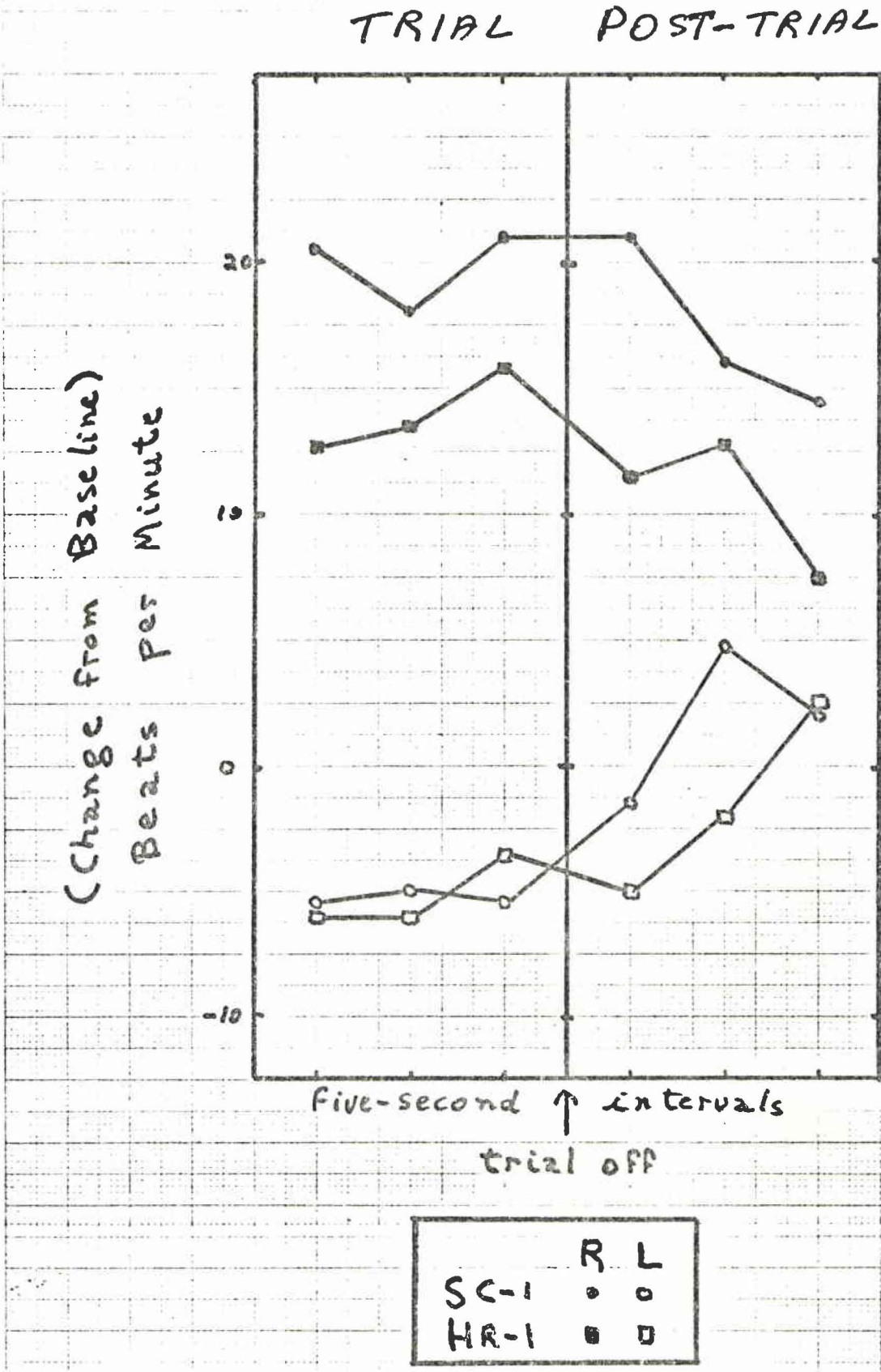
TRIAL
POST-TRIAL
20
10
0
-10
(Change from Baseline)
Beats per Minute
Five-second intervals
trial off
R L
SC-1
HR-1

Figure 7

trials, in both groups. An analysis of variance employing Groups, Types of Trial, and Time (last trial point versus third post-trial point) as variates evaluated the reliability of these changes. This analysis confirmed the reliability of the differences in the recovery functions following the two types of trial, in that a reliable Types of Trial by Time intereaction was obtained ($F_{1,8} = 26.40$, $p < .005$). Finally, also arguing that the lowering in heart rate which was observed on Lower trials is genuine is the fact that the decreases in heart rate which were produced when heart rate was rewarded were reliably larger than those which were produced when skin conductance was rewarded (T (10) = 11, $p < .05$; one-tail test). On the other hand, the magnitude of the heart rate decreases which were obtained on Lower trials was found to vary inversely with the duration of the preceding inter-trial interval. Performance on Lower trials which followed 30-, 40-, and 60-second inter-trial intervals was compared. The trials selected for this comparison were all from the last half of day 4 for the SC-1 group and from the last half of day 2 for the HR-1 group. The resulting Inter-Trial-by-Seconds interaction proved highly reliable, in the direction of smaller decreases in heart rate on trials which followed longer inter-trial intervals ($F_{10,90} = 20.05$, $p < .001$). A similar analysis carried out for Raise trials yielded a non-significant interaction ($F < 1$). These data indicate that heart rate decreases were more evident at shorter inter-trial intervals, where the pre-trial baseline was more likely to have been elevated by a preceding Raise trial.

Figure 8 examines acquisition effects in heart rate. The figure depicts the mean change in heart rate from baseline on the first trial and, subsequently, over the four blocks of three test trials. An analysis of variance employing Groups and Types of Trial as variates was

Figure 8. Acquisition of heart rate control. The difference between heart rate during the last 10 seconds of the trial and the baseline period is plotted for the very first trial (a test trial), and for successive blocks of test trials over two training days. Groups receiving heart rate training first (HR-1) and second (SC-1) are shown separately.

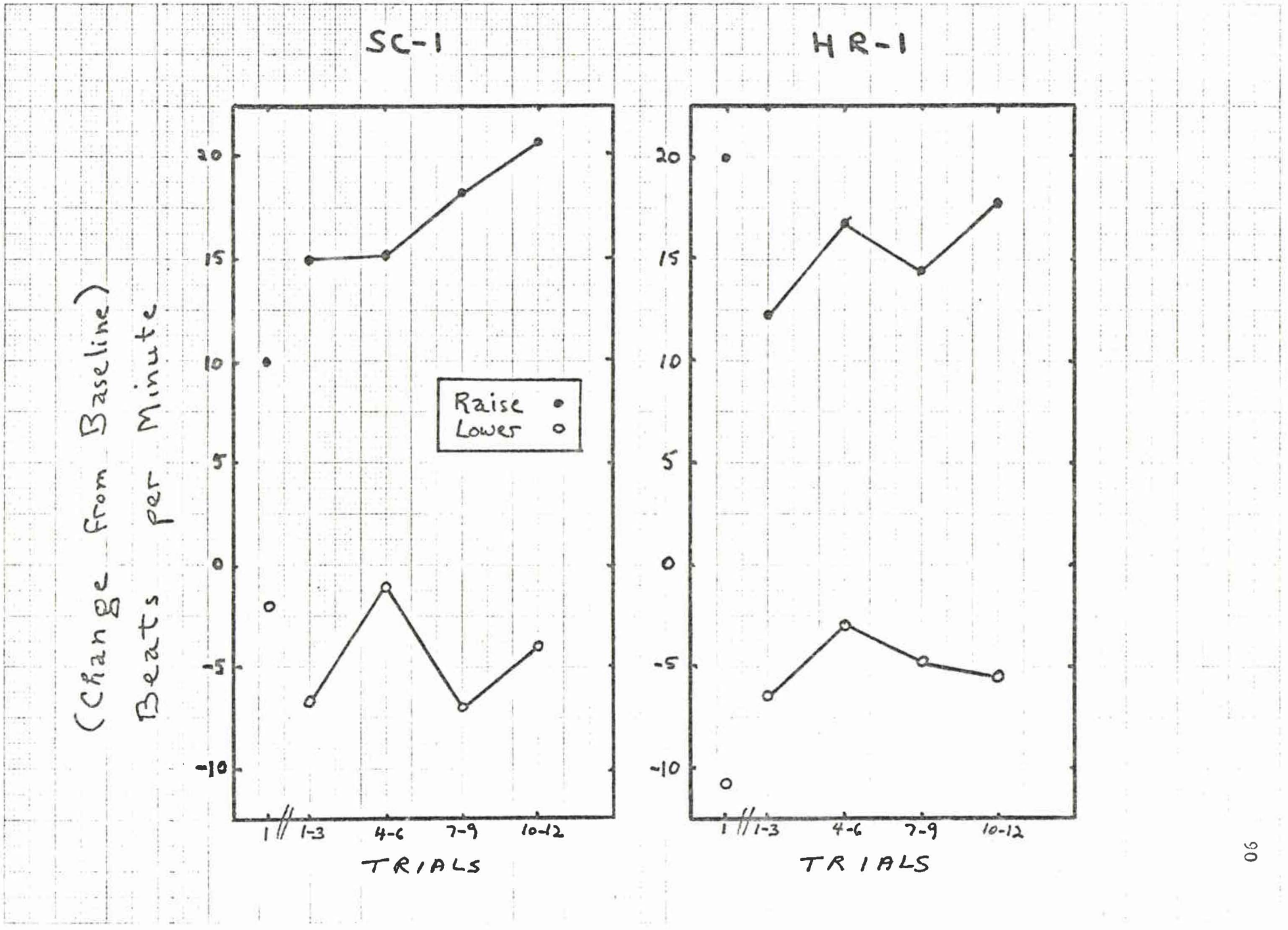
SC-1
HR-1
(Change from Baseline)
Beats per Minute
20
15
10
5
0
-5
-10
Raise
Lower
1
1-3
4-6
7-9
10-12
TRIALS

Figure 8

applied to performance on the first trial. This analysis yielded a reliable main effect of Types of Trial ($F_{1,8} = 17.61$, $p < .001$), and a Groups-by-Types of Trial interaction which approached statistical significance ($F_{1,8} = 3.50$, $p < .10$). These results indicate that both groups exerted some control over heart rate on the very first trial to control heart rate, and that this control tended to be more apparent in the HR-1 group. Acquisition effects were evaluated statistically through an analysis of variance which employed Groups, Types of Trial, and Trial Blocks as variates. While the Types of Trial main effect proved reliable ($F = 30.73$, $p < .01$), none of the interactions involving the Trial Blocks factor approached reliability. Thus, this analysis yielded no evidence that bidirectional control increased over the course of training, in either group.

The data presented so far were all obtained on test trials. The analysis which follows compared performance on test and training trials to determine the extent to which transfer took place. The relevant data are presented in Figure 9, which depicts the mean change in heart rate observed in the two groups on Raise and Lower trials over the four blocks of training, on test trials, and on training trials. Analysis of variance employing Groups, Feedback (present/absent), and Trial Blocks as variates were applied to performance on Raise and Lower trials separately. None of the main effects or interactions approached reliability, on either Raise or Lower trials. Performance was essentially the same on test trials, when the feedback stimulus was not presented, as on training trials, when the feedback stimulus was presented.

Both the number of presentations of the feedback stimulus on each day, and the total duration of feedback presentations per day, were recorded for all subjects. Analyses of variance employing Groups, Types of Trial, and Days as variates were applied to both the duration and the frequency data. No main effect or interaction approached reliability

Figure 9. A comparison of heart rate control on test trials (when the feedback stimulus was never presented) and on training trials (when the feedback stimulus was always presented). Groups receiving heart rate training first (HR-1) and second (SC-1) are plotted separately.

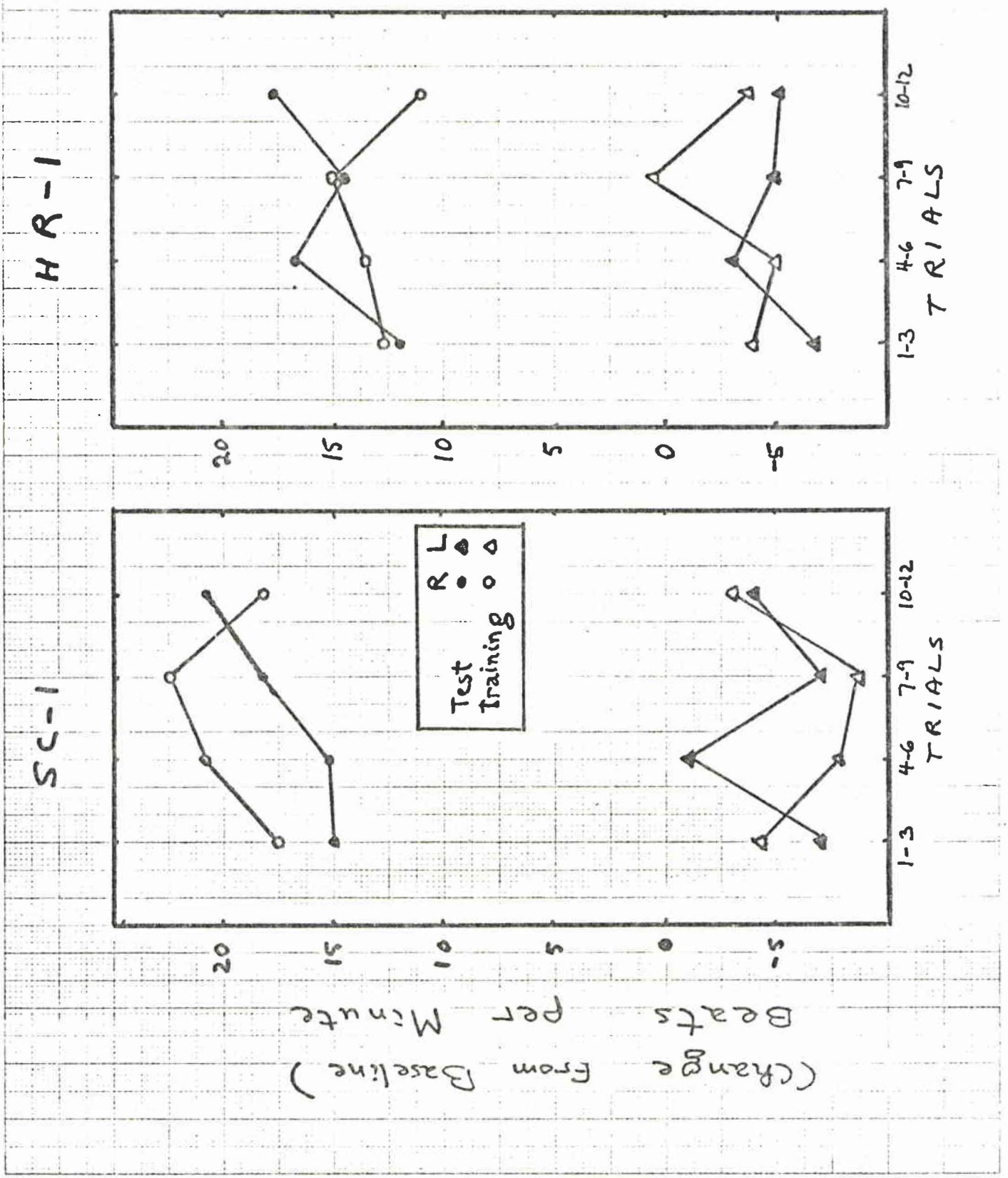
HR-1
SC-1
(Change From Baseline)
Beats per Minute
20
15
10
5
0
-5
1-3
4-6
7-9
10-12
TRIALS
R L
Test
Training

Figure 9

with respect to either frequency or duration.

The two right-hand panels of Figure 5 examine the response profiles generated on Raise and Lower trials when voluntary control was exerted over heart rate. The SC-1 and HR-1 groups have been combined for purposes of this graph, since they did not differ reliably with respect to any of the variables which were recorded. Inspection of Figure 5 indicates that rather different response profiles were generated on Raise and Lower trials. Statistical analyses indicated that the trials were differentiated reliably with respect to the two autonomic variables (for skin conductance, $\underline{T}$ (10) = 0, $\underline{p} < .01$; for heart rate, $\underline{T}$ (10) = 1, $\underline{p} < .001$, one-tail test), as well as with respect to body movement ($\underline{T}$ (9) = 0, $\underline{p} < .01$). None of the other differences approached statistical significance.

Examination of the questionnaire results revealed no differences between the two groups with respect to age,or educational level. The overall mean perceived success at controlling heart rate was rated at 5.0 on a 7-point scale (1 = not at all successful; 7 = very successful). Subjects perceived Lower trials to be somewhat more difficult than Raise trials, the former rating a mean of 4.4, the latter a mean of 5.5 on a 7-point scale of difficulty (1 = impossible; 7 = could control activity at will), but the difference in ratings was not reliable ($\underline{T}$ (9) = 9, $\underline{p} > .10$). Differences between the two groups did not approach significance with respect to either the success or the difficulty scales.

Finally, Raise and Lower trials could be differentiated reliably with respect to all of the eleven affective scales (minimum $\underline{T}$ (9) = 3.5, $p < .05$), Raise trials being rated as generally more tension-producing and arousing than Lower trials. The two groups responded similarly on

the scales, and in no case did they differ at a level approaching reliability.

Comparison of skin conductance and heart rate control

The results presented thus far have focussed on the questions of (i) whether control was established over skin conductance and heart rate, (ii) how this control developed over the course of training, (iii) the extent to which transfer took place from training to test trials, and (iv) the nature of the changes which took place in other systems when subjects attempted to gain control over skin conductance and heart rate. This section compares voluntary control of the two responses, with respect to several characteristics.

One difference between the two responses concerns how voluntary control was evidenced over the course of training. Comparison of Figures 3 and 8 shows that although voluntary control of heart rate was apparent and reliable on the first test trial, voluntary control of skin conductance was not. Voluntary control of skin conductance appeared to develop during subsequent training on this response, with a tendency toward better bidirectional performance in the group that received prior training on heart rate. The voluntary control of heart rate that was evident on the first trial of training on this response was not augmented by subsequent feedback training.

Voluntary control of the two responses was also associated with different response patterns. Analyses reported earlier (pp. 81 and 94; Figure 5) established that voluntary changes in skin conductance were accompanied by changes in an autonomic function (heart rate), whereas voluntary changes in heart rate were accompanied by changes in both autonomic (skin conductance) and somatic (body movement) functions. Comparisons of the response profiles produced in the two conditions revealed that

the differences in heart rate and body movement generated between Raise and Lower trials in the heart rate target condition were reliably larger than those generated in the skin conductance target condition, while those in respiration frequency approached reliability (for heart rate, $\underline{T}$ (10) = 6, $\underline{p} < .025$, one-tail test; for body movement, $\underline{T}$ (9) = 4, $\underline{p} < .05$; for respiration frequency, $\underline{T}$ (10) = 9, $\underline{p} < .06$).

The skin conductance and heart rate target conditions were also compared with respect to the subjects' responses on the questionnaire. Subjects perceived themselves more successful at controlling heart rate than at controlling skin conductance: control over heart rate received a mean rating of 5.0 and control over skin conductance a mean rating of 3.3 on the 7-point success scale, and this difference was reliable ($\underline{T}$ (10) = 5, $p < .02$. With respect to the affective scales, all 11 scales differentiated reliably between Raise and Lower trials when heart rate was reinforced, whereas only 7 did so when skin conductance was reinforced. However, in no case was the difference in ratings between Raise and Lower trials reliably different when changes in heart rate were rewarded than when changes in skin conductance were rewarded.

The effect of prior training on the other response was not particularly substantial for either response system. Skin conductance control on the first test trial was not reliably different for subjects who had, or had not, received prior heart rate training. However, statistical analyses suggested that subsequent acquisition of conductance control was more evident on Raise trials for subjects who had previously been trained to control heart rate (p. 74: Figure 3). On the other hand, initial control of heart rate tended to be greater for those subjects who were not pre-trained on skin conductance, but this effect was only marginally significant (p. 90; Figure 8). The generally insubstantial nature

of these transfer-of-training effects was further indicated by the fact that no relationship was found between success at controlling skin conductance and success at controlling heart rate. Product-moment correlations were computed between the magnitude of the skin conductance changes on the last day of training to control skin conductance, and the magnitude of the heart rate changes on the last day of training to control heart rate. These were computed separately for Raise and Lower trials. In neither case did the resulting correlation coefficient approach reliability (for Raise trials, $r = -.171$; for Lower trials, $r = -.001$; in both cases, $p > .10$).

Finally, the skin conductance and heart rate target conditions were compared with respect to the frequency and duration of feedback presentations. Analyses of variance employing Target Response, Types of Trial, and Days as variates were applied to both the frequency and duration data. The Target Response main effect proved highly reliable with respect to feedback frequency: feedback was presented more

frequently when heart rate was reinforced than when skin conductance was reinforced ($F_{1,9} = 75.16$, $p < .001$). However, the Target Response variable did not interact with the Types of Trial variable. Contrary to what was observed with respect to frequency, duration of feedback presentations did not vary with the nature of the rewarded response. However, a reliable Target Response-by-Types of Trial interaction was obtained with respect to duration ($F_{1,9} = 10.05$, $p < .05$), in the direction of longer feedback presentations on Raise trials in the skin conductance than in the heart rate target condition, and shorter feedback presentations in the skin conductance than in the heart rate target condition, on Lower trials.

Discussion

The primary goal of Experiment 1 was to develop a procedure for bringing skin conductance and heart rate under voluntary control. The results showed that subjects were able to generate reliable differences in skin conductance between Raise and Lower trials when skin conductance was the target response, and in heart rate when heart rate was the target response. Furthermore, there was evidence that control over both responses took place on both Raise and Lower trials, although cardiac control was more apparent on Raise trials. The differences which were obtained between Raise and Lower trials in skin conductance and in heart rate could not be attributed to differences in feedback parameters, since neither frequency nor duration of feedback presentations differentiated reliably between the two types of trial. Moreover, control over both skin conductance and heart rate was present on test trials, as well as on training trials. There was transfer from training to test trials.

Experiment 1 also examined and compared the correlates of control over skin conductance and heart rate. When changes in skin conductance were rewarded, reliable differences were produced between Raise and Lower trials not only with respect to skin conductance, but also with respect to another autonomic response, heart rate. When changes in heart rate were rewarded, reliable differences were produced between Raise and Lower trials not only with respect to heart rate, but also with respect to skin conductance and gross body movement. Bidirectional differences in heart rate and body movement were larger when heart rate was the target response than when skin conductance was the target response. Differences in skin conductance and heart rate control were accompanied further by differences in a number of affective dimensions, which were somewhat (though not reliably) more pronounced in the heart rate target than in the skin conductance target condition. Finally, the questionnaire results indicated that subjects found heart rate control easier to produce than skin conductance control

A third goal of Experiment 1 was to determine whether transfer-of-training occurred when subjects were pre-trained on a second autonomic response. The effect of pre-training on a second response appeared to be relatively small. Initial control of skin conductance was not reliably better when subjects were pre-trained on heart rate, although acquisition of conductance control was subsequently manifested primarily in this group. Initial control of heart rate, on the other hand, was diminished by prior training on skin conductance, but this effect was only marginally significant. Between-subjects correlations relating voluntary skin conductance and heart rate control were low and statistically unreliable.

These results have implications for the neural mechanisms involved in the performance of the voluntary changes in skin conductance and heart rate. The fact that both autonomic responses recorded in this experiment differentiated reliably between Raise and Lower trials in both target conditions suggests some overlap between these mechanisms. On the other hand, the facts that somatic correlates of control were present in the heart rate target condition but not in the skin conductance target condition, and that there was little transfer-of-training and no correlation between the degree of control over skin conductance and the degree of control over heart rate, indicate that the mechanisms involved in the performance of the electrodermal and cardiac responses are not identical. Thus, these results suggested the involvement of overlapping but not identical processes in the performance of the voluntary skin conductance and heart rate responses. These processes will be examined further in Experiment 2.

The present study combined instructions, strategy suggestions, and feedback in an attempt to demonstrate voluntary control. Although the presence of heart rate control on the first test trial indicates that exteroceptive feedback was not necessary for the performance of this response, it was generally not possible to assess the contribution of these experimental variables to electrodermal or cardiac control in this study. The contribution of each variable was examined in Experiment 2, which is presented in the next chapter.

Chapter 4: Experiment 2

Experiment 1 developed and tested a procedure for establishing voluntary control of the cardiac and the electrodermal systems. The control which was obtained over skin conductance and heart rate in Experiment 1 could have resulted from the operation of one or more of three variables which were employed in that procedure to bring about effective control. The first goal of Experiment 2 was to evaluate the contributions of each of these three variables to voluntary control over skin conductance and heart rate. The experiment examined the relative importance of (i) simple instructions to control one or the other system, (ii) the suggestion of behavioural strategies expected to facilitate control over skin conductance and heart rate, and (iii) the provision of feedback for responses of appropriate direction and magnitude, to voluntary control of skin conductance and heart rate.

Experiment 1 also examined response correlates of skin conductance and heart rate control. It was found that changes were in no case confined to the target system. Response correlates were also examined in Experiment 2. However, this experiment examined response profiles generated under different experimental conditions, and determined whether the response profiles produced when skin conductance and heart rate are brought under voluntary control depend upon how training is carried out. Hence, a second goal of Experiment 2 was to compare the response profiles generated when subjects were simply instructed to increase and decrease skin conductance or heart rate, with those generated when subjects were also provided with strategy suggestions, or feedback, or both.

A final goal of Experiment 2 was to compare the control which was obtained over skin conductance with that obtained over heart rate. This entailed a comparison of the effects of the different training variables (instructions, strategies, and feedback) on the two response systems, and a comparison of the response profiles generated in the skin conductance and heart rate target conditions. These comparisons have implications for the mechanisms which are involved in the acquisition and performance of voluntary electrodermal and cardiac changes.

Experiment 2 utilized 8 groups of subjects in a 2 x 4 factorial design. Four groups were concerned with control over skin conductance, and 4 others with control over heart rate. Homologous skin conductance and heart rate target groups were treated identically, with the single exception that subjects were attempting to control skin conductance in one case and heart rate in the other.

All groups were instructed, i.e. were informed of the response to be controlled and the direction of the changes to be produced. Subjects in the Instructions (I) group received no further information. They were asked to raise, for example, their heart rate on Raise trials, and to lower it on Lower trials. Subjects in the Instructions-Strategies (IS) group, however, were also provided with potentially useful behavioural strategies. These suggested strategies were similar to those which were utilized in Experiment 1, and were the same for heart rate and skin conductance. They are given in Appendix A. Comparison of the degree of control exerted by groups I and IS evaluates the effectiveness of the strategies in establishing and maintaining voluntary control.

Subjects in the Instructions-Feedback (IF) group did not receive any strategy suggestions, but rather were provided with feedback whenever

they successfully raised their heart rate (or skin conductance) on Raise trials, and lowered it on Lower trials. Comparing groups I and IF therefore evaluates the effect of feedback training in establishing and sustaining voluntary control[4].

Finally, subjects in the Instructions-Strategies-Feedback (ISF) group were provided with all three training variables. Subjects in this group were treated essentially as were those in Experiment 1. Hence, this group constituted a partial replication of Experiment 1, the major differences being that subjects in Experiment 2 received either skin conductance or heart rate training, but not both, and that they also received one more day of training on the target system than did those in Experiment 1. Comparison of groups IF and ISF, like that between groups I and IS, provides information about the role of strategies in establishing voluntary control. Comparing groups IS and ISF, on the other hand, yields information about the role of feedback in maintaining voluntary control.

Method

Subjects

A total of 64 subjects served in this experiment, each experimental group comprising 8 subjects. All subjects were male, and all were naive to the experimental situation. All were volunteers, recruited by means of advertisements placed around the McMaster University campus. They ranged between 17 and 39 years of age, with a mean age of 24.3 years.

Apparatus and electrophysiological recordings

All apparatus and electrophysiological recording facilities were as in Experiment 1.

Procedure

All subjects were run for three days, which were almost always con-

secutive, and they were run at approximately the same time each day. Subjects earned $3.00 per day for their participation, plus a bonus. In order to equate the groups for possible motivational effects of earning bonus money, eight possible 3-day combinations of daily bonuses were composed arbitrarily, and one subject in each group was assigned to one of these combinations based on his performance on the first day.

As in Experiment 1, subjects were provided with a standard set of instructions at the beginning of each day. These contained information about the general purposes of the study and the functions of the various recording devices, and specified the target response system. Information was also provided about the contingencies which nominally would be in effect during the session. In addition, subjects who received strategies, feedback, or both, were appropriately instructed. The instructions given the various groups are presented in Appendix A. Having read the instructions, the experimenter left the room, and the experiment began two to three minutes later.

As in Experiment 1 each session comprised 41 trials, including 20 Raise and 21 Lower trials (or the reverse). Trial duration and inter-trial intervals were as in Experiment 1, each trial lasting 30 seconds, and inter-trial intervals ranging from 30 to 60 seconds, with a mean of 40 seconds. Trials were presented in a mixed order and were randomized with respect to inter-trial intervals. The first and last Raise and Lower trials were always test trials. The same three trial sequences were used that were used in Experiment 1 (see Appendix B). Groups were matched for sequence of trial presentations, which was counterbalanced across days and between subjects within each group.

The criteria used for providing feedback to those subjects receiving feedback, i.e. those in the IF and ISF groups, differed somewhat from the criteria used in Experiment 1. A change in the criterion-setting procedure was felt necessary because, on several occasions in Experiment 1, it had been observed that skin conductance tonic level sometimes shifted considerably during the interval between two Raise or two Lower trials. This resulted in the subject being rewarded either continuously or not at all during the subsequent trial, regardless of his behaviour on that trial. It was felt that basing the criterion on behaviour immediately preceding trial onset, rather than on the previous trial, would alleviate this problem. Thus, while in Experiment 1 criterion for a given Raise or Lower trial was based on performance on the previous like trial, the criterion used in Experiment 2 was based on behaviour in the period between 30 and 10 seconds before the start of the trial. Otherwise, procedures for determining performance criteria were as in Experiment 1, the only difference being in the reference period[5].

At the end of day 3, subjects were asked to complete a questionnaire identical to that used in Experiment 1, except for the addition of questions about their smoking habits, and about whether they engaged in meditation or yoga.

Data analysis

Measurements of skin conductance, heart rate, gross body movement, respiration frequency and amplitude, and eye movements, were taken as in Experiment 1. These data were analyzed as described previously.

Results

Control over skin conductance

Figure 10 presents the mean skin conductance during test trials

Figure 10. Mean skin conductance on test trials on the last day of training for subjects in the I, IS, IF, and ISF groups attempting to control skin conductance. Skin conductance was measured at the midpoint of consecutive five-second intervals, beginning 30 seconds before the start of the trial onset, and ending at the end of the trial.

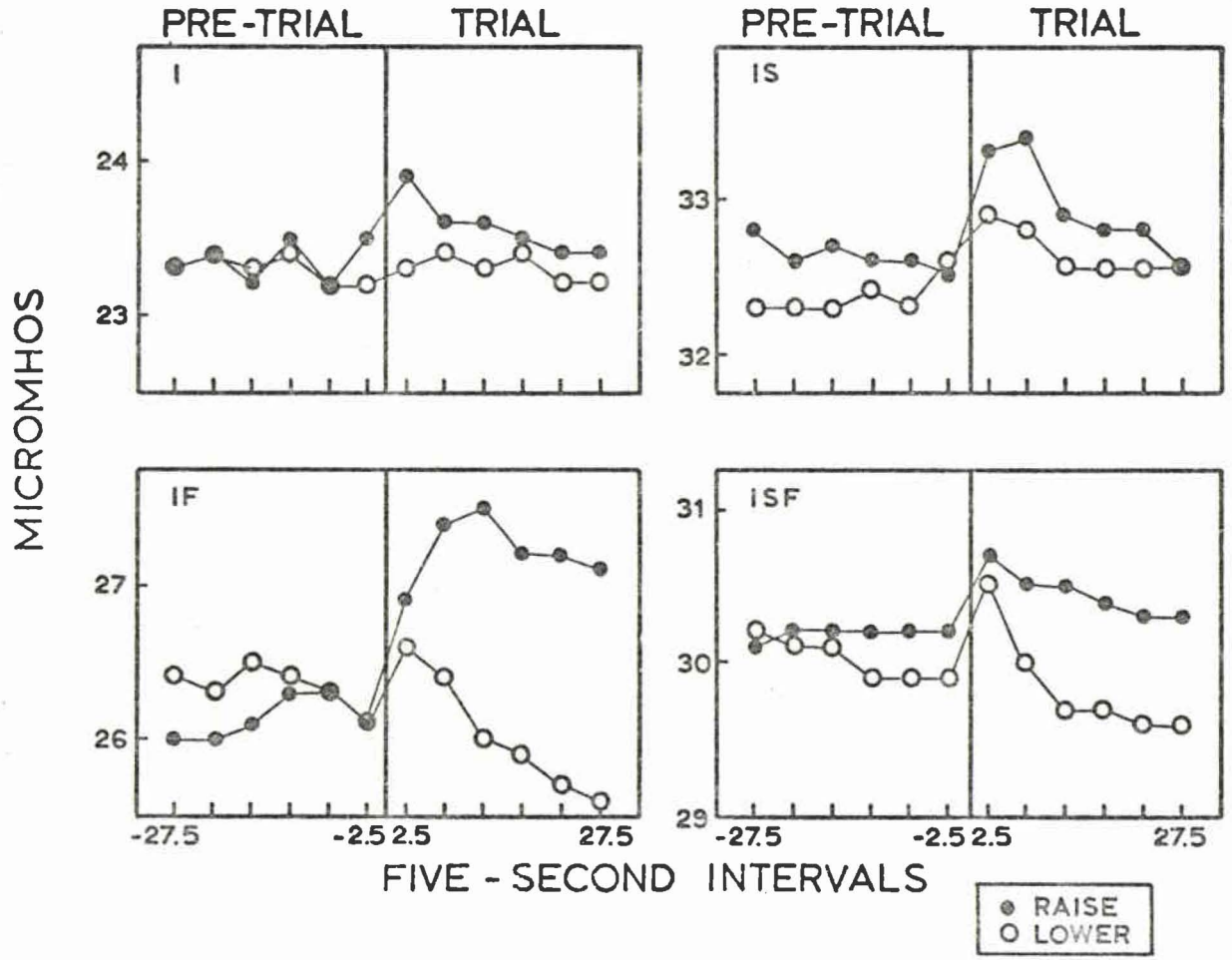
PRE-TRIAL
TRIAL
I
IS
IF
ISF
MICROMHOS
24
23
33
32
27
26
31
30
29
-27.5
-2.5
2.5
27.5
FIVE - SECOND INTERVALS
RAISE
LOWER

Figure 10

for each of the four groups which received skin conductance training, on the last day of training. Skin conductance is plotted as in Experiment 1, every five seconds during the 30-second periods preceding Raise and Lower trials (left-hand side of each panel) as well as during the trial periods (right-hand side of each panel).

As in Experiment 1, preliminary analyses were carried out on baseline skin conductance and on the response forms displayed during the trial periods. Analyses of variance using Types of Trial and Seconds as variates were applied to baseline skin conductance in each group. In no case did the Seconds main effect, the Types of Trial main effect, or the Types of Trial-by-Seconds interaction approach reliability. The difference in the response forms displayed on Raise and Lower trials was evaluated by determining, for each subject, which of the six trial measurements represented the maximum change in skin conductance in the targeted direction, and then comparing where this point occurred on Raise and Lower trials. There was a tendency for the maximum change from baseline in the targeted direction to occur earlier on Raise trials in all groups. However, this effect met or approached reliability only in the IF and ISF groups, the two groups in which skin conductance control was more evident (IF group, 7 of 7 subjects, sign test, $p < .02$; ISF group, 7 of 8 subjects, $p < .10$). As in Experiment 1, subsequent analyses focussed on the difference in performance between the last ten seconds of the trial period and the last ten seconds of the preceding baseline period.

Inspection of trial performance in Figure 10 suggests that control over skin conductance was not achieved to the same extent in all groups. This was examined by means of an analysis of variance applied to the difference in responding between the last ten seconds of the trial and pre-trial periods, in which Feedback (present/absent), Strategies (pre-

sent/absent), and Types of Trial served as variates. This analysis yielded two significant F's, a main effect of Types of Trial ($F_{1,28}$= 8.47, $p < .01$), as well as a reliable Feedback-by-Types of Trial interaction ($F_{1,28}$= 4.33, $p < .05$).[6] This interaction reflects the superior performance which is apparent under conditions of feedback in Figure 10, on both types of trial -- although, of course, in opposite directions on Raise and Lower trials. Performance was examined further by analyses of the difference in skin conductance between the last ten seconds of Raise and Lower trials in each of the four groups, and by comparing each group's performance to that of its appropriate control. Only in the two groups provided with feedback was performance reliably different on Raise and Lower trials (IF group, T (8) = 1, $p < .01$; ISF group, T (8)= 2, $p < .02$; one-tail tests). Moreover, the magnitude of the difference in skin conductance which was obtained between Raise and Lower trials was reliably larger in the IF and ISF groups than in the two groups not provided with feedback (Mann-Whitney U (N = 32) = 79.5, $p < .05$, one-tail test). This reflected a reliable difference between the performance of the I and IF groups (U (16) = 13.5, $p < .05$, one-tail test), but not between the performance of the IS and ISF groups (U (16) = 26.5, $p > .10$).

These results, then, indicate that control over skin conductance was achieved only when feedback was provided. Other analyses examined whether the skin conductance differences which were obtained between Raise and Lower trials under conditions of feedback reflected primarily a raising of skin conductance on Raise trials, a lowering on Lower trials, or both. While perusal of Figure 10 suggests that control was more prominent on Raise trials, statistical analyses revealed that control was in fact reliable in both directions in the IF group (Raise:

$\underline{T}$ (8) = 4, $\underline{p} < .025$; Lower: $\underline{T}$ (8) = 1.5, $\underline{p} < .01$; one-tail tests), and that it reached statistical significance only on Lower trials in the ISF group ($\underline{T}$ (8) = 5, $\underline{p} < .05$, one-tail test). Moreover, several considerations suggest that the lowering effect was genuine. First, analyses of variance revealed no reliable trends during the pre-trial periods that might have contributed to a gradual lowering of baseline skin conductance. Second, decreases in skin conductance were no greater on Lower trials which followed short inter-trial intervals, where recovery functions might have been more evident, than on trials which followed longer inter-trial intervals (Fs < 1 for both the IF and ISF groups). Third, decreases in skin conductance which were produced in the IF group trained to control skin conductance were reliably larger than those produced in the IF group trained to control heart rate ($\underline{U}$ (16) = 13.5, $\underline{p} < .05$, one-tail test). Finally, recovery functions following trial offset were examined to determine whether the lowering effect was genuine. These are portrayed in Figure 11. Although not reliable, there was a tendency for skin conductance to recover to a common, intermediate baseline following trial offset in the group (IF) in which the largest skin conductance changes were observed. Considered collectively, these several analyses suggest that bidirectional control was established over skin conductance, at least in the IF group.

Figure 12 examines acquisition effects. The figure depicts the mean change in skin conductance on Raise and Lower trials over the three days of training, for each of the four groups attempting to control skin conductance.

Figure 11. Skin conductance before and after trial offset on the last day of training to control palmar sweating. Only the groups provided with feedback (IF and ISF) are shown. Skin conductance is plotted as a change from the pre-trial baseline. Data are from test trials.

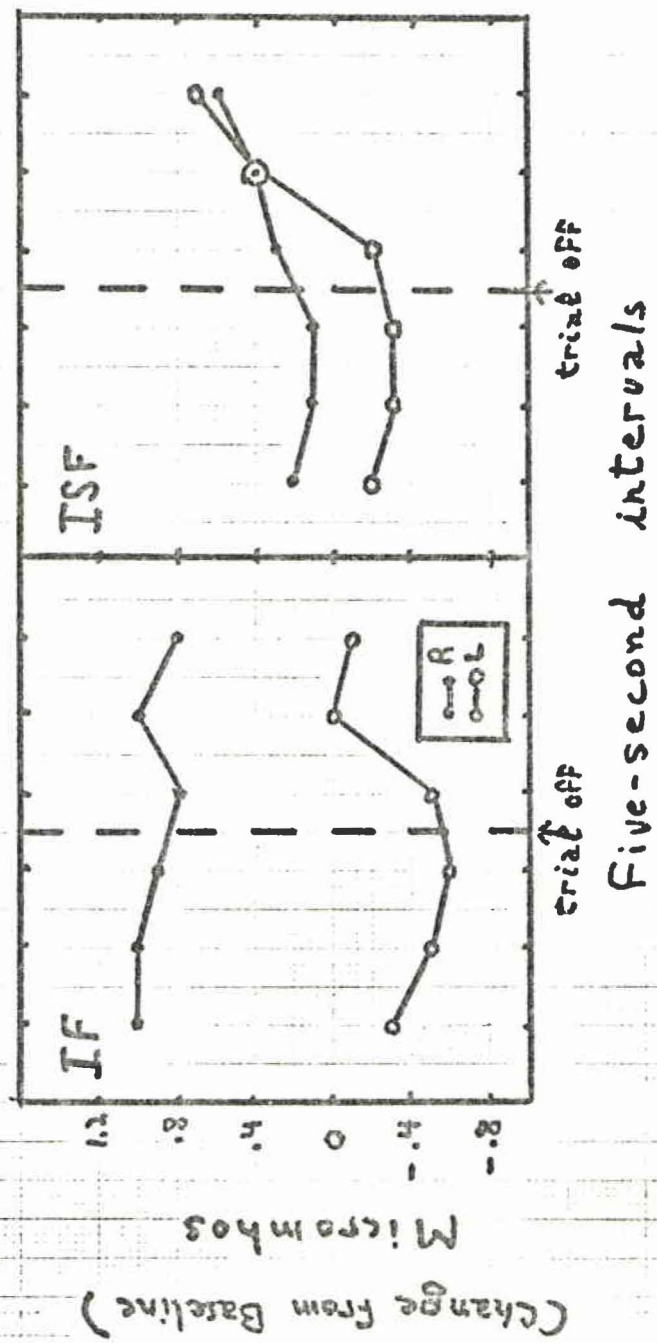
PRE-TRIAL
TRIAL
POST-TRIAL
PRE-TRIAL
TRIAL
POST-TRIAL
IF
ISF
trial OFF
trial OFF
R
L
Five-second intervals
Change from Baseline
Micromhos

Figure 11

Figure 12. Acquisition of skin conductance control. The difference between skin conductance in the last 10 seconds of the trial and pre-trial periods is plotted for test trials on the 3 training days.

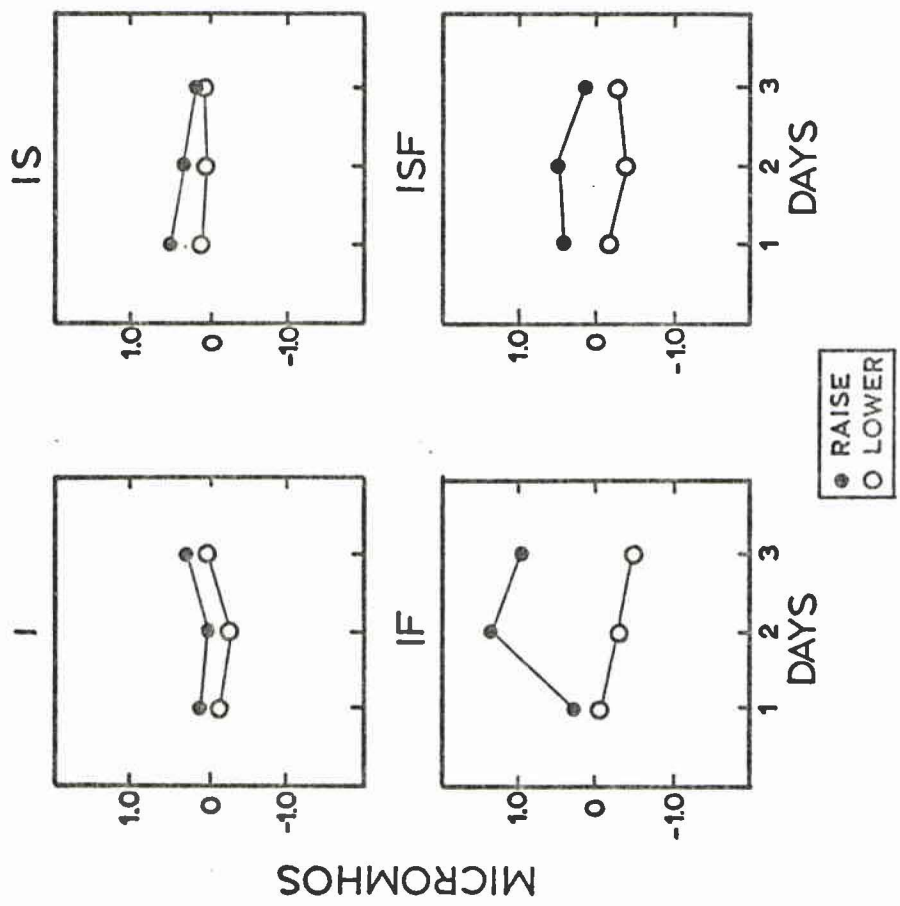

Figure 12

Acquisition effects were examined by means of analyses of variance, employing Days and Types of Trial as variates, which were applied to each group separately. Only in the IF group did the Days-by-Types of Trial interaction prove reliable ($F_{2,14} = 8.32$, $p < .01$). Moreover, post-hoc Newman Keuls analyses revealed that Raise trial performance was reliably different on day 1 than on day 3 ($p < .01$ in both cases), and that the difference between Lower trial performance on day 1 and Lower trial performance on day 3 approached statistical reliability ($p < .07$). Thus, while there was evidence for acquisition only in the IF group, it appears that acquisition took place on both types of trial in this group, although it was more evident on Raise trials.

The data presented so far were all gathered on test trials. The next analysis examined the extent to which transfer took place from training to test trials. The pertinent data are presented in Figure 13, which depicts the mean skin conductance changes observed in the IF and ISF groups over the three days of training, on test trials and on training trials. Performance on test and training trials was compared by analyses of variance which employed Days and Feedback (present/absent) as variates, and which were applied separately to Raise and Lower trials and to the IF and ISF groups. The only F statistic which proved reliable was with respect to the Feedback-by-Days interaction in the ISF group on Lower trials ($F_{2,14} = 8.14$, $p < .01$). This reflected an inferior

Figure 13. A comparison of skin conductance control on test trials (when the feedback stimulus was never presented) and on training trials (when the feedback stimulus was always presented). The unit employed is the difference between skin conductance in the last 10 seconds of the trial and pre-trial periods.

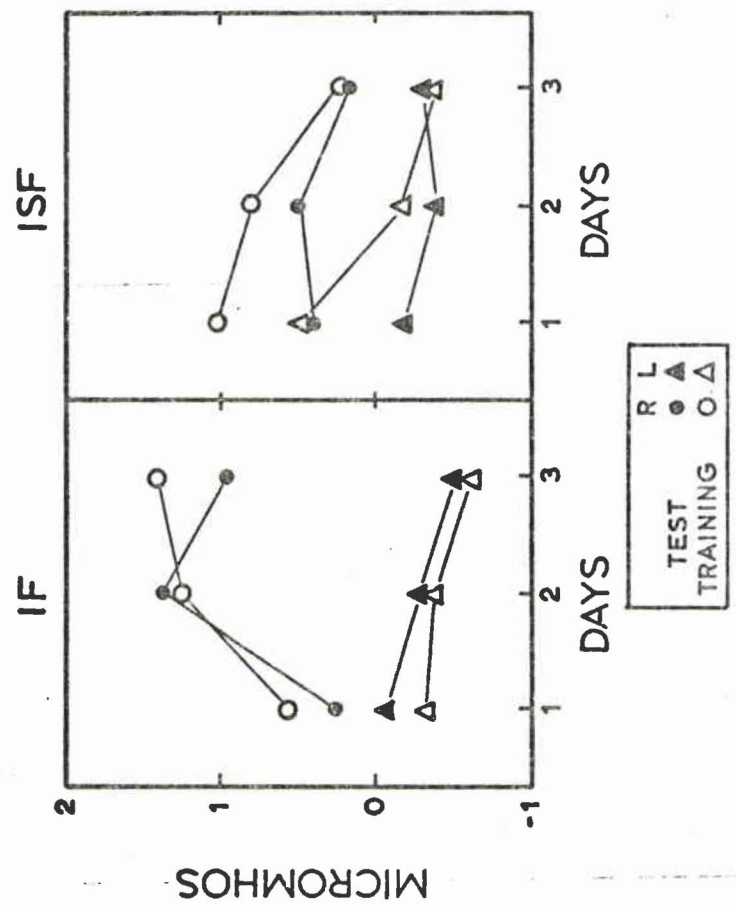

Figure 13

performance on training trials compared to test trials on day 1, an effect which evaporated over days. The fact that a similar (albeit unreliable) pattern was also obtained on Raise trials in this group suggests that orienting responses to feedback presentation were particularly large in these subjects early in training (possibly as they attempted to follow the strategy suggestions), but that these habituated over days.

The results presented so far have focussed primarily on the effects of feedback on electrodermal control. They indicate that, while providing subjects with instructions alone did not result in any degree of electrodermal control, providing them with both instructions and feedback resulted in substantial electrodermal control. What about the effects of strategies? The analysis of variance reported earlier (p.109), which employed Feedback, Strategies, and Types of Trial as variates yielded neither a reliable Strategy main effect nor a Strategy by Types of Trial interaction. This is consistent with what is apparent from perusal of Figure 10. Inspection of that figure indicates that the performance of the I and IS groups did not differ. Nor did the addition of strategies under conditions of feedback appear to improve performance. In fact, non-parametric analyses suggested the opposite effect. The IF and ISF groups differed almost reliably with respect to the difference in the electrodermal changes produced on Raise and Lower trials ($\underline{U}$ (16) = 18, $\underline{p} < .06$, two-tail test), in the direction of poorer performance by subjects given strategy suggestions. Thus, it is clear that the addition of strategies did not improve performance. In fact, provision of strategy information tended to impair performance under

feedback conditions.

Both the number of presentations of the feedback stimulus on each day, and the total duration of feedback presentations per day were recorded for those subjects receiving feedback, i.e. those in the IF and ISF groups. Analyses of variance employing Groups, Types of Trial, and Days as variates were applied to both the duration and the frequency data. These analyses failed to reveal either reliable Groups or Types of Trial main effects or interactions, with respect to either frequency or total duration of feedback presentations. However, statistically significant main effects of Days were obtained with respect to both frequency ($F_{2,28} = 3.49$, $p < .05$) and total duration of feedback presentations ($F_{2,28} = 4.27$, $p < .05$) in the direction of more and longer feedback presentations as a function of days.

Figure 14 examines response profiles. The upper half of the figure presents the median changes observed on Raise trials in all of the variables recorded in this experiment, on test trials of the last day of training, in each of the four groups attempting to control skin conductance. The lower half of Figure 14 presents the corresponding changes on Lower trials. These data were evaluated by 3-way analyses of variance employing Feedback, Strategies, and Types of Trial as variates, as was done for skin conductance. These indicated that reliable differences between the two types of trial were present with respect to all of the variables except respiration frequency and eye movement (for skin conductance, $F_{1,28} = 8.47$, $p < .01$; for heart rate, $F_{1,28} = 12.22$, $p < .01$; for body movement, $F_{1,28} = 4.42$, $p < .05$; for respiration amplitude, $F_{1,28} = 7.21$, $p < .05$). However, with the exception of skin

Figure 14. Changes in skin conductance (SC), heart rate (HR), gross body movement (GBM), respiration frequency (RF), respiration amplitude (RA), and eye movement (EM), on Raise (upper half) and Lower (lower half) test trials, on the last day of training for the subjects in each of the groups attempting to control skin conductance. All measures are based on the difference between performance during the last 10 seconds of the trial and pre-trial periods for the subjects who yielded the median difference in skin conductance between Raise and Lower trials. Skin conductance is plotted in micromhos. Heart rate is in beats per minute. Respiration frequency is plotted as respiratory period in milliseconds (negative up). Body movement, respiration amplitude, and eye movement are in arbitrary units.

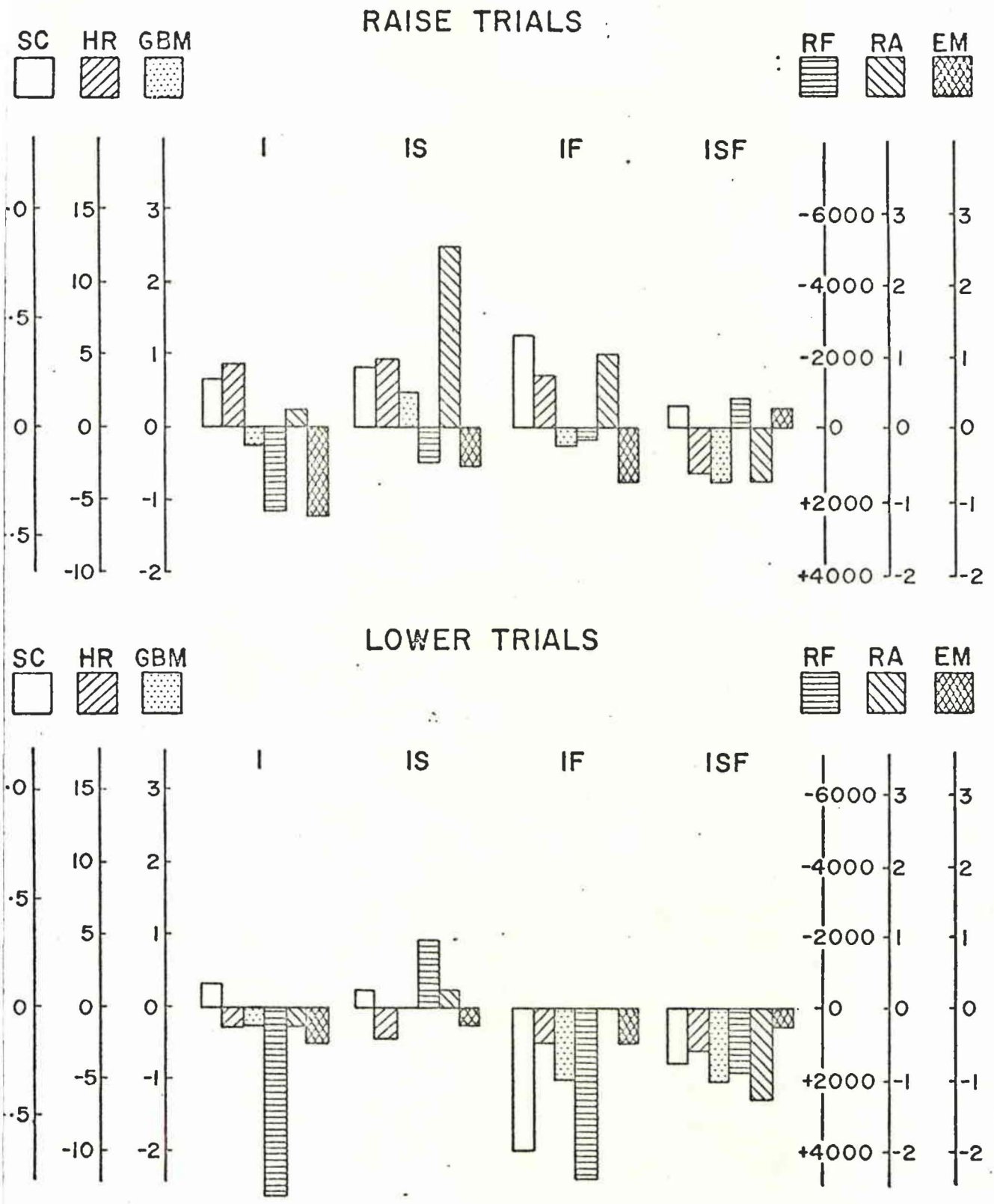
RAISE TRIALS
SC
HR
GBM
RF
RA
EM
I
IS
IF
ISF
·0
·5
·5
15
10
5
0
-5
-10
3
2
1
0
-1
-2
-6000
-4000
-2000
0
+2000
+4000
LOWER TRIALS

Figure 14

conductance, which was mentioned earlier, in no case was any main effect of Feedback or any interaction of Feedback and Types of Trial reliable. Further analyses examined the differences between the two types of trial in each group individually. Reliable differences were obtained only in the IS and ISF groups, in the IS group with respect to heart rate ($\underline{T}$ (8) = 4, $\underline{p} < .05$) and respiration amplitude ($\underline{T}$ (8) = 0, $\underline{p} < .02$), in the ISF group with respect to respiration frequency ($\underline{T}$ (8) = 3, $\underline{p} < .05$). None of the differences between Raise and Lower trials proved reliable in the IF group. Thus, although differences between Raise and Lower trials were generated in systems other than the electrodermal, these differences were not larger in the group showing larger skin conductance changes.

Relationships between skin conductance and non-target behaviours were also investigated by means of product-moment correlations. These were computed between all six recorded variables on test trials on the last day of training, for each subject, and for Raise and Lower trials separately[7]. The means of these within-subject correlations on Raise trials are presented in Table 1 for groups I, IS, IF, and ISF. (The corresponding data for Lower trials were similar and are not presented). Examination of Table 1 indicates that the obtained correlations are generally low. In fact, only 10% of the correlations were statistically significant at the .05 level. There was little tendency for reliable correlations to involve particular response variables or training groups.

Table 1

Correlation matrix for subjects receiving skin conductance training (Raise trials)

I group

	SC	HR	GBM	RF	RA
HR	.05				
GBM	.05	.21			
RF	-.02	.21	-.02		
RA	.24	.48	.08	-.27	
EM	.07	.17	.16	.16	-.11

IS group

	SC	HR	GBM	RF	RA
HR	.47				
GBM	-.01	.14			
RF	-.01	.30	.00		
RA	.27	.28	.26	-.06	
EM	.18	.04	.27	-.18	.16

IF group

	SC	HR	GBM	RF	RA
HR	.21				
GBM	.16	.39			
RF	.08	.21	-.04		
RA	.22	.25	.19	.06	
EM	.00	-.10	-.02	-.16	.07

ISF group

	SC	HR	GBM	RF	RA
HR	.17				
GBM	-.02	.42			
RF	.00	-.17	.15		
RA	.15	.08	.39	.00	
EM	-.13	.21	.01	-.26	.05

Analysis of the questionnaire data revealed no reliable differences between any of the groups with respect to age, educational level, smoking habits, or meditational habits. The overall mean perceived success in controlling skin conductance ("sweating") was rated as 4.7 on a 7-point scale (1= not at all successful; 7= very successful), and group differences on this variable were not statistically significant. Subjects perceived Raise and Lower trials to be of comparable difficulty, both types of trials rating a mean of 4.9 on a 7-point scale of difficulty (1= impossible; 7= could control activity at will). Moreover, when asked to compare directly the two types of trials with respect to the difficulty in controlling the target behaviour, subjects found Raise and Lower trials to be equally difficult (mean rating = 4.025 on a 7-point scale of difficulty, with 1= Raise much more difficult than Lower, and 7= Lower much more difficult than Raise). In no case were reliable group differences observed with respect to the difficulty scales. Finally, most subjects experienced positive attitudes toward the experiment, as 31 of 32 indicated that they would be willing to participate in a similar experiment in the future.

Table 2 presents the probability levels at which each of the 11 affective scales differentiated between Raise and Lower trials for each group attempting to control skin conductance. These p values are based on Wilcoxon analyses of the difference in ratings for Raise and Lower trials, In general, it can be seen that Raise and Lower trials were

Table 2

p values for difference in ratings between Raise and Lower trials, for groups attempting to control skin conductance

Group	A	B	C	D	E	F	G	H	I	J	K
I	.01		.06	.025	.01			.025		.01	.01
IS	.005	.005	.005			.025				.01	.005
IF	.005	.005	.005	.01			.01			.025	.01
ISF	.025		.06	.025				.01		.025	.025

A - tense-relax

B - comfortable-uncomfortable

C - work-rest

D - mind-muscles

E - sexual-asexual

F - exciting-dull

G - pleasant-unpleasant

H - hot-cold

I - easy-difficult

J - flaccid-strained

K - anxious-calm

differentiated with respect to the affective dimension(s) measured by the scales, although it is clear that some scales were better discriminants than others. Particularly good discriminators were scales A (tense-relax), C (work-rest), J (strained-flaccid), and K (anxious-calm).

Inspection of Table 2 fails to reveal any systematic relationship between the affective variables and either the feedback or the strategies variable. This was confirmed by statistical tests. One-way analyses of variance were carried out with respect to each affective scale, with Groups as variates. These analyses were carried out on the ratings for Raise trials, on the ratings for Lower trials, and on the difference between Raise and Lower ratings on each scale. None of the comparisons proved reliable.

Control over heart rate

Figure 15 presents the mean heart rate for each of the four groups which received heart rate training, on test trials of the last day of training. Heart rate is plotted, as was skin conductance earlier, every five seconds during the 30-second periods preceding Raise and Lower trials (left-hand side of each panel) as well as during the trial periods (right-hand side of each panel).

As with skin conductance, preliminary analyses were carried out on baseline heart rate and on the response forms displayed during the trial periods. Analyses of variance using Types of Trial and Seconds as variates were applied to baseline heart rate in each group. In no case did either the Types of Trial main effect or the Types of Trial by Seconds interaction approach reliability. The difference in response forms displayed on Raise and Lower trials was evaluated by determining, for each subject, which of the six trial measurements represented the maximum change in heart rate in the targeted direction, and then comparing

Figure 15. Mean heart rate on test trials of the last day of training for subjects in the I, IS, IF, and ISF groups attempting to control heart rate. Heart rate was measured at the midpoint of consecutive five-second intervals, beginning 30 seconds before the start of the trial, and ending at the end of the trial.

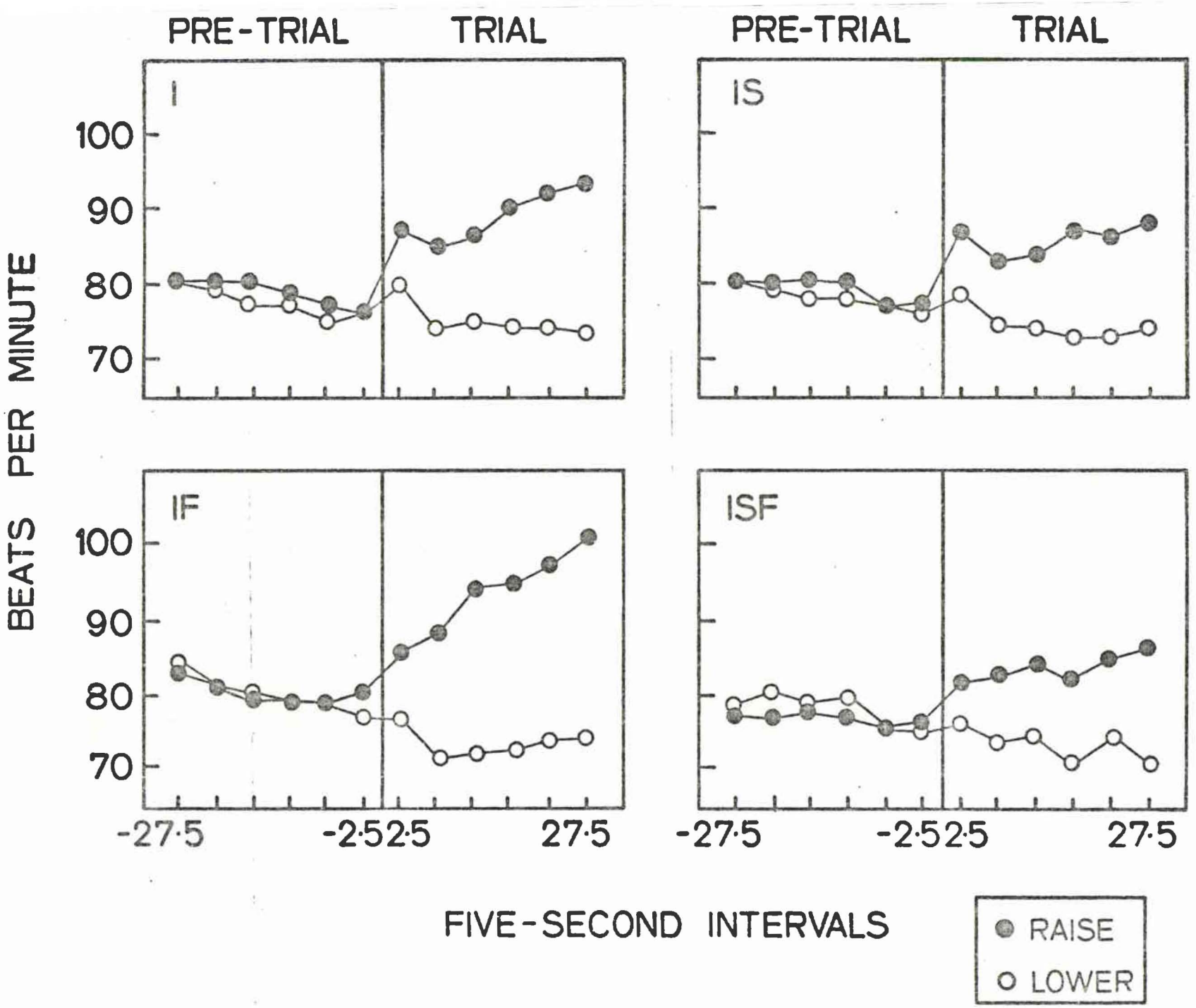
PRE-TRIAL
TRIAL
PRE-TRIAL
TRIAL
I
IS
IF
ISF
100
90
80
70
BEATS PER MINUTE
-27·5
-2·52·5
27·5
-27·5
-2·52·5
27·5
FIVE-SECOND INTERVALS
RAISE
LOWER

Figure 15

where this point occurred on Raise and Lower trials. Only in the IF group was there a tendency for the maximum change from baseline in the targeted direction to occur earlier on Lower trials than on Raise trials, but this effect only approached reliability (sign test, 7 of 8 subjects, $p < .10$). As with the skin conductance analyses, subsequent analyses focussed on the difference in performance between the last ten seconds of the trial period and the last ten seconds of the preceding baseline period.

Inspection of trial performance in Figure 15 suggests that control over heart rate was not achieved to the same extent in all groups. This was examined by means of an analysis of variance applied to the difference in heart rate between the last ten seconds of the trial and pretrial periods, in which Feedback (present/absent), Strategies (present/absent), and Types of Trial were employed as variates. This analysis yielded two significant F's, a main effect of Types of Trial ($F_{1,28} = 47.38$, $p < .001$), and a main effect of Strategies ($F_{1,28} = 5.45$, $p < .05$). These results indicate that voluntary control of heart rate was demonstrated, and that it was more substantial in the two groups (I and IF) that were not provided with strategy suggestions. Performance was examined further by analyses of the difference in heart rate between the last ten seconds of Raise and Lower trials in each of the four groups, and by comparing each group's performance with that of its appropriate control. Performance was reliably different on Raise and Lower trials in all four groups (I and ISF, $T(8) = 0$, $p < .005$; IS and IF, $T(8) = 1$, $p < .01$, all one-tail tests). Furthermore, the magnitude of the difference in heart rate which was obtained between Raise and Lower trials was

reliably larger in the I and IF groups taken together than in the IS and ISF groups (U (32) = 76, $p < .05$, two-tail test), although neither the difference between the performance of the I and IS groups nor that between the IF and ISF groups proved reliable.

These results, then, indicate that control over heart rate was achieved in all four groups, but that it was more substantial in the groups not provided with strategy suggestions. Other analyses examined whether the heart rate differences which were obtained between Raise and Lower trials reflected primarily a raising of heart rate on Raise trials, a lowering on Lower trials, or both. As in Experiment 1, control was more apparent on Raise trials, as the increases in heart rate observed on Raise trials proved reliable in all groups (I and ISF, T (8) = 0, $p < .005$; IS and IF, T (8) = 1, $p < .01$; all one-tail tests). The decreases in heart rate observed on Lower trials proved reliable only in the IS and IF groups (IS, T (8) = 5, $p < .05$; IF T (8) = 2, $p < .01$). However, these lowering effects are confounded by reliable trends in heart rate during the pre-trial periods, which were present in three of the groups (Seconds main effect, I, $F_{5,35} = 7.37$, $p < .001$; IS, $F_{5,35} = 4.79$, $p < .005$; ISF, $F_{5,35} = 4.36$, $p < .005$). Nevertheless, several considerations suggest that the lowering effects may have been genuine. First, the decreases in heart rate were no greater on Lower trials which followed short inter-trial intervals, where recovery functions might have been more evident, than on trials which followed longer inter-trial intervals (Inter-trial interval main effects, Fs < 1 for both the IF and ISF groups). Second, decreases in heart rate which were produced in the IF group trained to control heart rate were reliably larger than those produced in the IF group trained to control skin conductance (U

(16) = 8.5, $p < .01$, one-tail test), although the pre-trial trends were identical in the 2 conditions. However, such reliable differences were not obtained between any of the other homologous groups. Finally, an examination of the recovery functions following trial offset also suggests that the lowering effect was genuine. These are examined in Figure 16. There was a tendency for heart rate to increase following Lower trials, and to decrease following Raise trials, in all groups. These tendencies were confirmed statistically through an analysis of variance utilizing Groups, Types of Trial, and Time (last trial point versus third post-trial point) as variates, which yielded a reliable interaction between Types of Trial and Time ($F_{1,28} = 51.28$, $p < .001$). Thus, there was clear evidence of heart rate control on Raise trials in all groups, and there was also evidence of heart rate control on Lower trials, which was clearest in the IF group.

Figure 17 examines acquisition effects. The figure depicts the mean change in heart rate on Raise and Lower trials over the three days of training, for each of the four groups attempting to control heart rate. Only in the I and IF groups was there any indication of acquisition. Acquisition effects were examined through analyses of variance, employing Days and Types of Trial as variates, which were applied to each group separately. Only in the I group did the Days-by-Types of Trial interaction reach statistical significance ($F_{2,14} = 5.05$, $p < .05$). Post hoc Newman Keuls analyses indicated a reliable acquisition from day 1 to days 2 and 3 on Raise trials, but yielded no evidence of acquisition on Lower trials. Thus, while Figure 17 suggests that

Figure 16. Heart rate before and after trial offset on the last day of training to control cardiac responding. Heart rate is plotted as a change from the pre-trial baseline. Data are from test trials.

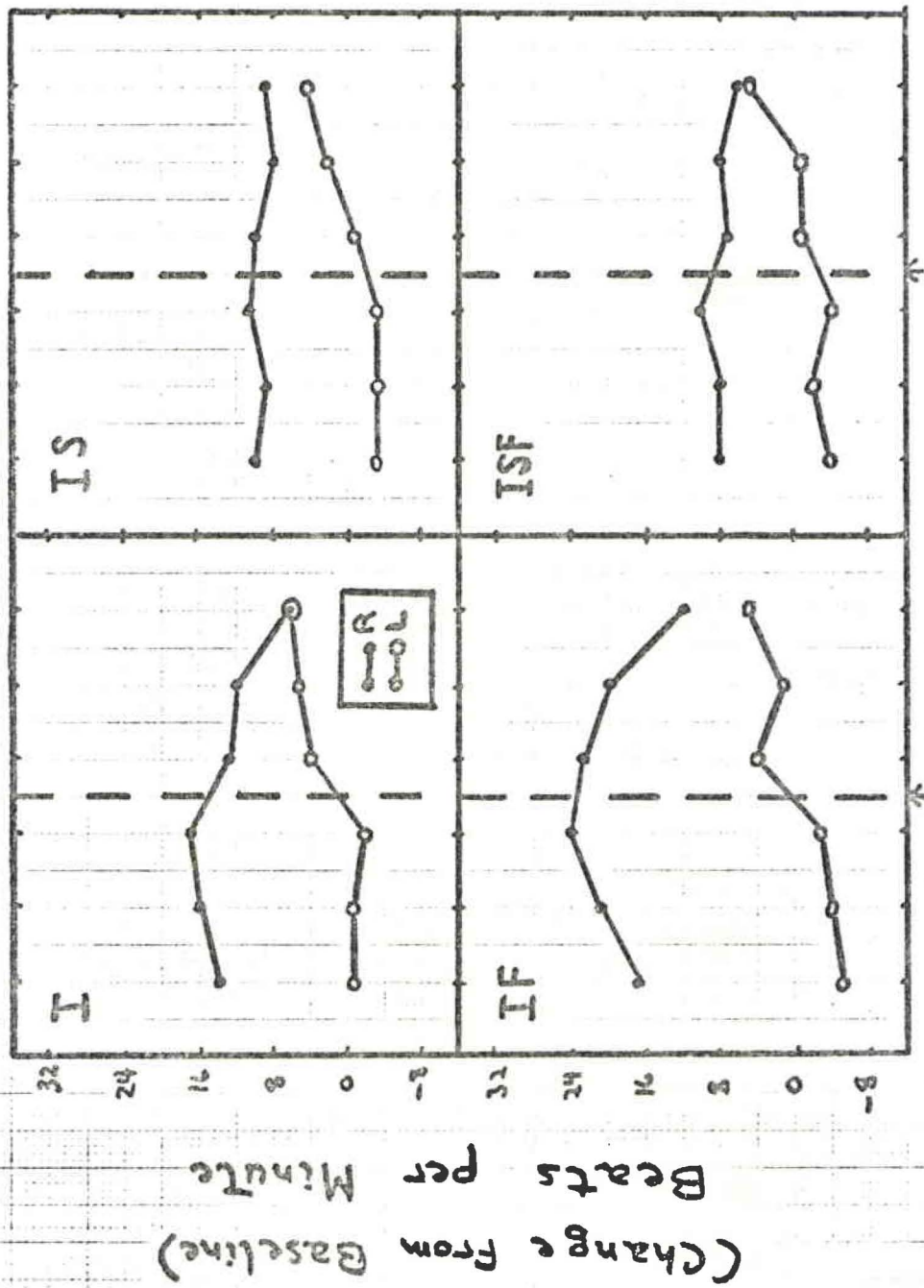
PRE-TRIAL
TRIAL
POST-TRIAL
IS
ISF
I
IF
R
L
32
24
16
8
0
-8
trial off
trial off
Five-second intervals
(Change From Baseline)
Beats per Minute

Figure 16

Figure 17. Acquisition of heart rate control. The difference between heart rate in the last 10 seconds of the trial and pre-trial periods is plotted for test trials on the 3 training days.

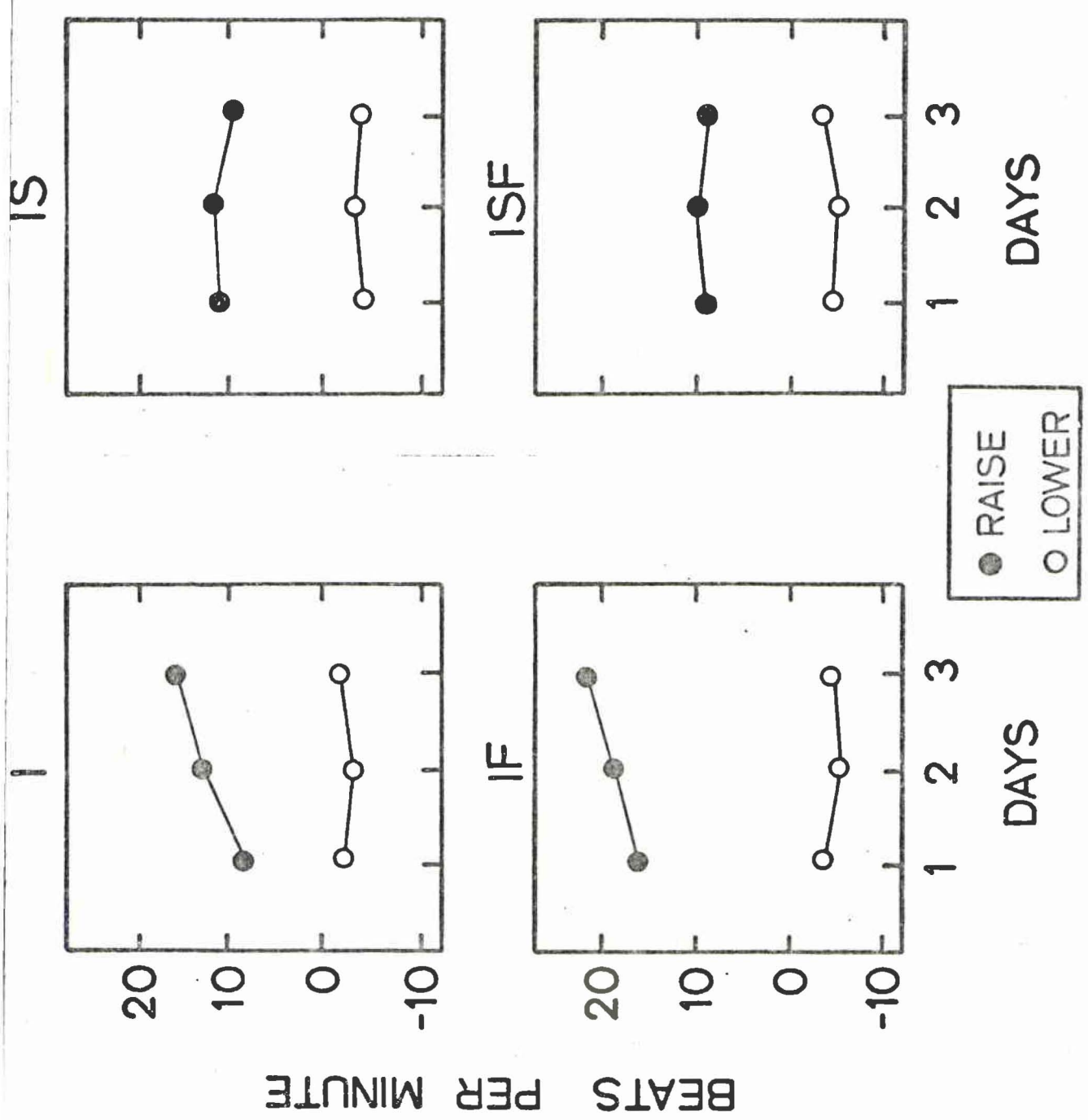

Figure 17

heart rate control improved over days in both the I and IF groups, only in the former and only on Raise trials was this trend reliable.

Since substantial control over heart rate was apparent on day 1 in all groups, it is reasonable to ask whether any acquisition might have taken place within the session on this day. Figure 18 presents the heart rate performance of the four groups receiving heart rate training on successive Raise test trials on day 1. It is clear that substantial increases in heart rate were produced in all groups on the very first trial, and that these were of similar magnitude in all groups. To examine whether group differences subsequently developed over the course of day 1 (as the figure suggests), an analysis of variance using Groups and Trials as variates was employed. This analysis yielded neither a significant Groups main effect nor a significant Groups-by-Trials interaction (both $\underline{F}s < 1$).

The data presented so far were all gathered on test trials. The next analysis examined the extent to which transfer took place from training to test trials. The pertinent data are presented in Figure 19, which depicts the mean heart rate changes observed in the IF and ISF groups over the three days of training, on test trials and on training trials. Performance on test and training trials was compared by analyses of variance which employed Days and Feedback (present/absent) as variates, and which were applied separately to Raise and Lower trials and to the IF and ISF groups. Performance on Lower trials was reliably better on training trials than on test trials in both groups (IF, $\underline{F}_{1,7} = 5.95$, $\underline{p} < .05$; ISF, $\underline{F}_{1,7} = 7.25$, $\underline{p} < .05$). Moreover, performance on Raise trials was better on training trials than on test trials at a level ap-

Figure 18. Acquisition functions in heart rate on Raise trials on the first day of training. Responding is plotted as the difference in heart rate between the last 10 seconds of the trial and pre-trial periods, on successive test trials. Only Raise trials are presented.

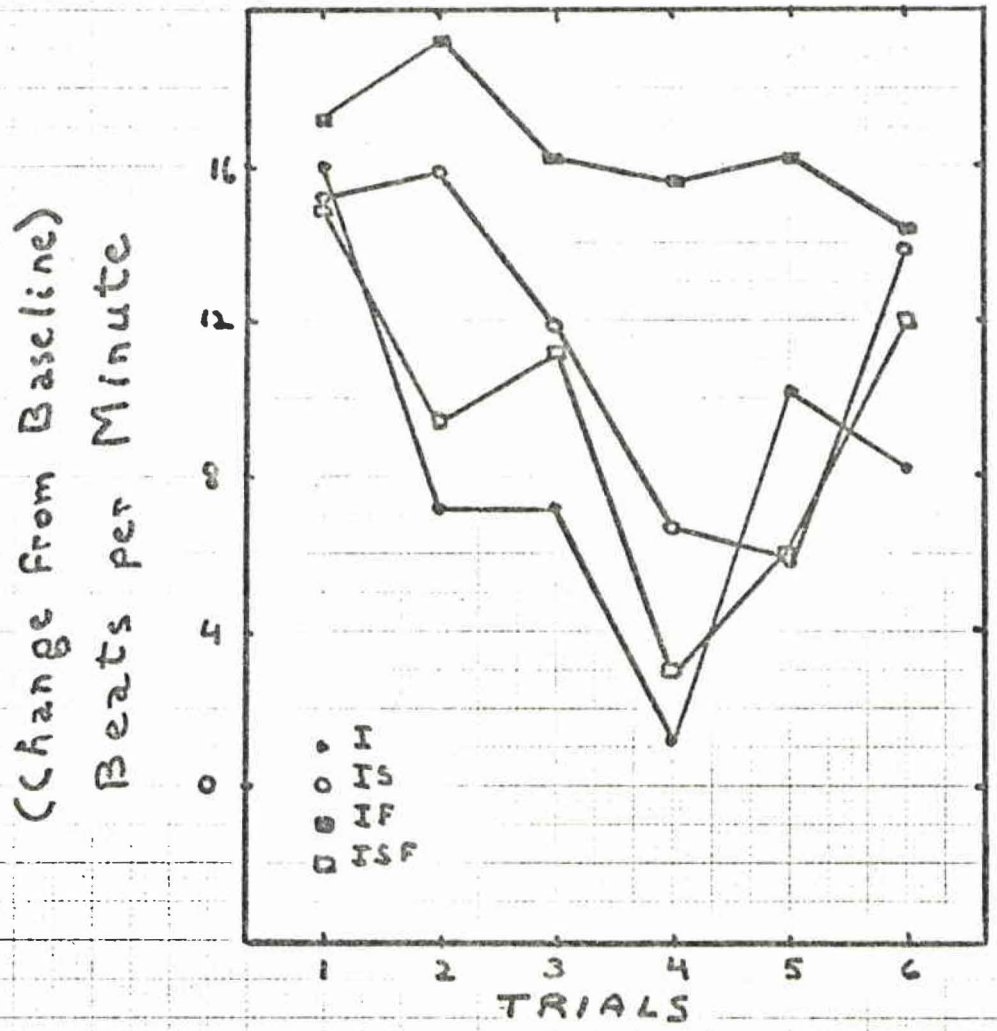
(Change from Baseline)
Beats per Minute
12
8
4
0
I
Is
IF
ISF
1
2
3
4
5
6
TRIALS

Figure 18

Figure 19. A comparison of heart rate control on test trials (when the feedback stimulus was never presented) and on training trials (when the feedback stimulus was always presented). The unit employed is the difference between heart rate in the last 10 seconds of the trial and pre-trial periods.

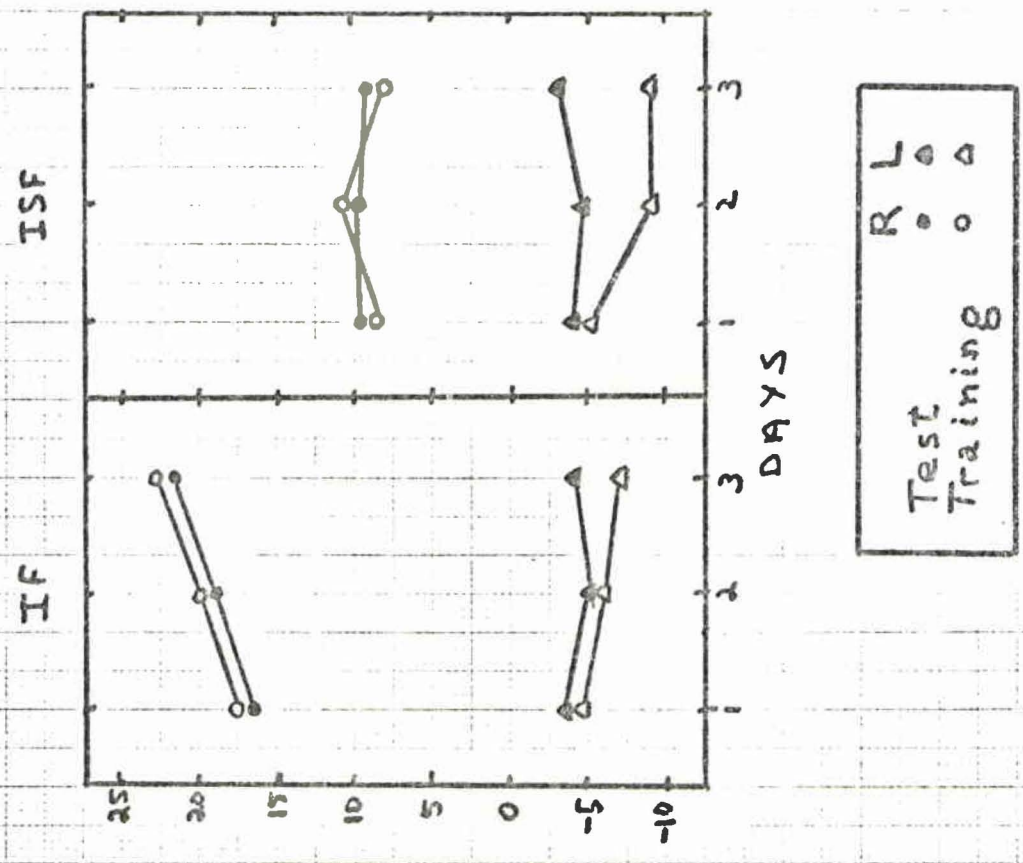
ISF
IF
Beats per Minute
25
20
15
10
5
0
-5
-10
1
2
3
DAYS
R L
Test
Training

Figure 19

proaching statistical significance in the IF group ($F_{1,7} = 5.27$, $p < .06$), but not in the ISF group. In no case did a Days main effect or a Days by Feedback interaction approach reliability, indicating that the generally superior performance on training trials was maintained throughout training.

In view of the fact that performance appeared somewhat better on training trials than on test trials, comparisons between all four groups receiving heart rate training, which were reported earlier based on performance on tests trials only, were recomputed using the training trials performance of those subjects provided with feedback, i.e. those in the IF and ISF groups, for the third day of training. However, as was the case when performance on test trials was examined, the Feedback main effect and Feedback-by-Types of Trial interaction failed to reach significance.

Improved performance on training trials indicates that provision of feedback contributed to voluntary control, even though between-groups comparisons failed to provide reliable evidence for feedback effects. It is possible that between-groups Feedback effects might have been obtained had voluntary control in the absence of feedback not been so substantial. This possibility was examined by determining whether subjects who profited most from provision of feedback were those who evidenced the least control of heart rate on the first test trial. For this purpose a median split was applied to the IF and ISF groups on the basis of the magnitude of the bidirectional heart rate difference manifested on the first Raise and Lower test trials. Performance on the last pair of Raise and Lower test trials was then examined. The

data are presented in Table 3. It is apparent that subjects least able to control heart rate prior to feedback training did not profit more from such training, than subjects who manifested substantial control of heart rate on the first test trial pair.

Both the number of presentations of the feedback stimulus on each day, and the total duration of feedback presentations per day, were recorded for those subjects receiving feedback, i.e. those in the IF and ISF groups. Analyses of variance employing Groups, Types of Trial, and Days as variates were applied to both the duration and the frequency data. These analyses failed to reveal either reliable Groups or Types of Trial main effects or interactions, with respect to either frequency or total duration of feedback presentations. However, a statistically reliable main effect of Days was obtained with respect to total duration of feedback presentations, in the direction of longer feedback presentations as a function of days ($F_{2,28} = 3.96$, $p < .05$).

Figure 20 examines response profiles. The upper half of the figure presents the median changes observed on Raise trials in all of the variables recorded in this experiment, on test trials of the last day of training, in each of the four groups attempting to control heart rate. The lower half of Figure 20 presents the corresponding changes on Lower trials. Overall statistical comparisons between the response profiles generated on Raise trials and those generated on Lower trials in the different groups were accomplished by means of analyses of variance employing Feedback, Strategies, and Types of Trial as variates, as was done for heart rate. These indicated that reliable differences between Raise and Lower trials were present with respect to all of the nontarget

Table 3

	First trial pair / Last trial pair	
Good performers	27.4	27.1
Poor performers	6.0	6.4

Initial and terminal performance for subjects in the IF and ISF groups showing substantial heart rate control (good performers) and minimal heart rate control (poor performers) on the first Raise-Lower test trial pair. The measure reported is the bi-directional heart rate difference in beats per minute.

Figure 20. Changes in skin conductance (SC), heart rate (HR), gross body movement (GBM), respiration frequency (RF), respiration amplitude (RA), and eye movement (EM), on Raise (upper half) and Lower (lower half) test trials, on the last day of training for the subjects in each of the groups attempting to control heart rate. All measures are based on the difference between performance during the last 10 seconds of the trial and pre-trial periods, for the subjects who yielded the median difference in heart rate between Raise and Lower trials. Skin conductance is plotted in micromhos. Heart rate is in beats per minute. Respiration frequency is plotted as respiration period in milliseconds (negative up). Body movement, respiration amplitude, and eye movement are in arbitrary units.

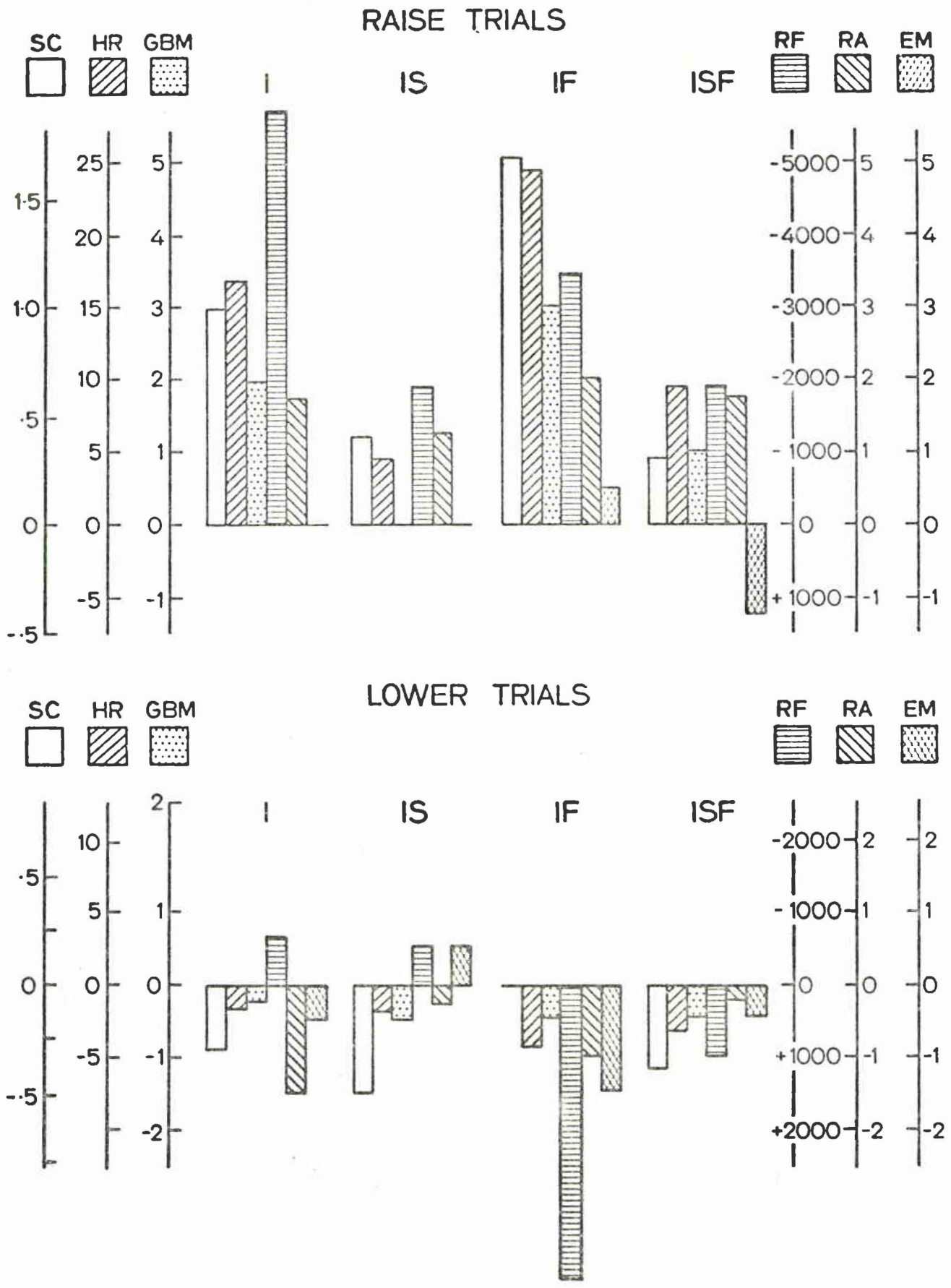
RAISE TRIALS
SC
HR
GBM
I
IS
IF
ISF
RF
RA
EM
1·5
1·0
·5
0
-·5
25
20
15
10
5
0
-5
5
4
3
2
1
0
-1
-5000
-4000
-3000
-2000
- 1000
0
+ 1000
LOWER TRIALS
SC
HR
GBM
I
IS
IF
ISF
RF
RA
EM
·5
0
-·5
10
5
0
-5
2
1
0
-1
-2
-2000
- 1000
0
+1000
+2000

Figure 20

variables except eye movement (Types of Trial main effects: skin conductance, $F_{1,28} = 21.23$, $p < .001$; body movement, $F_{1,28} = 46.70$, $p < .001$; respiration frequency, $F_{1,28} = 7.85$, $p < .05$; respiration amplitude, $F_{1,28} = 42.16$, $p < .001$). Moreover, with respect to both skin conductance and body movement, a reliable main effect of Strategies was also obtained, which parallelled that obtained with respect to heart rate (for skin conductance, $F_{1,28} = 7.33$, $p < .05$; for body movement, $F_{1,28} = 6.71$, $p < .05$). Thus, response correlates were more evident under conditions (I and IF) that produced the largest heart rate changes.

Further analyses examined the differences between the two types of trial in each group individually. Raise and Lower trials could be differentiated reliably with respect to all variables in both the I and the IF groups (for I, minimum T (8) = 3.5, $p < .05$, with respect to skin conductance; for IF, minimum T (8) = 2.5, $p < .05$, with respect to eye movement). In the IS group, Raise and Lower trials could be differentiated reliably with respect to heart rate (T (8) = 2, $p < .01$, one-tail test), respiration frequency (T (8) = 2, $p < .02$), and respiration amplitude (T (7) = 2, $p < .05$), while in the ISF group, only heart rate differentiated reliably between Raise and Lower trials (T (8) = 1.5, $p < .01$, one-tail test). Perusal of Figure 20 suggests that most of these differences between Raise and Lower trials resulted primarily from increases on Raise trials rather than decreases on Lower trials.

Relationships between heart rate and non-target behaviours were also investigated by means of product-moment correlations. These were computed between all six recorded variables, on test trials of the last day of training, and for Raise and Lower trials separately. The means

of these within-subject correlations which were computed on Raise trials are presented in Table 4 for the four groups attempting to gain control over heart rate[7]. (The corresponding data for Lower trials were similar and are not presented). The correlations were generally low; only 7% of them were statistically significant at the .05 level. There did not appear to be any systematic differences between groups or response systems with respect to the magnitude of the correlations obtained.

Inspection of the results of the questionnaire data revealed that no reliable differences were obtained between any of the groups with respect to age, educational level, smoking habits, or meditational habits. The overall mean perceived success in controlling heart rate was rated as 4.75 on a 7-point scale (1 = not at all successful; 7 = very successful), and group differences with respect to this variable were not statistically significant. Subjects perceived Raise and Lower trials to be of comparable difficulty, Raise trials rating a mean of 4.725, and Lower trials a mean of 5.0 on a 7-point scale of difficulty (1 = impossible; 7 = could control activity at will). Moreover, when asked to compare directly the two types of trial with respect to the difficulty in controlling the target behaviour, subjects found Raise and Lower trials to be about equally difficult (mean rating of 3.85 on a 7-point scale, on which 1 = Raise much more difficult than Lower; 7 = Lower much more difficult than Raise). In no case were

Table 4

Correlation matrix for subjects receiving heart rate training
(Raise trials)

I group

	SC	HR	GBM	RF	RA
HR	.05				
GBM	-.07	.26			
RF	.00	.28	.11		
RA	-.09	.10	.07	.10	
EM	.04	.18	.12	.01	.02

IS group

	SC	HR	GBM	RF	RA
HR	.13				
GBM	.03	-.02			
RF	.26	.18	.08		
RA	-.04	.00	.21	-.02	
EM	.04	-.05	.00	-.05	.18

IF group

	SC	HR	GBM	RF	RA
HR	.34				
GBM	.16	.11			
RF	-.12	-.16	-.07		
RA	-.03	.03	.10	-.17	
EM	-.15	.04	.06	-.01	-.03

ISF group

	SC	HR	GBM	RF	RA
HR	.61				
GBM	.22	.34			
RF	-.28	-.02	.06		
RA	.22	.16	.33	.06	
EM	-.07	.05	.07	.11	.16

reliable group differences obtained with respect to the difficulty scales. Finally, most subjects experienced positive attitudes toward the experiment, as 30 out of the 32 indicated that they would be willing to participate in a similar experiment in the future.

Table 5 presents the probability levels at which each of the 11 affective scales differentiated between Raise and Lower trials for each group attempting to control heart rate. These p values are based on Wilcoxon analyses on the difference in ratings for Raise and Lower trials. In general, it is clear that Raise and Lower trials were differentiated with respect to the affective dimension(s) measured by the scales, although some scales were clearly better discriminators than others. Particularly good discriminators were scales A (tense-relax), C (work-rest), G (unpleasant-pleasant), I (difficult-easy), J (strained-flaccid), and K (calm-anxious).

Inspection of Table 5 fails to reveal any systematic relationship between the affective variable and either the Feedback or the Strategies variable. This was verified by statistical tests. One-way analyses of variance were carried out with respect to each affective scale, with Groups as the treatment variable. These analyses were carried out on the ratings for Raise trials, on the ratings for Lower trials, and on the difference between ratings for Raise and Lower trials, on each scale. None of the comparisons proved statistically reliable.

Comparison of skin conductance and heart rate control

Previous sections have focussed on electrodermal and heart rate control separately. This section compares voluntary control over the two systems, primarily with respect to the effects of the training variables, and with respect to the response profiles which were produced.

Table 5

p values for difference in ratings between Raise and Lower trials, for groups attempting to control heart rate

Group	A	B	C	D	E	F	G	H	I	J	K
I	.005		.01			.01	.01		.025	.01	.06
IS	.005	.06	.005			.01	.01	.025	.005	.005	.005
IF	.005	.005	.005	.025	.06		.005		.005	.01	.005
ISF	.01	.025	.01			.025	.01	.025	.01	.01	.01

A - tense-relax

B - comfortable-uncomfortable

C - work-rest

D - mind-muscles

E - sexual-asexual

F - exciting-dull

G - pleasant-unpleasant

H - hot-cold

I - easy-difficult

J - flaccid-strained

K - anxious-calm

Figure 21 displays the response profiles produced in all eight groups in this experiment. Each bar represents the median difference generated between Raise and Lower test trials, on day 3. The response profiles generated in both the I and IF groups which did not receive strategy suggestions are presented in the upper half of the figure. The response profiles generated in the four homologous groups provided with strategies are displayed in the lower half of the figure.

The major findings of interest are found in the upper half of Figure 21, which portrays the performance of groups that were not provided with explicit strategy suggestions. Inspection of these data shows that skin conductance and heart rate were affected differently by instructions and feedback under this condition. Instructing subjects to control sweating (Group I/SC) did not produce reliable differences in skin conductance between Raise and Lower trials (see previous analyses, p.109). On the other hand, instructing them to control heart rate (Group I/HR) resulted in a substantial difference in heart rate between Raise and Lower trials ($p < .005$, see previous analyses, p.129). The difference in voluntary control which is apparent when these two groups are compared is an effect of referencing a particular response system, since the instructions given in the two target conditions were identical except for the response the subjects were asked to control (Appendix A). Further inspection of the upper half of Figure 21 shows that provision of feedback was necessary for the development of skin conductance control (Group IF/SC versus Group I/SC, $p < .05$, analyses previously reported, p.109). On the other hand, provision of feedback did not facilitate heart rate control, owing to

Figure 21. Bi-directional differences in skin conductance (SC), heart rate (HR), gross body movement (GBM), respiration frequency (RF), respiration amplitude (RA), and eye movement (EM) on the last day of training for the subjects provided with strategy suggestions (lower half) and for those not so provided (upper half). Groups I/SC, IS/SC, I/HR, and IS/HR were given instructions to control skin conductance and heart rate, respectively. Groups IF/SC, ISF/SC, IF/HR, and ISF/HR were also given feedback. All measures are based on the difference between the last 10 seconds of the trial and pre-trial periods for the subjects who yielded the median difference in the target response between Raise and Lower trials. Skin conductance is plotted in micromhos. Heart rate is in beats per minute. Respiration frequency is plotted as respiration period in milliseconds (negative up). Body movement, respiration amplitude, and eye movement are in arbitrary units.

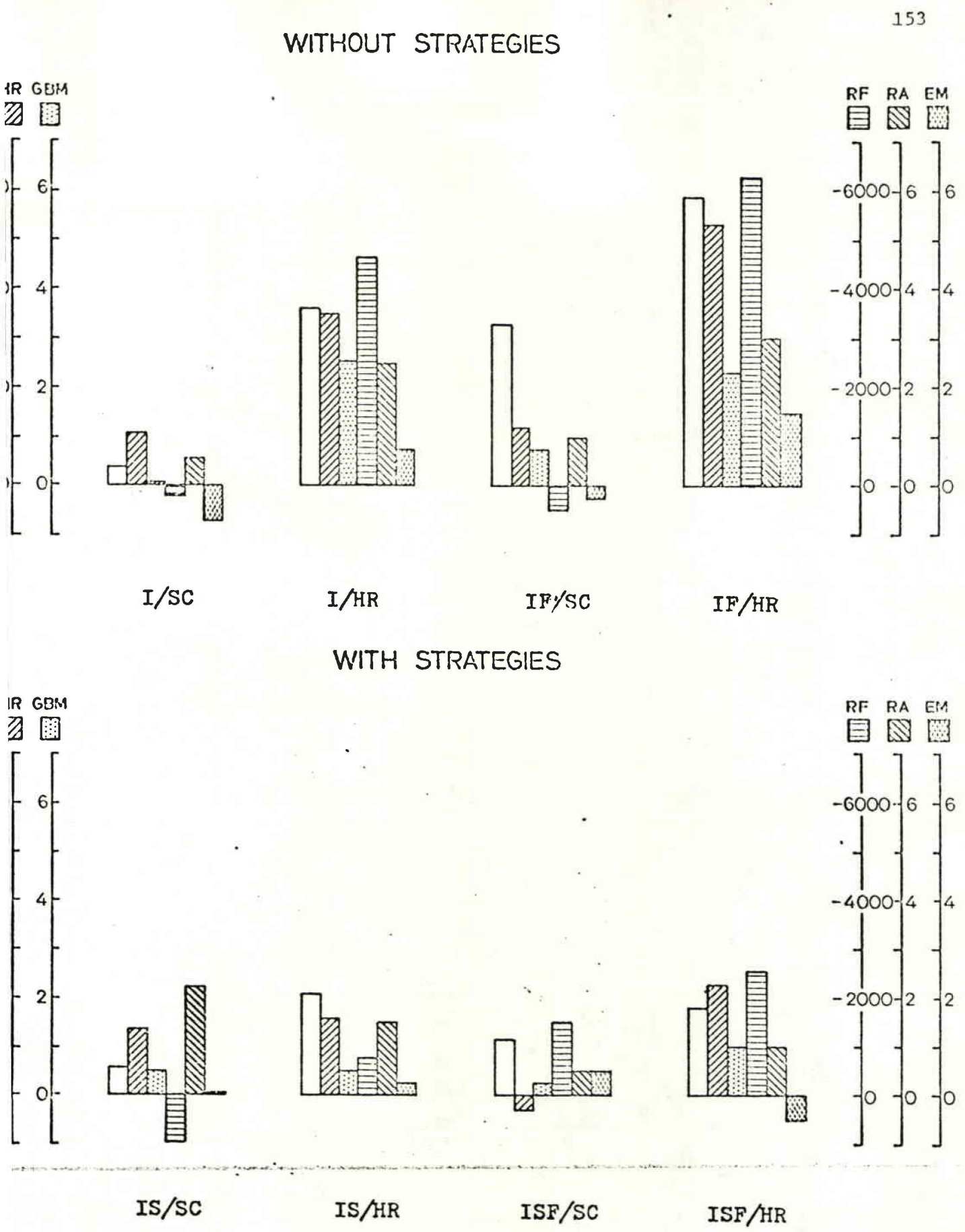
WITHOUT STRATEGIES
HR GBM
RF RA EM
-6000
-4000
-2000
0
6
4
2
0
I/SC
I/HR
IF/SC
IF/HR
WITH STRATEGIES
IR GBM
RF RA EM
-6000
-4000
-2000
0
6
4
2
0
IS/SC
IS/HR
ISF/SC
ISF/HR
GROUP

Figure 21

substantial control evidenced by subjects given instructions alone (Group HRI/FB versus Group HRI, analyses previously reported, p.129).

Consideration of the same comparisons in the lower half of Figure 21 shows that these effects were generally less evident when the groups were provided with strategy suggestions. Provision of strategies tended to have a deleterious effect on control of both response systems (previous analyses, pp.118 and 129).

Comparison of the response profiles in the upper half of Figure 21, where voluntary control was most evident, shows that control of skin conductance and heart rate was associated with different response profiles. The performance of voluntary heart rate changes was clearly associated with autonomic, somatomotor, and respiratory correlates. Comparison of the four heart rate conditions in Figure 21 shows that these correlates were more apparent in the groups (I/HR and IF/HR) that showed the largest heart rate changes (see previous analyses, p.146). On the other hand, voluntary changes in electrodermal responding appeared to be more specific. The correlates shown in the upper half of Figure 21 when feedback was given for electrodermal responding (Group IF/SC) were unreliable and similar to those seen in subjects given skin conductance instructions alone (Group I/SC) or provided with strategy suggestions (Groups IS/SC and ISF/SC, lower half of Figure 21). Amplification of electrodermal control by feedback was not associated with amplification in the magnitude of changes observed in other response systems (see previous analyses, p.122).

Direct comparisons of the response profiles generated in the two target conditions confirmed the view that correlates were generally

more evident when heart rate was the target response. Comparison of response patterns for the two groups that received instructions alone (I/SC versus I/HR, upper half of Figure 21) revealed reliably larger changes under conditions of heart rate training in skin conductance (U (16) = 9, $p < .01$), heart rate (U (16) = 9, p .01), gross body movement (U (16) = 6, $p < .005$), and respiration amplitude (U (16) = 9.5, $p < .01$). The same comparison for the two groups that received both instructions and feedback (IF/SC versus IF/HR, upper half of Figure 1) revealed larger changes under conditions of heart rate training with respect to heart rate (U (16) = 12, $p < .02$), gross body movement (U (16) = 13.5, $p < .05$), respiration amplitude (U (16) = 9, $p < .01$, and eye movements (U (16) = 10.5, $p < .02$). Thus, autonomic, somatomotor, and respiratory correlates were more prominent when heart rate was the target response.

A comparison of Tables 2 and 5 permits an evaluation of the differences in the questionnaire responses of subjects attempting to control skin conductance and heart rate. As was noted earlier, in neither the skin conductance nor the heart rate target condition did group differences materialize with respect to any of the affective scales. Comparing Tables 2 and 5 suggests that Raise and Lower trials could be differentiated on the basis of at least two more scales in the heart rate target condition than in the skin conductance target condition (scales G and I). The reliability of this observation, and the possibility of other affective differences between the two conditions, were examined through t-tests on the difference in ratings between Raise and Lower trials. Only with respect to scale I could the two target

conditions be differentiated reliably ($t_{62} = 7.08$, $p < .001$). There was no reliable difference between the two target conditions with respect to any of the scales measuring success at controlling the target response.

The skin conductance and heart rate target conditions were also compared with respect to the frequency and duration of feedback presentations. Analyses of variance employing Target Condition, Types of Trial, and Days as variates were applied separately to the frequency and duration data. For purposes of these analyses the IF and ISF groups in each condition were combined. The main effect of Target Condition proved reliable with respect to both frequency and duration (for frequency, $F_{1,30} = 48.47$, $p < .001$; for duration, $F_{1,30} = 5.45$, $p < .05$). However, these effects reflected trends in opposite directions, for while the feedback stimulus was presented longer in the skin conductance target condition, it was presented more often in the heart rate target condition. The Days effect proved reliable with respect to both frequency ($F_{2,60} = 3.01$, $p < .05$) and duration ($F_{2,60} = 7.82$, $p < .01$), in the former case reflecting reliable differences between days 1 and 2 and between days 1 and 3 ($p < .01$ in both cases, Newman Keuls), and in the latter case, between days 1 and 3 ($p < .01$). Thus, feedback was presented both more frequently and for longer periods of time as a function of days, in both target conditions.

Discussion

The goals of Experiment 2 were to (i) examine the role of instructions, strategy suggestions, and feedback in electrodermal and cardiac control, (ii) examine the response patterns which occur when voluntary

control is established over these two response systems, and (iii) compare voluntary control of electrodermal responding and heart rate. This section summarizes the main findings and considers similarities between these and the results of the previous experiment. Implications of the results for the question of how voluntary control is established over electrodermal and heart rate responding are discussed in the next chapter.

The main results with respect to skin conductance were as follows. Subjects were unable to comply with instructions to increase or decrease palmar sweating. Adding the suggestion that increases and decreases in sweating are usually associated with "getting tense and excited" on increase trials, and with "relaxing" on decrease trials, did not improve performance measurably in this study. On the contrary, if anything, strategy suggestions tended to diminish voluntary control when feedback was provided. Addition of exteroceptive feedback established voluntary control of skin conductance on both test and training trials by the third day of training. Control was evident on both increase and decrease trials and was most prominent when strategy suggestions were not provided. Voluntary control was associated with small but reliable bi-directional differences in heart rate, gross body movement and respiration amplitude, and with bi-directional differences on several of the affective scales. These correlates were equally evident in all groups but were not augmented by feedback training, as was voluntary control of skin conductance.

The results for heart rate were different. Substantial control of heart rate was evidenced by subjects who received instructions alone. Moreover, the magnitude of control increased reliably and substantially

over days in this condition, reaching 18 beats per minute by the third day of training. Addition of feedback improved control slightly, but insignificantly, when groups with and without feedback were compared. However, subjects appear to have utilized exteroceptive feedback to guide their performance, since control was significantly, although only slightly, better on training trials where feedback was present, than on test trials where feedback was removed. Addition of strategy suggestions did not improve, but rather diminished, voluntary control of heart rate. Bi-directional heart rate changes were associated with reliable bi-directional differences in skin conductance, gross body movement, respiration amplitude, and respiration frequency. These correlates were substantially larger than those seen when skin conductance was the target response, and two of the variables (skin conductance and gross body movement) were reliably larger under the experimental conditions (no strategy suggestions) that produced the largest heart rate changes. Voluntary control of heart rate was also associated with differences in a variety of affective scales, but unlike autonomic and somatomotor correlates, these were not enhanced by the experimental variables that enhanced the magnitude of voluntary heart rate change.

There are some similarities between the results of Experiments 1 and 2 worth noting. The ISF groups in the two studies were near replications of one another. In both experiments, little control of skin conductance was evident on early test trials, although voluntary control of heart rate on these trials was substantial. Voluntary control was subsequently demonstrated over both response systems, and transfer from training to test trials was nearly complete. Another result common to both studies was that voluntary heart rate changes were more

closely associated with somatomotor correlates than were voluntary electrodermal changes. On the other hand, there were some discrepancies between the two studies. Comparison of median performance (cf Figures 5 and 21) indicates that subjects in the skin conductance conditions performed similarly in the two experiments, while subjects in the previous study appeared better at controlling heart rate. Another difference was that feedback facilitated control of heart rate on Lower trials in the ISF group of Experiment 2, but not in Experiment 1, when training and test trials were compared. The reason for these discrepancies is unclear.

Chapter 5: General Discussion

This thesis was concerned with the general question of how voluntary control is established over skin conductance and heart rate. Throughout the thesis, voluntary control has been understood in terms of Brener's operational definition of a voluntary response as one that can be systematically influenced by instructions (Brener, 1974a). It was suggested in the introductory chapter that the problem of how voluntary autonomic control is established encompasses three questions. First, what are the experimental determinants of control? Second, what other behaviours are affected when voluntary control is exerted over skin conductance or heart rate? And third, how do subjects learn to comply with instructions to alter skin conductance or heart rate? The relevance of the thesis findings to each of these questions is discussed in this chapter.

Determinants of Control

It was suggested in Chapter 1 that a voluntary control procedure can be viewed as consisting of the following elements. First, subjects are instructed to produce changes in an autonomic response. Typically these instructions include both a directional requirement and a reference to a particular response system (for example, Brener, 1974a; Lang and Twentyman, 1974). Second, some investigators (for example, Klinge, 1972) have provided their subjects with explicit response strategies designed to facilitate compliance with the instructions. Third, subjects are provided with exteroceptive feedback contingent upon successful performance. The role of each of these variables in voluntary electrodermal and cardiac control is discussed in this section, in the light of the

results of Experiment 2 in which the contribution of each variable to control of skin conductance and heart rate was assessed. Since the role of these variables was found to depend upon which response system was considered, the two systems are discussed separately.

Skin Conductance

The present Experiment 2 appears to have been the first to examine the effects of instructions to control palmar sweating. Subjects provided with only instructions to increase and decrease palmar sweating did not exhibit reliable differences in autonomic, somatomotor, or respiratory response variables between Raise and Lower trials. The simple command to increase and decrease palmar sweating does not appear to produce substantial changes in electrodermal function.

A second variable which was examined in Experiment 2 was the provision of strategy suggestions. As were the subjects provided with instructions alone, those provided with instructions and strategies were told to try to increase and decrease palmar sweating. However, these latter subjects were also told that increases in sweating were usually associated with being tense and excited, with moving or thinking about moving, and with paying attention to internal events, whereas decreases in sweating were associated with relaxation, with immobility, and with paying attention to the external environment. Combining these strategy suggestions with simple instructions to control sweating did not result in any greater control of skin conductance than was observed when instructions alone were provided, under either feedback condition. Failure of the strategy suggestions to facilitate control does not appear to have been due to a failure to attempt to comply with the suggestions, or to the possibility that the

strategies which were suggested were irrelevant to electrodermal performance. Rather, it appears that subjects attempted to comply with the strategy suggestions, but that this attempt interfered with electrodermal control. This is suggested by a comparison of the IF and ISF groups that received skin conductance training, where it was found that subjects given feedback and strategy suggestions tended to perform more poorly at controlling skin conductance ($p = .06$, p.118) than did subjects given feedback alone. The same strategy suggestions produced a reliable decrement in control irrespective of feedback condition when heart rate was the target response ($p < .05$, p. 129).

Although the strategy suggestions employed in Experiment 2 did not facilitate electrodermal control in either feedback condition, there is evidence which suggests that other strategy instructions might have had this effect. In two experiments, Klinge (1972) examined electrodermal control in subjects told to "think emotional thoughts" on Raise trials, and to "relax" on Lower trials. She observed that subjects produced reliably more electrodermal phasic responses on Raise trials than on Lower trials in each study. Klinge's strategy suggestions referred only to thinking and relaxing, whereas those given the subjects in the IS group of the present study where control was not evidenced made reference also to manipulations of body movement and to paying attention to internal versus external events. It is possible that these additions may have led the subjects to focus on response strategies that were less appropriate for the production of electrodermal responses.

The present results are congruent with those of Klinge (1972) with respect to the contribution of feedback to control of electrodermal res-

ponding. In two different experiments, Klinge observed that provision of feedback for electrodermal changes enhanced electrodermal control. Subjects provided with feedback for electrodermal changes produced reliably greater bi-directional differences in electrodermal response frequency (mean: 3.0 responses/minute) than did subjects who were told simply to "think" and "relax" on Raise and Lower trials (mean: 1.0 responses/minute). In the present Experiment 2, reliable bi-directional differences in skin conductance were observed only in the two groups that underwent feedback training (IF and ISF). The combined performance of these two groups differed reliably from the combined performance of the two groups (I and IS) that did not undergo feedback training ($p < .05$, p.109). Failure of the latter two groups to evidence substantial control indicates that experience with an explicit feedback contingency was necessary for the establishment of learned electrodermal changes under the conditions of the present study.

Heart Rate

In addition to evaluating the contributions of instructions, strategy suggestions, and feedback to skin conductance control, the thesis examined the effects of these experimental variables on heart rate control. Subjects provided only with instructions to produce increases and decreases in heart rate exhibited substantial, reliable, heart rate control. On the third day of training, these subjects generated mean bi-directional heart rate differences of almost 20 beats per minute. Furthermore, the effects of instructions to increase and decrease heart rate were not confined to heart rate. Subjects in the I/HR group also produced large, reliable, bi-directional differences in all other responses which

were recorded (skin conductance, body movement, respiration frequency, respiration amplitude, eye movements) and in several of the affective scales.

These findings both replicate and extend earlier observations by other investigators. That subjects can control heart rate when receiving only instructions to do so is in keeping with a number of previous reports (Bergman and Johnson, 1971; Blanchard and Young, 1972; Brener, 1974a; Brener et al., 1969; Levenson, 1974; Ray, 1974; Schwartz, 1974). However, the magnitude of heart rate control reported here is greater than that observed in most previous studies. Brener (1974a) and Schwartz (1974) have reported heart rate bi-directional differences of 7 and 8 beats per minute in response to instructions alone, compared to 20 beats per minute in the present study. The discrepancy between the magnitude of these changes may be due in part to the way in which the responses were calculated. In the present work, heart rate changes from pre-trial to trial periods were based on the difference in performance during the last ten seconds of both periods. As can be seen from examination of Figure 15, this measure yields a maximum estimate of the degree of heart rate control. Estimating heart rate control on the difference between mean trial performance and mean pre-trial performance, as Schwartz and Brener appear to have done, yields a mean bi-directional difference of 10 beats per minute on the third day of training, a value which is more in keeping with theirs.

Another finding which emerged from an examination of the performance of the I/HR group was a reliable acquisition effect in the degree of heart rate control. Bi-directional heart rate differences averaged about 10 beats per minute on the first day of training but increased

subsequently, reaching almost 20 beats per minute by the third day of training. These findings are consistent with the observations of Brener (1974a) and Brener et al. (1969). They reported that bi-directional heart rate differences of 2 beats per minute were produced on the first day of training, and that these increased reliably to 3 beats per minute on the second day of training, in subjects provided with instructions alone. The present work extends these findings by indicating that most of the acquisition takes place on Raise trials .

The instructions given to I/HR subjects included both a directional requirement and a reference to a particular response system. Studies reviewed earlier (p.33-34) indicated that both the directional component (Brener and Goesling, 1969) and the reference component (Blanchard et al., 1974a), by themselves, can induce heart rate changes. The present data cannot provide information on the role of the directional component alone. However, they can provide evidence on the importance of the reference component of the instructions. The effect of this component is evident when the response patterns which were observed in the I/SC and the I/HR groups are compared (Figure 21 and p.155). Reliably larger bi-directional differences in heart rate, skin conductance, gross body movement, and respiration amplitude were produced in the latter group than in the former group. The only difference between the two groups was that subjects in the I/SC group were asked to control palmar sweating, whereas subjects in the I/HR group were asked to control heart rate. It is apparent that the effect of designating heart rate as the target response is not the same as designating palmar sweating as the response to be changed, in an instructional manipulation.

A second variable which was examined in Experiment 2 was the suggestion of behavioural strategies. Subjects provided with strategy suggestions were told to raise and lower heart rate, and were also informed that increases in heart rate were usually associated with being tense, with moving, and with paying attention to internal events, whereas decreases in heart rate tended to be associated with relaxation, with immobility, and with paying attention to the external environment. The addition of this strategy information to simple instructions to control heart rate did not result in any greater control over heart rate than was observed when instructions alone were provided. In fact, the opposite seemed to be the case. Providing subjects with strategy suggestions had a deleterious effect on heart rate control. This was evidenced by the fact that the two groups that did not receive strategy suggestions (I and IF) generated reliably larger bi-directional heart rate differences ($p < .01$, p.130) than did the two groups that received strategy suggestions (IS and ISF). This effect on the strategy suggestions appeared to be evident primarily on Raise trials.

These results were somewhat surprising in view of some observations reported by Brener (1973) and Obrist et al. (1975). Obrist et al. (1975) found a negative relationship between the extent to which motor and respiratory changes were constrained and the magnitude of heart rate increases. Brener (1973) indicated that he had gathered similar data. In Experiment 2, somatomotor and respiratory activities were not restricted. Rather, it was explicitly suggested to the subjects that the use of such strategies might be effective in controlling the target response. However the suggestions also made reference to other activities, such as getting tense and excited or paying attention to internal

and external cues, that may have been less effective in producing or facilitating cardiac change.

A final variable which was manipulated in Experiment 2 was the provision of exteroceptive feedback. Its effects were slight. Exteroceptive feedback was clearly not essential for heart rate control, since substantial control was apparent in the groups that did not undergo feedback training. Moreover, although comparisons between the groups provided with feedback (IF and ISF) and those not so provided (I and IS) indicates that performance on the heart rate task was generally better under feedback conditions, this effect was not reliable. Failure to find feedback effects cannot be attributed to a failure to attend to the feedback stimulus when it was presented, as there was evidence which suggested that the subjects used the feedback stimulus to guide their performance on the heart rate task. Subjects appeared to perform slightly better on training trials, when the feedback stimulus was presented, than on test trials, when it was not. The difference between test and training trial performance averaged less than two beats per minute, but it was reliable on Lower trials in both feedback groups ($p < .05$, p.136) and nearly so on Raise trials in the IF group ($p = .06$, p.141).

Thus, the present data do not point to exteroceptive feedback as an important determinant of heart rate performance. In this respect, they conflict with the observations of a number of other investigators, who observed better heart rate control in subjects provided with heart rate feedback than in subjects not so provided (e.g. Blanchard and Young, 1974b; Brener, 1974a; Brener et al., 1969). How can this discrepancy be accounted for?

One possible reason why feedback effects were not substantial in the present work may stem from the fact that comparatively few constraints were placed on the subject's behaviour. Although some restrictions were necessarily implicit in the experimental situation (for example, subjects remained seated in the test chair), subjects were neither asked to refrain from moving nor to refrain from breathing irregularly, as has usually been done in studies of learned autonomic control (cf. Chapter 2). It is possible that feedback effects are manifested only under conditions where the subject's behaviour is constrained considerably. There is a problem with this view, however, in that some of the experiments reporting no effect of feedback were carried out on subjects whose behaviour was constrained considerably (e.g. Johnston, in press; Levenson, 1974). In Levenson's experiment, for example, subjects were required to breathe through a respirometer and to maintain respiratory parameters within certain limits. They were also provided with exteroceptive feedback for respiratory changes to help them control their breathing. Under these conditions, cardiac control was no more substantial in subjects provided with exteroceptive feedback for heart rate changes than in subjects not so provided. Policies with respect to constraints may be a factor in the production of feedback effects, but this variable alone does not appear to fully explain discrepant findings.

Another possible reason why feedback effects were not substantial in the present work may lie in the degree to which subjects could control heart rate without feedback training. It is possible that feedback effects would have been more apparent, had heart rate control in the absence of feedback not been so great. Providing feedback for heart rate

changes may have little effect on the performance of subjects who exert substantial heart rate control prior to training, but it may assist subjects who perform poorly before exposure to the feedback contingency. However, the results of Experiment 2 do not support this possibility. The subjects who exhibited poor heart rate control early in training were compared with those who exhibited substantial control early in training. This comparison revealed that subjects in the former group benefited no more from feedback training than did subjects who controlled heart rate effectively before feedback training began (Table 3).

Still another possible reason why feedback effects were slight in this experiment may be that the feedback parameters employed here were not optimal for heart rate control. There is evidence that some feedback arrangements are more effective in fostering heart rate control than others (Blanchard and Young, 1974b; Lang and Twentyman, 1974). Lang and Twentyman (1974), for example, compared the heart rate performance of subjects receiving analog feedback (i.e., following every heart beat) with that of subjects receiving binary feedback (i.e., only when their heart rate exceeded a pre-determined criterion). Analog procedures were generally more effective in bringing about heart rate control. Subjects provided with analog feedback generated bi-directional heart rate differences which were double the magnitude of the increases generated by subjects provided with binary feedback (9.5 beats per minute versus 4.2 beats per minute). Similar results were reported by Blanchard et al.(1974b). In this thesis, subjects were provided only with binary feedback. It is possible that exteroceptive feedback would have proven a more powerful determinant of heart rate performance if it had been provided in analog fashion. It should be noted, however, that some of

the studies in which substantial feedback effects were observed employed feedback of a binary nature (Brener, 1974a; Brener et al., 1969) For examply, Brener (1974a) reported mean heart rate bi-directional differences of 15 beats per minute in subjects provided with exteroceptive binary feedback, as compared with differences of 5 beats per minute in subjects provided only with instructions to increase and decrease heart rate. Thus, it appears that failures to provide evidence of feedback effects cannot be attributed simply to the use of ineffective, binary feedback contingencies.

It seems evident that no single factor adequately accounts for discrepancies in the present literature with respect to heart rate feedback effects. However, the explanations considered above are not mutually exclusive; it is possible that an adequate account of feedback effects lies in some combination of them. It is also possible that other, as yet unspecified, variables may prove to play an important role in modulating the extent to which exteroceptive feedback affects heart rate control. Attention will return to this question in the last section of this chapter.

Comparison of skin conductance and heart rate

In sum, the present findings suggest that the effects of instructions to control the target response depend upon the nature of the target response system. Providing subjects with only instructions to produce increases and decreases in sweating were ineffective in generating appropriate changes in skin conductance. On the other hand, providing them with only instructions to produce increases and decreases in heart rate resulted in substantial heart rate control, and this control increased over days.

Like the provision of instructions, the provision of feedback exerted different effects on electrodermal and cardiac control. Feedback

training clearly was important to the development of electrodermal control. In fact, in the present experimental situation, feedback training appeared necessary for the development of control over skin conductance when skin conductance was specified as the target response. However, such was not the case with respect to control over heart rate. While performance on the heart rate task was enhanced slightly when the feedback stimulus was presented, the performance of the subjects who underwent feedback training did not differ significantly from that of the subjects who did not undergo such training.

Finally, unlike the provision of either instructions or feedback, the suggestion of behavioural strategies likely to facilitate control appeared to exert similar effects on control of skin conductance and heart rate. In both cases, these effects consisted of decrements in the level of control, where control was obtained in the first place.

Response Profiles

The first goal of the thesis was to assess the role of instructions, strategy suggestions, and exteroceptive feedback in electrodermal and cardiac control. The findings of the thesis with respect to this goal were discussed in the previous section. The second major goal of the thesis was to examine what other behaviours are affected when control is exerted over skin conductance and heart rate. The findings of the thesis as regards this second goal are discussed in this section. Moreover, this section compares the skin conductance and heart rate target conditions with respect to the nature of the response profiles generated, and with respect to the nature of the relationship between target and non-target responses.

Skin conductance

Changes in skin conductance in the groups attempting to control skin conductance were accompanied by changes in other response systems. In both Experiment 1 and Experiment 2, changes in heart rate accompanied electrodermal changes. Furthermore, in Experiment 2, in which more subjects were studied, changes in body movement and respiration amplitude also accompanied the skin conductance changes. Bi-directional differences in several affective scales were observed in both experiments. These findings replicate those of Klinge (1972), who observed that learned changes in skin conductance were accompanied by changes in heart rate and respiratory functions.

However, in the present work, there was evidence that the changes observed in non-target systems were unnecessary for the performance of the electrodermal response. Provision of feedback, which enhanced the magnitude of the skin conductance changes, had no effect on any of the other responses. This suggests that the observed changes in non-target systems may be ascribed either to the directional component of the instructions or possibly to an effect,on response strategies,of referring to sweating, rather than to an intrinsic relationship of these responses to electrodermal behaviour.

These results have implications for the neural processes involved in the performance of voluntary electrodermal changes. These processes do not appear to involve non-specific somatomotor activity or respiratory functions. This is supported by the fact that bi-directional changes in motor and respiratory functions were not augmented by provision of feedback for electrodermal changes. It is possible

that the processes involved in the production of skin conductance changes were specific to the neural fibres which control sweat secretion and reabsorption. However, there are two other possibilities which are consistent with what is known of the neural organization of the electrodermal system.

The first possibility is that electrodermal changes may be associated with movements specific to the arms or hands, particularly the hand from which target skin conductance was recorded. This possibility is a plausible one in view of the evidence reviewed in Chapter 2 (Culp and Edelberg, 1966) which indicated localized augmentation of electrodermal responding by specific movements of the hands and feet. Unfortunately, this possibility cannot be tested post hoc, since electromyographic activity from the relevant areas was not recorded. Nonetheless, if indeed specific hand movements were implicated in the performance of increases and decreases in skin conductance, these movements must have been relatively slight. Had they been of substantial magnitude, they would likely have influenced the gross body movement measure, and would have appeared as artifacts in the electrodermal records.

Another possibility is that skin conductance changes may be associated with changes in a non-specific arousal process that is known to affect electrodermal activity independently of somatomotor activity and heart rate (Roberts, 1974; Roberts and Young, 1971). While the function of this process in behaviour is not known, the fact that the process appears most evident under conditions of aversive stimulation suggests that it may serve a protective function, with increased sweating serving to protect the skin from injury. This process may correspond to what Edelberg (1972b) recently identified as a "defensive reaction". Electro-

dermal responses considered by Edelberg to be generated by this "defensive" process were characterized by long recovery times, presumably as a consequence of inhibited sweat reabsorption. Increased epidermal hydration following inhibition of reabsorption may protect the skin from injury (Edelberg, 1972a).

Heart rate

As with control over skin conductance, the changes in heart rate observed in the groups attempting to control heart rate were accompanied by changes in other responses. In both Experiment 1 and Experiment 2, changes in heart rate were accompanied by changes in skin conductance and body movement. Furthermore, in Experiment 2, in which more subjects were studied, changes in respiration frequency and respiration amplitude also accompanied the heart rate changes. In both experiments, bi-directional differences in heart rate were accompanied by differences on several affective scales. These findings corroborate previous reports of changes in motor variables (Brener, 1974a; Obrist et al., 1975), as well as in respiratory variables when voluntary heart rate changes are produced (e.g. Lang, 1974; Levenson, 1974; Wells, 1973). In addition, these findings indicate that electrodermal and affective differences parallel and voluntary production of heart rate differences.

Contrary to what was the case with respect to skin conductance, there was evidence that the changes observed in non-target response systems (particularly electrodermal and somatomotor responses) were intrinsically involved in the performance of the heart rate changes. This evidence is two-fold. First, in both Experiment 1 and Experiment 2, the magnitude of the changes observed in skin conductance and body movement was related

to the degree of heart rate control, when heart rate was the target response. Providing subjects with strategy suggestions, which reliably diminished heart rate control, also reliably diminished the magnitude of the bi-directional differences in skin conductance and body movement. This effect was also observed with respect to respiration functions, although it was not reliable with respect to these functions. Second, the somatomotor and respiratory changes observed in the groups attempting to control heart rate were larger than those observed in their homologous counterparts attempting to control skin conductance. Only the affective dimensions appeared clearly unrelated to either training or target variables, and thus independent of cardiac functioning. This suggests that bi-directional differences in electrodermal, somatomotor, and respiratory functions are intrinsically related to the performance of the heart rate response, whereas affective differences are not. The latter may simply be a consequence of the directional component of the instructions, as was suggested with respect to these differences when skin conductance was the target response.

These conclusions are consistent with other evidence which indicates that procedures designed to minimize somatomotor and respiratory changes during heart rate control tasks also minimize the degree of heart rate control on Increase trials (Black, 1974b; Brener, 1973; Obrist et al.. 1975). Obrist et al., for example, reported that subjects given no somatomotor restrictions produced larger heart rate increases than subjects told not to move and not to breathe irregularly while attempting to increase heart rate. Moreover, these latter subjects produced larger heart rate increases than subjects given the same somatomotor and respiratory constraints together with practice at implementing them. Brener (1973) indicated that he had gathered similar data. Finally, Black (1974b)

attempted to train rats to produce heart rate increases while holding still. He found that, although animals could be trained to do this, they nonetheless often adopted somatomotor strategies. For example, one animal engaged in somatomotor activity for some time and then stopped suddenly. Since, unlike movement, heart rate decelerated only gradually, the animal technically performed correctly, i.e. he held still while maintaining heart rate above baseline. Black noted that rats could be trained to produce genuine increases in heart rate while holding still, but that in such cases the heart rate increases which were produced were smaller than those which were produced when the animals were also moving.

In addition to these studies concerned with manipulations of motor functions during heart rate control tasks, there is considerable evidence of cardio-somatic and cardio-respiratory coupling in a variety of situations (see Chapters 1 and 2, or Obrist et al., 1970). Thus, the present findings are consistent with other data suggesting that changes in somatomotor and/or respiratory functions are involved in the performance of heart rate changes.

Comparison of the skin conductance and heart rate conditions

In sum, a comparison of the response profiles obtained in the skin conductance and heart rate target conditions indicates that in neither case were changes confined to the target system. However, two differences pertaining to the response profiles were found between the two target conditions. First, both the extent and the magnitude of the changes in non-target somatomotor, and respiratory responses were greater in the heart rate than in the skin conductance target condition. In both Experiment 1 and Experiment 2, Raise and Lower trials could be differentiated reliably with respect to at least one more response when

heart rate was controlled than when skin conductance was controlled. Moreover, comparisons of homologous I and IF skin conductance and heart rate groups indicated that bi-directional differences in heart rate, body movement, and respiration amplitude were reliably greater in the heart rate target condition than in the skin conductance target condition (p.155). Second, the nature of the relationship between target and correlated responses appears to be different for skin conductance and heart rate. The response profiles observed when skin conductance was controlled seemed to reflect, not any intrinsic relationship between skin conductance and its correlates, but rather a non-specific effect of instructions to increase and decrease a particular response. On the other hand, the response profiles observed when heart rate was controlled appeared to reflect an essential relationship between heart rate and at least some of its correlates.

These results indicate that the neural systems involved in the production of skin conductance and heart rate changes are different. While the production of heart rate changes appears to involve alterations in general somatomotor and/or respiratory functions, the production of skin conductance changes does not appear to involve these general somatomotor or respiratory functions. Rather, the latter may involve localized hand movements that were too small to be detected in the present experiments, or changes in a non-motor arousal process. Further work will be required in order to decide between these two alternatives.

Acquisition of Voluntary Control

One major finding of Experiment 2 was that instructing subjects to

control heart rate was sufficient to produce heart rate control, whereas instructing them to control palmar sweating was not sufficient to bring about electrodermal control. The subjects were able to exert control over skin conductance, however, when exteroceptive feedback for electrodermal changes was provided in addition to instructions to control sweating. On the other hand, exteroceptive feedback was not necessary to demonstrate heart rate control, and contributed only minimally to cardiac control when it was provided. These effects may perhaps be more understandable if more were known about the process by which control is acquired over skin conductance and heart rate. This section considers one account of the nature of this acquisition process.

This view holds that the same basic process is involved in acquiring control of skin conductance that is involved in acquiring control of heart rate. In both cases, subjects generate changes in a number of response systems which they believe to be related to the target behaviour. For example, in attempting to control heart rate, subjects may produce changes in somatomotor activity and respiration, in the hope that these will lead to corresponding changes in heart rate. Feedback arising from the occurrence of heart rate changes is utilized to identify those response strategies which are effective in producing cardiac change. These strategies are identified on the basis of temporal conjunctions between feedback deriving from heart rate changes and afferentation deriving from performance of response strategies used by the subjects to produce target responding.

If learning to control skin conductance and heart rate involves this same acquisition process, why is control of the two responses affected differently by the provision of instructions alone, and also by

the provision of exteroceptive feedback? The "discrimination" account of the acquisition process just described would attribute these effects to differences between the two systems with respect to the discriminability of afferentation generated as a consequence of changes in target responding prior to feedback training. As noted in Chapter 1, present knowledge of the neural organization of the electrodermal system (Kuno, 1953; Wang, 1964) suggests that little or no afferentation is generated by palmar or plantar sudomotor effector action in man and other mammalian species. There is evidently no afferentation arising directly from sudomotor activation itself, since the innervation of palmar and plantar sweat glands appears to be purely efferent. It is probable that other receptor systems in skin such as those involving temperature and tactile sensation are affected by extreme variations in epidermal hydration consequent upon sudomotor function, but these extremes are unlikely to be encountered under the usual conditions of psychophysiological experimentation. Afferentation arising as a consequence of myocardial contraction, on the other hand, appears to be more extensive and derives in part from baroreceptors in the carotid sinus and aortic arch, and possibly from receptor systems in the heart and vasculature as well. Extensive afferentation that may be more discriminable than these sources may derive indirectly from mechanoreceptors in muscle and overlying skin which respond to pressure changes generated by ventricular contraction. Thus, subjects may be able to distinguish between high and low heart rates on the basis of these interoceptive sources in the absence of exteroceptive feedback, and they may use this skill to identify response strategies that are effective in producing appropriate heart rate changes.

On the other hand, if little afferentation is associated with sweat gland activity, subjects may be unable to discriminate changes in the level of electrodermal activity in the absence of exteroceptive feedback. Provision of exteroceptive feedback may then be necessary to identify the response strategies which are effective in producing electrodermal changes.

This discrimination analysis made no reference to the subjects' knowledge of effective response strategies, or to their ability to change target behaviour, prior to feedback training. However, inspection of Figure 18 (p.138) shows that subjects evidenced changes in heart rate exceeding 12 beats per minute on the first test trial, under both feedback conditions. This suggests that performance in the present experiments may have been affected by transfer to the experimental task of information (or misinformation) about the response, that was mediated by the verbal labels used to designate the response systems in the instruction. If subjects adopted a particular strategy from prior knowledge that it works, a discrimination analysis assumes that subjects have discriminated variations in visceral responding and have identified activities that produce them in their natural environment. If, on the other hand, subjects act upon misinformation communicated by the verbal labels, as might have happened when instructed to change palmar sweating[8], discrimination theory explains why this information persists as a basis for performance until corrected by an explicit feedback contingency. Both the presence and absence of prior knowledge about a response are explained as a consequence of whether interoceptive referents are available to serve as a basis for learning about the response prior to explicit

feedback training. Thus, it is possible that the differential effect of verbal instructions to change palmar sweating and heart rate may derive from differences in the discriminability of response-related afferentation, even though a portion of the effect is accountable to transfer from previous learning that is mediated by the verbal labels used to reference the response systems in the instructions the subjects received.

An attractive feature of this "discrimination" hypothesis is that it provides an integrated explanation, not only of the different effects of instructions on electrodermal and cardiac control, but also of a number of other observations which might otherwise appear unrelated. For example, it can explain the acquisition function in heart rate in the heart rate target group provided with instructions alone. The identification of strategies effective in producing heart rate changes is a process which may well undergo refinement with practice. No external feedback is needed for this refinement to take place since reliable feedback from an interoceptive source is available.

The "discrimination" hypothesis can also explain why exteroceptive feedback contributed only minimally to heart rate control, but appeared critical for electrodermal control, in the present studies. Since accurate, interoceptive feedback on heart rate changes is already available to the subjects, adding exteroceptive feedback yields little new information, and consequently does not lead to a substantial improvement in performance. On the other hand, since little afferentation appears to be generated by sweat gland effectors, exteroceptive feedback provides the subjects' only reliable measure of skin conductance activity. It

functions as a substitute for the interoceptive feedback which is not available or not identified, and enables the identification of response strategies which are effective in producing appropriate changes in skin conductance.

The deleterious effect which the suggestion of strategies seemed to exert on both electrodermal and cardiac control can also be understood in terms of the above hypothesis. The hypothesis views the establishment of learned control essentially as a process of identifying appropriate response strategies. Strategy suggestions should facilitate control to the extent that they direct the subject's attention to behaviours which are closely related to target responding. As argued earlier, the strategy suggestions given in the present studies may have been ineffective in this respect. Where the subject had access to feedback on changes in the target response from interoceptive and/or exteroceptive sources, the strategy suggestions may also have interfered with control by diverting attention away from sources of afferentation that were related to target behaviour.

The heuristic value of the "discrimination" hypothesis is not limited to providing an account of the present findings. For example, the hypothesis suggests one plausible explanation of why exteroceptive feedback has been observed to contribute to performance in some experiments on heart rate control, but not in others. It seems reasonable to suppose that the discriminability of heart rate changes is a positive function of their magnitude. When few constraints are placed on the subjects' behaviour, as in the present experiments, subjects may engage in response strategies which lead to large heart rate changes that are discriminable on the basis of interoceptive afferentation. Under these

conditions, adding exteroceptive feedback to instructions to control heart rate adds little new information and does not lead to improved performance. Exteroceptive feedback would also not be expected to facilitate heart rate control under conditions where the subjects' behaviour is severely constrained, as in Levenson's studies (1974), because subjects are prevented from engaging in response strategies that are effective in producing heart rate changes, whether exteroceptive feedback is given or not. It is only under conditions where the subjects' behaviour is constrained moderately, such as in Brener's (1974) experiments, that adding exteroceptive feedback to instructions to control heart rate would be expected to lead to an improvement in performance. Under these conditions, the heart rate changes produced by subjects given instructions alone would be expected to be of relatively small magnitude, and are perhaps not easily discriminated. Hence, exteroceptive feedback could serve as a more reliable index of cardiac activity than the available interoceptive feedback, and could enable the subjects to identify the somatomotor strategies which are effective in bringing about heart rate changes. Thus, the "discrimination" hypothesis would lead one to predict that exteroceptive feedback should affect heart rate control only under conditions where the subjects' behaviour is constrained to a moderate extent. The published evidence appears to be compatible with this view.

Thus, the "discrimination" hypothesis presented earlier appears to be of some heuristic value, not only in that it helps understand the results of this thesis, but also in that it integrates other discrepant findings. The hypothesis calls attention to the study of visceral discriminations, and suggests that the study of these phenomena

may prove important in understanding the process(es) underlying the acquisition of control of autonomic responses. There are several questions that may be asked with respect to the phenomena of visceral discriminations, at a number of different levels. Three questions that seem particularly important are the following. First, can subjects discriminate changes in electrodermal activity and heart rate? Second, what is the source of the afferentation the subject has discriminated? Third, what is the relationship between the ability to discriminate and to control autonomic responses?

There is as yet only a small but developing literature on these questions. Most of the published research concerns the question of whether subjects can discriminate the occurrence of target responding. In one experiment, Brener and Jones (1974) presented subjects with 10-second trains of vibratory stimuli in which stimulus pulses were either contiguous with discrete heart beats (contingent trials) or independent of cardiac action (noncontingent trials). The subject's task was to indicate which type of trial he had received. Subjects successfully discriminated between the two types of trial, but only after they had received approximately 60 trials of discrimination training in which they were told whether their choice following each trial was correct. Discrimination training with a control group showed that subjects could not discriminate trial types on the basis of properties of the exteroceptive feedback alone. These results indicate that subjects can discriminate the occurrence of correlates or consequences of cardiac action. However, they do not provide information about which aspect of response state the subjects were discriminating. Subjects may have perceived the occurrence of discrete heart beats, as Brener and Jones intended, or they

may have detected changes in heart rate or the occurrence of somatomotor events that altered the frequency of cardiac contraction. It should be noted that subjects were told their task was to detect discrete heart beats, and that somatomotor and respiratory maneuvers were explicitly discouraged. These features of the test situation probably shifted the subjects away from discriminating changes in heart rate, particularly since somatomotor constraints may have restricted the range of rates available for discrimination.

The question of whether subjects can discriminate events relating to electrodermal responding has also received some attention. Baron (1966) asked subjects to indicate which of a series of exteroceptive stimulus presentations coincided with spontaneous skin resistance responses (GSRs). He found that four of ten subjects chose correctly at frequencies significantly greater than chance, when the discrimination test was given after 20 minutes of explicit feedback training in which subjects practised producing GSRs. No evidence was presented as to whether subjects could discriminate electrodermal responding prior to feedback training. Stern (1972) addressed this question, using a discrimination test similar to Baron's. Stern's subjects were apparently unable to discriminate GSR-contingent tones when tested prior to explicit exteroceptive feedback training for production of the response ($d' = .07$). Subjects who did undergo such training performed better on the discrimination task ($d' = .35$), but neither the performance of each group singly nor the difference between them was evaluated statistically. It should be noted that feedback training lasted only 15 minutes in Stern's experiment and 20 minutes in Baron's. In Experiment 2 of the present thesis, substantial control of electrodermal responding was not apparent

until after approximately 40 minutes (1 day) of training. Stern (1972) and Baron (1966) might have obtained evidence for better discrimination had they trained their subjects longer prior to discrimination testing.

These studies suggest that subjects are capable of discriminating afferentation related to the performance of cardiac and electrodermal responding. They also suggest that the ability to discriminate may be a product of explicit feedback training. It is apparent, however, that data pertaining to autonomic discrimination are generally lacking. Little is known about the variables that affect performance on discrimination tasks, or about how prior training on such tasks contributes to subsequent control of the response during biofeedback training (Brener, 1974). There are also insufficient data to judge the difficult question of whether differences in the intrinsic discriminability of autonomic responses are a factor in determining whether provision of an exteroceptive feedback contingency contributes to learned control of these responses. Little is known about the basis of the discriminations which have been reported. Nevertheless, the data presently available on discrimination encourage further study insofar as they suggest that discriminations occur as a result of biofeedback training, and that discriminability can be assessed. This would appear to be a profitable area for further experimental and theoretical work (Brener, 1974a).

Chapter 6: Summary

This thesis was concerned with the general question of how voluntary control, defined as compliance with a verbal instruction to change a response, is achieved over skin conductance and heart rate. This question encompasses three separate but related issues. First, what features of the experimental situation contribute to the establishment of electrodermal and cardiac control? Second, what other response systems are affected when control is achieved over electrodermal and cardiac functions, and how are changes in these systems related to the occurrence of the target response? Finally, how do subjects learn to comply with an instruction to control skin conductance and heart rate, and is the acquisition process different for the two responses? Following a preliminary experiment in which a procedure for producing voluntary control of heart rate and skin conductance was developed, a more extensive experiment addressed these issues.

One goal of the experiments was to assess the contribution of (i) instructions to change the response, (ii) suggestion of response strategies, and (iii) provision of exteroceptive feedback for successful performance, to control of electrodermal and heart rate responding. Verbal instructions to increase and decrease palmar sweating were insufficient to generate bi-directional control of skin conductance. However, electrodermal control was established when exteroceptive feedback for skin conductance responding was provided in addition to instructions to control sweating. Strategy suggestions appeared to inferfere with electrodermal control, when given to subjects that received feedback for

electrodermal responding. The results with respect to heart rate were different. Instructions to increase and decrease heart rate were sufficient to generate highly reliable changes in this response. Adding feedback to these instructions had little effect on performance. However, adding strategy suggestions interfered with performance irrespective of whether feedback was also provided.

A second goal of the experiments was to examine the changes which take place in other response systems when skin conductance and heart rate are controlled. Control over skin conductance was not confined to the electrodermal system. Bi-directional differences in skin conductance were accompanied by bi-directional differences in heart rate, gross body movement, respiration amplitude, and in a number of affective scales. However, these correlates did not appear to be intrinsically related to the skin conductance changes, as they were not augmented by feedback as was skin conductance responding. Correlated responses were also found when heart rate was the target response. These included changes in skin conductance, gross body movement, respiration frequency, respiration amplitude, and several affective scales. Contrary to the results obtained when skin conductance was the target response, there was evidence that the autonomic, somatomotor, and respiratory correlates of heart rate changes were intrinsically related to the performance of heart rate response. Correlates of cardiac responding were affected by the same variables that influenced the magnitude of voluntary heart rate change.

Finally, the thesis attempted to develop some understanding of the process(es) involved in the acquisition of skin conductance and heart rate control. It was hypothesized that the same basic process was

involved in the acquisition of electrodermal and cardiac control. In both cases, it was suggested that feedback deriving from the target response was utilized to identify appropriate response strategies. Since there appear to be a number of interoceptive sources of information about changes in cardiovascular function that may be intrinsically discriminable, subjects may have been able to identify response strategies which led to appropriate heart rate changes, without feedback training. On the other hand, there appear to be few interoceptive sources of information about changes in electrodermal function. Thus, exteroceptive feedback may have been necessary to identify response strategies that led to control of this response system.

Footnotes

1. Brener (1974a) has also reported a relationship between heart rate and respiratory variables. However, his method of recording respiration is quite idiosyncratic, and does not distinguish between changes in respiration rate and changes in respiration amplitude. Likewise, Schwartz (1974) reported a relationship between heart rate and respiratory variables, but did not elaborate on the nature of this relationship.
2. Monetary incentive was employed in view of some evidence (Lang, 1974) that heart rate control was better when it was provided than when it was not. This evidence was not reviewed in Chapter 2, since the provision of monetary incentive was not a variable which was manipulated in the present experiments.
3. These differences are small, and their basis is unclear, particularly as such differences were not observed in Experiment 2, where the same sequences of trial presentations were used. To evaluate whether these differences may have spuriously influenced heart rate control, product-moment correlations were computed between the degree of heart rate control on Raise and Lower trials and baseline heart rate. These proved unreliable. Thus, there is little reason to believe that the small differences in baseline heart rate in the SC-1 group affected the major results of the experiment.
4. However, such a comparison, if feedback training were to have an effect, could not yield information about the feature(s) of feedback training which is/are important, viz. its informative of incentive properties, for instance.

5. In fact, this new procedure for setting criterion was not without pitfalls. While it worked quite well in general, it had a tendency to make the criterion too easy to meet on Raise trials, particularly when heart rate was the target response. In order to maintain equivalent densities of feedback presentation on Raise and Lower trials, it was at times necessary to make the criterion more difficult than it would normally have been on those Raise trials.
6. It should be noted that in the present circumstances the main effect for Feedback is not the appropriate test for the effects of feedback training. Although a reliable main effect of Feedback would indicate that feedback training had some effect on electrodermal control, the absence of a reliable main effect of Feedback could not be taken as evidence that feedback training was ineffective. If there were a substantial degree of symmetry between Raise and Lower trial performance, a reliable main effect of Feedback would not obtain, even though feedback contributed to performance on both types of trial. In such a case the Feedback-by-Types of Trial interaction would be a more appropriate test for the effects of feedback training.
7. Each of the 1920 correlations that were computed was based on six pairs of measures taken on the third day of training, where control appeared established. These six pairs were based on the difference in performance between the last ten seconds of the trial and baseline periods on the six Raise and Lower test trials, for each subject. In retrospect, computing correlations in this way probably underestimated the congruence between at least some of the responses. Correlations based on more measures taken over acquisition, and based on

performance on both Raise and Lower trials, would probably have been more substantial. Likewise, directional correlations, that take only response direction and occurrence into account (Roberts and Young, 1971), also would likely have been more substantial.

8. Inspection of Group I/HR in Figure 21 shows that subjects evidenced substantial control of skin conductance when they were instructed to control heart rate. Thus it is clear that untrained subjects possess the ability to alter electrodermal responding, but that they fail to utilize this skill when the behavioural goal is specified as a change in "palmar sweating". Reference to palmar sweating may have switched the subjects on to thermoregulatory strategies that would not likely have influenced sudomotor outflow to volar surfaces, since palmar glands do not appear to respond to thermal stimulation except under rather extreme conditions (see Chapter 1).

9. Readers interested in these questions might find some of the work on EEG discrimination and control a particularly useful background (e.g. Black, Cott and Pavloski, in press; Kamyia, 1967; Rosenfeld and Hetzler, 1973).

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APPENDICES

Appendix A

Following are the instructions given the subjects in the various experimental groups in Experiments 1 and 2.

The instructions were given after the subject was seated and fully electroded. Prior to receiving these instructions, each subject had been told, as he entered the experimental chamber, that a number of electrodes would be placed on him, and that these electrodes would record his heart rate, his sweating activity, and his eye movements; that, in addition, a thing would be placed around his chest to measure his respiration, and, finally, that he would be sitting on a cushion which would record his gross body movement.

Subjects in Experiment 1 received the same instructions on days 1 and 2, but different instructions on days 3 and 4. Subjects in Experiment 2 received the same instructions every day.

Instructions to subjects receiving skin conductance training first in Experiment 1. Days 1 and 2.

In this experiment, we will try to see whether you can control certain responses which are normally thought of as being involuntary. In this case, we are interested in finding out whether you can control the amount of sweating that is generated by your fingers, or what we call the galvanic skin response. Here is what the response looks like (show sample of electrodermal record). When a person sweats a lot, he or she exhibits a lot of activity, as you can see here. On the other hand, when a person is not sweating very much, his record looks rather flat, like this. During the experiment, you will be asked to try to produce a lot of activity when this Raise or R light will be on (point), and as little activity as possible when this other Lower or L light (point) will be on. How well you manage to do this will determine how much money you'll get.

Each of these lights will go on periodically, and every time each light goes on, it will be on for 30 seconds. When the lights are not on, you are not required to do anything, just sit here quietly. When the R light goes on (point), it will mean that you should try to raise your level of activity, that you should produce as much activity as possible. When the L light goes on (point), you should try to lower your activity level, to respond as little as possible.

Every time you comply with these instructions, a soft tone will come on from behind you. I'll be keeping a record of how long the tone is on, and I'll be paying you according to how long you manage to keep the tone on. You'll be getting paid at a rate of 10¢ per minute when this tone is on. A typical subject can expect to earn between

70¢ and a dollar in bonus money, every day. This is in addition to the $3. a day that you'll be getting for participating in the study, and I'll tell you at the end of today how much you've earned.

You may wonder how you can go about responding correctly. A lot of sweating is usually associated with thinking or with being tense and emotionally active, while no sweating is usually associated with relaxing or with being emotionally quiet. During Raise trials, then, you might try to think or to be emotionally involved. And during Lower trials, you might try to relax, to be unemotional. This is just to help you, and I should point out that although these strategies work for many people, they may not necessarily work for you. You are encouraged to try a number of different things, and if you find something that works, stick with it.

Let me recapitulate. Every time that you are performing correctly while the Raise or Lower light is on, a soft tone will go on, to tell you that you are doing alright. Every time this tone is on, you'll be earning bonus money. The longer you keep the tone on during each trial the better you're doing, and the more money you're accumulating.

Periodically, there is going to be a trial when, even though you will be performing correctly, the tone will not go on at all. You can expect this to happen about every five trials, but these trials won't be indicated in any way. This is so that we can see how well you can control your sweating when you're not getting any feedback. Even though you won't be getting any feedback on those trials, you should keep trying because I'll still be monitoring your performance on these trials, and you'll still be getting paid whenever the tone would normally have been on. O.K.?

I am now going to leave you, and in a couple of minutes the Raise and Lower lights will start coming on periodically. The experiment will last about 45 minutes, and then I'll come back and take the electrodes off. O.K.? Do you know what to do? Do you have any questions? If you need to communicate with me at any time during the experiment, you may speak up, and I'll hear you through an intercom. O.K.?

One more thing: In addition to following the instructions I'll also ask you not to touch or play with the electrodes because it generates noise in the recordings.

Instructions on Day 3

In the first part of the experiment, we attempted to find out whether you could control the amount of sweating generated by your fingers. From now on, we will try to see whether you can control your heart rate. The rules are essentially the same as they were in the first part of the study, except that you should now try to control your heart rate rather than your galvanic skin response. During the experiment you will be asked to raise your heart rate whenever the Raise or R light will be on, and to lower your heart rate whenever the Lower or L light will be on.

As before, each of these lights will go on periodically, and every time each light goes on, it will be on for 30 seconds. When the lights are not on, you are not required to do anything, just sit here quietly. When the R light goes on, it will mean that you should try to raise your heart rate. When the L light goes on, it will mean that you should try to lower your heart rate.

As before, every time you comply with the instructions a tone will go on. I'll be keeping a record of how long the tone is on, and I'll be paying you according to how long you manage to keep the tone on. You'll be getting paid at a rate of 10¢ per minute when this tone is on, the same as you were during the first part of the study. A typical subject can expect to earn between 70¢ and a dollar in bonus money, in addition to the $3. that you'll be getting for participating in the study, and I'll tell you at the end of today how much you've earned.

How do you go about changing your heart rate? Increases in heart rate are <u>usually</u> associated with a number of things. They tend to be associated with being tense and excited, with moving or thinking about moving, and with rejecting the environment and paying attention to internal events. Decreases in heart rate, on the other hand, tend to be associated with relaxation, with immobility, and with taking in the environment, paying attention to external cues. During Raise trials, then, you might try to get excited, and to pay attention to your own bodily processes. And during Lower trials you might try to relax, and to pay attention to what is going on around you. Again, as in the first part of the experiment, this is just to help you, and although these strategies work for many people, they may not necessarily work for you. You are encouraged to try a number of different things, and if you find something that works, stick with it.

As in the first part of the study, periodically there is going to be a trial when, even though you'll be performing correctly, the tone will not come on at all. You can expect this to happen about every five trials, but these trials won't be indicated in any way. This is

so that we can see how well you can control your heart rate when you're not getting any feedback. Even though you won't be getting any feedback on these trials, you should keep trying because I'll still be monitoring your performance on these trials, and you'll still be getting paid accordingly.

O.K.? Do you know what to do? Do you have any questions?

Again, in addition to following the instructions, I'll ask you not to touch or play with the electrodes because it creates noise in the recordings.

Instructions on Day 4

Today again we will try to see whether you can control your heart rate, and the rules are the same as they have been all along.

As before, each of these lights will go on periodically, and every time each light goes on, it will be on for 30 seconds. When the R light goes on, it will mean that you should try to raise your heart rate. When the L light goes on, it will mean that you should try to lower your heart rate.

As before, every time you comply with these instructions, a tone will go on. I'll be keeping a record of how long the tone is on, and I'll be paying you according to how long you manage to keep the tone on. You'll be getting paid at a rate of 10¢ per minute when this tone is on, the same as you have been all along. A typical subject can again expect to earn between 70¢ and a dollar in bonus money, and I'll pay you at the end of today's session.

How do you go about changing your heart rate? Increases in heart rate are usually associated with a number of things. They tend to be

associated with being tense and excited, with moving or thinking about moving, and with rejecting the environment and paying attention to internal events. Decreases in heart rate, on the other hand, tend to be associated with relaxation, with immobility, and with taking in the environment, paying attention to external cues. During Raise trials, then, you might try to get excited, and to pay attention to your own bodily processes. And during Lower trials, you might try to relax, and to pay attention to what is going on around you. Again, this is just to help you, and although these strategies work for many people they may not necessarily work for you. You are encouraged to try a number of different things, and if you find something that works, stick with it.

As before, periodically there is going to be a trial when even though you will be performing correctly, the tone will not come on at all. You can expect this to happen about every five trials, but these trials won't be indicated in any way. This is so that we can see how well you can control your heart rate when you're not getting any feedback. Even though you won't be getting any feedback on these trials you should keep trying because I'll still be monitoring your performance on these trials, and you'll still be getting paid accordingly.

O.K.? Do you know what to do? Do you have any questions?

Again, in addition to following the instructions I'll ask you not to touch or play with the electrodes because it creates noise in the recordings.

Instructions to subjects receiving heart rate training first in Experiment 1. Days 1 and 2.

In this experiment, we will try to see whether you can control certain responses which are normally thought of as being involuntary. In this case, we are interested in finding out whether you can control your heart rate. During the experiment you will be asked to try to raise your heart rate when this (point) Raise or R light will be on, and to lower your heart rate whenever this other (point) Lower or L light will be on. How well you manage to do this will determine how much money you'll get.

Each of these lights will be on periodically, and every time each light goes on, it will be on for 30 seconds. When the lights are not on, you are not required to do anything, just sit here quietly. When the R light goes on (point), it will mean that you should try to raise your heart rate. When the L light goes on, you should try to lower your heart rate.

Every time you comply with these instructions, a tone will come on from behind you. I'll be keeping a record of how long the tone is on, and I'll be paying you according to how long you manage to keep the tone on. You'll be getting paid at a rate of 10¢ per minute when this tone is on. A typical subject can expect to earn between 70¢ and one dollar in bonus money. This is in addition to the $3. a day that you will be getting for participating in the study, and I'll tell you at the end of today how much you've earned.

You may wonder how you can go about responding correctly. Increases in heart rate are usually associated with a number of things. They

tend to be associated with being tense and excited, with moving or thinking about moving, and with rejecting the environment and paying attention to internal events. Decreases in heart rate, on the other hand, tend to be associated with relaxation, with immobility, and with taking in the environment, paying attention to external cues. During Raise trials, then, you might try to get excited, and to pay attention to your own bodily processes. And during Lower trials, you might try to relax, and to pay attention to what is going on around you. This is just to help you, and I should point out that although these strategies work for many people, they may not necessarily work for you. You are encouraged to try a number of different things, and if you find something that works, stick with it.

Let me recapitulate. Every time that you are performing correctly while the Raise or the Lower light is on, a soft tone will come on to tell you that you are doing alright. Every time this tone is on you will be earning bonus money. The longer you keep the tone on during each trial, the better you are doing, and the more money you are accumulating.

Periodically, there is going to be a trial when even though you will be performing correctly, the tone will not go on at all. You can expect this to happen about every five trials, but these trials won't be indicated in any way. This is so that we can see how well you can control your heart rate when you're not getting any feedback. Even though you won't be getting any feedback on these trials, you should keep trying because I'll still be monitoring your performance on these trials, and you'll still be getting paid whenever the tone would normally have been on.

I am now going to leave you, and in a couple of minutes the Raise and Lower lights will start coming on periodically. The experiment will last about 45 minutes, and then I'll come back and take the electrodes off. O.K.? Do you know what to do? Do you have any questions? If you need to communicate with me at any time during the experiment, you may speak up, and I'll hear you through the intercom. O.K.?

One more thing. In addition to following the instructions, I'll also ask you not to touch or play with the electrodes, because it creates noise in the recordings.

Instructions on Day 3

In the first part of the experiment, we attempted to find out whether you could control your heart rate. From now on, we'll try to see whether you can control the amount of sweating generated by your fingers, or what we call the galvanic skin response. Here is what the response looks like (show record). When a person sweats a lot, he or she exhibits a lot of activity, as you can see here. On the other hand, when a person is not sweating very much, his record looks rather flat, like the one on the bottom. During the experiment, you will be asked to try to produce a lot of activity when the Raise or R light will be on, and as little activity as possible when the Lower or L light will be on.

As before, each of these lights will go on periodically, and every time each light goes on, it will be on for 30 seconds. When the lights are not on, you are not required to do anything, just sit here quietly.

When the R light goes on, it will mean that you should try to raise your level of activity. When the L light goes on, you should try to lower your level of activity.

As before, every time you comply with the instructions a tone will go on. I'll be keeping a record of how long the tone is on, and I'll be paying you according to how long you manage to keep the tone on. You'll be getting paid at a rate of 10¢ per minute when this tone is on, the same as you were during the first part of the study. A typical subject can expect to earn between 70¢ and a dollar in bonus money, in addition to the $3. that you will be getting for participating in the study, and I'll tell you at the end of today how much you've earned.

How do you go about changing your sweating activity? A lot of sweating is usually associated with thinking, or with being tense and emotionally active, while no sweating is usually associated with relaxing or with being emotionally quiet. During Raise trials, then, you might try to think or to be emotionally involved, and during Lower trials you might try to relax, to be unemotional. Again, as in the first part of the experiment, this is just to help you, and although these strategies work for many people, they may not necessarily work for you. You are encouraged to try a number of different things, and if you find something that works, stick with it.

As in the first part of the study, periodically there is going to be a trial when, even though you will be performing correctly, the tone will not come on at all. You can expect this to happen about every five trials, but these trials won't be indicated in any way. This is so that we can see how well you can control your sweating when you're

not getting any feedback. Even though you won't be getting any feedback on these trials you should keep trying because I'll still be monitoring your performance on these trials, and you'll still be getting paid accordingly.

O.K.? Do you know what to do? Do you have any questions?

Again, in addition to following these instructions, I'll ask you not to touch or play with the electrodes because it generates noise in the recordings.

Instructions on Day 4

Today again we will try to see whether you can control the amount of sweating generated by your fingers, and the rules are the same as they have been all along.

As before, each of these lights will go on periodically, and every time each light goes on, it will be on for 30 seconds. When the R light goes on, it will mean that you should try to raise your level of sweating activity. When the L light goes on, it will mean that you should try to lower your activity level.

As before, every time you comply with these instructions, a tone will go on. I'll be keeping a record of how long the tone is on, and I'll be paying you according to how long you manage to keep the tone on. You'll be getting paid at a rate of 10¢ per minute when this tone is on, the same as you have been all along. A typical subject can again expect to earn between 70¢ and a dollar in bonus money, and I'll pay you at the end of today's session.

How do you go about changing your sweating activity? A lot of sweating is usually associated with thinking, or with being tense and emotionally active, while no sweating is usually associated with

relaxing or with being emotionally quiet. During Raise trials, then, you might try to think or to be emotionally involved, and during Lower trials, you might try to relax, to be unemotional. Again, this is just to help you, and although these strategies work for many people, they may not necessarily work for you. You are encouraged to try a number of different things, and if you find something that works, stick with it.

As before, periodically there is going to be a trial when, even though you'll be performing correctly, the tone will not go on at all. You can expect this to happen about every five trials, but these trials won't be indicated in any way. This is so that we can see how well you can control your sweating when you're not getting any feedback. Even though you won't be getting any feedback on these trials, you should keep trying because I'll still be monitoring your performance on these trials, and you'll still be getting paid accordingly.

O.K.? Do you know what to do? Do you have any questions?

Again, in addition to following the instructions, I'll ask you not to touch or play with the electrodes because it generates noise in the recordings.

Instructions to Ss in the Instruction groups (I) Experiment 2

In this experiment, we are trying to find out whether people can control certain responses which are normally thought of as being involuntary. In this case, we are interested in finding out whether you can control (your heart rate) / (the amount of sweating that is generated by your fingers). During the experiment, you will be asked to try to raise your heart rate/sweating activity when this (point) Raise or R light will be on, and to lower your heart rate/sweating activity whenever this other (point) Lower or L light will be on. How well you manage to do this will determine how much money you'll get.

Each of these lights will be on periodically, and every time each light goes on, it will be on for 30 seconds. When the lights are not on, you are not required to do anything, just sit here quietly. When the R light goes on (point), you should try to raise your heart rate/sweating activity. When the L light goes on (point), you should try to lower your heart rate/sweating activity level.

How well you manage to comply with these instructions will determine how much money you'll get. I'll be keeping a record of how well you do, and I'll be paying you accordingly. A typical subject can expect to earn between 70¢ and one dollar in bonus money. This is in addition to the $3. a day that you will be getting for participating in the study, and I'll tell you at the end of today how much you've earned.

I am now going to leave you and in a couple of minutes the Raise and Lower lights will start coming on periodically. The experiment will last about 45 minutes, and then I'll come back and take the electrodes

off. O.K.? Do you know what to do? Do you have any questions? If you need to communicate with me at any time during the experiment, you may speak up, and I'll hear you through the intercom. O.K.?

One more thing. In addition to following the instructions, I'll also ask you not to touch or play with the electrodes, because it creates noise in the recordings.

Instructions to Ss in the Strategy groups (IS) Experiment 2

In this experiment, we are trying to find out whether people can control certain responses which are normally thought of as being involuntary. In this case, we are interested in finding out whether you can control (your heart rate) / (the amount of sweating that is generated by your fingers). During the experiment, you will be asked to try to raise your heart rate/sweating activity when this (point) Raise or R light will be on, and to lower your heart rate/sweating activity whenever this other (point) Lower or L light will be on. How well you manage to do this will determine how much money you'll get.

Each of these lights will go on periodically, and every time each light goes on, it will be on for 30 seconds. When the lights are not on, you are not required to do anything, just sit here quietly. When the R light goes on (point), you should try to raise your heart rate/sweating activity. When the L light goes on (point), you should try to lower your heart rate/activity level.

You may wonder how you can go about responding correctly. Increases in heart rate/sweating are usually associated with a number of things. They tend to be associated with being tense and excited, with moving or thinking about moving, and with rejecting the environment and paying attention to internal events. Decreases in heart rate/sweating, on the other hand, tend to be associated with relaxation, with immobility, and with taking in the environment, paying attention to external cues. During Raise trials, then, you might try to get excited, and to pay attention to your own bodily processes. And during Lower trials, you might try to relax, and to pay attention to what is going

on around you.

How well you manage to comply with these instructions will determine how much money you'll get. I'll be keeping a record of how well you do, and I'll be paying you accordingly. A typical subject can expect to earn between 70¢ and one dollar in bonus money. This is in addition to the $3. a day that you will be getting for participating in the study, and I'll tell you at the end of today how much you've earned.

I am now going to leave you and in a couple of minutes the Raise and Lower lights will start coming on periodically. The experiment will last about 45 minutes, and then I'll come back and take the electrodes off. O.K.? Do you know what to do? Do you have any questions? If you need to communicate with me at any time during the experiment, you may speak up, and I'll hear you through the intercom. O.K.?

One more thing. In addition to following the instructions, I'll also ask you not to touch or play with the electrodes, because it creates noise in the recordings.

Instructions to Ss in the Feedback groups (IF) Experiment 2

In this experiment, we are trying to find out whether people can control certain responses which are normally thought of as being involuntary. In this case, we are interested in finding out whether you can control (your heart rate) / (the amount of sweating generated by your fingers). During the experiment, you will be asked to try to raise your heart rate/sweating activity when this (point) Raise or R light will be on, and to lower your heart rate/sweating activity whenever this other (point) Lower or L light will be on. How well you manage to do this will determine how much money you'll get.

Each of these lights will go on periodically, and every time each light goes on, it will be on for 30 seconds. When the lights are not on, you are not required to do anything, just sit here quietly. When the R light goes on (point), you should try to raise your heart rate/sweating activity. When the L light goes on (point), you should try to lower your heart rate/activity level.

Every time you comply with these instructions, a soft tone will come on from behind you. Whenever this tone is on, you'll be earning bonus money. I'll be keeping a record of how long the tone is on, and I'll be paying you according to how long you manage to keep the tone on. You'll be getting paid at a rate of 10¢ per minute when this tone is on. A typical subject can expect to earn between 70¢ and one dollar in bonus money. This is in addition to the $3. a day that you'll be getting for participating in the study, and I'll tell you at the end of today how much you've earned. So the longer you keep the tone on during each trial, the better you are doing.

Periodically, there is going to be a trial when, even though you will be performing correctly, the tone will not go on at all. You can expect this to happen about every five trials, but these trials won't be indicated in any way. This is so that we can see how well you can control your heart rate/sweating activity when you're not getting any feedback. Even though you won't be getting any feedback on these trials, you should keep trying because I'll still be monitoring your performance on these trials, and you'll still be getting paid whenever the tone would normally have been on. O.K.?

I am now going to leave you and in a couple of minutes the Raise and Lower lights will start coming on periodically. The experiment will last about 45 minutes, and then I'll come back and take the electrodes off. O.K.? Do you know what to do? Do you have any questions? If you need to communicate with me at any time during the experiment, you may speak up, and I'll hear you through the intercom. O.K.?

One more thing. In addition to following the instructions, I'll ask you not to touch or play with the electrodes, because it creates noise in the recordings.

Instructions to Ss in the Strategy-Feedback groups(ISF)Experiment 2

In this experiment, we are trying to find out whether people can control certain responses which are normally thought of as being involuntary. In this case, we are interested in finding out whether you can control (your heart rate) / (the amount of sweating that is generated by your fingers). During the experiment, you will be asked to try to raise your heart rate/sweating activity when this (point) Raise or R light will be on, and to lower your heart rate/sweating activity whenever this other (point) Lower or L light will be on. How well you manage to do this will determine how much money you'll get.

Each of these lights will go on periodically, and every time each light goes on, it will be on for 30 seconds. When the lights are not on, you are not required to do anything, just sit here quietly. When the R light goes on (point), you should try to raise your heart rate/sweating activity. When the L light goes on (point), you should try to lower your heart rate/activity level.

You may wonder how you can go about responding correctly. Increases in heart rate/sweating are usually associated with a number of things. They tend to be associated with being tense and excited, with moving or thinking about moving, and with rejecting the environment and paying attention to internal events. Decreases in heart rate/sweating, on the other hand, tend to be associated with relaxation, with immobility, and with taking in the environment, paying attention to external cues. During Raise trials, then, you might try to get excited, and to pay attention to your own bodily processes. And during Lower trials, you might try to relax, and to pay attention to what is going on around you.

Every time you comply with these instructions, a soft tone will come on from behind you. Whenever this tone is on, you'll be earning bonus money. I'll be keeping a record of how long this tone is on, and I'll be paying you according to how long you manage to keep the tone on. You'll be getting paid at a rate of 10¢ per minute when this tone is on. A typical subject can expect to earn between 70¢ and one dollar in bonus money. This is in addition to the $3. a day that you'll be getting for participating in the study, and I'll tell you at the end of today how much you've earned. So the longer you keep the tone on during each trial, the better you are doing.

Periodically, there is going to be a trial when, even though you will be performing correctly, the tone will not go on at all. You can expect this to happen about every five trials, but these trials won't be indicated in any way. This is so that we can see how well you can control your heart rate/sweating activity when you're not getting any feedback. Even though you won't be getting any feedback on these trials, you should keep trying because I'll still be monitoring your performance on these trials, and you'll still be getting paid whenever the tone would normally have been on. O.K.?

I am now going to leave you, and in a couple of minutes the Raise and Lower lights will start coming on periodically. The experiment will last about 45 minutes, and then I'll come back and take the electrodes off. O.K.? Do you know what to do? Do you have any questions? If you need to communicate with me at any time during the experiment, you may speak up, and I'll hear you through the intercom. O.K.?

One more thing. In addition to following the instructions, I'll ask you not to touch or play with the electrodes, because it creates noise in the recordings.

Appendix B

Following is the sequence of trial presentations used in both experiments. A and B trials can refer to either Raise and Lower trials, or the converse. Test trials are marked by an asterisk (*).

1. A*	11. A*	21. A	31. A
2. B*	12. B	22. B	32. B*
3. B	13. B	23. B*	33. B
4. A	14. B	24. A	34. B
5. A	15. A	25. A*	35. A
6. B	16. A	26. B	36. A
7. A	17. A*	27. A	37. A
8. B*	18. B	28. B	38. B
9. A	19. A	29. A*	39. A
10. B	20. B*	30. B	40. B*
			41. A*

Appendix C Post-experimental questionnaire

Subjects in Experiment 1 were asked to fill such a questionnaire at the end of days 2 and 4; subjects in Experiment 2 were asked to fill it at the end of day 3. The questionnaires were identical in the two studies, but for two minor differences. First, questions 13 and 14 did not appear on the questionnaires distributed in Experiment 1. And second, the words "the tones" in question 4 were replaced by either "your sweating activity" or "your heart rate" for subjects in the I and IS groups in Experiment 2.

Post Experimental Questionnaire

1. Name:_____

2. Age:_____

3. a. Year:_____

   b. Course:_____

4. Do you feel that you were successful overall in controlling the tones? Answer in one of the boxes on the scale below.

   Not at all successful |_____|_____|_____|_____|_____|_____|_____| Very successful

5. How difficult was it to perform correctly on Raise trials?

   Impossible |_____|_____|_____|_____|_____|_____|_____| Could do it at will

6. How difficult was it to perform correctly on Lower trials?

   Impossible |_____|_____|_____|_____|_____|_____|_____| Could do it at will

7. Which was more difficult to do, Raise or Lower?

   R very much more difficult than L |_____|_____|_____|_____|_____|_____|_____| L very much more difficult than R

8. What did you do when the R light came on?
   a. Any special kinds of thoughts?

   b. Any special kinds of movements?

8. c. Other?

9. What did you do when the L light came on?
   a. Any special kinds of thoughts?

   b. Any special kinds of movements?

   c. Other?

10. What did you do when neither light was on?

   a. Any special kinds of thoughts?

10. b. Any special kinds of movements?

c. Other?

11. Rate the R light on the following dimensions:

tense	——|——|——|——|——|——|——	relax
comfortable	——|——|——|——|——|——|——	uncomfortable
work	——|——|——|——|——|——|——	rest
mind	——|——|——|——|——|——|——	muscles
sexual	——|——|——|——|——|——|——	asexual
exciting	——|——|——|——|——|——|——	dull
pleasant	——|——|——|——|——|——|——	unpleasant
hot	——|——|——|——|——|——|——	cold
easy	——|——|——|——|——|——|——	difficult
flaccid	——|——|——|——|——|——|——	strained
anxious	——|——|——|——|——|——|——	calm

12. Rate the L light on the following dimensions:

tense	——|——|——|——|——|——|——	relax
comfortable	——|——|——|——|——|——|——	uncomfortable
work	——|——|——|——|——|——|——	rest
mind	——|——|——|——|——|——|——	muscles
sexual	——|——|——|——|——|——|——	asexual
exciting	——|——|——|——|——|——|——	dull
pleasant	——|——|——|——|——|——|——	unpleasant
hot	——|——|——|——|——|——|——	cold
easy	——|——|——|——|——|——|——	difficult
flaccid	——|——|——|——|——|——|——	strained
anxious	——|——|——|——|——|——|——	calm

13. Do you smoke? _____

If so, how much? _____

14. Do you engage in meditation or yoga? _____

If so, how often? _____

15. Do you have any comments about the experiment?

16. Would you be willing to participate in another experiment like this in the future? _____

THANK YOU VERY MUCH FOR YOUR PARTICIPATION IN THIS STUDY.