INDIVIDUAL DIFFERENCES IN HEART RATE REGULATION: AN EXPLORATORY INVESTIGATION

by

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Abstract

Demonstrations of heart rate regulation in humans have frequently revealed marked individual differences in performance. These differences appear to be reliable over time and have prompted several investigations aimed at their elucidation. These investigations have, however, dealt primarily with psychological (personality) variables, neglecting potentially important physiological variables. The present study was primarily concerned with an investigation of two such variables: tonic heart rate preceeding regulation trials (initial level) and heart rate reactivity. The psychological variable of incentive was also examined.

Exploration of the effects of initial level across subjects involved a comparison of absolute level scores (uncorrected) and transformed level scores (corrected) which adjusted for individual differences in the upper and lower bounds of cardiac rate. Reactivity was estimated, prior to the attempted heart rate regulation, using a respiratory manipulation to elicit both acceleratory and deceleratory heart rate responses. The relationships between these physiological variables and heart rate increase and decrease regulation were assessed in separate multiple regression analyses for each direction.

The regulation procedure itself involved five 4 min trials for each direction of attempted regulation with

visual, analogue feedback being available on the last four trials. The incentive manipulation was introduced in conjunction with the regulation segment of the experiment: for incentive subjects a monetary reward was offered for the best overall regulation performance and for no-incentive subjects an identical sum of money ($20) was awarded according to a random draw.

Analyses of the regulation data revealed significant heart rate changes in both directions, with increases being larger than decreases. Changes in both directions were, however, relatively small in comparison with other reports in the literature. This difference was attributed to the fact that, in the present study, instructions to the subjects may have more effectively restricted somatic changes. The incentive manipulation proved ineffective, likely for reasons specific to the particular incentive condition used in this study.

The multiple regression analyses revealed non-significant overall regression effects, however some interesting trends did appear. Uncorrected and corrected initial level showed some relationship to increase and decrease performance respectively and these relationships were discussed in the context of the Law of Initial Values (Wilder, 1957).

The correlation between decrease reactivity and decrease regulation also approached significance; it was suggested that this relationship might reflect the involvement of a respiratory strategy in regulation.

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I wish to thank the members of my committee, Dr. Bram Goldwater, Dr. Loren Acker and Dr. Derek Ellis for their dedicated contributions to my graduate education. Their influence extends far beyond this thesis.

A special note:

My efforts to test the extent of my supervisor's patience and tolerance of graduate students can be considered nothing short of valiant. But alas, the quest was in vain. The limits of his calm enduring are as the horizon to the wanderer - unattainable.

Dedication

Theses are very often dedicated to the spouse of the author; in this case to my wife Pam. The reasons for this become eminently clear in the writing of same.

Also to Hersch - if only he knew what he was in for.

Introduction

The primary concern of the present investigation was an exploratory examination of the contributions of two cardiac variables to individual differences in heart rate regulation. These variables were (a) tonic initial or pre-regulatory level and (b) reactivity to a somatic manipulation. Heart rate regulation was trained with the aid of proportional, bi-directional meter feedback, a paradigm generally viewed within the framework of instrumental conditioning.

The research strategy adopted here may be considered 'exploratory' in the sense that (a) it is correlational and (b) does not set out to test any specific, previously formulated models of the relationships of the above variables to heart rate regulation. The correlational approach was adopted of necessity since the variables are not readily amenable to experimental manipulation, at least not in a manner which might not also directly influence performance on the regulation task. With regards to hypothesis testing, current notions of the roles of these variables in heart rate are insufficiently precise to permit an approach in which experimental conditions are so arranged as to test specific predictions of alternate models.

The present study also included an assessment of the effects of motivational level on heart rate regulation, motivation being indirectly manipulated by an offer of monetary reward.

Evaluation of this effect does not, however, relate to individual differences in performance in the present investigation.

The remainder of the introductory section will provide, first, a comprehensive literature review relating to individual differences in heart rate regulation and subsequently, a more detailed presentation of the variables of interest here.

Individual Differences in Heart Rate Regulation: Literature Review

In the now considerable literature on heart rate regulation (Blanchard & Young, 1973) it is apparent that although such regulation has been clearly demonstrated, the effect is subject to considerable variability. Engel and his associates (Engel & Hansen, 1966; Engel & Chism, 1967) and Blankstein (1972) have explicitly noted a considerable degree of inter-individual variability in the degree of heart rate regulation achieved by their subjects. Such variability is also evident in a series of studies by Brener et al (Brener & Hothersall, 1966, 1967; Brener, Kleinman & Goesling, 1969) in which the performance of individual subjects is reported.

Engel & Hansen (1966) and Engel & Chism (1967) describe almost identical studies of heart rate slowing and speeding respectively. In the speeding study, while all experimental subjects showed increases relative to

their initial levels, these changes ranged in magnitude from 4.2 to 9.8 beats per minute (bpm). In the slowing study Engel & Hansen (1966) report that only five out of 10 subjects showed a learning effect; the learners showed decreases averaging around two bpm, while the non-learners showed increases of about the same magnitude.

Brener & Hothersall (1966, 1967), in two studies also having similar paradigms, trained subjects in bi-directional heart rate regulation using binary auditory feedback. Inter-individual differences are evident with respect to both the magnitude and the consistency of the resulting changes.

Other studies reporting on individual subjects who have received long-term training (10 - 18 weeks) provide further evidence of considerable variability. Stephens, Harris & Brady (1972) trained four subjects using both auditory and visual feedback as well as a monetary reinforcer. These subjects showed increases ranging from 15 to 34 bpm. Decreases were attempted by only two of these subjects, one showing a mean decrease of 22 bpm at the end of training and the other showing only a six bpm change.

Another study involving relatively intensive training has been reported by Wells (1973). Three of nine subjects showed increases of less than seven bpm throughout the entire experiment, consisting of approximately eight hours of feedback training. The remaining six subjects all

reached a level of at least 15 bpm over their pre-trial rates, with one subject showing a maximum increase during one session of 35 bpm. Again, during decrease trials, the changes were much smaller, with three of the subjects showing no mean decreases on any of the sessions. One subject demonstrated a decrease in only one session and the remainder of the subjects showed small changes, never exceeding 3.1 bpm.

Given that numerous instances of individual differences in heart rate regulation may be found, it should be pointed out that these differences also appear to be reliable over time. This reliability provides some assurance that the differences are, in fact, related to the individual's capacity for heart rate regulation rather than to some more transient aspects of the subject's internal state or the experimental situation.

In the above study by Wells (1973), in which subjects were given 13 training sessions all on different days, it appears from an examination of the heart rate increases that these individual differences were not due simply to initial differences in the rate of acquisition of heart rate regulation. All subjects who showed any clear learning did so by the seventh to the ninth sessions and subsequently retained their ability to the termination of the experiment. The remaining subjects showed only minimal changes throughout the entire experiment. No reliable differences were evident for heart rate decreases in this

study as all subjects performed very poorly in that the changes were small and inconsistent.

Further evidence of the consistency of such individual differences is found in Blankstein (1975). The performance of subjects in feedback assisted heart rate regulation in a one year follow-up was reported. Sixteen subjects were classified as either good bi-directional controllers (regulators) of poor bi-directional controllers on the basis of their performance in the earlier Blankstein & Egner (1973) study. Subjects were then ranked separately for increases and decreases according to their performance in both sessions. The Spearman rank order correlation co-efficients for increases and decreases between Year I and Year II were $r = +.92$, $p<.01$ and $r = +.74$, $p<.01$ respectively. Thus individual differences in ability to learn heart rate regulation remain stable over long periods of time.

Individual Difference Variables: Psychological

Observations such as these have led to the examination of various subject variables which might account for this variability, including personality traits (Fotopolous, 1970; Ray & Lamb, 1974; Ray, 1974; Blankstein & Egner, 1973), motivation (Cox & Siprelle, 1971), autonomic awareness (Bergman & Johnson, 1971; Blanchard, Young & McLeod, 1972) and sex (Young & Blanchard, 1972).

Fotopolous (1970) grouped subjects according to their scores on the Rotter (1966) Internal - External Locus of

Control Scale. This scale is based on the proposition that the effects of reinforcement will depend, in part, on whether or not the individual perceives the reinforcement as being contingent on his own behavior. That is, an individual designated as an Internal may be characterized as having the belief that the events and course of his life are determined by him. An External, on the other hand, would be inclined to believe that control of these events resides outside himself.

This instrument has been found to reliably differentiate subjects in a variety of situations (Throop & MacDonald, 1971) and its usefulness in heart rate control was consequently investigated. Fotopolous (1970) instructed her subjects to increase their heart rates under conditions of feedback and no-feedback and found that without feedback the Internal Locus of Control subjects were able to produce greater heart rate increases than the Externals. No differences were found in the feedback condition. These findings were, unfortunately, confounded by initial differences in baseline heart rates between the two groups. Her results were, nevertheless, replicated and extended by Ray & Lamb (1974) and Ray (1974). In neither of these two studies were there any initial heart rate differences and the Internals were again better able to increase their heart rates. In addition, the Externals were found to be more proficient at heart rate slowing. This interaction was found under both feedback and no-feedback conditions.

It would appear, therefore, that the Internal - External Locus of Control dimension is related to heart rate regulation, however there is some question as to the basis for this relationship. The fact that Fotopolous (1970) reported the relationship only under no-feedback conditions and in the Ray & Lamb (1974) and Ray (1974) studies no Locus of Control x Direction x Feedback interaction was reported suggests that the basis for the relationship is not due to differences related to the perception of the reinforcement as the construct suggests.

Those studies concerned with autonomic awareness (Bergman & Johnson, 1971; Blanchard, Young & McLeod, 1972; Green & Nielsen, 1966), as measured by the Autonomic Perception Questionnaire (APQ) (Mandler, Mandler & Uviler, 1958) reported results contradictory to the authors' expectations. On the assumption that the ability to detect heart rate variability facilitates heart rate regulation, it was predicted that high APQ subjects (ie. high in awareness of autonomic functioning) would show better heart rate control. Bergman & Johnson (1971), who divided their subjects into three groups (high, medium and low APQ) obtained the best performance from the medium group, and Blanchard et al (1972), using two groups, reported the low group to be superior. Green & Nielsen obtained the same results as the latter investigators in a study in which instrumental conditioning of non-specific galvanic skin response (GSR) emission was compared for high and low APQ

subjects.

These apparently contradictory findings may perhaps be better understood through a re-interpretation of the APQ as an indicator of anxiety. An investigation of the effects of anxiety are consistent with this view. Blankstein & Egner (1973) grouped subjects according to the Spielberger State-Trait Anxiety Inventory (Spielberger, Gorsuch & Lushene, 1968), into high, medium and low anxious subjects. Increase performance was generally superior to decrease performance and the low anxious group was better than both the medium and high groups which did not differ significantly from each other.

Another variable which has on occasion been suggested to be important to heart rate regulation studies is motivation. Wells (1973) reports large magnitude heart rate changes produced by subjects that the experimenter considered "to have the greatest motivation and interest" at the time of subject selection. Consideration of this variable was previously recommended by Headrick, Feather & Wells (1971). Some support of the effect of motivation may be found in the failure of Cox & Sipprelle (1971) to replicate the earlier Ascough & Sipprelle (1968) study involving verbal conditioning of heart rate increases and decreases. These latter experimenters used subjects who were true volunteers while those in the replication were coerced. Cox & Sipprelle (1971) subsequently found that the effect was replicable with volunteers or subjects

receiving an essentially token monetary reinforcement.

Individual Difference Variables: Sex

The final study of individual difference variables to be discussed here is that of Young & Blanchard (1972) in which the effect of sex of subjects was assessed. These authors reported that, while there was no difference in average heart rate decreases between males and females, average increase performance (measured as a change from resting level) was found to be significantly greater for males ($t = 2.25$, $p < .05$). Unfortunately no baseline data were reported.

This brief review highlights the study of individual differences in heart rate regulation to date. It is clear that reliable differences over time do exist and their elucidation will undoubtedly contribute to the development of procedures for the maximization of such regulation across subjects and may contribute some insight into the mechanisms of heart rate regulation.

The above studies, however, deal almost entirely with the relationships of psychological factors to autonomic regulation while inter-subject differences along physiological dimensions have received little attention. Physiological variables would, in particular, be more directly relevant to questions of mechanism and the individual difference variables discussed below are those with which this study was primarily concerned.

Individual Difference Variables: Physiological

A. Initial level. One seemingly obvious variable of interest would be the initial or pre-regulatory level of activity in the system of interest. In the context of heart rate regulation, it is proposed that pre-regulation heart rate for any given subject will determine to some extent the degree of change possible (in either direction) within the system. This general effect has previously been described as the Law of Initial Values (Wilder, 1957). Heart rate is considered to fluctuate within some range of values determined by a negative feedback (homeostatic) system, and changes from an initial level near either limit cannot be expected to be large, if they are in the direction approaching that limit.

These considerations may then apply particularly to the training of heart rate decreases. A review of the literature clearly indicates that, with a few notable exceptions (Scott, Blanchard, Edmundson & Young, 1972; Stephens et al, 1972), heart rate decreases have been very small relative to the changes reported for heart rate increases. In almost every report of human heart rate regulation, the subjects are selected from a normal college population and are given an initial adaptation period, generally of 10 to 20 minutes duration, from which baseline levels are determined. Furthermore, these subjects are generally in a semi-reclined position in a comfortable chair, in a sound attenuated and softly

lit room. It is reasonable to assume, therefore, that unless the task or some other aspect of the experimental situation induces considerable arousal, these subjects are initially, and remain, relatively relaxed. Autonomic (and central) indicators of arousal would be expected to show appropriately low levels of activity and thus it seems highly unlikely that decrease training through feedback would produce further changes of the order shown for heart rate increases.

Gatchel (1974), in a discussion of differences between heart rate speeding and slowing, considered the above hypothesis, however he subsequently rejected it, partially on the basis of a nonsignificant correlation between initial level and slowing performance ($r = .36$, $p<.10$). Although not significant, there does appear to be a tendency for high resting rate to be associated with greater slowing performance. More reliable, however, was his finding of a significant relationship between initial heart rate level and average speeding performance during feedback trials ($r_1 = -.47$, $p<.01$; $r_2 = -.52$, $p<.01$) [1]. Low resting rate was associated with greater performance.

Lang & Twentyman (1974) briefly offered the same suggestion of finding small decreases as a result of low resting levels. These authors note that "the human pulse

[1] Gatchel (1974) presented data from two experiments, but only heart rate speeding was studied in the second.

almost never falls more than 20 beats below these base rates, whereas an increase of more than 100 bpm would not exceed the normal range in work or emotional arousal (p. 628)". However, it appears that these authors also dismissed this alternative in favor of one suggesting different physiological regulation mechanisms, a suggestion for which they presented little justification.

Differences in heart rate increase and decrease regulation also caused Engel & Chism (1967) to remark that

"One factor which may be important between learning heart rate speeding and heart rate slowing is the degree to which a S can emit a change. Within the conditions of this study of heart rate speeding there is no boundary to S's ability to emit the correct response, ie. S would be rewarded for any increase in rate over his operant level, and since it is well known that it is possible for HR to increase considerably from one's resting level, the reward conditions of this study are easy to fulfill (p. 425)".

Further tentative support for this initial level hypothesis may be found in those studies which do report considerable changes in heart rate on an individual basis. Scott et al (1972) reported a trained decrease averaging eight bpm from an initial level of 88 bpm in a subject with a history of tachycardia. Changes in the increase direction were also reported for two subjects, one producing an increase of 16 bpm from an initial level of 75 bpm and the other showing a change of 31 bpm from an initial rate of about 56 bpm. Stephens et al (1972) also reported a considerable decrease (22 bpm) for a subject whose resting rate was 91 bpm. Furthermore, of the four subjects trained, the largest increase (34 bpm) was shown by the subject having the lowest resting rate (48 bpm).

Thus the findings reviewed above offer some support for the initial level hypothesis, suggesting that it may have been prematurely dismissed by Gatchel (1974) and Lang & Twentyman (1974). However, it appears that the correlations reported by Gatchel (1974) constitute the only statistical evaluation of the hypothesis in the heart rate regulation literature.

It is important to note that in each of the above studies any given subject's baseline activity could be expressed only relative to that of other subjects in the study. This point is central to any examination of an LIV relationship and requires further discussion.

Systematic investigation of the relationship between initial level and conditioning effects demands an assessment of a subject's resting level relative to his own range of activity rather than relative to the group range. The LIV and its present analogue refer to the properties of an individual nervous system and comparisons across subjects in this regard are unlikely to provide appropriate data since inter-subject variation in initial level may be confounded with individual differences in range (Bridger & Reiser, 1959; Block & Bridger, 1962). Thus, for example the same heart rate baseline might represent a relatively low level for one subject and a relatively high level for another in terms of their respective ranges of normal functioning. Conversely, different initial levels might represent relatively equivalent levels of functioning for

individuals with different ranges. Block & Bridger (1962) demonstrated that the relationship between the magnitude of change and pre-stimulus level across a group is considerably different from that determined for each of the individual subjects who composed the group.

In light of this consideration, the correlations reported by Gatchel (1974) may represent less than optimal evaluations of the relationship. These correlations do not reflect an individual's rate relative to his own physiological range, but rather relative to the group. Thus they contain some amount of between-subjects variability unrelated to the operation of the LIV. For example, Lykken, Rose, Luther & Maley (1966), in a discussion of arousal as reflected in skin potential (SP), concluded that

"the limits within which SP varies for a given individual are determined by structural and physiological factors probably unrelated to the underlying variable of interest... (p. 481)".

Similar inter-individual variability surely exists in the cardiovascular system and any attempt to demonstrate a relationship between initial level and heart rate control will require an approach whereby this variability is removed.

Lykken and his associates (Lykken, 1965; Lykken et al, 1966; Lykken & Venables, 1971; Lykken, 1972) have reported a technique developed specifically for this purpose. These authors recommended a transformation of the measured values of a physiological system into values which reflect the activity levels of the system with respect to its own structurally defined properties, that is, those

which determine the range of possible values that might be obtained for a given individual. The upper and lower bounds of the range may be estimated and this range applied to the measured values of physiological functioning as a correction having the form

$$\frac{p_{ix} - p_{i(min)}}{p_{i(max)} - p_{i(min)}}$$

where p_{ix} is the uncorrected value of the measure for $subject_i$ in $situation_x$, and where $p_{i(max)}$ and $p_{i(min)}$ are estimates of that subject's maximum and minimum output levels respectively.

The effectiveness of this correction has been demonstrated in several instances (Lykken et al, 1966; Lykken, 1972), its application resulting in considerably increased orderliness of the data and increased power of the significance tests.

Thus, given the current status of the initial level hypothesis in the heart rate regulation literature and the availability of a technique offering increased precision of analysis, a principal objective of the present research is a more precise investigation of the effects of initial level on the magnitude of feedback assisted heart rate changes.

Initial level, then, constitutes one parameter of physiological functioning which may be related to the previously discussed variability in the acquisition or demonstration of heart rate regulation.

B. Reactivity. A second physiological variable examined in the present study was that of heart rate reactivity or lability, as measured with an eliciting stimulus (the "reactivity stimulus").[2] The investigation of stimulus-elicited cardiac responses as a predictor of heart rate regulation is suggested by the following very general lines of reasoning.

The present argument is predicated on a conceptualization of heart rate changes under operant conditioning procedures as involving relatively discrete, phasic "responses"; the more of them and the greater their magnitude, the better the regulation performance. Heart rate is, of course, continually variable and subject, under normal conditions, to a variety of both external and internal influences. It is assumed here that these same influences or mechanisms are operative also under conditions of heart rate regulation, but that the conditioning procedures promote a more systematic operation of specifically relevant mechanisms. Thus, an analysis of cardiac regulation in operant terms implicitly involves (a) the view that any and all changes in the

[2] While an investigation of heart rate reactivity and regulation had not been reported in the literature prior to the commencement of the present study, such a report did appear during the period of data collection. Bell & Schwartz (1975) employed tasks involving environmental intake and rejection (Lacey, Kagan, Lacey & Moss, 1963) to produce heart rate decreases and increases respectively. No significant relationship was found between the magnitude of heart rate changes to these reactivity tasks and the magnitude of changes under feedback conditions. Heart rate responses to cognitive tasks or to outward directed attention would not, therefore, seem predictive of regulation performance.

appropriate direction, relative to pre-regulation baselines, are "responses", and (b) the assumption that these "responses" are present at all times, ie. they are not new "responses" having their origin in the feedback situation.

Besides being implicit in the operant model, the assumption that the regulation (conditioning) involves responses already present in the subject's repertoire receives additional explicit support in the fact that the demonstration of regulation may require only the instructions to regulate heart rate, ie. without feedback (eg. Bell & Schwartz, 1973, 1975), and that where both instructions and feedback are given from the outset, a significant level of regulation performance is often observed on the very first trial.

The hypothesis that the responses which occur under the regulation condition might be related to the responses elicited by the reactivity stimulus is predicated on the assumption that there exists a general factor of cardiac lability; specifically, it is assumed that subjects may be ranked according to the magnitude of their heart rate response to a particular stimulus and that this ranking remains relatively constant across different stimuli (see, for example, the discussion of individual response-stereotypy in Sternbach, 1966). Thus, individuals whose autonomic responding is characterized by large cardiac responses relative to other subjects, might also be expected to show large magnitude changes under conditions of analogue

feedback, particularly if the feedback situation is viewed simply as "setting the occasion" for the production of pre-existing responses. Moreover, to the extent that heart rate regulation in the feedback situation does involve learning to increase the emission of appropriate responses, it would seem reasonable that those subjects who show a tendency or capacity for the production of relatively large changes in heart rate might, under conditions of analogue feedback, have a greater opportunity for proportionally larger magnitude reinforcement.[3]

Since the present investigation involves an attempt to demonstrate bi-directional heart rate regulation, and since it has been suggested that different mechanisms may be involved in increases and decreases (Lang & Twentyman, 1974; Gatchel, 1974; Bell & Schwartz, 1975), it is desirable to obtain reactivity measures in both directions as well.

While a variety of stimuli are known to elicit a heart rate acceleration, the conditions producing a discrete deceleratory response are more limited.

Researchers have for a number of years reported on heart rate decelerations concommitant with 'environmental

[3] Under conditions of analogue feedback the reinforcer is defined in terms of degrees of meter needle deflection and thus the magnitude of the reinforcer is directly proportional to the magnitude of the response. The reinforcement value of any given degree of needle deflection (in other words, the status of a given needle deflection as a reinforcer) will, of course vary as a function of the individual's reinforcement history.

intake' (Lacey et al, 1963; Lacey & Lacey, 1970; Obrist, Webb, Sutterer & Howard, 1970a, 1970b; Chase, Graham & Graham, 1968). That is, attention directed outward is associated with a deceleratory heart rate response. The commonly used reaction time paradigm may be viewed as such an instance of outward directed attention, however the heart rate responses are often of small magnitude (around 2 bpm) (Johnson & May, 1969; Connor & Lang, 1969) and would also require a large number of trials to obtain a relatively stable estimate of reactivity.

A more reliable and more dramatic deceleratory heart rate response has recently been demonstrated by Furedy & Poulos (1974) with a simple respiratory manipulation involving three seconds of even exhalation, four seconds of even inhalation and 30 seconds of breath-holding. This respiratory pattern produced a reliable bi-phasic heart rate response, the decelerative component averaging approximately nine bpm relative to the rate at pre-manipulation exhalation. The initial acceleratory component averaged about 14 bpm.

Motivation

One final question to which the present research addressed itself was that of motivation. As previously mentioned, a number of investigators (Wells, 1973; Headrick et al, 1971; Cox & Siprelle, 1971) considered a high level of motivation important to the effectiveness of feedback training. Motivation level is included here as an

independent variable, in part to reproduce the effects reported by Cox & Siprelle (1971) and further to assess the efficacy of a different type of incentive condition.

Monetary reinforcers have frequently been used in training heart rate regulation (Engel & Hansen, 1966; Engel & Chism, 1967; Scott et al, 1972; Lang, 1974; Cox & Siprelle, 1971). In these studies subjects have generally been awarded small amounts of money ranging from 1/10¢ to 1/2¢ per second of correct responding and some cumulative indication of the amount of money the subject had acquired was presented to him on a counter. The present study, however, contains a variation of an approach suggested by some research carried out by Dr. L. Rosenblood (personal communication). In this case, a money incentive was presented in the form of a $20 prize to be awarded the subject showing the best overall performance. This procedure, it was hoped, would prove effective on the assumption that the possibility of winning a relatively large sum of money would be more motivating for the subject than the more usual procedure of either offering very small monetary reinforcers or simply paying the subject $1.50 - $2.00 per hour (or per session).

Summary of Research Objectives

The following constitutes a brief overview of the objectives and research strategy of the present investigation:

1. A demonstration of bi-directional heart rate regulation. Instructions to alter heart rate and visual, analogue feedback were provided. Performance was assessed in an analysis of variance.
2. An examination of the effects of motivation on heart rate regulation. A monetary incentive was available for some subjects contingent on their regulation performance. A no-incentive control group was included. The incentive - no-incentive factor was included in the above ANOVA.
3. An examination of the relationship of (a) initial level (corrected and uncorrected), and (b) cardiac reactivity to a respiratory manipulation to each direction of heart rate regulation. These relationships were examined in correlational analyses.

Method

Subjects

Subjects were solicited from the college population by an advertisement which included mention of a feedback experiment and a $20 prize. Twenty-four male subjects were selected, all having been screened for histories of cardiovascular or respiratory illness.

Apparatus and Physiological Recording

Electrodes and site preparations. The electrode sites were scrubbed lightly with Brāsivol (Medium), a surfactant cleansing base containing a mild abrasive (fused aluminum oxide particles). The sites were then cleansed with isopropyl alcohol and Beckman Ag-AgCl disc electrodes containing Beckman electrode paste were applied.

Heart rate. Electrode placement for the electrocardiogram (EKG) was on the sternum in order to eliminate, as much as possible, any movement artifact. The signal was fed into a signal shaping device to further reduce the intrusion of movement artifact and the output of this device triggered a Grass Model 7P4C Tachograph, whose output is a direct measure of rate in bpm. From the tachograph the signals were fed through two parallel DC Driver amplifiers and recorded simultaneously on two polygraph channels, one for scoring purposes and one for control of feedback to the subject. That is, this second channel made possible the rest period adjustments to be described below in the

Procedure section.

Feedback was presented via an Armaco UM3 ±500 microampere meter placed about four feet directly in front of the subject. The meter face measured approximately 11 x 7 cm. Calibration of the meter was such that full scale deflection to either side of centre represented a change of 20 bpm and the scale was divided into units of 1 bpm. The meter was concealed behind smoked plexiglass allowing the meter face to be viewed only when it was illuminated. The "Increase", "Decrease" and "Rest" signals were presented to the subject by the illumination of the appropriate sign directly above the feedback meter (see Figure 1).

Muscle Activity. Muscle activity was recorded through a Wide Band AC Preamplifier & Integrator from the right forearm extensor. It was initially intended that the electromyographic (EMG) activity be recorded as an integrated function, however, equipment difficulties necessitated the recording of raw EMG activity, permitting only very gross quantification.

Respiration. A Beckman Strain Gauge strapped around the subject's chest provided the input for a Grass Low Level DC Preamplifier, Model 7P1A.

Respiration was recorded but was not scored. It served primarily as a visual check on the subject and as a basis for respiratory cycle time units for heart rate scoring during the reactivity task.

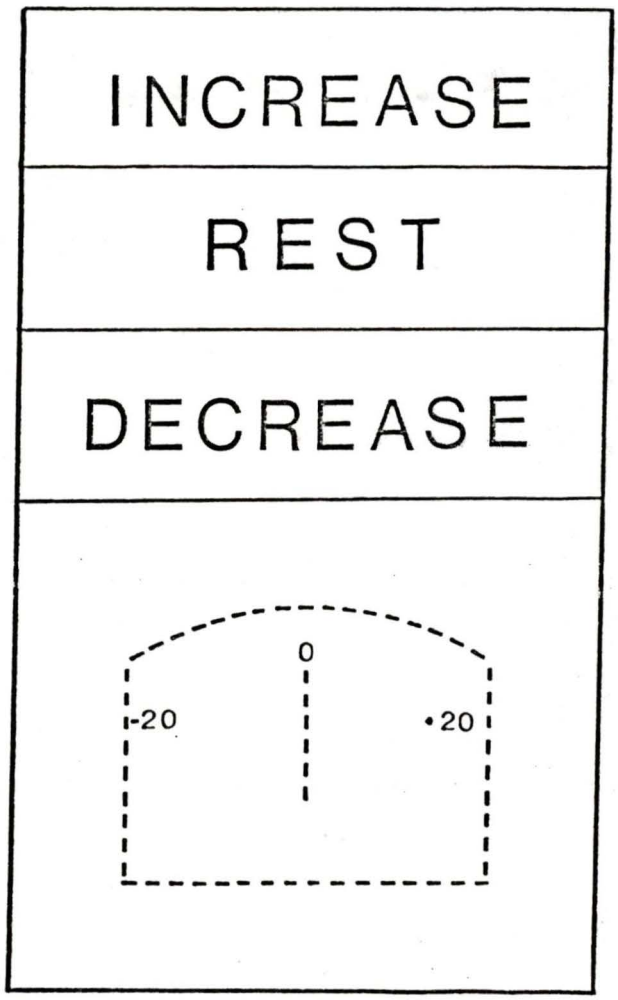

Figure 1. Arrangement of feedback meter and signal lights

Range Determination

Estimate of Maximum. The upper estimate of a subject's range was determined by a method previously used by Lykken et al (1966). This consists simply of having the subject inflate a balloon until it bursts. The maximum heart rate attained is then taken to represent the upper limit of cardiac functioning.

Subjects were cued as to when to begin balloon inflation and were instructed to use regular, paced breaths, avoiding hyperventilation.

Estimate of Minimum. For the lower bound it seemed reasonable to simply obtain a measure of the subject's heart rate during a time when it would be expected to be at its lowest point in its daily rhythmic fluctuation.

On the basis of two studies in which subjects were monitored every six hours (Adkins, 1964; Málek, 1962), heart rate appears to be at its lowest point in the early hours of the morning and reaches its peak about mid-morning. Some pilot data taken while lying in bed just before going to sleep and just after awakening also indicated that heart rate was reliably lower in the morning. Thus subjects were instructed to monitor their radial pulse each morning (while still lying in bed) for a period of one week prior to the training session and the lowest reported heart rate was used as the estimate of the lower limit.

Reactivity

The respiratory manipulation of the present experiment was somewhat different from that reported by Furedy & Poulos (1974) (previously described on p. 19). Some pilot data of the present investigation indicated that neither the prolonged exhalation nor the breath-holding appeared to be necessary to the production of the previously described bi-phasic response pattern. Subjects were therefore simply told to maintain a regular and shallow respiration pattern and then, on cue, to take a slow, deep breath, filling their lungs to capacity. They were not required to hold their breath and could then continue to breathe as prior to the cue. This sequence was repeated six times for each subject.

Using this technique, two reactivity measures were obtained. During the initial acceleration to the deep breath the heart rate associated with the shortest R-R interval was obtained. From this value was subtracted the heart rate over the last complete respiration cycle preceeding the deep inspiration. This comprised the reactivity-up score. The reactivity-down score was derived by subtracting the lowest heart rate (longest R-R interval) obtained within 10 seconds after the peak acceleration from the pre-inspiration value. These two reactivity scores were calculated only for the last two respiration sequences (ie. #5 and #6) and the mean reactivity score in each direction was used in the analyses.

Heart Rate Regulation

Each heart rate regulation trial was of a four minute duration and was preceeded by a 2 min baseline interval. For all trials except the first, this latter interval was comprised of a 30 sec time-out followed by a 90 sec rest period; the first trial was preceeded by a 2 min rest period. Regulation trials were scored for the average rate over the 4 min period. Pre-trial base levels were obtained by scoring heart rate over the last 30 seconds of the preceeding rest period.

A separate no-feedback control group was not included in the design since the primary emphasis of the study was the examination of individual differences in heart rate regulation rather than a specific demonstration of the source of such regulation in instrumental conditioning or feedback training. Moreover, such a control group would have doubled the number of subjects required. Instead, a crude within-subjects control was added to the design in the form of two initial regulation trials without feedback, one for each direction of regulation. These two preliminary trials involved only instructions to increase or decrease heart rate; the remaining trials included both feedback and instructions.

Incentive

Subjects were informed by the recruitment notices that a $20 prize was being offered for participation in this study. They were not told, however, of the specific conditions of the award until the introduction of the

incentive manipulation in the instructions immediately prior to the regulation phase of the experiment.

There were, in fact, two $20 prizes and under the incentive condition the money was to be awarded to the subject showing the best overall performance on the regulation task. The no-incentive condition was one in which the prize was awarded on the basis of a random draw and was in no way contingent upon performance.

Procedure

Upon arrival at the laboratory, the subject was seated in a comfortable chair and given general instructions regarding the experiment. More specific instructions were given immediately prior to each subsequent phase of the experimental procedure.

All the necessary subject and equipment preparations were made and on completion a two minute baseline was taken. After this period the subject was instructed as to the respiration task and the six respiration sequences or trials were obtained. Following this, instructions were given for the balloon task which was initiated by a signal from the experimenter and terminated with the bursting of the balloon. Subjects were then allowed a five minute recovery period which ended the pre-regulation segment of the experiment.

Following this period, instructions on heart rate regulation and the use of the feedback apparatus, together with appropriate briefing on the $20 prize were presented,

subjects having been previously assigned to the incentive or no-incentive group on an alternate basis.

Subjects were told to attempt, during each 4 min regulation trial, to either increase or decrease their heart rate according to the condition in effect for that trial as signalled by the "Direction" sign above the meter. They were instructed to attend both to the magnitude and the consistency of heart rate changes, using the meter for feedback when provided. The deliberate use of muscle tension or respiratory changes was discouraged, and this restriction was emphasized by informing the subject that when marked changes in muscle tension during a regulation trial were observed on the polygraph, the experimenter would note these and draw them to the subjects attention during the subsequent inter-trial baseline period. Subjects were also informed as to the sequence of regulation, time-out and rest periods and were further provided with a summary chart of this sequence for reference. The subject was permitted to make postural adjustments during the time-out period, but was instructed to sit as quietly as possible during the 90 seconds of rest period which immediately preceeded a trial.

Each subject received a total of 10 regulation trials, five in the increase direction and five in the decrease direction. The direction of attempted regulation was alternated across trials, with order counterbalanced across subjects. The first two trials were without feedback, the meter face being dark; the meter was illuminated, feedback

thereby being provided, for the remaining eight trials. The meter illumination was always turned off during pre-trial baselines. Increase or decrease trials were signalled by illumination of the appropriate Direction sign and rest periods by illumination of the "Rest" sign, while time-out was indicated by turning off the feedback meter and all signal lights. A brief summary of the experimental procedure is presented in Table 1.

Table 1

Summary of the experimental procedure

Pre-regulation procedure:

general instructions

hook-up procedures

2 min baseline

respiratory (reactivity) task

balloon inflation

5 min recovery period

feedback and incentive instructions

Regulation procedure:

Trial 1:	2 min rest period	
	4 min regulation, no feedback	1 increase & 1 decrease with order counter-balanced.
Trial 2:	30 sec time-out	
	90 sec rest	
	4 min regulation, no feedback	
Trials 3-10:	30 sec time-out	alternate increase & decrease trials with order counter-balanced.
	90 sec rest	
	4 min regulation, with feedback	

Results

Regulation Performance and Incentive

Analysis of the regulation and incentive effects was carried out using a mixed ANOVA design with incentive (2; incentive vs no-incentive) as the between-groups factor and regulation (2; pre-trial rest period vs trial period), direction (2; increase vs decrease) and trials (5) as the within-groups factors. Since there was no indication that feedback had any effect over and above that of instructions alone on the first increase and decrease trial, no separate analysis of feedback vs no-feedback was conducted.

The mean values for each rest and regulation period are reported separately for increase and decrease trials in Table 2. The results of the ANOVA are summarized in Table 3 and the significant trends are graphically displayed in Figures 2-5.

In the ANOVA, the effect of primary importance with regards to a demonstration of heart rate regulation is that of the direction x regulation interaction ($F = 35.99$, $df = 1,22$; $p = .000$). This interaction is largely attributable to the elevated heart rate under the increase regulation periods, showing an average change over trials of about 2.5 bpm (see Figure 2), an effect also indicated by the significant main effect of regulation ($F = 7.10$, $df = 1,22$; $p = .014$). Analysis of the increase simple main effect shows this change to be highly significant

Table 2

Performance on heart rate regulation task: Mean heart rate levels (pre-trial rest and regulation trial) and change scores. Data collapsed across the non-significant between-groups effect.

	Decrease			Increase		
	Rest	Regul.	HR Change	Rest	Regul.	HR Change
Trial 1	70.05	69.35	- .70	69.11	72.06	2.95
2	69.12	67.74	-1.38	68.93	71.16	2.23
3	67.93	67.47	- .46	67.68	69.08	1.40
4	66.72	65.41	-1.31	64.85	67.63	2.78
5	65.54	65.26	- .28	64.51	67.78	3.27
Mean	67.87	67.05	- .82	67.02	69.54	2.52

Table 3

Analysis of Variance: Incentive (2) x Regulation (2) x Direction (2) x Trials (5).

Source	df	MS	F	P
Incentive (Incent.)	1	1120.27	.48	.494
Error	22	2317.69		
Regulation (Regul.)	1	87.31	7.10	.014
Regul. x Incent.	1	.60	.05	.828
Error	22	12.17		
Direction (Dir.)	1	80.93	6.91	.015
Dir. x Incent.	1	22.32	1.91	.181
Error	22	11.71		
Trials (Tr.)	4	345.99	31.27	.000
Tr. x Incent.	4	6.55	.59	.670
Error	88	11.06		
Regul. x Dir.	1	337.18	35.99	.000
Regul. x Dir. x Incent.	1	.14	.02	.905
Error	22	9.37		
Regul. x Tr.	4	4.93	1.44	.227
Regul. x Tr. x Incent.	4	2.97	.87	.487
Error	88	3.42		
Dir. x Tr.	4	6.49	1.07	.376
Dir. x Tr. x Incent.	4	5.87	.97	.429
Error	88	6.06		
Regul. x Dir. x Tr.	4	4.44	.95	.437
Regul. x Dir. x Tr. x Incent.	4	5.05	1.09	.369
Error	88	4.65		

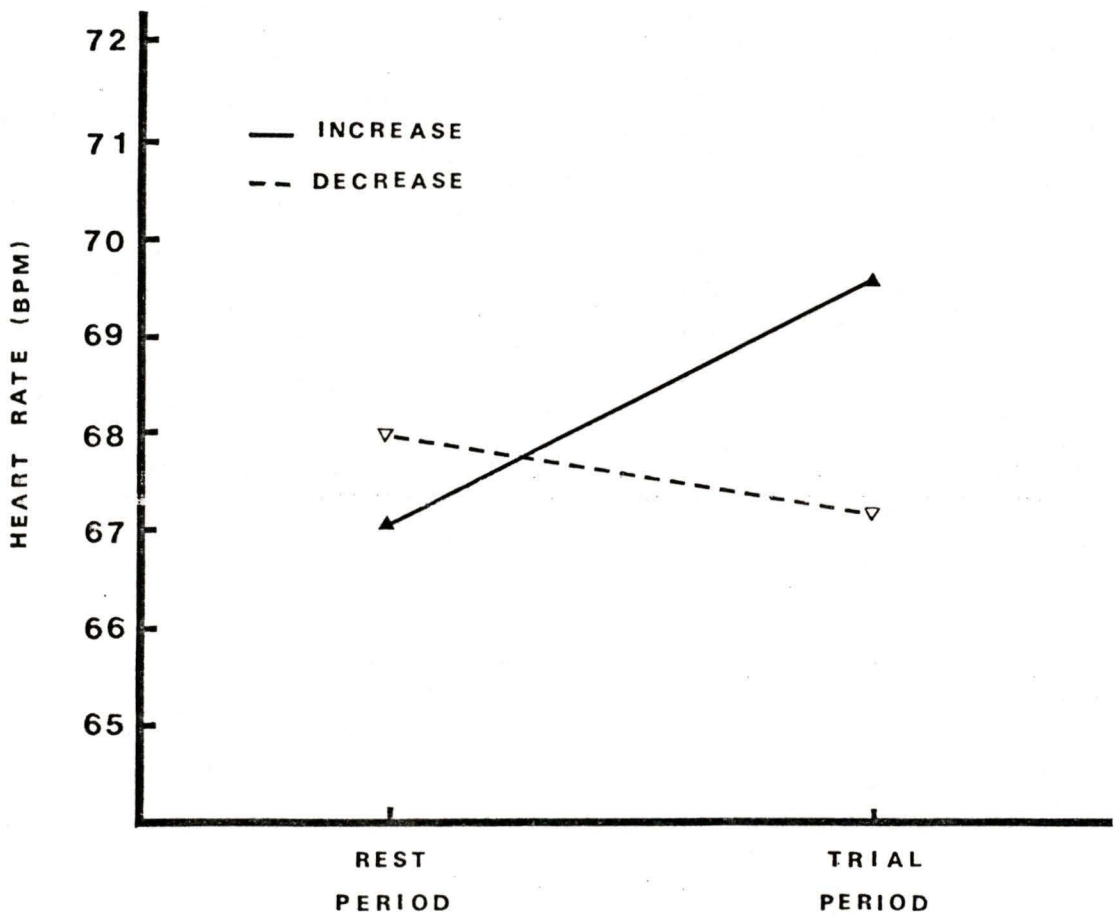

Figure 2. The regulation by direction interaction.

($F = 25.89$, $df = 1,22$; $p = .000$). Heart rate changes in the decrease direction were quite small, averaging only .82 bpm, but were found to be significant in the analysis of this simple main effect ($F = 5.98$, $df = 1,22$; $p = .023$). Thus, heart rate changes in both directions are significant. These changes are not, however, associated with a learning effect over trials (see Figure 3), as indicated by the failure of either the regulation x trials or the regulation x direction x trials interactions to achieve significance. The finding of a significant main effect of trials ($F = 31.27$, $df = 4,88$; $p = .000$) simply reflects heart rate adaptation over time (Figure 4).

Since there was an overall trend for heart rate levels to decline over trials the question arises as to whether the decrease effect is in fact an effect of regulation, or, on the other hand, simply a reflection of this same overall adaptation trend. In order to obtain a measure of adaptation the average pre-trial rest score for the last trial was subtracted from that of the first trial, yielding a value of approximately 4 bpm. This represents a drop of 4 bpm over a period of 54 minutes or about .07 bpm per minute. Thus, over a 4 minute period heart rate might be expected to drop about .28 bpm. This is clearly less than the average decrease regulation change of .82 bpm. Furthermore, examination of the change in heart rate averaged over all five decrease trials on a minute by minute basis (Figure 5), it is clear that, in fact, the entire decrease

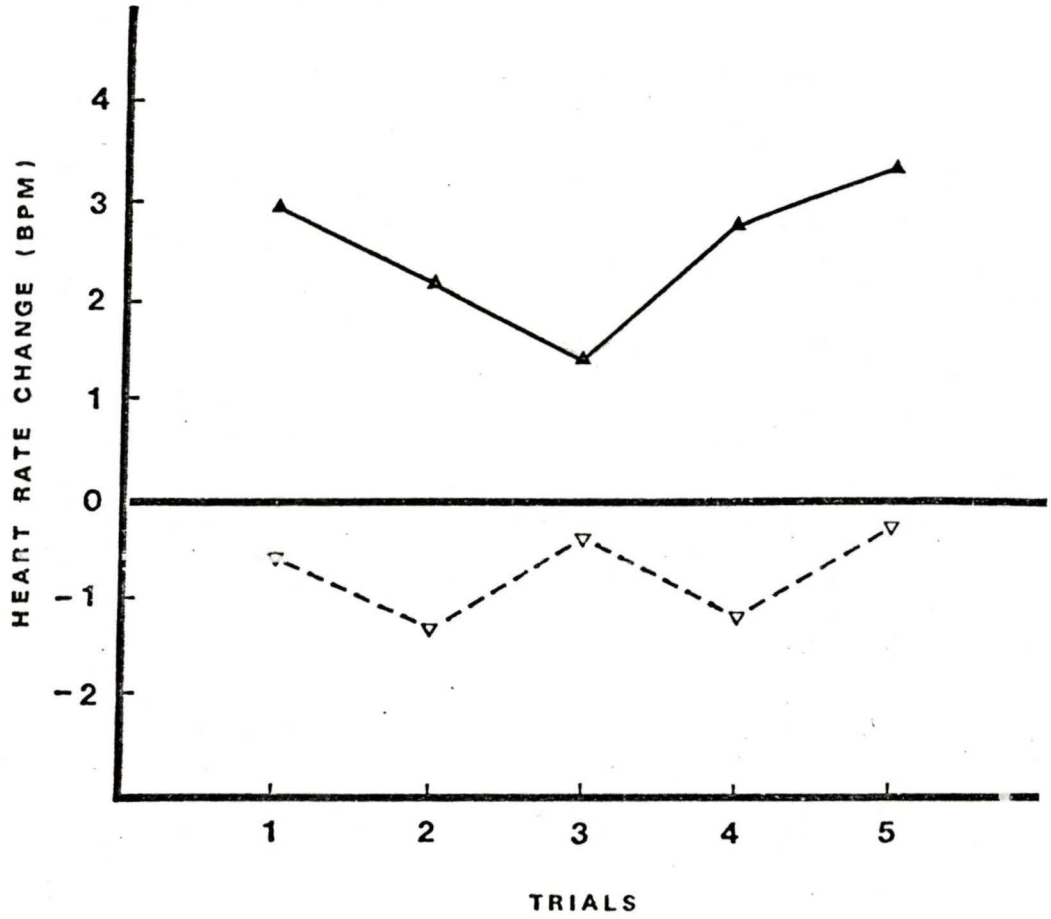

Figure 3. Average change in heart rate from pre-trial rest period to regulation period over trials.

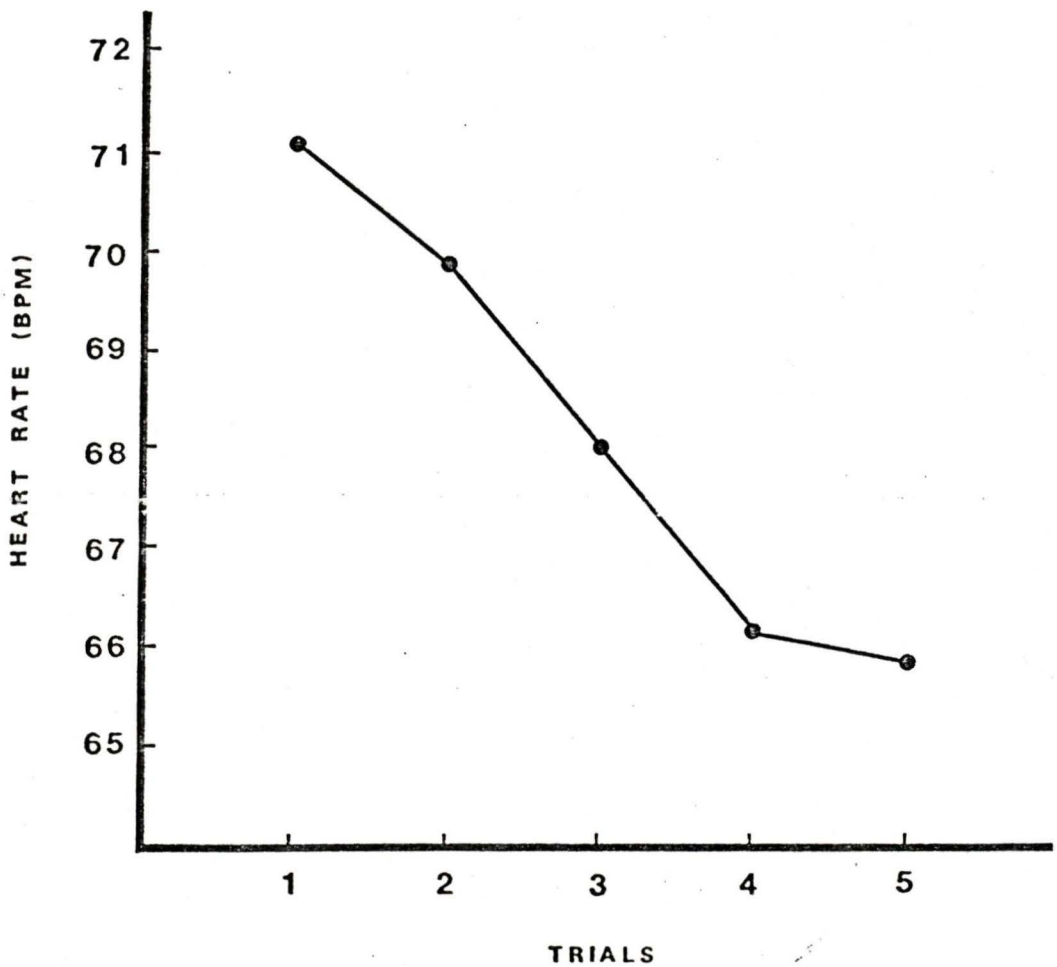

Figure 4. Heart rate over trials averaged across the direction and regulation factors.

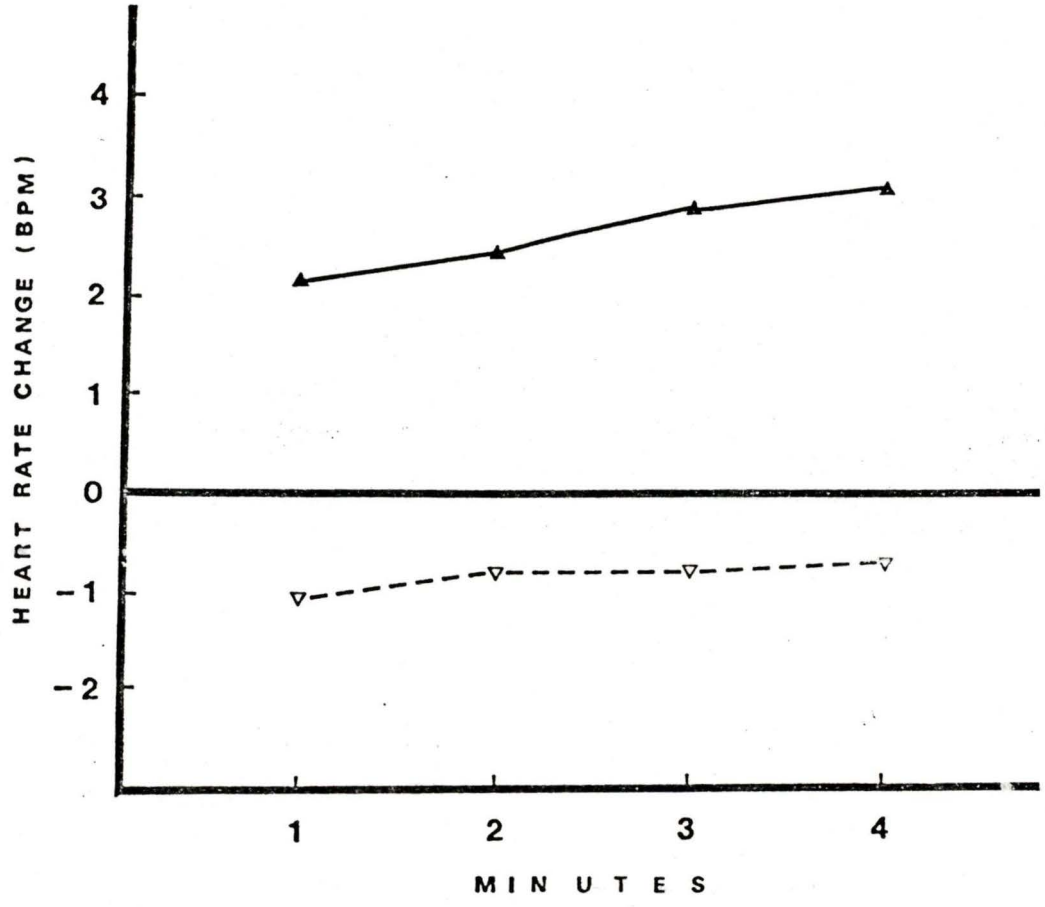

Figure 5. Average change in heart rate from pre-trial rest during each minute within regulation trials.

effect, actually of greater than 1 bpm, occurred in the first minute, with heart rate gradually increasing slightly over the remainder of the trial.

Another simple main effect analysis was performed on the pre-trial rest scores of the regulation factor indicating a significant direction effect ($F = 6.80$, $df = 1,22$; $p = .016$). The resting heart rates prior to a decrease trial were found to be an average of .86 bpm higher than those antecedent to an increase trial. The nature of this effect was explored by an examination of heart rate levels in the periods between successive trials, for each consecutive 30 second period. These data are presented in Figure 6.

The incentive condition failed to produce either a significant main effect or interaction with any other variable.

Correlational Analyses: Multiple Regression

Multiple regression analyses were conducted using the following variables:

Predictors: Respiration (reactivity) increases
Respiration (reactivity) decreases
Corrected initial level
Uncorrected initial level

Criteria: Regulation period increase performance
Regulation period decrease performance

For the purpose of the regression analyses, the heart rate regulation performance score was the heart rate change

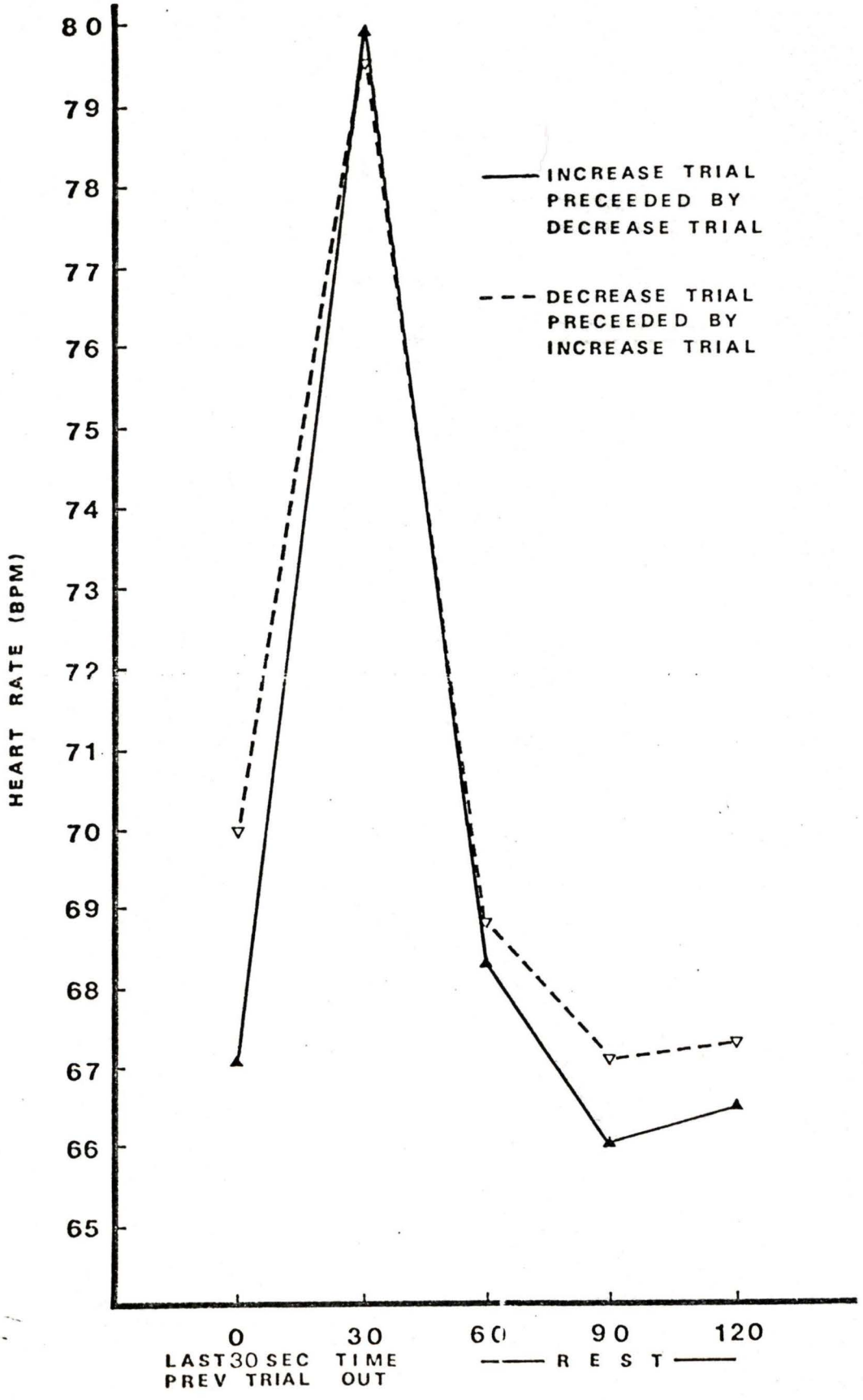

Figure 6. Average heart rate for 30 sec periods between trials.

in bpm from the final 30 sec rest to the average of the 4 min trial period for the subject's best trial in each direction. The corrected and uncorrected heart rate levels are those of the same 30 sec rest period immediately preceeding that trial.

Separate analyses using all four predictors were performed for each direction of attempted heart rate regulation. Subjects with missing data were not included in these analyses, leaving 21 subjects in each. The correlation matrices and significance tests are given in Tables 4 and 5. In neither case was there a significant relationship between the weighted combination of predictors and the criterion. Ordinarily a non-significant Regression would preclude examination of individual relationships, however, given the exploratory approach adopted here, the pertinent relationships may be pointed out.

For the increase analyses, uncorrected pre-trial period heart rate level revealed a correlation of -.42 ($p<.05$, one-tailed) with the criterion, whereas that for corrected level was only .02. Still within this analysis, the reactivity score (ie. respiration increase) also showed a negligible correlation.

In the decrease analysis, uncorrected pre-trial rest period heart rate correlated -.31 (nonsignificant) with the criterion, yet the corrected pre-trial heart rate produced a correlation of -.49 ($p<.025$, one-tailed). The correlation of the criterion with the reactivity measure

Table 4

Multiple Regression - Increase

	React. Decr.	React. Incr.	Corr. HR Level	Uncorr. HR Level	Regulation Perf.Incr.
Reactivity Decr.	1.000				
Reactivity Incr.	.123	1.000			
Correct. HR Level	.032	.021	1.000		
Uncorrect. HR Level	- .060	- .341	.282	1.000	
Regul. Perf. Incr.	- .149	.122	.024	- .417	1.000

Criterion: Regulation Performance Increase

Name	Coefficient	St. Error	Beta-Weight	T-Value	Variation
Reactivity Decr.	- .099	.121	- .181	- .820	2.229
Reactivity Incr.	- .019	.187	- .024	- .106	1.996
Correct. HR Level	.074	.103	.166	.725	.074
Uncorrect. HR Level	- .175	.089	- .482	-1.969	18.608

Source	DF	Sum. Sq.	Mean Sq.	F	P	R^2
Regression	4	60.262	15.066	1.187	.354	.229
Error	16	203.005	12.688			
Total	20	263.267				

Table 5

Multiple Regression - Decrease

	React. Decr.	React. Incr.	Corr. HR Level	Uncorr. HR Level	Regulation Perf.Incr.
Reactivity Decr.	1.000				
Reactivity Incr.	.123	1.000			
Correct. HR Level	- .271	.087	1.000		
Uncorrect. HR Level	- .236	- .228	.274	1.000	
Regul. Perf. Incr.	.390	.218	- .492	- .307	1.000

Criterion: Regulation Performance Decrease

Name	Coefficient	St. Error	Beta-Weight	T-Value	Variation
Reactivity Decr.	.089	.082	.229	1.089	15.199
Reactivity Incr.	.116	.117	.206	.996	2.934
Correct. HR Level	- .127	.064	- .423	-1.971	18.163
Uncorrect. HR Level	- .024	.057	- .090	- .419	.681

Source	DF	Sum. Sq.	Mean Sq.	F	P	R^2
Regression	4	49.392	12.348	2.35	.099	.370
Error	16	84.246	5.265			
Total	20	133.638				

(respiration decrease) was +.39 ($p<.05$, one-tailed).

Of particular interest, then, are the relationships between (a) the uncorrected level and the criterion in the increase analysis, (b) corrected level and the decrease criterion and (c) decrease reactivity and the decrease criterion.

Discussion

The findings of the present investigation will first be discussed briefly with reference to the summary of research objectives presented on p. 21. A more detailed discussion of the various aspects of the data and their implications will follow.

1. Bi-directional heart rate regulation was indicated by the ANOVA, although heart rate increases were considerably larger than decreases. Differences in pre-trial resting levels between increase and decrease trials raised some questions in regard to these conclusions. Feedback did not appear to be essential to cardiac regulation, at least not to the level of performance achieved in this study. This level of performance appears relatively small in comparison to previous literature reporting similar paradigms.
2. The incentive manipulation employed in the present study failed to enhance regulation performance.
3. The overall regression effect in both the increase and the decrease analyses failed to attain significance. As a result, extra caution had to be used in interpreting the individual correlations. Examination of these relationships revealed a significant correlation between uncorrected initial level and increase regulation and also between corrected initial level and decrease regulation.

Each of these relationships may have different implications for the LIV. A moderate, but significant correlation was also found between decrease reactivity and decrease regulation performance. This relationship may reflect the involvement of somatic processes in cardiac regulation.

Regulation Performance

Effect of Instructions

The first issue worthy of some discussion is that pertaining to the effects of instructional set in heart rate regulation. Although in the present study the distinction between feedback and no-feedback conditions reflected an experimental manipulation and an expectation of differential control performance, this distinction was not maintained in the analyses. The data provided no evidence of a specific feedback effect and all trials were therefore taken to reflect the same control phenomenon, that is, the heart rate changes during the trial periods are apparently entirely an effect of instructional set.

While in most studies the effects of feedback and instructional set are confounded, a review of the literature does reveal various types of comparisons relating to instruction effects and a discussion of the findings of the present investigation may benefit from an examination of these data.

Bergman & Johnson (1971) reported two experiments in

which subjects were given only instructions to change their heart rate during the presentation of a tone (ie. without feedback). In the first experiment, the heart rate increase group and the heart rate decrease group both demonstrated significantly altered heart rates relative to the control group which was simply told "we will be measuring the reaction of your HR to the tone (p. 263)". In the second experiment, identical to the first except that each "instructions" group was now to attempt bi-directional regulation, the decrease effect failed to replicate.

The Bergman & Johnson data may have suffered from the use of a particularly short trial period. This period was indicated by a ó sec tone and the heart rate response was scored only for the first six beats after tone onset, which may account for the relatively small magnitude of the changes (one to two beats per minute). Furthermore, the instructions to the groups were such that the tone acquired signal status for the two instructions groups since it was a stimulus to which a response was required. This was not the case for the control group. Any differences in the unconditioned heart rate responses to these two types of stimuli, either in topography or in habituation, may have contributed confounding elements.

A subsequent report (Berman & Johnson, 1972) in which only heart rate increases were attempted, indicated that subjects receiving only instructions to raise heart

rate performed significantly better than the control subjects. In this study the trial period was increased to 20 seconds and the control group differed from the previous control groups in that subjects were now told to regulate an internal response although the specific response was still not known to them.

Also included in this study was an external reinforcement condition consisting of a .5 sec light with each heart beat whenever heart rate was 10% above the baseline rate. Subjects under this condition, however, did not perform any better than those receiving only increase instructions.

Another study involving an assessment of instructional effects is reported by Johns (1970). A comparison of two groups of subjects instructed to regulate their heart rates in both directions under feedback and no-feedback conditions revealed significant decreases for the no-feedback groups and significant increases for the feedback groups. Subjects under an uninstructed condition failed to show any significant increase-decrease differences under either feedback or no-feedback. Interpretation of the data from these uninstructed groups is difficult, however, since it is not clear whether or not these subjects were, in fact, told to control the "feedback" tones.

Blanchard, Scott, Young & Edmundson (1974) in their investigation of instructional effects, report that subjects instructed to regulate their heart rate, but not given

feedback, were unable to show any significant increase-decrease differences. Another group given the same instructions, but with the addition of feedback, did show such a difference. It appears that this group produced significant changes in both directions (from rest), however no statistical tests of these were made. A further group instructed simply to regulate some internal response, and given feedback, was also able to show a significant increase-decrease difference, however this effect was due almost entirely to the heart rate increases.

Between-group comparisons revealed significantly better performances in both directions for the group given feedback and instructed to regulate their heart rate as opposed to the group given the same instructions without feedback. The former group, when compared to the group told to regulate "some internal response", was superior only for decreases. Thus it appears from this study that feedback was necessary for the production of heart rate decreases, but that subjects were able to produce significant increases even when the specific response system was unknown to them, provided they had feedback. The decreases reported by Johns (1970) under the regulate instructions, no-feedback conditions were not replicated by these authors.

The above studies have been concerned with between-groups comparisons, however a few additional investigations may be found which involved a pre-feedback, instruction-only condition similar to that of the present research.

Stephens, Harris, Brady & Shaffer (1975) have reported changes in both directions under no-feedback conditions (average increase of about 6 bpm and decrease of about 2 bpm). These changes were not tested for statistical significance and their importance may therefore be questionable, particularly in light of the considerable variability in performance across subjects evident in this study.

Further, perhaps more convincing evidence, comes from a series of studies by Bell & Schwartz (1972, 1975), however only significant increases were claimed for these. In the earlier of these two studies only increases were attempted and subjects given instructions to "control and raise" their heart rate produced increases of six to seven bpm. In their more recent investigation Bell & Schwartz again reported a significant increase (seven bpm) for subjects in their pre-feedback condition, however their subjects had no success in decreasing under this same condition. Both studies reported better regulation with the addition of feedback, including successful decrease regulation in the latter.

Finally, reference may be made to Lang & Twentyman (1974) who reported that "speeding and slowing instructions alone produced no significant differential change in heart rate from base level (p. 621)".

Thus, the literature on the effects of instructional set reveals numerous inconsistencies and seems to indicate

that under some conditions instructions alone will produce significant heart rate increases, decreases, or both. The fact that such an effect is found in the present study and has been reported elsewhere in the literature suggests strongly that subjects draw on already existing behavioral repertoires for changing heart rate, likely those involving subtle somatic changes (respiratory, muscle tension) or cognitive, affective imagery or both. This is also consistent with the observation that heart rate changes have often been demonstrated on the very first feedback trial (Bell & Schwartz, 1975).

The inconsistencies in the literature, on the other hand, suggest the need for a more precise delineation and comparison of the conditions under which these effects are found. The exact nature of the instructions to the subject, trial lengths, respiration and muscle tension controls are all of critical importance. Furthermore, particular concern must be directed to appropriate reference points against which to measure any heart rate changes, whether these be separate control groups or "rest" conditions in a within-subjects design.

Differences in Resting Levels Prior to Increase Trials vs Decrease Trials

The importance of the baseline conditions used to evaluate performance is highlighted in the present study, where pre-trial rest period, against which heart rate changes were evaluated, were found to differ significantly

between increase and decrease trials. That is, the rest levels preceeding decrease trials were on average higher than those preceeding increase trials. Because the direction of this difference would serve to facilitate appropriate changes during the subsequent heart rate trial periods, it is necessary to exercise considerable caution in interpreting the small decrease effect. This difference was not present preceeding the very first trial; it apparently developed as a result of the attempt to regulate heart rate.

Since increase and decrease trials were alternated it is possible that the rest difference reflects an anticipatory effect. Subjects might have engaged in differential preparatory activities or they might simply have responded differently to the anticipation of increases and decreases, perhaps as a result of the apparently different degree of difficulty of the two tasks. On the other hand, since the recovery from the time-out period showed a similar trend for both curves, except for the difference between both initial and final levels, it is also conceivable that the rest difference might have been due to a carry-over effect (see Figure 6). That is, the difference in pre-trial rest values might be in some way a function of the comparable difference at the end of the preceeding trial. Viewed simply as a carry-over effect, then, the data suggest that the period following an increase trial was of insufficient length to permit

full recovery from the previous tonic elevation. The phasic acceleration during the time-out period is superimposed on, but apparently independent of this slower recovery process.

These explanations are, of course, purely speculative and cannot be investigated further in the context of the present study. This type of effect should, however, be avoided in subsequent studies through randomly determined increase-decrease sequences and longer inter-trial rest periods. In this way both anticipatory and carry-over effects could be eliminated.

Magnitude of the Regulation Effects

Another aspect of the present findings deserving of some discussion relates to the magnitude of the observed effects. The heart rate changes, in addition to being produced apparently without the necessity of feedback, were relatively small, averaging less than 1 bpm for decreases and only about 2.5 bpm for increases. Other studies in the literature which are roughly comparable in terms of modality of feedback, actual time in feedback training and having fully informed subjects (Gatchel, 1974; Lang & Twentyman, 1974; Bell & Schwartz, 1975; Blanchard et al, 1974) report increases averaging between 6 and 7 bpm and decreases of about 3 bpm.

There remain, of course, several procedural and instrumentational differences between these studies cited for comparison and the present investigation. One

difference which may be particularly relevant to the reduced magnitude of the observed heart rate changes may be the degree to which somatic changes were inhibited. The typical procedure with regards to the control of somatic changes has been to provide instructions to the subject to the effect that heart rate regulation is to be attempted by "mental means only" and that subjects are to breathe normally and avoid any excessive movements and/or changes in muscle tension. Three of the above studies report the use of such instructions, yet none recorded EMG activity.

In the present investigation, EMG activity was recorded and reported to the subjects. When substantial and prolonged changes in EMG were observed on the polygraph record, this was indicated to the subject immediately after the trial in which they occurred. Thus subjects were initially given instructions to the effect that EMG changes were undesirable and were subsequently informed of any such changes throughout the session.

It is therefore proposed that this procedure led to a more effective suppression of EMG changes and thus smaller heart rate changes. Unfortunately, direct evidence of this effect cannot be obtained from the present study, since muscle activity was not experimentally manipulated. In fact, even a correlational correspondence could not be demonstrated since equipment malfunctions prevented the recording of integrated EMG. The raw EMG, while providing

a rough indication of muscle activity, is not amenable to precise quantification. It does appear, however, from a visual inspection of the available data, that in the majority of subjects, resting EMG levels were maintained. Additional confirmation of the importance of somatic activity in heart rate regulation may be found in the literature.

The issue of somatic mediation has been debated for a number of years (Katkin & Murray, 1968) and initial work seemed to indicate an independence of somatic and autonomic processes (Trowill, 1967; Miller & DiCara, 1967; DiCara & Miller, 1968; Brener & Hothersall, 1967). Considerable evidence has, however, been presented by Obrist and his associates (Obrist, Webb, Sutterer & Howard, 1970) in support of a close correspondence between cardiac and somatic activity based on a central, metabolically relevant cardiac-somatic linkage. This relationship, originally proposed on the basis of findings from classical conditioning and reaction time paradigms, is also evident in a more recent study involving operant heart rate conditioning (Obrist, Galosy, Lawler, Gaebelein, Howard & Shanks, 1975). As opposed to assessing the concomittance between heart rate and some other somatic measure(s), as is most often the case, Obrist et al directly assessed the effectiveness of the heart rate reinforcement contingency under varying conditions of somatic control. This control was effected through instructions and, in one case, instructions plus

paced respiration. Seven groups of subjects were run, five of which received reinforcement and feedback. Of these five, three groups were reinforced for heart rate increases and differed in the extent to which somatic activity was controlled: no control, minimal control and maximal control (including paced respiration). The other two groups were to produce heart rate decreases and received either maximal somatic control instructions or no control instructions. The remaining two groups were non-contingent controls, one with maximum somatic control instructions and one with minimum control instructions.

The results of this study clearly indicate that the contingency and feedback modified somatic activity (chin EMG) in a direction consistent with the direction in which heart rate was to be modified, and further, that suppression of somatic activity through instructional control resulted in markedly reduced heart rate performance. The magnitude of the base level-regulation differences in the increase groups was a direct function of somatic control, with the largest difference seen with no control instructions (17.1 bpm) and the smallest seen with maximum instructional control (6.1 bpm). In the decrease groups there were smaller base level-regulation differences with an overall deceleratory effect seen only in the maximum instructional somatic control condition. This latter group, however, was not significantly different from the maximum control, non-contingent group.

It may be noted that even the maximum somatic control

group showed average changes over the experimental session of greater magnitude than those of the present study. This difference, however, is likely attributable to the use of an avoidance paradigm which was, in fact, used by Obrist et al with the expectation that it would provide larger changes and more uniform motivational levels among the subjects. The fact that even under the maximum instructional control condition changes in the order of 6 bpm were in evidence need not necessarily imply that these changes were produced independently of somatic changes. The relatively more powerful contingencies of this study may have led to the involvement of muscles other than those of the chin. Furthermore, it was reported that despite even the paced respiration in the maximum instructional control condition, the increase group had a significantly higher incidence of increases in both respiratory frequency and amplitude than the maximum instructional control decrease group, and that the former group showed a significantly greater incidence of both increases and decreases in respiratory frequency relative to the maximum instructional control, non-contingent group.

Clearly, the restriction of somatic activity inhibits heart rate regulation performance and it seems reasonable to conclude that the more stringent somatic controls of the present study, relative to those generally found in the literature, were responsible for the relatively small

magnitude heart rate changes. Such an effect might actually have been predicted, even in the absence of the Obrist et al data. The typical instructions to breathe normally and to avoid changes in muscle tension implicitly acknowledge that large heart rate changes could be produced through the unrestricted use of somatic mediators. Further, it might follow from this that the greater the restrictions on somatic activity, the smaller the heart rate regulation effects. It should be pointed out, however, that the imposition of restrictions on somatic activity is nevertheless justifiable in that heart rate changes resultant to gross somatic changes were not of interest to the present investigation. It is by no means clear that heart rate regulation is entirely somatically mediated and therefore, those heart rate changes, albeit small ones, that might have occurred in the absence of changes in somatic activity were the intended objects of study.

Incentive Manipulation

It is to some extent surprising that the incentive manipulation had no effect on heart rate conditioning. Monetary rewards have frequently been added to the conditioning procedure since the feedback itself and/or compliance with the experimenter's instructions may not provide particularly powerful reinforcement for all subjects, as has been clearly demonstrated by the previously cited investigations of Cox & Sipprelle (1971). A more

generalized reinforcer is, therefore, thought to enhance performance, however the effect has not been systematically assessed in the context of heart rate regulation. Nevertheless, an indication is obtained from a study by Lang (1974), who reported a marked improvement in heart rate increase performance subsequent to the introduction of a condition in which small monetary rewards were made contingent upon performance.

The present failure to find a similar effect appears to be due to the characteristics of the incentive condition. This condition differs from those generally used in that the subject is rewarded not simply on the basis of his own performance, but rather on the basis of his performance relative to the other subjects of the group. Thus the effectiveness of a reinforcer would seem to be, in part, a function of the perceived probability of being reinforced, and subjects in the present study may not have considered winning the prize a likely occurrence. In fact, the spontaneous report of one subject at the end of the session indicated that he did not believe that any prize was actually being offered, but that it was simply a ploy to attract subjects to the experiment (another declaration of the fact that university students expect to be deceived in psychology experiments).

Correlational Analyses: Multiple Regression

The predictors used in the multiple regression analyses were of two types: (a) those pertaining to initial level and (b) those providing some measure of reactivity. Each will be dealt with separately in this discussion.

Initial Level

The relationship of initial level to response magnitude has been the subject of considerable investigation and is generally discussed in the context of the Law of Initial Values (Wilder, 1957). To review briefly, this "law" predicts that pre-stimulus levels in a system are inversely related to the magnitude of response to a function-raising stimulus. The opposite relationship is expected with a function-lowering stimulus.

In the present data, the tests of the overall regressions (increase and decrease) fail to support the LIV. Neither the corrected nor uncorrected pre-trial levels appear to be reliably predictive of the magnitude of the heart rate regulation effects. The failure of this weighted combination of predictors in the multiple regression equation to account for a significant proportion of the variance (R^2), according to statistical decision rules, makes any further interpretation of the observed relationships tenuous. The possibility must be considered, however, that the statistical procedure used here to evaluate the relationships may have been insensitive to their presence. That is, while initial level, in either corrected or un-

corrected form, may independently show a moderate relationship with the heart rate training effects, this relationship may have been obscured in the test of the regression equation. The inclusion, in a multiple regression analysis, of predictors which contribute very little to the regression sum of squares will (a) alter the degrees of freedom for the regression and error terms and (b) may augment the error variance. Thus, in the present case, either or both of these may have been sufficient to produce an insignificant F for the regression.

It is recognized that the appropriate procedure would now be replication and examination of only the most promising of the presently observed relationships. However, given the previously expressed exploratory intent of this investigation, and the fact that replication is not feasible at this time, some further attempts at interpretation of the present data seemed to be justified.

Examination of the correlations of the corrected and uncorrected initial level with the criterion, in both directions, reveals an interesting anomaly. For the increase direction, the uncorrected level correlated -.42 with the criterion whereas the corrected level correlation was negligible (.02). It was reported earlier that Gatchel (1974) found significant correlations of uncorrected initial level with heart rate speeding performance ($r = -.47$, $p<.01$; $r = -.52$, $p<.01$), but not with slowing performance ($r = .36$, $p<.10$). The uncorrected level correlations of the

present study appear to follow the same pattern.[4] It is interesting, however, that the corrected level correlations show the reverse trend.

The point has previously been made that the LIV refers to the properties of an individual nervous system. That is, it is most appropriately assessed within subjects. The range correction employed here serves to approximate a within-subjects condition by expressing each subject's initial level as a point within his particular range of functioning. Therefore, if the uncorrected initial level correlation, for heart rate increases, reflects processes underlying the LIV, a larger magnitude correlation should have been in evidence for the corrected initial level. If the present findings can be accepted with any degree of confidence, this appears not to be the case. The data suggest rather, that while uncorrected initial level may show a moderate and perhaps significant relationship with response magnitude, this does not reflect an LIV phenomenon.

Further to this interpretation is the expectation that an LIV relationship would be seen maximally under conditions where a subject's initial level approaches the upper or the lower limit (depending on the direction of the response) of his range (Lykken & Venables, 1971). Given that in the present case initial levels for heart rate increases were

[4] The decrease correlation reported by Gatchel is positive whereas those reported here are negative. The direction of the relationship is, however, the same in both cases. The negative sign simply reflects the expression of decrease performance as negative change scores.

all within the lower third of their respective ranges, the absence of homeostatic restraints is not surprising.

For heart rate decreases, as in the case of the increase trials, initial levels all fell within approximately the lower third of their respective estimated ranges. Under such conditions a decrease response would be expected to more readily engage homeostatic mechanisms, as indeed was found to be the case, corrected initial level showing a correlation of -.49 with decrease performance.

The uncorrected level correlation was substantially reduced for decreases. It is not known what factor or factors are responsible for the relationship between uncorrected initial level and magnitude of conditioned heart rate changes, but it does appear that they reflect processes differentially related to direction of change. This deduction must be qualified, however, by the narrow range of initial levels found under the conditions of the present study. Perhaps the uncorrected initial level correlation manifests itself under conditions where homeostatic constraints are weak, independent of the direction of the response.

Reactivity

Both increase and decrease reactivity scores were included in each of the multiple regression analyses. It is quite clear from the data that reactivity in the direction opposite to that of the conditioned heart rate

changes is of no predictive value. Of the two remaining reactivity correlations, only that between respiratory decreases and conditioned decreases is of sufficient magnitude to warrant any additional discussion. This correlation, accounting for only slightly less variance (15%) than the corrected initial level (18%), suggests that there exists a tendency for those subjects showing greater magnitude decreases under the conditions of the respiratory task to show greater heart rate decreases during conditioning.

Such a relationship may be indirectly indicative of some degree of respiratory involvement in the conditioning of heart rate decreases. The heart rate response to the respiratory task prior to regulation reflects the individual's cardiac sensitivity to respiratory influences and the correlation between the decrease reactivity scores and the decrease regulation performance implies the involvement of such respiratory influences during regulation. While respiratory changes were unfortunately not assessed in this investigation, previous studies have noted their influence on heart rate level and on heart rate variability (Sroufe, 1971; Levenson & Strupp, 1972; Levene, Engel & Pearson, 1968). Heart rate decreases tended to show a strong association with decreased rate and amplitude of respiration. It is conceivable, therefore, that subjects may have manipulated both these respiratory parameters, perhaps even engaging in more

complex manipulations of the inhalation/exhalation ratio, and possibly succeeding in producing small phasic decelerations which would result in small average decreases across the four minutes of each trial.

The fact that this respiratory reactivity measure accounts for only a small proportion of the observed decrease effect may be a result of the influence of other factors (aside from initial level) such as cognitive strategies (eg. greater attention to feedback meter or other environmental stimuli), small changes in muscle tension between the rest periods and the trial periods, differences in the degree to which respiratory manoeuvres are employed, or simply a low correspondence between the magnitude of changes produced under the gross respiratory reactivity manipulation and the much more subtle and perhaps more complex changes during conditioning. Furthermore, if it happens that subjects were indeed able to produce small, phasic, deceleratory changes, the relatively more tonic measure of conditioning performance may have served to reduce the relationship between conditioning and reactivity.

Finally, some mention might be made of the absence of this relationship for heart rate increases. This absence may be attributable to the comparatively greater influence of other factors, particularly muscle tension. A strategy involving an increase in muscle tension may readily elevate heart rate. The only way in which muscularly

mediated heart rate decreases could be produced within a trial would be through a reduction in muscle tension. However, to the extent that subjects were already relaxed during the rest period, the effectiveness of such a strategy in the production of heart rate decreases would have been considerably reduced. Respiratory changes, therefore, would have become prime candidates for the manipulation of heart rate, especially in light of their capacity to produce decelerations. In the case of the reactivity task, it is true that the respiratory manipulation resulted in a bi-phasic response, however subjects may have been able, through their own experimentation, to discover a particular respiratory pattern associated predominantly with heart rate decelerations.

Some evidence that subjects do appear to use alterations in respiratory patterns to produce heart rate decreases may be found in data reported by Blankstein (1975). The heart rate changes in this study for good and poor controllers were accompanied by respiratory changes. Increased heart rate was associated with increased respiratory rate, however respiratory changes did not differentiate between good and poor controllers. In the case of heart rate decreases, however, only the good controllers showed decreases in respiratory rate. The respiration rate of the poor controllers showed a very slight average increase.

Conclusions

In view of the rather lengthy discussion, the major findings relating to heart rate regulation and their implications are reviewed to provide an averall perspective of the present research.

Perhaps the most salient feature of the data of the present study was the evidence suggesting an apparently central role of somatic activity in heart rate regulation. This was suggested first by the fact that while significant regulation was demonstrated, no difference existed between the feedback and no-feedback conditions. Similar findings have been reported in the literature and such data strongly suggest that subjects use previously learned somatic manipulations to produce the desired heart rate changes. It seems unlikely that such regulation is achieved through a regulatory system specific to cardiac functioning since subjects are not normally able to discriminate heart rate changes and have not previously had exteroceptive feedback available to them. Somatic changes are, however, associated with readily discriminable subjective sensations (interoceptive feedback) and the influence of muscular effort or tension on heart rate is known to all.

Somatic involvement was again suggested by the relatively small magnitude of the heart rate changes of the present study. It was proposed that a more effective restriction of muscular involvement in regulation than is generally seen in the literature, was responsible for

these small magnitude heart rate changes. Some convincing data in this regard are found in the Obrist et al (1975) study from which the authors conclude that the heart rate reinforcement contingencies and feedback "effectively modified somatic activity and in a direction consistent with the direction HR was to be modified (p. 453)". Furthermore, varying degrees of supression of muscle activity resulted in corresponding reductions in magnitude of heart rate change.

A final indication of somatic involvement in the production of heart rate changes comes from the reactivity-regulation correlation for heart rate decreases. More specifically, with regards to respiration, the reactivity correlations appear to reflect a greater role for respiratory strategies during decrease regulation than during increase regulation.

Thus, under the conditions of the present investigation and, it appears from studies such as that of Obrist et al (1975), generally under the conditions of the typical feedback paradigm, somatic systems play a significant role in heart rate regulation.

Another finding of interest was that pertaining to corrected and uncorrected initial level scores, the former being related to decrease regulation and the latter to increase regulation. The possibility that these reflect fundamentally different phenomena may be deserving of further scrutiny. The corrected level correlation was

thought to reflect the influence of the LIV, however the relationship between uncorrected level and regulation was suggested to reflect processes other than the LIV and may be interesting in their own right as regards their contribution to heart rate regulation.

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Instructions

Part I

The present experiment consists of two major parts. The first part will involve a variety of simple tasks for you to perform and the second part will consist of the actual feedback training. In order to keep things as simple and clear as possible, you will be given the instructions in small segments corresponding to the various tasks you are to perform.

Before we go on, however, I would like to thank you for your participation and hope that you will give each of the subsequent tasks your undivided attention as each is of the utmost importance to the experiment.

A number of physiological responses will be recorded and the experimenter will attach three pairs of electrodes and a device for measuring respiration. One pair of electrodes will be placed on your chest to detect your heart beat, a second pair on your hand will detect changes in skin resistance, which are related to changes in sweat gland activity, and the third set will relay information concerning muscle tension. The respiration device will be attached, via a belt, around the chest.

When all the recording devices have been attached and the experimenter has had an opportunity to ensure that all signals are being recorded properly on the polygraph, you will be required to sit quietly - without moving - for a 2 minute period to allow for some baseline measurements.

This period will be indicated to you by the illumination of the REST light directly in front of you.

Further instructions will follow.

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At some time during the next minute or so, the REST light will come on briefly and be followed closely by a relatively loud tone. The tone will be very short, approximately 1 second in duration and will be presented within a few seconds of the onset of the REST light. No response is required on your part.

After this tone, the REST light will again be illuminated and will again mean that you are to sit quietly. with a minimum of movement - as you were in the previous 2 minute baseline period. In addition, you are now to concentrate particularly on ma ntaining a regular and shallow respiration pattern. It is important, however, that your breathing be only as shallow as is comfortable. After a short time the REST light will go off. This will be a signal for you to take a slow, deep breath, <u>filling your lungs to capacity</u>. Inhale as deeply and as slowly as you can through your nose and then continue breathing as before the light went off. The offset of the REST light is not a signal for you to immediately take a deep breath, but rather do it <u>when you are ready</u>. That is, only <u>after</u> you have finished your <u>normal exhalation</u>. The

offset of the REST light will simply be a signal for you to produce the inhalation within a few respiration cycles of the time that it went off.

A series of six such signals will be given about 30 - 45 seconds apart, each of these trials following exactly the same procedure. To review the sequence briefly: breathe normally and shallowly; when the REST light goes off, complete your normal exhalation and then take a slow deep breath; exhale and resume breathing normally and shallowly.

Further instructions will follow upon completion of this segment of the experiment.

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On the stool beside your chair you will find a balloon and a pair of goggles. You are to put on the goggles and then inflate the balloon until it bursts. This is to be done using regular, paced breaths, being careful not to hyperventilate. You may not stop for any length of time once you have begun. The balloon must burst, but you may be comforted to know that during preliminary testing, in no instance has the balloon struck the face of the subject upon bursting. The goggles are merely an added precaution.

Having burst the balloon, you are to remove the goggles and you will be given a 5 minute rest period.

This will again be signalled by the REST light. The subsequent offset of the REST light will indicate the end of Part I of the experiment and you will then receive the instructions for Part II.

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Part II

Part II of the experiment will be concerned with heart rate control through the use of biofeedback. Heart rate control appears to be a skill which is learned, like any other, through practice. Subjects sometimes find it a difficult skill to acquire and the changes may initially be very small. It is important, therefore, that you pay attention, not only to the magnitude of change, but also to the consistency of the change. Even small changes in heart rate, particularly if they are consistent, are valuable to your acquisition of the skill and, of course, greatest progress may be made only through constant effort.

It is important to stress, at this point, that your chances of winning the $20 prize are dependent on your performance on the feedback task. The prize will be awarded to the person showing the greatest overall control of his heart rate, so do the best you can, giving the task your full attention. You will be one of about 12 people competing for this prize.

During the remainder of the experiment, you will be required to attempt to control your heart rate. You will attempt to make it beat both faster and slower at designated times. The signal lights on the table in front of you will indicate when to INCREASE, DECREASE, or REST, and the meter directly below these lights will provide you with feedback of your heart rate. The face of the meter will be marked off in units ranging from -20 on the left to +20 on the right, with 0 in the centre. The meter will be adjusted so that deflections to the right of 0 will indicate that your heart rate has increased above your resting level, and deflections to the left of 0 will indicate that your heart rate has decreased relative to your resting level.

Heart rate control training will be provided in a series of trials, and the actual trial sequence will be as follows:

The first signal light to go on after these instructions will be a REST light. During the time this REST light is on, no feedback will be available to you and you are simply to sit quietly with an absolute minimum of muscle movement and breathe according to your normal pattern. There is no longer any requirement for you to breathe shallowly. After the REST period, either the INCREASE or DECREASE light will come on, indicating the direction in which you are to attempt to change your heart rate. You will continue to attempt to change your heart

rate in the appropriate direction until this direction light goes off. At this time you will have a short period in which to stretch, adjust your body position and make yourself comfortable. No lights will be on at this time. Following this adjustment period, the REST light will again be illuminated and you are again to sit quietly and breathe according to your normal, regular pattern. Each REST period will be followed again by a heart rate control period signalled by a direction light. This sequence - rest period, control period, adjustment period - will be repeated 10 times. During the first two heart rate control periods, however, no feedback will be presented. You are simply to attempt to demonstrate heart rate control without the benefit of the feedback. On the subsequent 8 trials the feedback meter will be activated in conjunction with the direction signal. That is, on all trials except the first two, the feedback meter and the INCREASE or DECREASE light will be activated simultaneously.

Once again, you will receive a total of 10 heart rate control trials. On the first two you will attempt to change your heart rate in the appropriate direction without the aid of the feedback. On the remaining 8 control trials, feedback will be presented. Each control period will be separated by an adjustment and rest period.

One final word of caution concerning the heart rate

control trials and the rest periods is in order. Under both of these conditions you are to sit quietly with an absolute minimum of muscle activity and you are to breathe in as regular a pattern as possible. At no time are you to attempt heart rate changes directly through changes in muscle tension or breathing. Heart rate control is to be achieved by mental means only. Muscle tension and respiration will be monitored throughout and heart rate changes resulting from muscle tension and/or respiratory changes will not be acceptable.

The instructions to the no-incentive subjects were identical to those for the incentive condition (above) except for the paragraph concerned with the $20 prize. This paragraph read as follows:

It is important to stress, at this point, that your chances of winning the $20 prize are in no way dependent upon your success on the feedback task. Of course, we want you to do the best you can, giving the task your full attention. The prize, however, will be awarded to one of about 12 participants on the basis of a draw and not on the basis of performance.

VITA

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Educational Institutions Attended, with Dates of Entering and Leaving:

UNIVERSITY OF TORONTO	1969	to	1973
UNIVERSITY OF VICTORIA, B.C.	1973	to	1976

Degrees, Diplomas, Etc., Awarded, with Dates and Names of Institutions:

B.Sc. (Honors)	1973	University of Toronto

Honors and Awards:

University of Victoria Graduate Scholarship 1973/74

University of Victoria Graduate Scholarship 1974/75

National Research Council Postgraduate Scholarship 1975/76

National Research Council Postgraduate Scholarship 1976/77

Publications:

Blankstein, K. R. & Egner, K. Effects of Trait Anxiety on Voluntary Heart Rate Control and Subjective Tenseness. Psychophysiology, 1974, 11, 241-242 (Abstract).

Blankstein, K. R., Zimmerman, J., & Egner, K. Within-Subjects Control Designs and Voluntary Bidirectional Control of Cardiac Rate: Methodological Comparison Between Pre-experimental and Pre-trial

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Title of Thesis/Dissertation

Individual Differences in Heart Rate Regulation:
An Exploratory Investigation.

Author _____

Karl Egner

June 3, 1976