EFFECTS OF CARDIAC DISEASE ON ACQUISITION OF HEART RATE CONTROL

A Dissertation

Presented to

the Faculty of the Department of Psychology

University of Houston

In Partial Fullfilment

of the Requirements for the Degree

Doctor of Philosophy

By

Edward P. Friedman

Fall, 1978

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ABSTRACT

The majority of studies of voluntary self-control of heart rate have been conducted using college students as subjects. These studies have shown that self-control of heart rate is possible; that the extent of control is augmented by the provision of feedback; and that cardiac acceleration is more easily acquired than cardiac deceleration.

Few researchers have studied the ability of persons other than young, healthy college students to alter their cardiac rate. Engel and his colleagues have utilized biofeedback training with patients suffering from cardiac disease, and have found that some were able to achieve a degree of cardiac control after a large number of training sessions. However, only Lang and his colleagues have systematically compared the response to brief cardiac biofeedback training of healthy young persons, healthy older persons, and older persons with heart disease.

Lang found a hierarchical ordering of these groups in terms of their ability to decelerate their heart rate, with the healthy young subjects doing the best and the subjects with ischemic heart disease doing the poorest. The hierarchical ordering was not found for heart rate acceleration, however; both the older subjects with heart disease, and the healthy older subjects, proved unable to accelerate their cardiac rate significantly.

The present study extended Lang's research by making a number of refinements in subject selection to achieve greater homogeneity of the patient group. Whereas Lang's patient group had in common only a history of ischemic heart disease, two heart disease groups were compared in the present research: patients who had suffered a first, uncomplicated myocardial infarction within the past month; and patients who had had one or more infarctions in the past, the most recent being no less than three months previously. No patients in the recent infarction group were taking cardiotropic drugs, and no patients in either the recent or past infarction group had had cardiac surgery. A control group of persons without known or detectable cardiac disease, matched in age to the two heart disease groups, was also studied.

All subjects were given three brief biofeedback training sessions, one in cardiac acceleration and two in cardiac deceleration. Feedback was provided by a cardiotachometer, which reflected changes in heart rate on a beat-by-beat basis. The sessions included periods during which subjects were instructed to increase or decrease their heart rate without the feedback device being activated, as well as periods during which continuous feedback was provided.

The results indicated that none of the three groups accelerated their heart rate significantly, either with or without feedback. This

is consistent with Lang's findings, and may reflect the effects of increasing fibrosis of the heart with age. Significant heart rate deceleration was achieved by the control group, however, and the past infarction group's deceleration closely approached significance. The amount of deceleration was significantly greater when feedback was provided than when it was not. The recent infarction group was not able to decelerate their heart rate significantly, in spite of being just as responsive as the past infarction group to cardiac deceleration during an orienting task.

Personality data indicated that anxiety was related to the ability to decelerate cardiac rate and that the control group was significantly less anxious than either of the two infarction groups. An explanation based upon the different effects of stress-aroused catecholamines on the scarred myocardium of the convalesced infarction patient and the irritable myocardium of the acute infarction patient was suggested; and possible motivational differences between these two groups were discussed.

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CHAPTER I

INTRODUCTION

Until slightly more than a decade ago it was generally assumed that autonomic functions such as heart rate were beyond the voluntary control of the individual. In recent years, however, it has been demonstrated that with effective training techniques, some degree of voluntary regulation of heart rate is indeed possible (Blanchard and Young, 1973; Engel, 1972). When supplied with information about their own heart rate, subjects have been taught to decrease, increase, and alternately decrease and increase their heart rate in the laboratory (Blanchard and Young, 1972; Brener and Hothersall, 1966; Engel and Hansen, 1966; Engel and Chism, 1967; Headrick, Feather and Wells, 1971; Levene, Engel and Pearson, 1968). Moreover, once having learned to regulate their heart rate with the aid of feedback, subjects are able to transfer this learning to non-feedback laboratory conditions (Bell and Schwartz, 1975). Despite speculation that these changes in heart rate are brought about by alterations in breathing or muscular activity, there is evidence that voluntary control of heart rate is possible in the absence of respiratory or somatic mediation (Manuck, 1976). Subjects in these studies for the most part have been young (usually of college age) and free of any history or symptoms of cardiac disease. These findings have both

necessitated a major change in thinking about the capacity of the individual to control and regulate his own autonomic processes, and initiated further research into the potential clinical applications of biofeedback training, i.e., the presentation to an individual of information (in visual or auditory form) about the ongoing activity of a physiological function, in order to help him bring it under his voluntary control.

Two questions have arisen with regard to the clinical applicability of cardiac biofeedback training: can persons with compromised or damaged hearts learn to control their cardiac functioning in the same way that young, healthy persons can; and will such control, if attainable, make a significant clinical difference to persons with cardiac disease? A thorough review of the literature in this area yields inconclusive results. Some researchers have demonstrated, for example, that patients with chronic atrial fibrillation can learn to control ventricular heart rate (Bleecker and Engel, 1973a); that patients with chronic tachycardia can achieve substantial reductions in heart rate (Scott, Blanchard, Edmunson and Young, 1973); and that training in cardiac control can be useful in reducing the frequency of premature ventricular contractions (Weiss and Engel, 1971) and of abnormally conducted heart beats, as in Wolff-Parkinson-White syndrome (Bleecker and Engel, 1973b). However, other studies

indicate that persons with ischemic heart disease are significantly less capable of learning heart rate control than persons of similar age who are free of cardiac disease (Lang, Troyer, Twentyman and Gatchel, 1975); and that patients with complete heart block, in which conduction between atria and ventricles is interrupted, are unable to learn consistent control of ventricular heart rate (Weiss and Engel, 1975).

Unfortunately, many of these studies suffer from significant methodological shortcomings such as absence or non-comparability of control groups, lack of homogeneity of patient groups, and failure to fully utilize those aspects of biofeedback training which have been found to be most facilitative of learned heart rate control (Blanchard, Scott, Young and Edmondson, 1974; Gatchel, 1974; Lang and Twentyman, 1974). The procedures and length of training utilized by different researchers have also varied widely. The purpose of the present research was to determine whether patients who have had myocardial infarctions could learn, with the aid of biofeedback, to control their own heart rate; and whether such control, if it could be learned, could be transferred to conditions where biofeedback was absent. These questions have direct clinical significance. One possible result of a myocardial infarction is the presence of a cardiac arrhythmia, such as tachycardia or premature ventricular contractions. If persons

who have suffered myocardial infarctions can be trained in heart rate control, it is possible that they will be able to reduce the frequency and/or the severity of these arrhythmias, and perhaps to decrease their dependence upon anti-arrhythmic medication. The accomplishment of either or both of these objectives would represent an important clinical contribution. Demonstration, in the laboratory, of the cardiac trainability of persons who have had myocardial infarctions is a necessary first step in investigating the application of cardiac biofeedback techniques to this clinical population.

A recent study has indicated that patients with ischemic heart disease are less capable of learning voluntary heart rate control than healthy control subjects matched to the patients on age (Lang, Troyer, Twentyman and Gatchel, 1975). However, this study did not make clear how many of the patients had suffered previous myocardial infarctions, how long before the study the infarctions occurred, whether those patients with a history of infarction performed differently from those who had ischemic heart disease but no history of documented infarction, and whether there were any differences in performance between patients who had and who had not undergone cardiac (i.e., open heart) surgery. In contrast, the subjects whose learning of voluntary heart rate control (cardiac acceleration and deceleration) in response to brief, intensive biofeedback training

were compared in the present study were: (1) patients who were currently recovering from a first myocardial infarction suffered no more than one month previously (i.e., who had passed the critical phase of the infarction, were no longer in the Coronary Care Unit, and were allowed to leave their ward for the study); (2) persons matched in age to the heart patients, who had no history or symptoms of heart disease; and (3) patients who had suffered a myocardial infarction a minimum of three months prior to the study. As additional experimental controls, the first (recent infarction) group was limited to those patients who were not taking any cardiotropic medications with a direct effect on heart rate; and both cardiac groups were limited to persons who had never had cardiac surgery.

The principal hypothesis which the present study tested was that patients who had suffered a recent myocardial infarction (i.e., who have sustained recent damage to the myocardium, or heart muscle, along which travel the electrical impulses which originate in the internal pacemakers of the heart and which cause it to contract) would be less able to learn voluntary control of heart rate than would both patients who had had infarctions in the past from which they had already convalesced, and persons of similar age who were free of known cardiac disease. In addition, the inclusion of several different conditions in the design of the study (to be described in a subsequent

chapter) made possible the testing of implicit exploratory hypotheses regarding the importance of feedback (compared to non-feedback) conditions for self-control of heart rate, and the relative ease of acquiring control in one direction (deceleration, or slowing) versus the other (acceleration, or speeding). Before describing in detail the methods by which the subjects for this study were selected and the procedures with which they were trained, however, a thorough review of previous research efforts and findings pertinent to the present study is in order. Following this, the methodology of the present research will be explained in detail.

CHAPTER II

REVIEW OF THE LITERATURE

Experimental Studies

An early attempt to use operant conditioning to control heart rate (HR) in humans was reported by Engel and Hansen (1966). These researchers utilized ten male college students as experimental subjects (Ss) and five male college students as control Ss. The Ss were told that the experiment involved conditioning, but were not informed that HR was the response being reinforced. Reinforcement was provided by a light which came on whenever a S's HR fell below the operant level and by a clock which accumulated the amount of time that the correct response was emitted, and which served as the basis for paying the Ss at the rate of one-half cent per second correct. The Ss were tested for six one-hour-sessions, with a higher rate of reinforcement being provided for the first two sessions in order to shape the desired response. Learning was assessed during the last four sessions, when an operant level HR was selected which would keep a S's light on 50 per cent of the time. Each session was divided into a 30-minute adaptation period, a five-minute period to establish the operant level HR, and a 25-minute training period. The experimental Ss were reinforced for slowing their HR on a beat-by-beat basis, while the control Ss were yoked to the experimental Ss (i.e.,

their reinforcement was based upon the performance of paired experimental Ss). Respiration strain gauges were used to measure changes in breathing, in order to assess whether any HR changes found were mediated through respiratory changes.

Five of the 10 experimental Ss showed evidence of learning to slow HR. There was no indication that the observed changes in HR were mediated through changes in breathing rate. Interestingly, all of the four experimental Ss who guessed that decreased HR was the correct response were non-learners. The yoked control Ss performed more poorly than either the successful or unsuccessful experimental Ss, although they reported using the same techniques as did the experimental Ss. Four of the five control Ss correctly guessed the correct response, but none guessed that response and reinforcement were dissociated. The researchers concluded that some normal Ss could be taught operantly to slow HR, but it was not made clear why only one-half of the experimental Ss met their criteria of learning, although they speculated that knowledge of the correct response may have interfered with learning.

The same procedure was also used, with similar (male, college student) Ss, to condition HR speeding by use of an operant paradigm (Engel and Chism, 1967). All 10 of the experimental Ss learned to increase HR, as did two of the five control Ss. Four of the five

experimental Ss (who were yoked to control Ss) showed significantly greater increases than the control Ss to whom they were yoked. In contrast to the earlier study of HR slowing (Engel and Hansen, 1966), in which the experimental Ss improved their performance from the beginning to the end of the training periods, neither the experimental nor the control Ss in this study showed improvement in their performance during the sessions, a finding which the researchers took as evidence that HR speeding was more easily learned than HR slowing. As in the HR slowing study, changes in breathing did not appear to mediate the observed changes in HR, either among the experimental Ss or among those control Ss who learned to speed HR.

A paradigm in which Ss were taught to alternately increase and decrease HR was employed by Levene, Engel and Pearson (1968), using five female college students as Ss. No control Ss were employed. Lights were used as discriminative cues for either increasing or decreasing HR and as an immediate reinforcer for the correct response. The Ss were not informed that HR control was the desired response. An accumulated-time-correct clock was employed, and the Ss were paid at the rate of one-fourth cent per second correct. Each session lasted about two hours, and the Ss received from six to 10 sessions, depending upon their speed of learning. On the first several days of training, slowing and speeding were trained separately (with a break

between the two parts of the experiment) until the Ss were able to keep the reward light on significantly more than 50 per cent of the time. Following this, the cue lights alternated automatically at one-minute intervals (with one 30-minute rest period) for one hour. Separate clocks accumulated time correct for the two tasks during the alternating sessions.

The results of this study showed that, consistent with what had previously been demonstrated (Engel and Chism, 1967), slowing was more difficult to learn than speeding, relative to resting HR levels. The researchers pointed out that the greater ease of acquisition of the speeding response might have resulted from speeding being an easier response to emit than slowing, or from the possibility that many Ss' reactions to the experimental situation included increased HR as part of an autonomic reaction pattern. Both the one S who correctly guessed that HR was being conditioned, and another who thought that blood flow was the desired response, had difficulty in learning HR slowing. Although several Ss showed changes in breathing between the slowing and speeding trials, they were not aware of these changes; and attempts to duplicate the HR changes by pacing the respiration rates of these Ss were not successful. Only two Ss were able to both increase and decrease HR consistently and significantly relative to their resting HR.

Other researchers have trained Ss to increase and decrease HR alternately, with results similar to those of Levene, Engel and Pearson (1968). Brener and Hothersall (1966), in a single-session design without any control Ss, found that their Ss were able to raise their HR above baseline on 85 per cent of beats, but were able to lower their HR's below baseline on only 55 per cent of beats. Brener and Hothersall did not inform the Ss of the desired response, and used a binary auditory type of feedback in contrast to the binary visual feedback used by Engel and his associates. Headrick, Feather and Wells (1971), using proportional visual feedback with uninformed Ss and a single session only, achieved modest increases, but no decreases, in HR. However, using informed Ss, proportional visual feedback, and twelve sessions, major HR increases and modest HR decreases were achieved. Finally, Blanchard and Young (1972), using proportional auditory and visual feedback with informed Ss in two separate (increase and decrease) sessions, found that their experimental Ss learned to increase HR more beats per minute (BPM) than they learned to decrease HR, and that the differences between the experimental and the control Ss (who received no feedback) were greater during the speeding than the slowing sessions.

While the Ss in all of the studies referred to above were instructed to remain as still as possible in order to avoid somatic mediation,

the possibility cannot be entirely discounted that subtle somatic changes (i.e., muscle activity not observable by the researchers) were responsible for the changes in HR which were found. Manuck's (1976) research addressed itself to precisely this issue; 15 college student subjects (eight females and seven males) were assigned to each of four conditions: paced respiration rate; electromyogram (EMG) feedback (to maintain constant muscle activity); respiration rate pacing and EMG feedback; and no somatic (i.e., respiration rate or EMG) controls. The Ss were given comparable opportunities to practice maintaining a constant somatic state prior to HR training, and were told that they would be paid 25 cents for each trial in which they successfully changed their HR without concomitant changes in respiration rate (RR) or muscle activity. Beat-by-beat feedback for HR control was provided by lighted numerals "2" through "8" (with "5" being equal to a S's baseline HR). Each S received 10 60-second acceleration and 10 60-second deceleration trials; these were randomly distributed and each was followed by a 60-second rest period. The Ss were not told which trials were rewarded until the entire session had been completed.

Manuck found that the Ss were able to produce changes in their HR in the direction of acceleration (the mean HR increase was 3.40 beats per minute, or BPM) but not in the direction of deceleration

(the mean HR decrease was only .30 BPM). Instructions not to alter RR or move about, and some motivation to follow these instructions (in the form of a monetary reward), were not sufficient to eliminate respiration changes, although EMG changes were adequately controlled by the Ss in the absence of EMG feedback during the acceleration trials. Increases in HR were maintained by Ss when RR was controlled by a pacing procedure, however, thus demonstrating that HR increases could be obtained in the absence of, and without mediation through, changes in respiratory or muscle activity. Since the Ss in this study were unable to produce significant decreases in HR, the feasability of decelerating HR without somatic mediation could not be assessed.

Stephens, Harris, Brady and Shaffer (1975) were able to demonstrate HR increases of as much as 46 BPM and HR decreases of as much as 14 BPM, with a mean increase of 11 BPM and a mean decrease of three BPM. The Ss were 11 females and 29 males, all of whom were either students or hospital personnel ranging in age from 19 to 33 years. Sessions were held weekly for five consecutive weeks, and lasted for 95 minutes. Two kinds of proportional feedback were provided: visual (a meter showing HR fluctuations) and auditory (a varying tone). In addition, binary feedback was provided during the last three sessions, in the form of a contingency light which also determined the Ss' rate of pay (one-half cent for each second that the contingency light was illuminated).

In addition to reporting large magnitude HR changes (average increases of 10 BPM and average decreases of four BPM were produced by 18 of the 40 Ss), Stephens and his co-researchers found that contrary to the opinions of other investigators (Engel, 1972; Bell and Schwartz, 1975) to the effect that HR speeding and slowing are different tasks controlled by different mechanisms, ability to raise and lower HR were positively correlated in their sample. They also found that those Ss with greater HR variability during baseline, rest, and post-trial periods also showed greater ability to both increase and decrease HR, and that HR increases during training periods were accompanied by increases in both systolic and diastolic blood pressure (BP). Neither rate nor amplitude of respiration appeared to be associated with changes in HR during trial periods.

Bell and Schwartz (1975), using 20 male college student Ss, and providing feedback by means of a meter which gave the Ss information about their HR on a beat-by-beat basis, added pre- and post-feedback voluntary control periods (i.e., instruction to raise or lower HR without feedback) to their single-session design. They also had the Ss take their own pulse for two consecutive days after the experiment, at a variety of times and in a variety of situations (e.g., while resting, before and after exercising, before and after performing a mental task, and before and after imagining an argument). They

found that the Ss were able to raise but not to lower their HR before feedback; that with feedback, the Ss were able to both raise and lower HR; and that the ability to raise and lower HR was maintained during the post-training transfer period. Decelerative changes were smaller than accelerative changes, a finding which Bell and Schwartz attributed to the fact that resting HR's in the laboratory were close to the low end of the Ss' HR range during a typical two-day period. No correlation was found to exist between magnitude of HR increases and magnitude of HR decreases.

One further finding of the Bell and Schwartz study which is of importance to single-session experiments is that the Ss failed to demonstrate improvement in their performance over the course of the feedback trials; feedback was not necessary for the Ss to raise their HR, and only minimum feedback was necessary for the Ss to lower their HR. Exposure to feedback as an aid in selecting an already learned but untested behavioral strategy for HR change thus appeared to be more important than more lengthy practice, at least for Ss who were fully informed about the response to be reinforced during the experiment.

It is apparent from the studies reviewed thus far that a number of methodological differences characterize research in this area. Among the more important of these differences are whether or not

the Ss are aware that HR is the response being conditioned; the nature of the feedback which the Ss receive (binary versus analogue, i.e., proportional); and the frequency with which the feedback is provided to the Ss (after each beat versus after a specified number of beats).

In several of the early studies with human Ss, the Ss were told only that the experiment involved conditioning, and were not told what response was being conditioned (Engel and Hansen, 1966; Engel and Chism, 1967; Brener and Hothersall, 1966). In a post hoc analysis of their data, Engel and Hansen (1966) suggested that knowledge of the correct response may have actually impeded learning, since those Ss who did not guess correctly that HR was being conditioned showed greater HR slowing than those Ss who did make the correct inference, i.e., that the desired response was a change in HR. Blanchard, Scott, Young and Edmundson (1974), using 64 Ss (32 male and 32 female, ages 16 to 45 years) whom they recruited from the students and staff of a medical center and two colleges, tested Engel and Hansen's hypothesis by assigning the Ss to one of four conditions: Informed-Feedback; Informed-No Feedback; Uninformed; and Misinformed (i.e., told that skin resistance was the response being conditioned). In order to make within- as well as between-group comparisons, all Ss were trained for two sessions with both raising and lowering trials in each session, so that some of the Uninformed

and Misinformed Ss could be correctly informed before the second session.

Between-group comparisons generally indicated that those Ss who were correctly informed that HR was the response being conditioned, and who received feedback of their own HR, were better able to lower their HR than were either the Uninformed or Misinformed Ss or the correctly informed Ss who did not receive feedback. Being correctly informed and receiving feedback also facilitated learning to raise HR, although not as consistently as it facilitated lowering. Within-group comparisons (Ss who were Uninformed or Misinformed in Session I and then correctly informed in Session II) also showed a trend for correct knowledge of the response to facilitate control of HR. These results strongly supported the contention that correctly informing the Ss of the response to be controlled and providing them with information (i.e., feedback) as they attempt to control this response are advantageous in modifying HR.

The nature of the feedback which the Ss receive is another dimension which differentiates cardiac conditioning studies. Two basic kinds of information can be provided: binary feedback (which informs a S only whether he is or is not keeping his HR above or below a criterion level) and analogue, or proportional, feedback (which provides a S with information about his degree of success or failure,

i.e., how far above or below the criterion level his own HR is). Lang and Twentyman (1974) compared these two types of feedback with a sample of 22 male college students and found that, although there were no apparent differences between the binary and analogue procedures insofar as training in HR slowing was concerned, analogue feedback was more effective in training the Ss to speed their HR. In addition to demonstrating the superiority of analogue feedback in training for HR acceleration, these researchers also found that HR slowing was learned more slowly than HR speeding; that the absolute change in HR was smaller on slowing than on speeding trials; and that slowing showed less evidence of transfer to no-feedback conditions, and greater evidence of rebound during rest periods, than did speeding. In addition to demonstrating that HR acceleration training was optimized by the use of analogue information, these results also indicated that acceleration and deceleration of HR may be controlled by different autonomic mechanisms.

Another study suggesting that the optimal type of information for facilitating HR control depends upon whether HR speeding or slowing is being trained was made by Bouchard and Corson (1976). They compared the effects of success and failure signals on the performance of 32 male college student Ss who attempted either HR speeding or HR slowing during a single experimental session. In addition to

receiving training in either slowing or speeding, Ss received either positive or negative feedback, i.e., were signalled either for their successes or for their failures (to control HR at the criterion level). The results indicated that those Ss who were signalled for their successes performed better on HR speeding trials than did those who were signalled for their failures; whereas the Ss who received failure signals were better able to slow their HR than the Ss who received success signals. In other words, HR acceleration training was facilitated by informing the Ss of their successes, and deceleration was facilitated by informing the Ss of their failures. In addition to suggesting a compatibility between the type of signal and the task being trained, this study supported the contention of Lang and Twentyman (1974) and of other researchers as well, that voluntarily increasing and decreasing one's HR are two biologically different tasks; and that, of the two, HR speeding is the easier to accomplish, in part because most Ss' resting HR is closer to the lower than the upper limit of the potential range of HR.

One further dimension along which studies of cardiac biofeedback differ is the frequency of the feedback, i.e., whether the Ss are provided with information about their HR after every beat or after a certain number of beats. Gatchel (1974) assigned 40 male college student Ss to three frequency-of-feedback conditions: after each

beat (i.e., continuous); after every five beats (approximating the length of a respiratory cycle); and after every 10 beats. All feedback was presented visually and was in analogue form. In addition to evaluating the effect of feedback frequency on the acquisition of HR speeding and slowing skills, Gatchel evaluated the relationship between frequency of feedback and transfer of training to no-feedback conditions. The Ss receiving feedback training were also compared with a control group who performed a visual tracking task while their HR was monitored. Both in speeding and in slowing sessions, all the Ss who received feedback performed better than the control Ss, supporting a general feedback effect. The effects of varying frequencies of feedback, however, differed from speeding to slowing sessions. Training in HR acceleration was facilitated by more frequent feedback, with the group receiving beat-by-beat feedback being clearly superior to those Ss who received feedback after every five beats, and the Ss receiving feedback after every 10 beats showing the poorest performance.

The effects of receiving different frequencies of feedback also persisted during the transfer-of-training periods following training in HR speeding. All feedback groups (continuous, five-beat, and ten-beat) demonstrated a significant improvement in the ability to accelerate HR over successive sessions of training in HR speeding.

During slowing sessions, however, there were no significant differences between those Ss who received feedback after each beat, after every five beats, and after every 10 beats. Furthermore, unlike HR acceleration, there was no improvement in the ability to slow HR with additional training sessions; and slowing was not maintained during transfer-of-training periods. The correlation between acceleration and deceleration was not significant ($r = -.11$), nor was that between HR variability during the initial rest periods and subsequent slowing performance ($r = .17$). However, there was a significant relationship between variability of HR during the initial rest period and average speeding performance with feedback ($r = .48$ in one experiment and .40 in another), suggesting that greater cardiac lability was associated with better performance on HR acceleration tasks.

Gatchel's findings support the contentions of other researchers (Engel, 1972; Bell and Schwartz, 1975; Lang and Twentyman, 1974) that voluntary speeding and slowing of HR are mediated by different mechanisms. At least until more is learned about the mechanisms involved, it may be best to analyze HR speeding and HR slowing as separate skills, with deceleration possibly being responsive to different kinds of feedback than acceleration (Bouchard and Corson, 1976), and less responsive than acceleration to the amount of infor-

mation provided by the feedback (Lang and Twentyman, 1974) and by its frequency (Gatchel, 1974).

Clinical Studies

One characteristic common to all of the studies referred to so far is that the Ss were uniformly young, healthy persons, frequently drawn from college student populations, and always free of any indications of cardiac disease. Studies of operant conditioning of older Ss, and of Ss with histories of heart disease, are fewer in number. Weiss and Engel (1971) carried out one of the first studies of HR conditioning in clinically ill Ss in order to determine whether learned HR control could facilitate the reduction of cardiac arrhythmias among patients with premature ventricular contractions (PVC's).

The Ss in this study were eight patients with PVC's, six males and two females. Their age ranged from 36 to 77 years. Four had suffered one or more myocardial infarctions at some time in the past. All the Ss were admitted to a hospital for the duration of the study, so that in addition to HR training in the laboratory, their electrocardiograms (EKG's) could be monitored at various times while they were on the ward by means of telemetry.

The training procedure in this study varied somewhat from patient to patient. The apparatus was the same for all patients: red and green lights which signalled the patients to slow or to speed

their HR; a yellow light which signalled correct responses on a continuous (beat-by-beat) basis; and a meter which accumulated time correct. The Ss were first trained to speed their HR; then to slow their HR; then to alternate speeding and slowing within a single session; and finally to maintain their HR within a certain range. During the last (range-maintaining) contingency, the feedback was gradually phased out, from an On:Off ratio of 1:1 to an On:Off ratio of 1:7. All the sessions were approximately 80 minutes in length. Depending upon how fast each S learned, the number of sessions per S varied from 22 to 71. Three Ss were also studied with intravenous administration of several autonomically active drugs after HR conditioning had been completed. Since the goal of the study was to see if the frequency of patients' PVC's could be reduced by utilizing HR control (speeding, slowing, and maintaining HR within a certain range), all the Ss were fully informed and involved throughout the research.

Five of the eight Ss decreased the frequency of their PVC's in association with HR control in the laboratory, four of them by voluntarily slowing their HR and one by voluntary HR speeding. Four of these five patients also showed decreased frequency of PVC's on the ward, where their EKG's were monitored by telemetry and four continued to have a lower frequency of PVC's at home following

the conclusion of the study, the longest followup being 21 months. The drug studies carried out with three of the Ss showed that decreased PVC frequency was associated with diminished sympathetic tone in one S and with increased vagal tone in another. One S, who successfully learned HR (and PVC) control and was able to transfer this control to his home, had no myocardial infarction during 21 months of followup, although he had had three infarctions in the 11 months preceding the study.

Bleecker and Engel (1973a) used the same procedure as Weiss and Engel (1971) in studying HR control among patients with chronic atrial fibrillation in order to determine whether patients with atrial fibrillation could learn to control their ventricular rate. The Ss were six patients (three males and three females) with atrial fibrillation and histories of rheumatic valvular heart disease, three of whom had undergone cardiac surgery. Their ages ranged from 28 to 62 years. The six patients were hospitalized for three weeks for the study, receiving from 40 to 69 sessions of training in speeding, slowing, alternately speeding and slowing, and maintaining HR within a given range (see Weiss and Engel, 1971). Five Ss also received cardiotropic drugs intravenously after HR control had been established.

The results of this study indicate that all of the Ss were able to control their HR differentially, i.e., to alternately speed and slow

their HR during a single training session. Two of the six Ss learned speeding more consistently than slowing; two learned slowing more consistently than speeding; and two were able to both speed and slow reliably. None of the drugs which were administered abolished the Ss' ability to voluntarily modify their HR, except for atropine, which abolished differential HR control in four Ss and reduced it in one other S. Two of the Ss, who demonstrated highly variable HRs, were given extensive training in an attempt to decrease their HR variability. Although both of these Ss showed significant reductions in HR variability during training sessions, neither demonstrated any reduction in day-to-day variability. In spite of the differences among the six Ss, the data indicated that "patients with AF (atrial fibrillation) can be operantly conditioned to modify VR (ventricular rate) differentially [Bleecker and Engel, 1973a, p. 170]." This study, together with the findings of a study of patients with third-degree heart block (which will be summarized shortly), suggested that the neural mediation of voluntary VR control by patients with chronic AF occurs at the level of the A-V (atrioventricular) node (Bleecker and Engel, 1973a; Weiss and Engel, 1975).

Bleecker and Engel (1973b) also reported an experiment in cardiac conditioning of a 29-year-old woman with Wolff-Parkinson-White syndrome (a disorder characterized by a type of intermittent

conduction) as well as sinus tachycardia and supraventricular tachycardia (200 to 240 BPM) of three years duration. The S's regular HR was 110 BPM at the time the study began. Normal conduction could be distinguished from Wolff-Parkinson-White conduction by the length of the PR intervals and the QRS durations on the patient's EKG.

The patient was trained first to decelerate her HR, then to accelerate her HR, and finally to alternate deceleration and acceleration, using the apparatus described in previous studies (Weiss and Engel, 1971). Following the training in HR control, the patient was trained in controlling the prevalence of normal and Wolff-Parkinson-White beats by the utilization of binary auditory feedback (an EKG tracing which selectively triggered a clicker from normal but not Wolff-Parkinson-White beats) and attempted to either increase or decrease the frequency of the sound, depending upon whether the instructions were to increase normal or Wolff-Parkinson-White conduction. Finally, training in increasing normally conducted beats was carried out without feedback. The patient received 26 slowing sessions, 15 speeding sessions, 21 sessions of alternating slowing and speeding, eight sessions of increasing normal conduction, three of increasing Wolff-Parkinson-White conduction, 22 of alternately increasing normal and Wolff-Parkinson-White conduction, and eight

of increasing normal conduction without feedback (for a total of 100 sessions).

The results indicated that she was able to decrease her HR by an average of 3.4 BPM and to increase her HR by 2.5 BPM; while alternating acceleration and deceleration, the difference between speeding and slowing segments averaged 5.5 BPM. She was also able to increase normal conduction significantly in four of eight sessions, and to increase Wolff-Parkinson-White conduction in two out of three sessions. Ability to increase normal conduction during alternating sessions depended upon the prevalence of normal beats during the baseline period, with the increase being greater in those sessions in which the frequency of normal beats had initially been smaller. Finally, the S achieved a mean proportional increase in normal conduction of 13 per cent during those sessions in which feedback was omitted. Followup after 10 weeks demonstrated that her ability to differentially modify cardiac function had been maintained. No further followup was conducted beyond this 10-week period.

In discussing the applicability of operant conditioning procedures to the treatment of patients with heart disease, Scott, Blanchard, Edmunson and Young (1973) pointed out that most researchers reported relatively small changes in HR (usually no more than six BPM), whereas to be of use to patients with chronic tachycardia, considerably

larger decrease in HR would be necessary. They recommended a flexible shaping procedure, the purpose of which was to maximize the Ss' contact with the contingency, as one way of increasing the amount of change in HR by operant conditioning procedures. The strategy of these researchers was to first test a new procedure in an analogue experiment with normal volunteer Ss and then, if the analogue experiment proved successful, to extend the procedure to a clinical experiment. In this case, the variable criterion shaping procedure was first used in an experiment to accelerate the HR of normal Ss, and was then utilized to decelerate the HR of patients with chronic tachycardia. The Ss were three female students (18, 20 and 20 years old) and one male student (22 years old), as well as a 46-year-old male patient with a 20-year history of tachycardia (referred by the Cardiology Department) and a 50-year-old male patient with a 26-year history of tachycardia (referred by the Psychiatry Department with a diagnosis of anxiety neurosis). Several variables were experimentally manipulated during the course of the study: the level of information of the Ss; the flexibility of the shaping procedure; and the type of reinforcement (access to the video portion of ongoing commercial television programs, and monetary reinforcement at the rate of one cent per 10 seconds of correct response).

The results of this study indicated clearly that a constant criterion shaping procedure (adjusted only after the Ss had met or surpassed the criterion on three consecutive trials) was less effective in producing large magnitude changes in HR, both in experimental and in clinical Ss, than a variable criterion procedure (altered on a minute-to-minute basis to maximize the Ss' contact with the contingency). The first patient, whose baseline HR was initially 89 BPM (a rate which did not change during 26 trials with the constant criterion procedure), showed a decrease in HR to an average of 72 BPM during 18 trials with the variable criterion procedure. This patient's HR stabilized at 77 BPM during the final baseline condition. The second patient, whose baseline HR was 96 BPM, decelerated his HR to 82 BPM after 19 trials with the variable criterion procedure (the constant criterion procedure was not utilized with this patient). During eight trials with return to baseline condition, this S's HR actually stabilized at a lower rate (78 BPM) rather than returning to its pathologically high initial baseline level. Both patients reported feelings of greater physical and psychological well-being, and one reduced his tranquilizer consumption from 40 mg to 10 mg per day. The large decreases in HR achieved by these patients may reflect, in part, the high initial value (89 BPM and 96 BPM) of their pre-training HRs. Large magnitude changes were also brought about by the

introduction of the variable criterion procedure with the four experimental Ss, who produced increases in HR of 16 BPM, 16 BPM, 30 BPM and 35 BPM, respectively. However, the real purpose of utilizing these experimental Ss was to provide analogue experiments before the clinical trials (described above) were undertaken. As did the clinical Ss, the experimental Ss maintained their changes in HR over at least three successive days of training.

In order to learn more about the limits imposed by cardiac damage on the ability to learn voluntary HR control, Weiss and Engel (1975) studied three Ss with complete heart block, a condition in which the atria and the ventricles beat independently of each other. This condition is caused by anatomical interruption of impulse conduction between the atria and the ventricles, and occurs most frequently in older persons but may also result from open heart surgery or a congenital disorder. Whereas these patients' atria were activated by the normal cardiac pacemaker and beat at a normal rate (usually 60 to 80 BPM), their ventricles were activated by a slower intraventricular pacemaker, so that their ventricular rate was significantly lower than their atrial rate.

Of the three Ss, two were elderly males (70 and 80 years old) with ventricular HR's of 38 and 32 BPM, respectively. The third was a 22-year-old male with a ventricular HR of 40 BPM. All

three had suffered from complete heart block for at least one year. The Ss received several training sessions daily, with a procedure similar to that described previously (Weiss and Engel, 1971), except that only one cue light (for speeding HR) was used instead of two. A feedback light was illuminated whenever a S's HR was above his resting baseline level. The Ss were also administered atropine (a vagal blocking agent) and isoproterenol (a beta-adrenergic stimulator) intravenously during the last part of the experiment.

The results indicated that, although beta-adrenergic stimulation and exercise increased the ventricular HR of all three Ss, none of them was able to produce consistent, voluntary increases in ventricular rate during the feedback training sessions. Since these same researchers had previously trained patients of similar age to attain some voluntary control over HR (Weiss and Engel, 1971; Bleecker and Engel, 1973a) using a practically identical training paradigm, they concluded that when impulse conduction was interrupted at the level of the A-V node, learned control of ventricular HR was no longer possible, and that appropriate neural innervation of the ventricles by an intact A-V node was a necessary condition for voluntary HR control. Whether an intact myocardium is also a necessary condition for learning voluntary control of HR has not yet been established.

Only one published study has systematically evaluated the effects of age and cardiac disease on ability to control HR by the use of a control group (Lang, Troyer, Twentyman and Gatchel, 1975). This study compared the performance of three groups of Ss: young college students (n = 20); middle-aged and elderly patients with ischemic heart disease (n = 30); and non-patients who were matched in age to the patient Ss (n = 20). The age of the patient Ss ranged from 42 to 71 years, the mean age being 58 years. Two-thirds had suffered previous myocardial infarctions. Data on the length of time since infarction, and whether some Ss had suffered more than one infarction, were not presented. None of the cardiac Ss was convalescent, all were ambulatory, and most were working. The age-matched control Ss were all free of any indication or symptom of cardiac disease, as were the student Ss.

Feedback on HR was presented to the Ss by a visual analogue display consisting of a line which swept horizontally across an oscilloscope, on which a fixed vertical ("target") line represented the Ss' median R-R interval during the criterion period. The horizontal line was initiated by an R wave and terminated by the fifth subsequent R wave, so that a faster HR resulted in a shorter line and a slower HR extended the line's length. The Ss' task was either to terminate the horizontal line before it reached the criterion

line (for speeding trials) or to extend it beyond the criterion line (for slowing trials). The target line was initially set at the median R-R interval of a one-minute period at the beginning of the session, during which the Ss were instructed either to increase or decrease their HR (depending upon whether it was a speeding or slowing session) without feedback. It was subsequently altered automatically to provide a "shaping" schedule such that the new target line would always be one-half of the distance between the previous target line and the median R-R interval on the last feedback trial, making the task more difficult for Ss who were doing well and easier for Ss who were doing poorly.

To control for the effects of attending to the visual display, a control ("tracking") task was included in which the horizontal line was controlled by a computer which caused it to terminate before or after the target line in random order; the Ss pressed a switch when it terminated on the designated side of the target line. Also included was a time estimation task (as a non-visual control for periods in which the Ss attempted to speed or slow their HR without feedback); the Ss pressed a switch every 10 seconds (according to their estimated times) for a one-minute period.

Each S was trained for a total of seven sessions. The first session was the same for each S, and consisted of screening and

orientation. The student control group and the age-matched control group were each randomly divided into two sub-groups of 10 Ss, and the heart disease group was divided into three sub-groups of 10 Ss. The college student and age-matched control Ss, and two of the three sub-groups of the patient group, were treated in the same fashion: one sub-group from each of the three groups received three sessions of tracking followed by three sessions of HR control. The other three sub-groups received the HR control tasks first and the tracking tasks second. The order of tasks within blocks of three sessions was held constant (slowing, speeding and slowing for the three HR control sessions, and tracking-right, tracking-left and tracking-right for the three tracking sessions). The third sub-group of the patient group received six sessions of training in HR control (two speeding and four slowing) and no tracking or time-estimation tasks; this sub-group was referred to as the "extended training" group. Each session was approximately 30 minutes in length, and the sessions were spread over a period of two to three weeks. The format of the sessions was standardized, and both the order of the tasks and the shaping of the criterion line during the training trials were controlled by an on-line computer.

The results indicated that the college student Ss performed best on both speeding and slowing tasks, the healthy middle-aged Ss

(the age-matched control group) did slightly less well, and the Ss with ischemic heart disease showed the least ability to learn to accelerate or decelerate HR. All three groups of Ss were able to slow their HR significantly compared to tracking, and the student Ss were also able to speed their HR relative to tracking. However, neither the patient Ss nor the age-matched control Ss were able to accelerate their HR significantly relative to their HR during the tracking task.

Analysis of variance showed that during the initial "try" period (without feedback), the student Ss were able to accelerate but not decelerate HR more than were the other two groups. When their HR during the transfer-of-training periods was compared with their HR during the time estimation task, all three groups appeared to sustain the changes in HR they had achieved during the training periods, with no significant differences between the groups. Subsequent analyses showed that the difference between the transfer period and the time estimation task period were significant for the slowing sessions but not for the speeding session. Extended training of 10 patient Ss (the sub-group which received six rather than three training sessions) did not improve these Ss' performance. Those college students who had slow basal HR's performed better on the speeding task, and those with fast basal HR's did better on the slowing trials. Basal HR was not correlated with performance for either of the two groups

of older Ss, whose initial HR's were also less variable than those of the student Ss.

The 30 patient Ss were also classified into three categories according to the effects (on HR) of the medication which they were taking at the time of the study: no medication, or medication with no known effect on HR; medication with possible effects on HR; and medication with definite effects on HR. No differences were found in basal HR among these three categories. The definite-effect Ss demonstrated poorer performance on acceleration and better performance on deceleration than did the no-effect Ss. When the Ss' resting HR variability was compared, both the no-effect and the definite-effect Ss had less variable HR's than the possible-effect Ss, perhaps because of the tendency on the part of physicians to not prescribe cardiotropic medication to patients with stable HR's (the first category) and to prescribe anti-arrhythmic medication for patients with highly varible HR's (the third category).

According to the researchers, their data "strongly suggest that the ability to change heart rate in response to instructions and feedback varies significantly with the age and disease state of the subjects . . . . the evidence suggests a hierarchical ordering of heart control ability on feedback tasks, with college students clearly superior to both groups, and the age-matched controls marginally

better than the patient subjects [Lang, Troyer, Twentyman and Gatchel, 1975, p. 443]." Although the patient Ss who were receiving cardiotropic medication performed differently in several respects from the other Ss, "a comparison of age-matched control subjects and the no-medication-effect patient group yielded a pattern of results similar to that shown by the entire patient sample . . . . [suggesting] that drugs were not a primary factor in producing the overall impression of poor psychological control of heart rate in ischemic heart disease [ibid., p. 444]." Speeding proved to be a more difficult task for the older Ss (both normals and patients) than slowing; and the patient Ss who received extended training in HR control (six rather than three sessions) did not profit from the additional training.

Three points need to be raised about this study. The first (which the researchers themselves mention) is that the college student Ss may have differed from the age-matched control Ss and the patient Ss in ways other than age and illness. No information was presented on the socioeconomic or educational levels of any of the three groups of Ss, except that the student Ss were undergraduates at the University of Wisconsin and the age-matched control Ss were recruited from Volunteers for Action. The second point is that no separate analyses were reported for type of ischemic heart disease, e.g., those patients who had had one (or more) myocardial infarctions versus

those who had no evidence of having had an infarct. The analysis by drug category was only an indirect comparison of patients with varying degrees of severity of disease, and no diagnostic data about the patients in the three drug categories were presented. Lastly, feedback was provided to these Ss on a non-continuous basis (the length of the five preceding R-R intervals was averaged on the oscilloscope display), which has been shown to be less effective in training HR control, particularly for speeding, than feedback provided on a continuous (i.e., beat-by-beat) basis (Gatchel, 1974).

A number of conclusions are apparent from this review of existing research in the area of learned cardiac control. First, young, healthy Ss can be taught to decrease, increase, and alternately decrease and increase their own HR when they are trained to do so using biofeedback paradigms of varying designs (Blanchard and Young, 1972; Brener and Hothersall, 1966; Engel and Hansen, 1966; Engel and Chism, 1967; Headrick, Feather and Wells, 1971; Levene, Engel and Pearson, 1968). However, it appears that acceleration and deceleration of HR may be very different tasks, and speeding may be easier for younger Ss to achieve than slowing (Bell and Schwartz, 1975; Engel, 1972), although this is not a unanimous opinion (Stephens, Harris, Brady and Shaffer, 1975). Furthermore, although some researchers have claimed that somatic activity mediates

between reinforcing stimuli and changes in HR (Obrist, 1973), it has been shown that voluntary changes in HR can occur in the absence of either somatic or respiratory mediation (Brener and Hothersall, 1968; Manuck, 1976). It has also been shown that the ability to profit from biofeedback training was facilitated when the Ss were informed that HR was the response being conditioned (Blanchard, Scott, Young and Edmundson, 1974); when analogue rather than binary feedback was provided (Lang and Twentyman, 1974); and when feedback of the Ss' HR was provided on a beat-by-beat basis (Gatchel, 1974). All of these variables appeared to be more important for training in HR acceleration than HR deceleration, again suggesting that these two tasks are based upon different physiological mechanisms.

Studies of HR control using older Ss and Ss with cardiac disease have indicated that patients could be taught to reduce the frequency of PVC's by learning to slow their HR, and that these effects were maintained for as long as 21 months after training (Weiss and Engel, 1971); that patients with chronic atrial fibrillation could be trained to control their ventricular HR (Bleecker and Engel, 1973a); that both cardiac arrhythmias and abnormalities in cardiac conduction were capable of modification with biofeedback procedures (Bleecker and Engel, 1973b); and that large magnitude decreases in HR could be attained by patients with chronic tachycardia by using a flexible-

criterion shaping schedule (Scott, Blanchard, Edmunson and Young, 1973). However, patients with ischemic heart disease are reported to have had significantly more difficulty learning to control their HR than persons of similar age who had no evidence of heart disease, and both had more difficulty than did healthy younger persons (Lang, Troyer, Twentyman and Gatchel, 1975). Patients in whom conduction between atria and ventricles had been interrupted by complete heart block could not learn voluntary control of ventricular rate with any consistency at all (Weiss and Engel, 1975).

What is needed are more studies of the clinical applicability of HR biofeedback procedures. These should have the following characteristics: greater comparability of patient and non-patient (control) groups; greater homogeneity of patient groups regarding type and severity of cardiac disease; and utilization of those features of previously tested paradigms which were shown to be the most facilitative of self-control of HR. Also needed are more long-term studies to assess both the extent to which voluntary HR control achieved in the laboratory is a transferable skill which can be utilized on an ad lib basis outside of the laboratory, and whether such ad lib utilization can be of clinical value to persons with different kinds of heart disease, or in the prevention of heart disease in symptom-free populations.

CHAPTER III

METHOD

Subjects

All 30 Ss in this study were present or former in- or outpatients at the Houston (Texas) Veterans Administration Hospital. There were 10 Ss in each of three subject groups: the Control group; the group of patients convalescing from a first, uncomplicated acute myocardial infarction suffered less than one month previously (the AMI group); and the group of patients and former patients with a history of at least one documented myocardial infarction in the past, the infarction (or the most recent, if more than one) having occurred no less than three months previously (the PMI group). All of the Ss were males.

Control Group. The Control Ss were selected, with the assistance of their physicians, from services other than Cardiology. One of the Control Ss was a patient on the Dermatology Service; the other nine were patients on the Pulmonary Service. More detailed information on these patients and their diagnoses is given in Table 1.

Ss in the Control group had to meet the following criteria: have no history, signs or symptoms of cardiac disease; not be taking any medication with a direct effect on heart rate (such as bronchodilators, which frequently induce tachycardia); have no history or current

TABLE 1

Characteristics of Control Group Subjects

Patient	Age/Race/Sex	Principal Diagnosis
N.E.	55 y/o WM	Psoriasis
J.W.	49 y/o BM	Tuberculosis
E.C.	50 y/o WM	Tuberculosis
B.G.	44 y/o WM	Tuberculosis
B. C.	56 y/o WM	R/O Ca Lung
B.H.	53 y/o WM	Tuberculosis
H.T.	49 y/o WM	R/O Ca Lung
J.B.	49 y/o BM	Tuberculosis
W.S.	48 y/o WM	Tuberculosis
P.H.	53 y/o BM	Silicosis

indications of organic brain disease or psychosis; and not be a current, untreated alcoholic (defined by the presence of alcoholic encephalopathy or neuropathy, or by alcoholic liver disease).

The ages of the Control Ss ranged from 44 to 56 years, with a mean age of 51.2 years (SD = 4.0). Their educational backgrounds varied from seventh grade to three years of college, with a mean of 10.4 years of school completed (SD = 2.8). Seven were white and three were black.

AMI Group. These Ss were selected from patients who were, or had recently been, hospitalized on the Coronary Care Unit (CCU). Patients on the CCU and the adjacent convalescent ward were routinely screened, and their physicians were approached if they met the following criteria: a documented acute, uncomplicated myocardial infarction at the time of the present admission; no history or electrocardiographic (EKG) evidence of a previous infarction; no cardiac surgery in the past; and none of the following medications with a direct effect on heart rate: digitalis; quinidine; procainamide; propranolol (Inderal); or any anti-hypertensive drug other than diuretics. Nitrates (including nitroglycerin tablets), which function as vasodilators and exert only an indirect effect on heart rate, and are frequently prescribed for angina pectoris (chest pain) due to ischemic heart disease (either with or without myocardial infarction), were permitted. Like the

Control (and the PMI) Ss, AMI Ss could not have a history or diagnosis of organic brain disease or psychosis, or of untreated alcoholism.

Patients who met these criteria and whose physicians approved of their participation in the study, were approached as soon as possible after being moved from the CCU into the adjacent convalescent ward. The study was described to them and, if they agreed to participate, they were asked to sign an Informed Consent Statement (Appendix A). Biofeedback training was begun as soon as the patient was permitted to leave the ward (in a wheelchair) to go to the laboratory. Two patients were not contacted until just before their discharge from the hospital, and in both these cases they came back to the hospital for the training sessions shortly after being discharged.

The AMI Ss ranged in age from 38 to 63 years, with a mean age of 54.3 years (SD = 7.7). Their educational backgrounds ranged from sixth grade to two years of college, with a mean of 9.5 years of schooling (SD = 3.6). Nine were white and one was black. The shortest amount of time between the occurrence of the myocardial infarction (MI) and the first training session was four days, and the longest was 29 days; the mean number of days between the MI and the beginning of training was 11.9 (SD = 7.4). Table 2 gives more detailed information on the Ss, the locations of their infarctions, and the medications (if any) they were taking at the time of the study.

TABLE 2

Characteristics of AMI Group Subjects

Patient	Age/Race/Sex	Principal Diagnosis	Time Since MI	Current Cardiac Medications
J.J.	52 y/o WM	Acute ASMI[1]	13 days	None
J.A.	63 y/o WM	Acute ALMI[2]	20 days	Isordil, 5 mg q 4h
G.D.	45 y/o WM	Acute SEMI[3]	29 days	Isordil, 5 mg q 2h Nitrobid, 6.5 mg hs
F.C.	61 y/o WM	Acute SEMI	8 days	None
A.M.	53 y/o WM	Acute IWMI[4]	8 days	Isordil, 5 mg q 6h
D.B.	38 y/o WM	Acute IWMI	4 days	Isordil, 5 mg q 4h
G.M.	60 y/o WM	Acute IWMI	11 days	Isordil, 5 mg q 4h
C.W.	57 y/o WM	Acute IWMI	8 days	None
B.S.	58 y/o BM	Acute SEMI	7 days	None
E.B.	56 y/o WM	Acute IWMI	11 days	None

[1] Anteroseptal myocardial infarction
[2] Anterolateral myocardial infarction
[3] Subendocardial myocardial infarction
[4] Interior wall myocardial infarction

PMI Group. All of the Ss in this group had suffered at least one documented infarction. Since this was intended to be a post-convalescent group, it was required that the MI (or, if the person had had more than one infarction, the most recent MI) have occurred at least three months before the inception of biofeedback training, 13 weeks being the accepted maximum length of time of recovery following a myocardial infarction. None of the PMI Ss could have had cardiac surgery, but no restrictions were placed on complications during recovery from infarction or on cardiotropic (or other) medications which the Ss could be taking at the time of their participation.

Seven of the 10 PMI Ss were currently participating, as either in- or outpatients, in a cardiac rehabilitation program at the hospital. These Ss were selected with the assistance of either the physician in charge of the program or the therapist who worked with the program's participants in their daily exercise sessions. All of the PMI Ss were ambulatory, and those who were inpatients (usually for purposes of stress testing to establish and build up exercise tolerance) were on a self-care ward during the week and went home on pass each weekend. The outpatients attended exercise sessions from two to five times a week. Two of the three PMI Ss who were not participants in the rehabilitation program were volunteer workers at the hospital, contacted with the cooperation of the volunteer office; and one was a

former CCU patient who had not been contacted early enough to be included in the AMI group.

The age range of the PMI Ss was from 40 to 72 years, with a mean age of 53.0 years (SD = 11.2). Their education ranged from sixth grade to two years of graduate study, with a mean educational level of 13.1 years (SD = 3.5). Five of the PMI Ss were white and five were black. Four had had more than one documented MI, one having had a total of three. The time between their infarction (or latest infarction) and their participation in the study varied from 14 weeks to three years, the mean time since infarction being 70.6 weeks (SD = 52.8). Seven PMI Ss were taking one or more of the cardiotropic drugs (digitalis, quinidine or propranolol) which precluded inclusion in the AMI group; the specific drugs and dosages, as well as other information on these Ss, appears in Table 3.

Group Comparisons. There were no significant differences among the three subject groups, either insofar as age or years of education is concerned (age, $F = 0.362$, $df = 2$, $p > .05$; education, $F = 3.182$, $df = 2$, $p > .05$).

Procedure

Selection of Subjects. All prospective Ss were selected with the assistance and consent of their physicians, and their current and past medical records were carefully reviewed to assure conformity with

TABLE 3

Characteristics of PMI Group Subjects

Patient	Age/Race/Sex	Principal Diagnosis	Time Since Last MI	Current Cardiac Medications
J.C.	40 y/o WM	S/P ASMI	20 weeks	Isordil, 5 mg q 4h
F.M.	56 y/o WM	S/P SEMI Hx MI, 1971	1 year	None
T.C.	72 y/o WM	S/P IWMI Hx ALMI, 1974	1 1/2 years	Digoxin, .25 mg qd Isosorbide, 10 mg qid Quinaglute, 1/2 tab q 8h
C.R.	48 y/o BM	S/P IWMI	2 1/4 years	Inderal, 20 mg qid
L.K.	43 y/o BM	S/P ALMI	1 1/2 years	Inderal, 80 mg qid Isordil, 5 mg q 4h
W.M.	58 y/o BM	S/P ASMI	3 years	Inderal, 20 mg qid Nitrobid, 1 tab qd
D.B.	68 y/o WM	S/P ALMI	3 years	Digoxin, .25 mg qd
R.C.	48 y/o BM	S/P IWMI Hx SEMI, 1975	5 months	Quinidine, 300 mg q 6h

(TABLE 3 - Cont'd)

Patient	Age/Race/Sex	Principal Diagnosis	Time Since Last MI	Current Cardiac Medications
W.M.	40 y/o BM	S/P IWMI Hx MI, 1972 & 1974	14 weeks	Digoxin, .25 mg qam Quinidine, 200 mg q 6h Isordil, 5 mg q 4h
J.Z.	57 y/o WM	S/P AWMI[2]	6 months	None

[1] Status Post

[2] Anterior wall myocardial infarction

the criteria for inclusion in the pertinent patient group. Prospective Ss who met the criteria were interviewed by the researcher, who described the study ("Explanation of the Research," Appendix A) and, if the prospect agreed to participate, had him sign an Informed Consent Statement (Appendix A).

Sequence of Training Sessions. Each S came to the laboratory for three training sessions. Session One was devoted to training in slowing heart rate; Session Two for speeding heart rate; and Session Three for slowing once more. The period of time over which the three sessions were spread unavoidably varied between Ss, ranging from 48 hours to 16 days. The mean length of time between the first and third sessions were: AMI group, 2.6 days (SD = .7); Control group, 3.2 days (SD = 1.3); and PMI group, 7.2 days (SD = 3.7). The dispersion of training for the PMI Ss was greater because several of them were outpatients or former patients, and could not come to the laboratory on consecutive days. These group differences in total time elapsed between the beginning and end of training were statistically significant ($F = 11.96$, $df = 2$, $p < .05$).

After the third session, each S was given two self-administered instruments, the Autonomic Awareness and Locus of Control questionnaires (Appendices B and C), to be completed and returned. An inkblot test (the Rorschach) was also given, either at that time or shortly after, by the researcher or an associate. These protocols were scored

blind by Dr. Sidney E. Cleveland. Three scores were obtained from the Rorschach protocols: an index of anxiety (Elizur, 1949; Holtzman, Thorpe, Swartz & Herron, 1961) and two dimensions of body image, Barrier and Penetration (Cleveland & Fisher, 1968). The inkblot method of assessing anxiety was chosen because of its unobtrusiveness, particularly with patients who had recently had MI's. All 30 Ss filled out the two questionnaires; however, two of the PMI Ss refused to take the Rorschach, and one PMI S had a cardiac arrest before a Rorschach protocol could be obtained from him.

Session One. The first session was devoted to training in slowing heart rate; however, Ss were not made aware of this until immediately before the first trial period in which they were instructed to alter their heart rate; nor were they informed about the order of the two subsequent training sessions.

Upon coming to the laboratory for the first time, each S received a brief explanation of the purpose of the various electrodes and transducers while the researcher was attaching them to the S. The transducers and feedback and recording instruments which were used will be described in detail in a subsequent section of this chapter. The total time involved in preparing the S for the session was approximately 15-20 minutes.

Once all the electrodes and transducers had been attached and checked, the first part of the Instructions (Appendix D) was read to

the S. The S's initial basal heart rate was measured by averaging over a 180-interbeat interval (IBI) period, and the cardiotachometer was then set so that the needle would rest at the mid-point of the dial when the S's heart was beating at this rate. Following this, the next part of the instructions was read, telling the S only to pay close attention to the needle "in order to get used to it." The visual display in the room where the S was sitting was then turned on, and the S's response to feedback in the absence of instruction to either slow or speed his heart rate was measured for a 180-IBI period. The visual display was then shut off and the last part of the instructions read to the S, explaining the operation of the feedback display and the purpose of the "Slow" and "Fast" instruction lights. When the apparatus and procedure had been explained, and the S indicated his understanding and readiness to proceed, the researcher went into the adjacent room for the remainder of the session.

The rest of the session consisted of four training periods (both with and without feedback) of varying lengths, each preceded by a 120-IBI rest period, which was used as a baseline from which to measure change in the following training period. Before the first training period, Ss were instructed to slow their heart rate without any feedback being provided, for a 180-IBI (instruction-only) period. Following this were two 480-IBI training periods during which the

instruction to slow was augmented by the visual feedback display. A brief intervention was made after the second instruction-with-feedback period, the intention of which was to counteract the expected tendency of heart rate to gradually decline during the course of the session as a result of habituation. This intervention consisted of the researcher entering the room in which the S was seated and asking a few brief questions. There was then another 180-IBI instruction-only training period; and immediately before the end of the session, final basal heart rate was measured by averaging over the last 180-IBI period.

Since the length of both rest and training periods was measured in IBI's rather than prescribed amounts of time, there was an inverse relationship between a S's heart rate and the length of the session. Most sessions lasted approximately 25 to 35 minutes, apart from time spent in attaching and checking electrodes and explaining the format of the experiment to the Ss. Most Ss were actually in the laboratory for a period of 45 minutes to an hour on each of the three sessions.

Session Two. The sequence of training periods in the second session was identical to that of the first, except that Ss were instructed to speed, rather than to slow, their heart rate. The fact that they were going to be told to alter their heart rate in the opposite direction from that which they had been told to do in the preceding session, however,

was not revealed until after the feedback-only (i.e., without instruction) period. The reason for this was to determine whether Ss would generalize their response to the visual feedback display from the first to the second session, by slowing their heart rate more during this segment of Session Two than during the identical segment of Session One. Following the feedback-without-instruction period of Session Two, Ss were informed that the remainder of the session would be devoted to training in heart rate speeding. The session then proceeded as in Session One, except that in place of the intervention which was made in the first session, Ss continued resting for an equivalent period at this point in Session Two.

Session Three. The third session, which was devoted to heart rate slowing once more, was identical to the first session but with one exception: instead of the feedback-only period, the S was instructed to work on a specified mental task until the researcher told him to stop (a 180-IBI period). The task consisted of making up as many sentences of five or more words as possible, in which the first letter of each word was an "r." The purpose of this task, adapted from the research of Lacey and his associates, was to determine whether Ss in the present study would respond similarly to those in Lacey and associates' research, with cardiac deceleration while attending to the external environment (feedback without instruction) and cardiac

acceleration while engaging in "mental work," such as the sentence construction task (Lacey, 1967; Lacey, Kagan, Lacey & Moss, 1963).

Instruments

Feedback. Ss were provided with information about changes in their cardiac rate by means of a visual analogue feedback device. This consisted of a meter (approximately eight inches in length) mounted in a console which faced the lounge chair in which Ss sat. The meter was marked at approximately one-quarter-inch intervals, but no numerical scale was provided. A pointer indicated changes in cardiac rate by moving to the left if heart rate decreased, and to the right if it increased. The feedback thus provided was continuous, and reflected the direction of change (downward or upward) of the immediately preceding two heartbeats. The pointer was set at the beginning of each session so that it was centered at the S's initial basal heart rate on that particular day. A battery-operated EKG simulator was used for this purpose, as well as for calibrating the cardiotachometer.

The cardiotachometer itself operated by measuring the length of successive R-R intervals and converting this into a cardiac rate. Because a new IBI was "recognized" by the appearance of another R-wave (usually the highest peak in a normal EKG record), EKG lead placement was designed to maximize the height of the S's R-wave, so that it would trigger the apparatus. Three chest leads were used

(one reference and two active electrodes), usually with the reference and negative electrodes in the left and right midclavicular positions, respectively, and the positive electrode along the left midaxillary line. Although this generally sufficed for the Control Ss, the electrodes did, on occasion, have to be moved around on AMI and PMI Ss to overcome weak EKG signals and to maximize the height of the R-wave.

On either side of the meter was a small (approximately one inch square) instruction light. When independently illuminated, the one to the left of the meter said "Slow" and the one to the right said "Fast." These were used to tell the Ss when they were supposed to alter their cardiac rate, and in which direction; and were turned on and off (as was the feedback meter) by the researcher, who was in an adjacent room.

Recording. In the adjacent room, in addition to the controls for setting and turning on and off the S's visual feedback display and instruction lights, was the recording apparatus. This was an eight-channel Brush recorder which simultaneously recorded EKG, heart rate, blood pressure (taken at 60-second intervals with an automatically inflating and deflating cuff), frequency and depth of respiration, digital venous return (measured with a finger plethysmograph) and electrodermal activity (GSR). Data on physiological activities other than cardiac rate were gathered for additional analyses separate from

the present study. Each channel was calibrated before every training session to assure accurate measurement.

Measurement of Heart Rate. For purposes of the data analysis (the results of which will be presented in the next chapter), heart rate was calculated by averaging over discrete 60-IBI periods. This method of measuring cardiac rate was used because the Brush recorder could be set to mark off 60-IBI segments (i.e., segments corresponding to 60 successive R-R intervals on the EKG). Heart rate then could be calculated by measuring the distance that the strip chart paper had travelled between the beginning and end of a segment, and applying a simple transformation to arrive at the average cardiac rate for that segment. Heart rates for longer periods (such as the 180-IBI basal periods, the 120-IBI rest periods, and the 180-IBI and 480-IBI training periods) were arrived at by calculating the average rate for each of the component 60-IBI segments, and then calculating the means of these respective rates.

CHAPTER IV

RESULTS

Data Analysis

The principal hypotheses, regarding patient group differences in ability to control heart rate, were tested using a mixed analysis of variance model. Data from each of the three sessions were analyzed separately. In addition, data from Sessions One and Three (the two slowing sessions) were combined in a single analysis of variance; and data from Sessions One and Two, and from Sessions Two and Three, were compared in separate analyses of variance in order to look at the differential effects of instructing Ss to slow and to speed their heart rate.

Additional hypotheses, regarding the role of personality variables (awareness of autonomic activity; Locus of Control; anxiety; and two body image dimensions, Barrier and Penetration), were tested with non-parametric analyses, since it was not felt that the measurement of these variables met the assumptions of parametric tests. The specific tests used to test these hypotheses will be detailed later in this chapter, when those portions of the results are presented.

Initial Heart Rate

Initial basal heart rate was averaged over a 180-interbeat interval (IBI) period at the beginning of each session, after all electrodes had

been attached and the Ss had been resting for approximately 15 minutes. None of the differences in initial heart rate, either across groups within each of the three sessions, or across sessions within each of the three groups, were significant at the .05 level (Table 4).

Change from Initial to Final Heart Rate

Basal heart rate at the end of each session (final basal heart rate) was obtained by averaging over the last 180-IBI period of the session, during which Ss were receiving neither instructions nor feedback. There was a significant mean decline of 2.55 beats per minute (BPM) in the heart rate of all 30 Ss from beginning to end of all three sessions ($p < .01$; Table 5). There were no significant differences in the amount of decline between groups, between sessions, or between groups within sessions.

Orienting, Mental Task, and Intervention

Orienting Response. After initial basal heart rate was obtained, Ss were instructed (in Sessions One and Two) to pay close attention to the visual display (meter) when it came on, ostensibly to become accustomed to watching it. At this point in Session One, Ss were naive as to the function of the meter; in Session Two, although they were no longer naive, the same instructions (simply to pay close attention) were given. The heart rate of all 30 Ss declined an average of 1.00 BPM during both of these 180-IBI periods ($p < .01$; Table 6). There

TABLE 4

ANOVA for Initial Basal Heart Rate:

All Groups, Sessions One, Two and Three

Source	df	MS	F
Between			
Groups (G)	2	691.15	1.29
Error	27	533.53	
Within			
Sessions (R)	1	21.32	1.10
R x G	2	12.49	0.64
Error	27	19.29	

TABLE 5

ANOVA for Initial and Final Basal Heart Rate:

All Groups, Sessions One, Two and Three

Source	df	MS	F
Between			
Groups (G)	2	1287.30	1.20
Error	27	1067.83	
Within			
Sessions (R)	1	30.59	0.84
R x G	2	13.53	0.37
Error	27	36.01	
Initial vs. Final (S)	1	292.86	38.9*
S x G	2	3.58	0.47
Error	27	7.52	
R x S	1	1.02	0.31
R x S x G	2	5.98	1.81
Error	27	3.28	

* $p < .001$

TABLE 6

ANOVA for Change in Heart Rate

During Feedback Without Instruction:

All Groups, Sessions One and Two

Source	df	MS	F
Between			
Groups (G)	2	728.13	0.92
Error	27	787.53	
Within			
Sessions (R)	1	0.01	0.01
R x G	2	10.14	0.40
Error	27	24.99	
Response to Meter (S)	1	30.00	21.94*
S x G	2	1.03	0.76
Error	27	1.36	
R x S	1	0.01	0.01
R x S x G	2	0.82	0.51
Error	27	1.59	

* $p < .001$

was no difference between the amount of decline shown during the feedback-without-instruction period in Session One, and the corresponding period in Session Two; nor were there any differences in the amount of decline shown by any of the three groups of Ss during either of the two sessions.

This decline in heart rate is consistent both with the expected effect of habituation to the experimental situation, and also with previous findings to the effect that attentiveness to the external environment is associated with a decrease in heart rate (Lacey, 1967). The fact that the mean decrease in heart rate for all 30 Ss during the feedback-without-instruction period in Session Two (1.02 BPM) was not significantly greater than the mean decrease during the same period in Session One (.98 BPM) did not, however, confirm the expectation that Ss would respond to the feedback meter alone with greater slowing the second time than the first because of its having become a conditioned stimulus for slowing of heart rate. (It will be recalled that although Ss were no longer naive as to the function of the meter at the beginning of the second session, they were uninstructed as to whether that session would involve slowing or speeding of heart rate, and presumably expected being instructed to slow.)

Response to Mental Task. Following the initial basal heart rate period in Session Three, Ss worked on the sentence construction task

described in the preceding chapter, in place of the feedback-without-instruction condition, for a 180-IBI period. The average heart rate of all 30 Ss increased slightly (.31 BPM) but not significantly during this task (Table 7), not supporting Lacey's hypothesis that attentiveness to the "internal environment" is associated with an increase in heart rate (Lacey, 1967). However, although the slight increase in heart rate during the mental task was not statistically significant, it was in the opposite direction from the downward change which occurred during the uninstructed feedback-only periods in Sessions One and Two, when the attention of the Ss was focused on the feedback meter.

Response to Intervention. A brief intervention was made between the rest period immediately following the second instruction-with-feedback period, and that immediately preceding the second instruction-without-feedback period, during Sessions One and Three. These interventions consisted of the experimenter entering the room where the S was sitting, asking whether the S had any questions, and obtaining some demographic data if time permitted; the total time elapsed was between one and two minutes. The purpose of the interventions was to increase the heart rate of the Ss in order to see whether, during the final instruction-only period, they were capable of slowing their hearts subsequent to social stimulation . During the corresponding

TABLE 7

ANOVA for Change in Heart Rate

During Sentence Construction Task:

All Groups, Session Three

Source	df	MS	F
Between			
Groups (G)	2	708.27	2.09
Error	27	337.70	
Within			
Response to Task (R)	1	1.47	0.46
R x G	2	1.83	0.58
Error	27	3.13	

period in Session Two, an extended rest of approximately the same length of time as the intervention was substituted.

The intervention just described failed to bring about the anticipated increase in heart rate during either Sessions One or Three; in both sessions, in fact, the mean heart rate of all 30 Ss declined slightly but not significantly from the rest period preceding the intervention to that following the intervention (Table 8). Although (as can be seen from Figures 1 and 2) the average heart rate of the PMI Ss actually did show a small increase following both interventions (contrary to the small decreases shown by the AMI and Control Ss), there was not a significant interaction between group and response to intervention on either session. The extended rest in Session Two (which is shown in Figure 3), on the other hand, was followed by a significant mean decline in heart rate, among all 30 Ss, of .60 BPM ($p < .05$; Table 9).

Response to Instruction and Feedback

Session One (Slowing). Response to instruction to slow heart rate (Training effect) was measured in terms of differences between Ss' heart rate during the training periods (two 180-IBI periods of instruction without feedback, and two 480-IBI periods of instruction with feedback) and the resting periods (120 IBI's with neither instruction nor feedback) immediately preceding the training periods. The repeated

TABLE 8

ANOVA for Change in Heart Rate

During Mid-Session Intervention:

All Groups, Sessions One and Three

Session One

Source	df	MS	F
Between			
Groups (G)	2	310.76	0.67
Error	27	461.31	
Within			
Response to Intervention (R)	1	0.41	0.19
R x G	2	1.42	0.66
Error	27	2.14	

Session Three

Source	df	MS	F
Between			
Groups (G)	2	531.64	1.58
Error	27	334.68	
Within			
Response to Intervention (R)	1	1.83	0.58
R x G	2	6.95	2.21
Error	27	3.13	

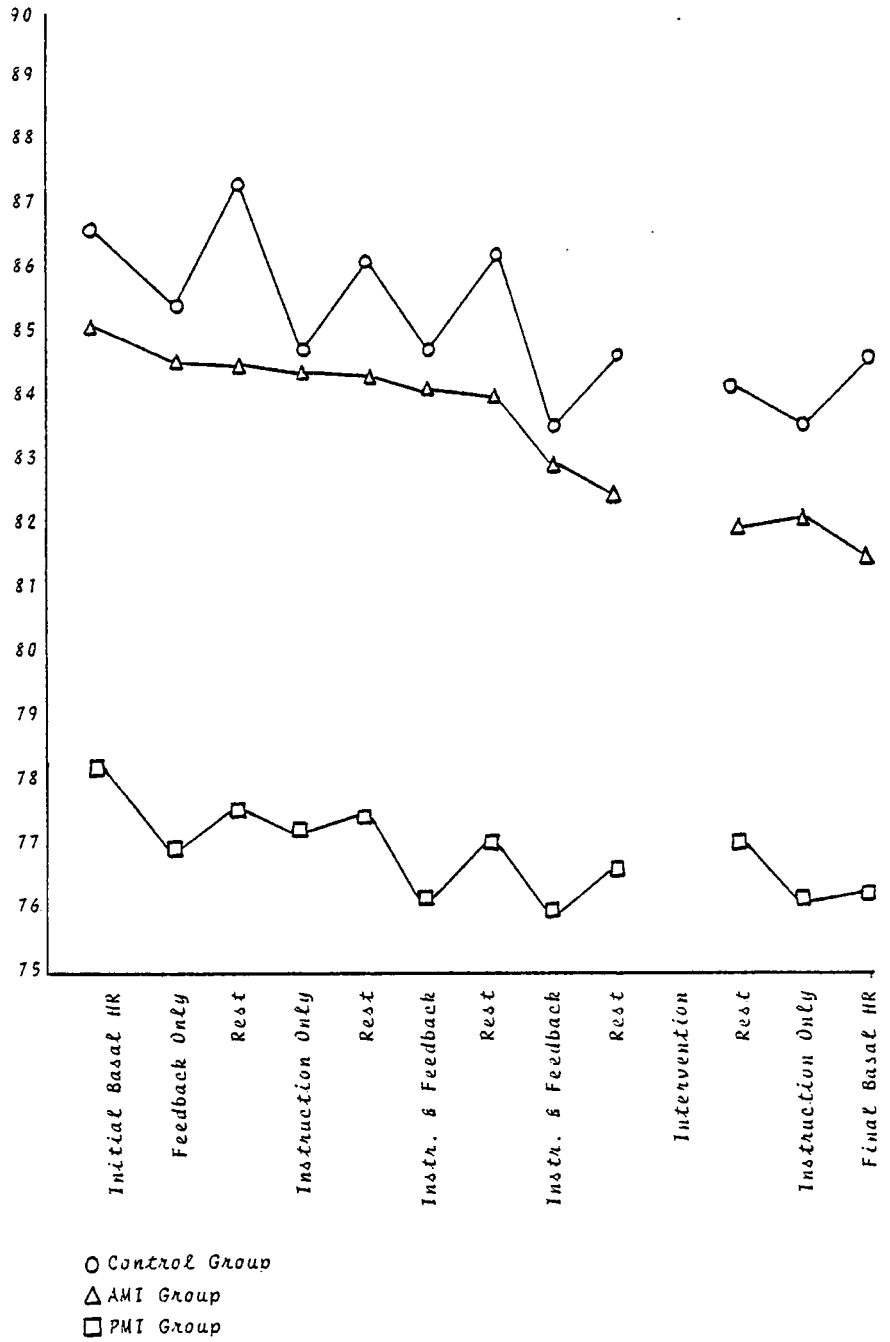

Figure 1

Changes in Mean Heart Rate During All Periods in Session One (Deceleration)

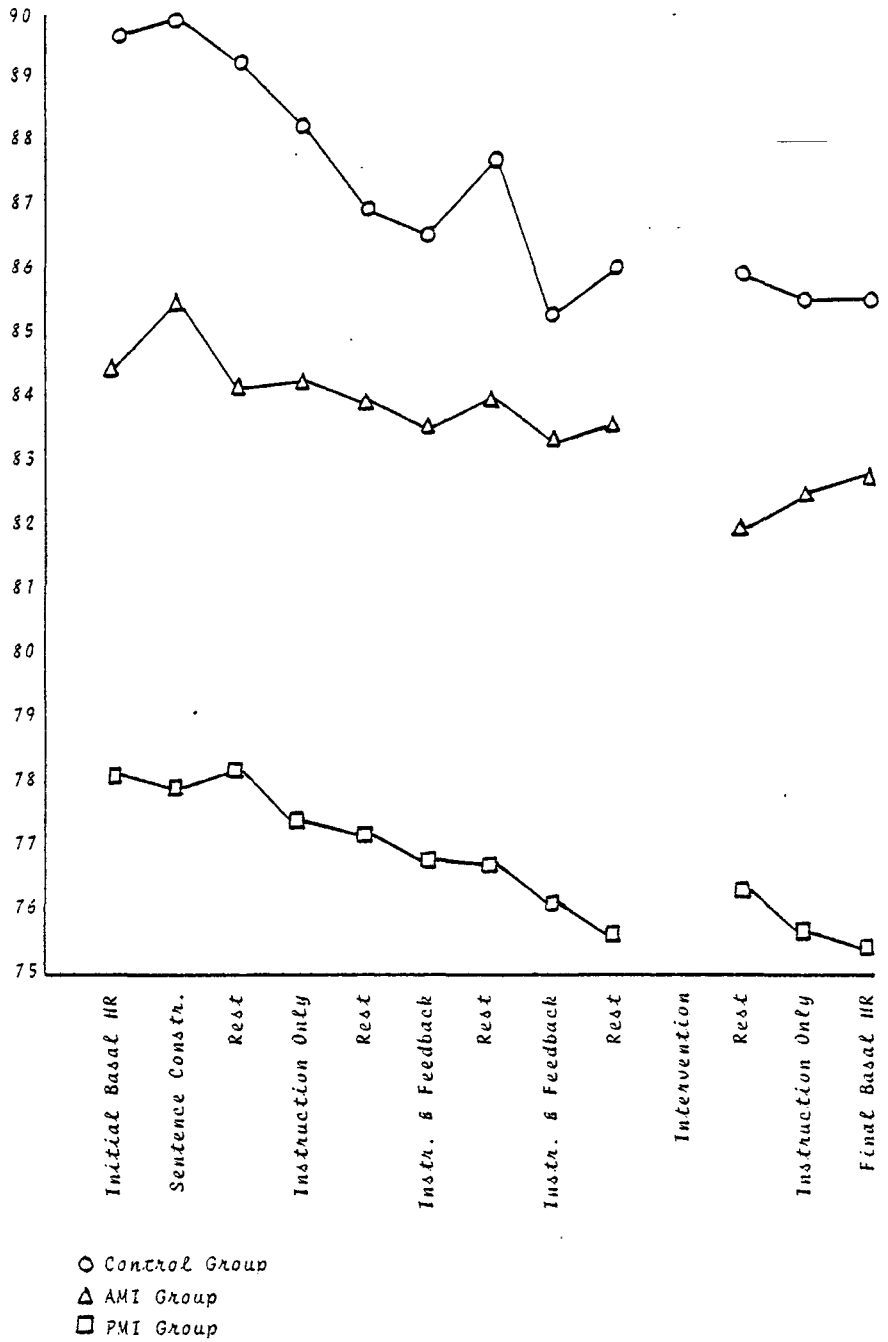

Figure 2

Changes in Mean Heart Rate During All Periods in Session Three (Deceleration)

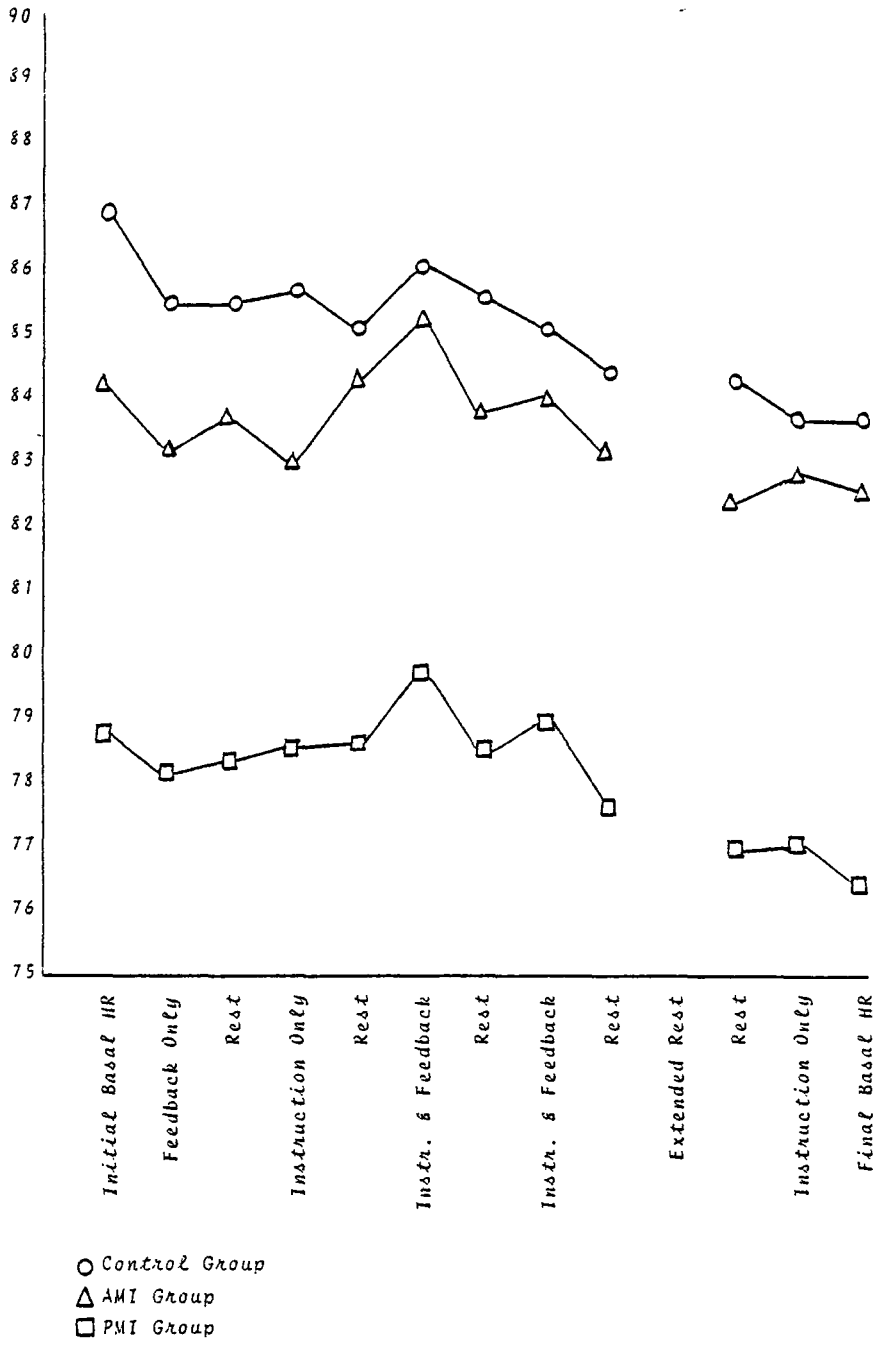

Figure 3

Changes in Mean Heart Rate During All Periods in Session Two (Acceleration)

TABLE 9

ANOVA for Change in Heart Rate

During Mid-Session Extended Rest Period:

All Groups, Session Two

Source	df	MS	F
Between			
Groups (G)	2	269.06	0.80
Error	27	333.88	
Within			
Effect of Extended Rest (R)	1	5.40	4.82*
R x G	2	0.95	0.84
Error	27	1.11	

*$p < .05$

measures ANOVA for the four training, and four associated resting, periods of Session One are summarized in Table 10.

First, it is apparent that there was a significant main effect of Training, as indicated by a mean difference of 1.02 BPM between resting and training periods ($p < .01$). In addition, there were two significant interactions meriting further exploration: between Training and Feedback ($p < .05$), and between Training and Group ($p < .05$).

The first of these two interactions (Training x Feedback) dealt with the question of whether or not the Training effect was more pronounced when instruction to slow heart rate was accompanied by visual feedback (the meter display) than when the instruction was presented alone, that is, without feedback. The mean difference between resting and training heart rate for the instruction-alone condition was .74 BPM; for the instruction-with-feedback condition it was 1.30 BPM. Clearly, the decrease in heart rate which Ss were capable of bringing about when instructed to do so was enhanced when the instruction was augmented by the provision of visual feedback.

The second significant interaction (Training x Group) indicated that the three groups of Ss did not all show resting-training heart rate differences (Training effects) of similar magnitudes. The mean resting-training difference for Ss in the Control group was 1.82 BPM, whereas for Ss in the AMI and PMI groups, the mean resting-training

TABLE 10

ANOVA for Change in Heart Rate

During Training Periods: All Groups, Session One

Source	df	MS	F
Between			
Groups (G)	2	1557.28	0.85
Error	27	1825.86	
Within			
Early vs. Late (R)	1	76.50	18.65*
R x G	2	5.70	1.38
Error	27	4.10	
Feedback vs. No Feedback (S)	1	1.13	0.41
S x G	2	4.66	1.69
Error	27	2.74	
R x S	1	22.50	7.30**
R x S x G	2	2.29	0.74
Error	27	3.08	
Resting vs. Training (T)	1	62.11	20.48*
T x G	2	10.93	3.60**
Error	27	3.03	
R x T	1	0.06	0.06
R x T x G	2	0.59	0.62
Error	27	0.95	
S x T	1	4.78	4.73 **
S x T x G	2	0.05	0.05
Error	27	1.01	

(TABLE 10 - Cont'd)

Source	df	MS	F
R x S x T	1	5.07	3.08
R x S x T x G	2	5.25	3.19
Error	27	1.64	

* $p < .001$
** $p < .05$

differences were .36 BPM and .88 BPM, respectively. Individual ANOVA's performed on each of the groups indicated that only the Control Ss showed a significant Training effect ($p < .01$; Table 11); the decrease in heart rate from resting to training shown by the PMI Ss closely approached significance ($p = .06$); and that of the AMI Ss was clearly not significant. Furthermore, paired comparisons among the three groups indicated that only the difference between the mean decrease of the Control Ss and that of the AMI Ss was significant ($p = .01$; Table 12); neither the AMI-PMI difference nor the PMI-Control difference reached significance.

Session Three (Slowing). As was found in Session One, there was also a main effect of Training in Session Three, although the mean decrease in heart rate from resting to training periods of .56 BPM ($p = .01$; Table 13) seen in this session was smaller than that seen in the first session (1.02 BPM). Also as in Session One, individual ANOVA's indicated that of the three groups, only the Control Ss significantly slowed their heart rate during the training periods ($p = .05$; Table 14). There was not, however, a Training x Group interaction in Session Three, indicating that the decrease in heart rate achieved by each of the three groups during the training periods in this session did not differ significantly from each other; nor was there a Training x Feedback interaction, indicating that the mean

TABLE 11

Individual Group ANOVA's for Change in Heart Rate

During Training Periods: Session One

AMI Group

Source	df	MS	F
Early vs. Late (R)	1	49.61	12.20*
Error	9	4.06	
Feedback vs. No Feedback (S)	1	7.08	1.52
Error	9	4.63	
R x S	1	13.77	3.63
Error	9	3.79	
Resting vs. Training (T)	1	2.52	1.51
Error	9	1.66	
R x T	1	0.45	1.01
Error	9	0.44	
S x T	1	2.17	3.06
Error	9	0.71	
R x S x T	1	1.20	0.58
Error	9	2.04	

Control Group

Source	df	MS	F
Early vs. Late (R)	1	32.51	9.39**
Error	9	3.46	

(TABLE 11 - Cont'd)

Source	df	MS	F
Feedback vs. No Feedback (S)	1	0.68	0.40
Error	9	1.68	
R x S	1	12.32	3.49
Error	9	3.52	
Resting vs. Training (T)	1	65.88	16.51*
Error	9	3.98	
R x T	1	0.54	0.37
Error	9	1.44	
S x T	1	1.01	0.83
Error	9	1.21	
R x S x T	1	13.61	6.16**
Error	9	2.20	

PMI Group

Source	df	MS	F
Early vs. Late (R)	1	5.77	1.20
Error	9	4.77	
Feedback vs. No Feedback (S)	1	2.70	1.40
Error	9	1.92	
R x S	1	0.99	0.51
Error	9	1.93	

(TABLE 11 - Cont'd)

Source	df	MS	F
Resting vs. Training (T)	1	15.57	4.52
Error	9	3.44	
R x T	1	0.25	0.26
Error	9	0.96	
S x T	1	1.71	1.54
Error	9	1.10	
R x S x T	1	0.78	1.13
Error	9	0.68	

* $p < .01$
** $p < .05$

TABLE 12

Paired Group Comparison ANOVA's for Change in Heart Rate During Training Periods: Session One

AMI and Control Groups

Source	df	MS	F
Between			
Groups (G)	1	88.50	0.04
Error	18	1775.14	
Within			
Early vs. Late (R)	1	81.22	21.58*
R x G	1	0.90	0.23
Error	18	3.76	
Feedback vs No Feedback (S)	1	6.08	1.92
S x G	1	1.68	0.53
Error	18	3.15	
R x S	1	26.08	7.13***
R x S x G	1	0.02	0.01
Error	18	3.65	
Resting vs. Training (T)	1	47.08	16.64*
T x G	1	21.31	7.53***
Error	18	2.82	
R x T	1	0.01	0.01
R x T x G	1	0.99	1.04
Error	18	0.94	
S x T	1	3.08	3.19
S x T x G	1	0.11	0.11
Error	18	0.96	

(TABLE 12 - Cont'd)

Source	df	MS	F
R x S x T	1	11.44	5.38**
R x S x T x G	1	3.36	1.58
Error	18	2.12	

AMI and PMI Groups

Source	df	MS	F
Between			
Groups (G)	1	1843.48	0.96
Error	18	1901.80	
Within			
Early vs. Late (R)	1	44.62	10.09**
R x G	1	10.76	2.43
Error	18	4.42	
Feedback vs. No Feedback (S)	1	0.51	0.15
S x G	1	9.26	2.82
Error	18	3.27	
R x S	1	11.07	3.86
R x S x G	1	3.69	1.28
Error	18	2.86	
Resting vs. Training (T)	1	15.31	5.99***
T x G	1	2.78	1.08
Error	18	2.55	
R x T	1	0.68	0.97
R x T x G	1	0.01	0.01
Error	18	0.70	

(TABLE 12 - Cont'd)

Source	df	MS	F
S x T	1	3.87	4.26
S x T x G	1	0.01	0.01
Error	18	0.90	
R x S x T	1	0.02	0.01
R x S x T x G	1	1.95	1.43
Error	18	1.36	

PMI and Control Groups

Source	df	MS	F
Between			
Groups (G)	1	2739.85	1.52
Error	18	1800.65	
Within			
Early vs. Late (R)	1	32.85	7.97***
R x G	1	5.43	1.32
Error	18	4.11	
Feedback vs. No Feedback (S)	1	0.33	0.18
S x G	1	3.05	1.69
Error	18	1.80	
R x S	1	10.15	3.72
R x S x G	1	3.16	1.15
Error	18	2.72	
Resting vs. Training (T)	1	72.76	19.58*
T x G	1	8.69	2.34
Error	18	3.71	

(TABLE 12 - Cont'd)

Source	df	MS	F
R x T	1	0.02	0.02
R x T x G	1	0.77	0.63
Error	18	1.20	
S x T	1	2.67	2.30
S x T x G	1	0.04	0.03
Error	18	1.16	
R x S x T	1	3.93	2.72
R x S x T x G	1	10.45	7.22***
Error	18	1.44	

* $p < .001$
** $p < .01$
*** $p < .05$

TABLE 13

ANOVA for Change in Heart Rate

During Training Periods: All Groups, Session Three

Source	df	MS	F
Between			
Groups (G)	2	2151.13	1.59
Error	27	1350.17	
Within			
Early vs. Late (R)	1	102.31	23.41*
R x G	2	1.53	0.35
Error	27	4.36	
Feedback vs. No Feedback (S)	1	0.63	0.15
S x G	2	6.53	1.57
Error	27	4.14	
R x S	1	57.91	35.24*
R x S x G	2	3.04	1.85
Error	27	1.64	
Resting vs. Training (T)	1	18.64	7.19**
T x G	2	5.36	2.06
Error	27	2.59	
R x T	1	0.67	0.46
R x T x G	2	1.05	0.71
Error	27	1.46	
S x T	1	2.88	3.17
S x T x G	2	1.70	1.87
Error	27	0.90	

(TABLE 13 - Cont'd)

Source	df	MS	F
R x S x T	1	5.43	5.31**
R x S x T x G	2	1.95	1.91
Error	27	1.02	

* $p < .001$
** $p < .05$

TABLE 14

Individual Group ANOVA's for Change in Heart Rate

During Training Periods: Session Three

AMI Group

Source	df	MS	F
Early vs. Late (R)	1	23.32	7.49**
Error	9	3.11	
Feedback vs. No Feedback (S)	1	5.10	2.00
Error	9	2.54	
R x S	1	17.86	8.52**
Error	9	2.09	
Resting vs. Training (T)	1	0.05	0.02
Error	9	1.99	
R x T	1	0.98	0.17
Error	9	0.54	
S x T	1	3.12	3.82
Error	9	0.81	
R x S x T	1	0.48	0.51
Error	9	0.93	

Control Group

Source	df	MS	F
Early vs. Late (R)	1	52.16	7.57**
Error	9	6.88	
Feedback vs. No Feedback (S)	1	7.93	1.63
Error	9	4.84	

(TABLE 14 - Cont'd)

Source	df	MS	F
R x S	1	38.64	16.42*
Error	9	2.35	
Resting vs. Training (T)	1	23.54	5.79**
Error	9	4.06	
R x T	1	2.66	1.35
Error	9	1.96	
S x T	1	2.88	5.47
Error	9	0.52	
R x S x T	1	8.71	6.33
Error	9	1.37	

PMI Group

Source	df	MS	F
Early vs. Late (R)	1	29.89	9.62*
Error	9	3.10	
Feedback vs. No Feedback (S)	1	0.66	0.13
Error	9	5.06	
R x S	1	7.50	15.54*
Error	9	0.48	
Resting vs. Training (T)	1	5.77	3.36
Error	9	1.71	
R x T	1	0.01	0.01
Error	9	1.86	

(TABLE 14 - Cont'd)

Source	df	MS	F
S x T	1	0.27	0.19
Error	9	1.38	
R x S x T	1	0.15	0.20
Error	9	0.75	

* $p < .01$
** $p < .05$

decrease in heart rate achieved by Ss under the instruction-only condition did not differ significantly from the decrease achieved when the same instructions were accompanied by the feedback display.

Sessions One and Three. In addition to the analyses of Sessions One and Three described above, data from these two sessions were put into a single ANOVA in order to compare the slowing performance of the Ss during these two sessions, and also to look at the results of combining data from the two slowing sessions in a single analysis. This analysis is summarized in Table 15.

As with both Sessions One and Three analyzed separately, there was a main effect of Training when the data from these two sessions were analyzed together. The mean amount of decrease in heart rate from resting to training periods for the two slowing sessions was .79 BPM ($p < .01$). However, there was no significant interaction between Session and Training, nor was there a significant Session x Training x Group interaction, indicating that the difference between the Training effects in Sessions One and Three was not significant, either for all 30 Ss or for any of the three groups.

There was a significant Training x Feedback interaction ($p < .01$), confirming the expectation that the amount of decrease in heart rate achieved during training periods would be greater when feedback accompanied instruction to slow heart rate than when the instruction was presented alone (1.04 BPM and .54 BPM, respectively). There

TABLE 15

ANOVA for Change in Heart Rate

During Training Periods: All Groups, Sessions One and Three

Source	df	MS	F
Between			
Groups (G)	2	3657.14	1.20
Error	27	3027.21	
Within			
Sessions (R)	1	188.50	1.26
R x G	2	51.27	0.34
Error	27	148.82	
Early vs. Late (S)	1	177.87	34.03*
S x G	2	3.55	0.68
Error	27	5.22	
R x S	1	0.93	0.28
R x S x G	2	3.68	1.13
Error	27	3.24	
Feedback vs. No Feedback (T)	1	0.03	0.01
T x G	2	8.53	1.96
Error	27	4.34	
R x T	1	1.72	0.67
R x T x G	2	2.66	1.04
Error	27	2.54	
S x T	1	76.32	33.03*
S x T x G	2	4.73	2.04
Error	27	2.31	

(TABLE 15 - Cont'd)

Source	df	MS	F
R x S x T	1	4.10	1.70
R x S x T x G	2	0.60	0.24
Error	27	2.41	
Resting vs. Training (U)	1	74.41	21.31*
U x G	2	15.74	4.50***
Error	27	3.49	
R x U	1	6.34	2.97
R x U x G	2	0.55	0.25
Error	27	2.13	
S x U	1	0.57	0.59
S x U x G	2	0.03	0.03
Error	27	0.96	
R x S x U	1	0.16	0.11
R x S x U x G	2	1.60	1.10
Error	27	1.45	
T x U	1	7.55	7.93**
T x U x G	2	0.83	0.87
Error	27	0.95	
R x T x U	1	0.12	0.12
R x T x U x G	2	0.92	0.95
Error	27	0.96	
S x T x U	1	10.50	6.35***
S x T x U x G	2	6.63	4.01***
Error	27	1.65	
R x S x T x U	1	0.01	0.01
R x S x T x U x G	2	0.58	0.57
Error	27	1.01	

* $p < .001$
** $p < .01$
*** $p < .05$

was also a significant Training x Group interaction ($p < .05$). Individual ANOVA's indicated that only the Controls showed a significant mean decrease (of 1.45 BPM) from resting to training periods ($p < .01$), whereas the mean decrease (of .71 BPM) among the PMI Ss only approached significance ($p = .06$), and the mean decrease (of .20 BPM) among the AMI Ss was not significant (Table 16). Paired comparisons among the three groups also indicated that only the Control and AMI groups differed significantly from each other in the amount of slowing achieved by each during the training periods ($p < .01$); neither the difference between the AMI and PMI groups, nor that between the PMI and Control groups, was significant (Table 17).

Session Two (Speeding). The data indicated that a significant Training effect did not occur during this session; the mean increase in heart rate of all 30 Ss between resting and training periods was only .34 BPM, and neither Training x Feedback nor Training x Group interactions were significant (Table 18).

Comparing the single speeding session with each of the two slowing sessions in turn, however, made it apparent that some difference in the Training effect was attributable to the different sets of instructions (i.e., to slow or to speed heart rate). Both when Sessions One and Two, and when Sessions Two and Three, were compared, there was a significant interaction between Session and

TABLE 16

Individual Group ANOVA's for Change in Heart Rate

During Training Periods: Sessions One and Three

AMI Group

Source	df	MS	F
Sessions (R)	1	12.76	0.05
Error	9	253.50	
Early vs. Late (S)	1	70.49	24.48*
Error	9	2.87	
R x S	1	2.45	0.57
Error	9	4.29	
Feedback vs. No Feedback (T)	1	12.10	2.33
Error	9	5.19	
R x T	1	0.08	0.04
Error	9	1.98	
S x T	1	31.50	9.68***
Error	9	3.25	
R x S x T	1	0.13	0.05
Error	9	2.63	
Resting vs. Training (U)	1	1.64	0.61
Error	9	2.65	
R x U	1	0.93	0.91
Error	9	1.01	
S x U	1	0.06	0.06
Error	9	0.93	

(TABLE 16 - Cont'd)

Source	df	MS	F
R x S x U	1	0.48	7.86***
Error	9	0.06	
T x U	1	5.25	7.89***
Error	9	0.66	
R x T x U	1	0.04	0.04
Error	9	0.86	
S x T x U	1	1.60	0.66
Error	9	2.40	
R x S x T x U	1	0.08	0.14
Error	9	0.57	

Control Group

Source	df	MS	F
Sessions (R)	1	262.14	4.79
Error	9	54.68	
Early vs. Late (S)	1	83.52	11.83
Error	9	7.05	
R x S	1	1.15	0.35
Error	9	3.29	
Feedback vs. No Feedback (T)	1	1.98	0.67
Error	9	2.93	
R x T	1	6.64	1.85
Error	9	3.58	

(TABLE 16 - Cont'd)

Source	df	MS	F
S x T	1	47.30	21.05*
Error	9	2.24	
R x S x T	1	3.66	1.00
Error	9	3.62	
Resting vs. Training (U)	1	84.09	24.86*
Error	9	3.38	
R x U	1	5.32	1.14
Error	9	4.66	
S x U	1	0.40	0.50
Error	9	0.79	
R x S x U	1	2.80	1.07
Error	9	2.61	
T x U	1	3.66	6.97**
Error	9	0.52	
R x T x U	1	0.24	0.19
Error	9	1.21	
S x T x U	1	22.05	11.16**
Error	9	1.97	
R x S x T x U	1	0.27	0.16
Error	9	1.60	

PMI Group

Source	df	MS	F
Sessions (R)	1	16.12	0.11
Error	9	138.29	

(TABLE 16 - Cont'd)

Source	df	MS	F
Early vs. Late (S)	1	30.97	5.39***
Error	9	5.74	
R x S	1	4.69	2.18
Error	9	2.14	
Feedback vs. No Feedback (T)	1	3.02	0.61
Error	9	4.91	
R x T	1	0.34	0.16
Error	9	2.06	
S x T	1	6.97	4.87
Error	9	1.43	
R x S x T	1	1.52	1.54
Error	9	0.98	
Resting vs. Training (U)	1	20.16	4.54****
Error	9	4.43	
R x U	1	1.19	1.66
Error	9	0.71	
S x U	1	0.18	0.15
Error	9	1.16	
R x S x U	1	0.08	0.04
Error	9	1.67	
T x U	1	0.30	0.18
Error	9	1.66	
R x T x U	1	1.68	2.04
Error	9	0.82	

(TABLE 16 - Cont'd)

Source	df	MS	F
S x T x U	1	0.12	0.20
Error	9	0.57	
R x S x T x U	1	0.81	0.94
Error	9	0.86	

* $p < .001$
** $p < .01$
*** $p < .05$
**** $p = .06$

TABLE 17

Paired Group Comparison ANOVA's for Change in Heart Rate During Training Periods: Sessions One and Three

AMI and Control Groups

Source	df	MS	F
Between			
Groups (G)	1	494.01	0.16
Error	18	2911.71	
Within			
Sessions (R)	1	195.31	1.26
R x G	1	79.60	0.51
Error	18	154.09	
Early vs. Late (S)	1	153.73	30.93*
S x G	1	0.27	0.05
Error	18	4.96	
R x S	1	0.12	0.03
R x S x G	1	3.48	0.91
Error	18	3.79	
Feedback vs. No Feedback (T)	1	2.14	0.52
T x G	1	11.93	2.93
Error	18	4.06	
R x T	1	4.09	1.47
R x T x G	1	2.62	0.94
Error	18	2.78	
S x T	1	78.01	28.37*
S x T x G	1	0.80	0.29
Error	18	2.74	

(TABLE 17 - Cont'd)

Source	df	MS	F
R x S x T	1	2.59	0.82
R x S x T x G	1	1.20	0.32
Error	18	3.13	
Resting vs. Training (U)	1	54.61	18.09*
U x G	1	31.12	10.31**
Error	18	3.01	
R x U	1	5.35	1.88
R x U x G	1	0.90	0.31
Error	18	2.84	
S x U	1	0.39	0.45
S x U x G	1	0.07	0.08
Error	18	0.86	
R x S x U	1	0.48	0.35
R x S x U x G	1	2.81	2.10
Error	18	1.33	
T x U	1	8.84	14.86*
T x U x G	1	0.07	0.12
Error	18	0.59	
R x T x U	1	0.24	0.23
R x T x U x G	1	0.04	0.03
Error	18	1.03	
R x S x T x U	1	0.32	0.29
R x S x T x U x G	1	0.02	0.02
Error	18	1.09	

(TABLE 17 - Cont'd)

AMI and PMI Groups

Source	df	MS	F
Between			
Groups (G)	1	3649.05	1.18
Error	18	3074.73	
Within			
Sessions (R)	1	28.79	0.14
R x G	1	0.09	0.01
Error	18	195.89	
Early vs Late (S)	1	97.46	22.61*
S x G	1	4.00	0.92
Error	18	4.31	
R x S	1	0.18	0.05
R x S x G	1	6.96	2.16
Error	18	3.22	
Feedback vs. No Feedback (T)	1	1.51	0.29
T x G	1	13.61	2.69
Error	18	5.05	
R x T	1	0.04	0.02
R x T x G	1	0.37	0.18
Error	18	2.02	
S x T	1	34.06	14.54*
S x T x G	1	4.41	1.88
Error	18	2.34	
R x S x T	1	1.27	0.70
R x S x T x G	1	0.37	0.20
Error	18	1.80	

(TABLE 17 - Cont'd)

Source	df	MS	F
Resting vs. Training (U)	1	16.65	4.69***
U x G	1	5.15	1.45
Error	18	3.54	
R x U	1	2.11	2.44
R x U x G	1	0.01	0.01
Error	18	0.86	
S x U	1	0.23	0.22
S x U x G	1	0.15	0.14
Error	18	1.04	
R x S x U	1	0.48	0.55
R x S x U x G	1	0.08	0.09
Error	18	0.86	
T x U	1	4.05	3.47
T x U x G	1	1.51	1.29
Error	18	1.16	
R x T x U	1	0.59	0.70
R x T x U x G	1	1.12	1.33
Error	18	0.84	
S x T x U	1	0.42	0.28
S x T x U x G	1	1.30	0.87
Error	18	1.49	
R x S x T x U	1	0.19	0.26
R x S x T x U x G	1	0.70	0.97
Error	18	0.71	

(TABLE 17 - Cont'd)

PMI and Control Groups

Source	df	MS	F
Between			
Groups (G)	1	6828.36	2.20
Error	18	3095.20	
Within			
Sessions (R)	1	204.16	2.11
R x G	1	74.11	0.76
Error	18	96.48	
Early vs. Late (S)	1	108.11	16.89*
S x G	1	6.38	0.99
Error	18	6.40	
R x S	1	5.25	1.93
R x S x G	1	0.59	0.21
Error	18	2.71	
Feedback vs. No Feedback (T)	1	4.95	1.26
T x G	1	0.05	0.01
Error	18	3.92	
R x T	1	1.98	0.70
R x T x G	1	5.00	1.76
Error	18	2.82	
S x T	1	45.30	24.63*
S x T x G	1	8.97	4.88***
R x S x T	1	4.95	2.14
R x S x T x G	1	0.23	0.10
Error	18	2.30	

(TABLE 17 - Cont'd)

Source	df	MS	F
Resting vs Training (U)	1	93.31	23.86*
U x G	1	10.95	2.80
Error	18	3.91	
R x U	1	5.77	2.14
R x U x G	1	0.74	0.27
Error	18	2.69	
S x U	1	0.56	0.57
S x U x G	1	0.02	0.02
Error	18	0.97	
R x S x U	1	0.96	0.45
R x S x U x G	1	1.92	0.89
Error	18	2.14	
T x U	1	3.04	2.77
T x U x G	1	0.92	0.84
Error	18	1.09	
R x T x U	1	0.32	0.31
R x T x U x G	1	1.54	1.56
Error	18	1.02	
S x T x U	1	9.45	7.40***
S x T x U x G	1	12.72	9.96**
Error	18	1.27	
R x S x T x U	1	0.07	0.05
R x S x T x U x G	1	1.01	0.82
Error	18	1.23	

* $p < .001$
** $p < .01$
*** $p < .05$

TABLE 18

ANOVA for Change in Heart Rate

During Training Periods: All Groups, Session Two

Source	df	MS	F
Between			
Groups (G)	2	1054.90	0.79
Error	27	1330.71	
Within			
Early vs. Late (R)	1	55.77	8.69**
R x G	2	0.15	0.24
Error	27	6.41	
Feedback vs. No Feedback (S)	1	60.30	21.68*
S x G	2	1.57	0.56
Error	27	2.78	
R x S	1	11.48	8.21**
R x S x G	2	0.97	0.69
Error	27	1.39	
Resting vs. Training (T)	1	6.97	1.95
T x G	2	1.33	0.37
Error	27	3.57	
R x T	1	5.92	3.78
R x T x G	2	0.97	0.62
Error	27	1.56	
S x T	1	2.46	1.22
S x T x G	2	0.22	0.11
Error	27	2.01	

(TABLE 18 - Cont'd)

Source	df	MS	F
R x S x T	1	2.30	1.80
R x S x T x G	2	0.03	0.02
Error	27	1.27	

* $p < .001$
** $p < .01$

Training ($p < .01$; Tables 19 and 20); whereas no such Session x Training effect was apparent when Sessions One and Three were compared, in which the instructions were the same, that is, to slow heart rate (Table 15). The presence of a significant Session x Training interaction when a comparison was made between sessions with different sets of instructions, but not when a comparison was made between sessions with the same set of instructions, indicated that the Training effect which occurred when Ss were instructed to slow their hearts differed significantly from that which occurred when Ss were instructed to speed their hearts.

Cardiac Control and Cardiac Lability

For purposes of investigating the relationship between ability to control heart rate and degree of cardiac lability, and also between ability to control heart rate and each of the personality variables measured, cardiac control was defined only in reference to heart rate slowing achieved with the assistance of feedback, that is, under circumstances most favorable to control. Differences between mean resting and instruction-with-feedback heart rate were computed both for Sessions One and Three separately, and for Sessions One and Three averaged. In addition, a classification of all Ss was made into Responder and Non-responder categories, on the basis of whether or not a S achieved a mean decrease of at least 1.00 BPM during the two

TABLE 19

ANOVA for Change in Heart Rate During Training Periods:

All Groups, Sessions One and Two

Source	df	MS	F
Between			
Groups (G)	2	2587.58	0.85
Error	27	3036.33	
Within			
Sessions (R)	1	47.25	0.39
R x G	2	24.60	0.20
Error	27	120.24	
Early vs. Late (S)	1	131.46	21.45*
S x G	2	3.41	0.55
Error	27	6.12	
R x S	1	0.81	0.18
R x S x G	2	2.43	0.55
Error	27	4.38	
Feedback vs. No Feedback (T)	1	38.98	13.33*
T x G	2	2.24	0.76
Error	27	2.92	
R x T	1	22.44	8.62**
R x T x G	2	3.99	1.53
Error	27	2.60	
S x T	1	33.07	12.40**
S x T x G	2	1.41	0.53
Error	27	2.66	

(TABLE 19 - Cont'd)

Source	df	MS	F
R x S x T	1	0.91	0.50
R x S x T x G	2	1.84	1.01
Error	27	1.81	
Resting vs. Training (U)	1	13.73	4.36***
U x G	2	9.76	3.10
Error	27	3.14	
R x U	1	55.35	16.01*
R x U x G	2	2.50	0.72
Error	27	3.45	
S x U	1	3.60	2.75
S x U x G	2	0.02	0.01
Error	27	1.30	
R x S x U	1	2.38	1.97
R x S x U x G	2	1.54	1.27
Error	27	1.20	
T x U	1	0.19	0.13
T x U x G	2	0.18	0.13
Error	27	1.37	
R x T x U	1	7.05	4.28***
R x T x U x G	2	0.19	0.05
Error	27	1.64	
S x T x U	1	7.10	3.79
S x T x U x G	2	2.66	1.42
Error	27	1.87	

(TABLE 19 - Cont'd)

Source	df	MS	F
R x S x T x U	1	0.27	0.25
R x S x T x U x G	2	2.62	2.50
Error	27	1.04	

* $p < .001$
** $p < .01$
*** $p < .05$

TABLE 20

ANOVA for Change in Heart Rate During Training Periods:

All Groups, Sessions Two and Three

Source	df	MS	F
Between			
Groups (G)	2	3091.49	1.22
Error	27	2527.06	
Within			
Sessions (R)	1	47.00	0.30
R x G	2	114.54	0.74
Error	27	153.82	
Early vs. Late (S)	1	154.58	22.78*
S x G	2	0.42	0.06
Error	27	6.78	
R x S	1	3.50	0.87
R x S x G	2	1.26	0.31
Error	27	3.99	
Feedback vs. No Feedback (T)	1	24.30	5.96***
T x G	2	6.08	1.49
Error	27	4.07	
R x T	1	36.63	12.81*
R x T x G	2	2.02	0.71
Error	27	2.85	
S x T	1	60.49	40.08*
S x T x G	2	2.87	1.90
Error	27	1.50	
R x S x T	1	8.91	5.81***
R x S x T x G	2	1.14	0.74
Error	27	1.53	

(TABLE 20 - Cont'd)

Source	df	MS	F
Resting vs. Training (U)	1	1.40	0.44
U x G	2	5.77	1.80
Error	27	3.19	
R x U	1	24.21	8.16**
R x U x G	2	0.92	0.31
Error	27	2.96	
S x U	1	5.29	2.33
S x U x G	2	2.02	0.89
Error	27	2.26	
R x S x U	1	1.30	1.72
R x S x U x G	2	0.01	0.01
Error	27	0.75	
T x U	1	0.01	0.01
T x U x G	2	1.47	1.05
Error	27	1.40	
R x T x U	1	5.33	3.50
R x T x U x G	2	0.44	0.29
Error	27	1.52	
S x T x U	1	7.40	6.57***
S x T x U x G	2	0.92	0.81
Error	27	1.12	
R x S x T x U	1	0.33	0.28
R x S x T x U x G	2	1.06	0.91
Error	27	1.17	

* $p < .001$
** $p < .01$
*** $p < .05$

instruction-with-feedback periods of Session One. A similar classification was also made on the basis of mean decrease during the same four periods of both Sessions One and Three. The Spearman rank-order correlation (for the entire sample) between cardiac control demonstrated on Session One and that demonstrated on Session Three was .42 ($p < .05$).

Since the Responder-Non-responder distinction was made on the basis of Ss' performance during Sessions One and Three only, cardiac lability was measured only during these two sessions as well. Lability was defined as the absolute difference between the highest and lowest heart rates recorded during the initial basal period of each of the sessions. The rank-order correlation between all 30 Ss' lability scores on Sessions One and Three was .40 ($p < .05$). There was not a significant difference (using the Kruskall-Wallis One Way Analysis of Variance by Ranks) among the lability scores of the three groups of Ss on either Sessions One or Three. The relationship between lability and cardiac control demonstrated during the two slowing sessions was different for each of the sessions. For Session One the rank-order correlation between lability and control was -.05 ($p < .05$); for Session Three the lability-control correlation was .69 ($p < .01$).

Cardiac Control and Personality

Several personality variables were also measured and related to

ability to control heart rate. These variables were: (1) anxiety; (2) awareness of autonomic reactivity; (3) Locus of Control; and two dimensions of body image, namely, (4) Barrier and (5) Penetration.

Anxiety. Anxiety was scored from the Ss' Rorschach protocols according to the scheme developed by Elizur (1949) and adopted by Holtzman et al. (1961). The scores were corrected for number of Rorschach responses. The Rorschach protocols were scored blind by Dr. Sidney E. Cleveland after they were obtained by the experimenter or an associate. The mean Anxiety scores of the three groups of Ss were: PMI, 7.6; AMI, 6.2; and Control, 3.1. Differences among these scores were found to be significant, using the Kruskall-Wallis test ($p = .02$). There was no difference (using the Mann-Whitney U test) between the Anxiety scores of Responders and Non-responders, regardless of whether the distinction was made on the basis of performance on Session One alone, or on average performance on Sessions One and Three. However, when the Ss were divided at the median score into Low Anxiety and High Anxiety groups (irrespective of sample group membership), and the training performance of these two groups during the two slowing sessions was compared, a different picture emerged.

On Sessions One and Three, there were differences, although not significant ones, between the mean decrease achieved by the Low and High Anxiety Ss during the instruction-with-feedback periods. For

Session One, the mean decrease was 1.80 BPM and .98 BPM, respectively ($t = 1.58$; $df = 25$; $p < .10$); for Session Three, it was 1.13 BPM and .41 BPM, respectively ($t = 1.33$; $df = 25$; $p = .10$). When the mean decrease from Sessions One and Three were averaged, and the Low and High Anxiety groups were compared, the difference between the mean decrease of 1.45 BPM for Low Anxiety Ss and of .70 BPM for High Anxiety Ss was found to be significant ($t = 1.95$; $df = 25$; $p < .05$). Although this was somewhat confounded by the fact that the Anxiety scores of the Control Ss were lower than those of both the AMI and PMI Ss, and it was the Control group which also showed a significant Training effect on both Sessions One and Three, it appeared nevertheless that the less anxious Ss, with the assistance of feedback, were able to slow their hearts significantly more than the more anxious Ss, at least when data from the two slowing sessions were combined.

Awareness of Autonomic Reactivity. This variable was measured with the self-administered Autonomic Awareness Questionnaire (Appendix B) and was scored twice: first, using the total of each S's weighted responses to all 21 questions concerning awareness of autonomic reactions at times of stress; and a second time, using the total of only those questions which related to possible cardiac symptoms (feelings in the chest; heart beating faster; heart beating harder than

usual). There were no differences among the three subject groups on either their Autonomic Awareness (total) or Autonomic Awareness (heart) scores (using the Kruskall-Wallis test); nor did Responders and Non-responders (on Session One or on Sessions One and Three averaged) differ on either of the two Autonomic Awareness scores (using the Mann-Whitney U test).

Locus of Control. This variable was measured by the Rotter I-E scale, as previously described (Appendix C). No differences in I-E scores were found either among the three groups of Ss (using the Kruskall-Wallis test), or between the Responder and Non-responder groups, both on Session One and on Sessions One and Three averaged (using the Mann-Whitney U test).

Barrier and Penetration. These two body image variables were scored by Dr. Sidney E. Cleveland in the manner described by Cleveland and Fisher (Cleveland and Fisher, 1968), using the Ss' Rorschach protocols. No differences were found, using the Median test, among either the Barrier or the Penetration scores of the three groups of Ss; nor did the Responder and Non-responder groups (on both Session One alone, and Sessions One and Three averaged) differ with respect to either Barrier or Penetration scores.

Summary

The principal finding of the present study was that a significant Training effect (that is, a change in heart rate in a desired direction

from resting to training periods) occurred during both of the slowing sessions, but not during the speeding session. On the first slowing session, but not on the second, there was also an interaction between the extent of the Training effect and the provision of feedback, such that the amount of change in heart rate that Ss achieved was greater when they received both instruction to slow heart rate and simultaneous visual feedback than when they received the same instruction but were not provided with the feedback. Moreover, the provision of feedback alone (that is, without any instruction to alter heart rate) was accompanied by a significant decrease in heart rate during those periods of both the first and second sessions in which Ss were provided only with the feedback.

Most important, follow-up analyses indicated that of the three groups of Ss, only the Control group showed a significant Training effect during both Sessions One and Three; the PMI group showed a near-significant Training effect during Session One but not Session Three; and the AMI group did not show an effect of Training during either of the two slowing sessions. Examination of the differences among the three groups in the amount of change attributable to Training which each group achieved revealed only one significant between-group difference in the magnitude of the Training effect: that between the AMI and Control groups on the first slowing session.

Not only were Ss unable to produce significant increases in heart rate when instructed to do so, either with or without feedback; but, in addition, heart rate did not increase significantly under two conditions intended to evoke acceleration of heart rate: an intervention during one of the resting periods in Sessions One and Three; and a mental (sentence construction) task at the beginning of the third session. Furthermore, there was a significant decline from initial to final basal heart rate during each of the three sessions, and the absence of an interaction between type of session (slowing or speeding) and difference between initial and final basal heart rate indicated that the decline during the single speeding session did not differ significantly from that during the two slowing sessions.

Lability of heart rate during the initial basal period was found to be significantly correlated with amount of slowing during the instruction-with-feedback periods of Session Three and Sessions One and Three averaged, but not of Session One. No subject group differences in lability during either Sessions One or Three were found.

Only one personality variable discriminated among the three groups of Ss: significant differences were found in Anxiety scores, with the PMI Ss being the most anxious and the Control Ss the least anxious. When all 30 Ss were divided into Responder and Non-responder categories on the basis of their ability to slow their hearts, no

differences were found in the ranked Anxiety scores of the Responder and Non-responder groups. However, when the Ss were classified into Low and High Anxiety categories, the less anxious Ss were significantly better able to slow their hearts (when the data from the two slowing sessions were averaged) than the more anxious Ss. None of the other personality variables (Autonomic Awareness, Locus of Control, and Barrier and Penetration scores) differentiated either among the original three groups of Ss (AMI, Control, and PMI) or between the Responder and Non-responder categories. The implications of these findings will be discussed in the next chapter.

CHAPTER V

DISCUSSION

In discussing the results presented in the last chapter, it might be wise to begin by stating the salient questions which have emerged from these findings.

The first (and most important) question involves accounting for the observed differences between the three groups in their ability to slow their heart rate voluntarily. What do the results of this study suggest regarding why the patients with heart disease (diagnosed myocardial infarctions) performed differently in the slowing sessions from the Control Ss, who had no detectable cardiac disease; and why did the performance of those heart patients who were still in the convalescent phase following a first infarction differ from the performance of those who had suffered one (or more than one) infarction at least three months before?

The next question relates to the entire sample of 30 Ss: why was the magnitude of change achieved by those Ss who successfully slowed their hearts so small? Although a significant Training effect was found in the slowing sessions, the mean decrease in heart rate was in the neighborhood of one to two beats per minute. What features of the procedure utilized in this experiment might account for the

small size of the changes which these Ss brought about, compared to those found in other studies?

The last question is why the Ss failed to demonstrate the ability to speed their heart rate voluntarily. Why was there no Training effect in the heart rate speeding session, and how consistent is this with what has been found by other researchers?

Group Differences in Slowing

It will be recalled that a significant Training effect was found in both slowing sessions, as well as when data from the slowing sessions were combined in a single analysis. The Control group, however, was the only one consistently to achieve significant voluntary decreases in heart rate; the voluntary slowing of the PMI Ss in Session One (and Sessions One and Three combined) closely approached significance; and the AMI Ss' slowing performance was negligible and not significant in either slowing session.

Whereas Lang et al. (1975) found that age-matched control Ss tended to be superior to Ss with ischemic heart disease when data on both slowing and speeding were analyzed together, the present study indicated a hierarchical ordering of subject groups for slowing only, with the Control Ss being the best slowers, followed by the PMI Ss and, in last place, the AMI Ss. Since the ages of the Ss did not differ significantly from group to group, there was apparently something

about having had a myocardial infarction that made the cardiac disease Ss (AMI and PMI) less capable of voluntarily slowing their heart rate than the non-cardiac (i.e., Control) Ss.

Furthermore, the recency of the infarction appeared to be relevant to this capability as well, since some degree of voluntary control was recovered after the post-infarction convalescent phase. This occurred even though four of the 10 PMI Ss had a history of having had more than one infarction in the past, suggesting the presence of more extensive myocardial damage than existed among the AMI Ss, who were all convalescing from an uncomplicated first infarction. How can these findings be accounted for, and how do they compare with the study of Lang and his associates (1975)?

One difference between the two studies was that the Control Ss in the present study were all inpatients at the time, whereas in the Lang study the controls were healthy volunteers. The selection of the Control Ss in the present study from a patient population, however, was intended as an additional control on the comparability of the three groups, by equating for the possible effects of patient status (a variable which was not controlled in the Lang study).

Both cardiac disease patient groups in the present study also differed from the heart disease group in Lang and associates' study in that the latter was less homogeneous than either the AMI or the

PMI group. In the Lang et al. study, the only apparent criteria for inclusion in the heart disease group were a history of ischemic heart disease, ambulatory status, and (if the prospective S had suffered an infarction) not being convalescent. Approximately one-third of that group had not had an infarction, and almost one-third had undergone revascularization (i.e., bypass) surgery. In contrast, none of the cardiac disease Ss in the present study had undergone cardiac surgery, and all had suffered documented infarctions. Of the 10 AMI Ss, eight were currently hospitalized with a diagnosis of acute myocardial infarction, and two had recently been discharged from the hospital with the same diagnosis. All were less than one month post-MI and were still convalescing; and none had a history (or any EKG evidence) of having had a past infarction. Any patient whose recovery was other than uncomplicated, or who was taking any medication with a direct effect on heart rate, did not meet the criteria for the AMI group. The AMI Ss, then, differed from Lang and associates' heart disease Ss both in terms of their greater homogeneity and in terms of the acuteness of their condition (i.e., the recency of the infarction).

The PMI Ss were all past the convalescent stage (at least three months post-MI) at the time of the study. Of the 10 PMI Ss, seven were participating (three as outpatients and four as inpatients) in a

cardiac rehabilitation program at the hospital. In addition, four PMI Ss had had more than one infarction; and only one (not a participant in the rehabilitation program) had returned to work. Because it would have eliminated too many prospective PMI Ss, no restrictions were placed on medications. The absence of exclusive drug criteria for the PMI group also made this group more similar to the post-MI patients usually encountered in medical practice.

Because Lang and his associates failed to specify the medical condition of the heart disease Ss in their sample, it is impossible to judge which group (his patient Ss, or the AMI and PMI Ss in the present study) was the more seriously ill. It is definite, however, that the AMI Ss were the more acutely ill; and because of the stringent criteria for their selection, they were more homogeneous (insofar as cardiac condition was concerned) than were Lang and associates' Ss. The PMI Ss more closely resembled the heart disease subjects in the Lang et al. study in that all the PMI Ss were post-convalescent and no restrictions were placed on what medications they could be taking. The PMI Ss were more varied than the AMI Ss as well, in terms of cardiotropic medications, number of past infarctions, past complications, and length of time since infarction (or last infarction).

The finding that only the Control Ss in the present study consistently achieved significant voluntary decreases in heart rate is

consistent with Lang and associates' finding that "patients with a history of heart disease profited least from instructions and training, and showed the poorest performance on both heart rate speeding and slowing [Lang, Troyer, Twentyman and Gatchel, 1975, p. 443]." Whereas the Lang et al. study showed that age as well as heart disease was a factor in the ability to control heart rate voluntarily (by demonstrating the superiority of college student Ss over older, healthy Ss), the present study gave further support to the hypothesis that cardiac disease would interfere with the voluntary ability to control heart rate when the effects of age were controlled.

The results of the two studies, however, do not help us to understand why the PMI Ss' ability to slow their heart rate approached significance more closely than that of the AMI Ss. The AMI group was selected from a much larger number of patients admitted with first myocardial infarctions. The criteria that the infarctions be uncomplicated and that the patients not be taking cardiotropic drugs (except for vasodilators) excluded the most seriously ill and limited the AMI group to patients with cardiac damage of mild to moderate severity. The PMI group, in contrast, included four persons who had had more than a single infarction; seven PMI Ss were taking one or more cardiotropic drugs with direct effects on heart rate; and several had histories of complications during recovery from infarction. Why did these persons do better on the slowing task than the less seriously, but more acutely, ill AMI Ss?

Clearly, the data implicated the recency of the infarction as somehow having limited the ability of the AMI Ss to lower their heart rate voluntarily. There were two principal ways in which the recency of an infarction could have interfered with the capability for cardiac self-regulation: because of purely physiological differences between these (AMI) Ss and the PMI Ss; or because of psychological (i.e., emotional and/or motivational) differences between the two groups, that interacted with the Ss' cardiac physiology in such a way as to cause the observed differences in performance.

The first possibility is that some structural or functional factor was related to the slowing performance of the AMI Ss being poorer than that of the PMI Ss. The PMI Ss' hearts were, however, more extensively damaged and functionally compromised than those of the AMI Ss, since several PMI Ss had had more than one infarction, and most required one or more cardiotropic drugs for cardiac arrhythmias or heart failure. Although the AMI Ss' hearts had not yet completely healed at the time of their participation in the experiment (causing their myocardia to be more irritable than the healed myocardia of the PMI Ss), none of them had an anatomic interruption between atria and ventricles such as complete heart block, which has been shown to be a condition precluding voluntary heart rate control (Weiss & Engel, 1975).

Such a physiological explanation would, therefore, have to postulate a temporary change in the heart, which either disappeared following convalescence from an infarction or to which patients became adapted, and which could account for the difference in slowing performance between the AMI and PMI Ss. The effects of such a temporary change in the heart would have had to be specific to the voluntary alteration of heart rate, since no differences were found among the three groups of Ss in the amount of heart rate slowing which occurred during the orienting periods; that is, the mean heart rate of the AMI group slowed as much as that of the PMI group when the change was evoked by an external stimulus, although the AMI Ss were unable to emit a similar slowing response voluntarily when instructed to do so. In this same regard, no differences in resting heart rate lability were found between the AMI and PMI groups. No structural or functional difference between these groups existed which could have accounted for this pattern of findings on a purely physiological basis. The fact that the AMI Ss responded no differently to stimulation (i.e., feedback without instruction) than did the PMI Ss, but were less able than the PMI Ss to emit voluntary decelerative changes in their heart rate, points toward a model which would postulate an interaction of psychological and physiological factors to explain why the AMI Ss, who were convalescing from a

first, uncomplicated myocardial infarction, should have been less able than the PMI Ss to slow their heart rate voluntarily.

Influence of Personality Variables. The present study was not the first to assess the relationship of personality variables to the ability to control heart rate voluntarily. Among the personality variables which have received the attention of other researchers are Locus of Control (Lang, Troyer, Twentyman and Gatchel, 1975; Ray, 1974) and awareness of autonomic activity (Bergman and Johnson, 1971; Blanchard, Young and McLeod, 1972; McFarland, 1975). With the exception of the Lang et al. study, however, this research has utilized college student Ss exclusively.

Ray (1974), studying students chosen on the basis of their Locus of Control scores, found that internal Locus of Control Ss were better able to increase their heart rate than external Locus of Control Ss, and external Locus of Control Ss were better able to decrease their heart rate than internal Locus of Control Ss. Lang, Troyer, Twentyman and Gatchel (1975) found a near-significant correlation of .40 between Locus of Control and the slowing performance of Ss with heart disease, but no correlation between Locus of Control and voluntary heart rate control in either of their control groups.

Ray's tentative explanation of the association between external Locus of Control and superior slowing performance was that these

Ss adopted a strategy of looking at objects in the laboratory in their attempt to slow their heart rate, thus serendipitously confirming the hypothesis of Lacey, Kagan, Lacey and Moss (1963) that attentiveness to the external environment would result in an involuntary deceleration of heart rate (a finding reported in the present study as well). However, in view of Lang and associates' (1975) failure to replicate consistently Ray's findings, the absence of a relationship in the present study between Locus of Control and the ability to control heart rate voluntarily is not surprising.

With regard to the relationship between awareness of autonomic activity and the ability to control heart rate voluntarily, Bergman and Johnson (1971) found that Ss with middle scores on the Autonomic Perception Questionnaire (APQ) were better able to both increase and decrease their heart rate than Ss with either high or low APQ scores. It is significant that this study was done without providing external feedback to the Ss, suggesting that "the greater degree of control displayed by middle APQ score Ss over the high and low APQ groups may have been due to the greater accuracy of these Ss in perceiving autonomic activity [Bergman and Johnson, 1971, pp. 188-189]." No provision was made in this experiment for determining whether the addition of external feedback would have affected the relationship between APQ score and ability to control heart rate,

although it could have been hypothesized that removing the necessity for Ss to rely on their own perceptions of autonomic functioning would have resulted in a levelling of differences between the high, middle and low APQ groups.

Blanchard, Young and McLeod (1972), employing visual feedback of heart rate, found that Ss with low scores on only those items of the APQ relating to heart activity were able both to raise and lower their heart rate successfully, but that Ss with high scores on the heart-awareness items were unable to alter their heart rate in either direction. Although these researchers did not offer an explanation for their findings, McFarland (1975) found that scores on the heart-awareness items of the APQ were very poorly correlated ($r = .13$) with scores on a task designed to measure the accuracy of a S's perception of his heart rate by having him press a button in rhythm with his estimation of his heartbeat. While no relationship was found between APQ score and heart rate control, a significant relationship was found between accuracy of heart rate estimation and the ability to increase (but not decrease) heart rate.

Unfortunately, none of these studies clarified the question of what was being measured by the APQ; although the finding that APQ score was not a good indicator of accuracy of autonomic perception (McFarland, 1975) suggested that the APQ was more a measure of

concern or anxiety about bodily functioning than of accuracy of awareness. Again, the absence of a relationship in the present study between autonomic awareness and voluntary heart rate control was not discrepant with the findings of earlier studies, in view of the inconsistent results obtained by these other researchers.

The Role of Anxiety. It was not a surprising finding in the present study that Ss with low Anxiety scores (obtained as unobtrusively as possible, i.e., from the Ss' Rorschach responses) decreased their heart rate during the training periods significantly more than Ss with high Anxiety scores. In addition to the common-sense appeal of this finding, it was found that the Control group, which was the only one to achieve a consistently significant mean decrease in heart rate, also had a significantly lower mean Anxiety score than either the AMI or the PMI groups (whose slowing performance was, respectively, not significant and almost significant). However, the association between level of anxiety and slowing performance could not explain why the AMI Ss, who actually had a slightly lower mean anxiety score than the PMI Ss, were not able to decrease their heart rate significantly, while the PMI Ss were able to decrease their heart rate to a degree closely approaching significance, although no difference was found between the AMI and PMI groups in how much deceleration of heart rate was evoked by the feedback-without-instruction (orienting)

condition. A tentative explanation of these findings would have to consider the different effects of equivalent amounts of anxiety on the hearts of Ss in the AMI and PMI groups.

During the period following the occurrence of, and until the completion of convalescence from, a myocardial infarction (usually considered to be three months post-infarction), that portion of the myocardium which has been damaged by the infarct is more irritable to the effects of catecholamines (produced by the adrenal glands, in response to stress or anxiety) than it either was before infarction, or will be after healing has taken place (Cromwell, Butterfield, Brayfield and Curry, 1977). Since heart muscle does not replace itself, the process of healing consists of the replacement of damaged (and irritable) muscle by scar tissue, which is not irritable. The effect of catecholamines on the heart is an increase in myocardial oxygen requirements due to the creation of a temporary myocardial hypoxia, thus causing an accelerated heart rate. While the principle is the same in the undamaged, the newly damaged, and the damaged-but-healed heart, the increased irritability of the recently damaged myocardium implies that the net effect of an equivalent amount of catecholamines will be greater heart rate acceleration (and greater refractoriness of the heart to deceleration) among patients who are still convalescing from an infarction than among those whose areas of infarction have healed.

It is precisely by opposing catecholamine stimulation of the heart (through its action in blocking beta-adrenergic receptors in the myocardium) that the cardiotropic drug, propranolol, brings about a reduction in heart rate (Pitt, 1976). Whether the exclusion from the AMI group of patients taking propranolol (Inderal) had the effect of making the AMI Ss even more refractory to training in heart rate deceleration than they might have been if patients receiving a beta-adrenergic blocking agent had been included in this group cannot be assessed. The three PMI Ss who were taking propranolol at the time of the experiment did not achieve a greater degree of slowing than the other PMI Ss, contrary to the finding of Lang and his associates (1975) that patients who were taking cardiotropic drugs with a definite effect on heart rate showed better slowing performance than patients taking drugs with no known effect on heart rate. However, as Lang et al. (1975) also pointed out, the possible interaction between the severity of a patient's condition and the type of medication prescribed by his physician make the interpretation of such drug effects extremely difficult; and the only way in which definitive answers could be given to the questions thus raised would be by controlled studies in which cardiotropic medication was either experimentally or randomly administered to patients whose cardiac condition was clearly comparable.

A final comment on the failure of the relationship between Anxiety score and cardiac control to explain the difference in the slowing performance of the AMI and PMI Ss is that the measurement of anxiety by Rorschach responses may not have provided an accurate reflection of the Ss' level of anxiety at the time that they were supposed to be attempting to lower their heart rate. The idea of altering one's heart rate (and of viewing an instrument which measures this alteration) may have had a very different meaning to those Ss who recently had been discharged from the Coronary Care Unit, where changes in heart rate were closely monitored and frequently viewed as cause for alarm, than to Ss participating in a cardiac rehabilitation program which emphasized altered heart rate as one of its goals. The mechanism by which such momentary changes in anxiety could have affected the potential for heart rate deceleration has already been discussed.

Magnitude of Change

Another aspect of the slowing performance of the Ss which requires discussion is that even the Control Ss, who showed a significant Training effect in both slowing sessions, did not produce a mean decrease in heart rate of over two beats per minute, even under the conditions most conducive to self-regulation (i.e., simultaneous instruction and feedback). The PMI Ss, who showed a near-significant Training effect, produced still smaller mean

decreases during the training periods. Why were these changes no larger than they were?

Although a definitive answer to this question cannot be given, it should be remembered that the Ss were trained to slow their heart rate during two sessions only, and that the instruction with feedback segment of each session was only 960 IBI's in length (slightly less than a mean of 11 1/2 minutes per subject). The amount of deceleration in heart rate which the Ss were able to achieve should be evaluated in light of the limited amount of biofeedback training which they received. Although Lang and associates (1975) gave their Ss short periods of feedback training (three minutes per session), and found that the heart patients who were given extended training (six sessions, instead of the three sessions given to the other Ss) did not profit from the additional training, other researchers who have reported success in training cardiac patients to control their heart rate have given as many as 69 training sessions to a single patient (e.g., Bleecker & Engel, 1973a).

The present study, therefore, should be considered as having demonstrated that some degree of heart rate slowing could be accomplished with Ss other than young, healthy college students, using only a brief training procedure. It showed that a significant voluntary deceleration of heart rate could be achieved by a group of middle-aged

hospitalized Ss free of known cardiac disease, and that a group of similarly middle-aged Ss with a history of one or more myocardial infarctions in the past, none of whom had had caridac surgery, was able to achieve a near-significant deceleration of heart rate, with only a mean of 23 minutes of biofeedback training per subject in slowing cardiac rate. Before concluding that the relatively modest reduction of heart rate found in this study represents the upper limit of what these Ss were capable of achieving, further research utilizing a larger number of training sessions and experimenting with varying training formats would have to be carried out.

Failure to Speed

The last aspect of the study to be discussed relates to the single speeding session. As was described in the previous chapter, no Training effect for speeding was found in this study, either for the entire sample or for any of the three subject groups. How does this finding compare with what has been reported by other researchers and, if it differs from what others have reported, what might be the reason?

For the most part, those experiments in which the Ss have been able to produce significant increases in heart rate were conducted with healthy, college-age Ss (Bell & Schwartz, 1975; Blanchard & Young, 1972; Brener & Hothersall, 1966; Engel & Chism, 1967; Gatchel, 1974; Headrick, Feather & Wells, 1971; Lang & Twentyman,

1974; Levene, Engel & Pearson, 1968; Manuck, 1976; Stephens, Harris, Brady & Shaffer, 1975). Bleecker and Engel (1973a) and Weiss and Engel (1971) attempted to train patients with chronic atrial fibrillation and premature ventricular contractions (PVC's) to both slow and speed their heart rate. In the first study, two out of six Ss were unable to speed their hearts; two were able to achieve modest increases in heart rate (one to three beats per minute); and the remaining two produced more substantial increases. The Ss, however, received from 10 to 21 training sessions in speeding alone, plus additional training sessions in slowing and in alternating speeding and slowing. In the second study, only one of the eight Ss was consistently able to speed, and this subject (for reasons unknown to the researchers) was able to produce heart rate increases during afternoon but not during morning sessions. The number of speeding sessions per subject in this study ranged from six to 14, the researchers (in both of the latter two studies) varying the number of sessions from subject to subject in order to maximize the attainment of clinical objectives (e.g., a reduction in the frequency of PVC's).

Because in the latter two studies, "the patient's responses at any stage of the study always dictated the procedure [Weiss and Engel, 1971, p. 302]," these studies resembled two series of replicated clinical case studies more than controlled experimental

studies. The only controlled experimental study of heart rate modification among patients with heart disease and a control group of persons of comparable age without heart disease was made by Lang and his associates (Lang, Troyer, Twentyman and Gatchel, 1975), who compared the performance of college students, patients with ischemic heart disease, and healthy older volunteers free of any history or symptoms of cardiac disease.

Whereas many researchers have reported that college student Ss more easily learned to speed than to slow their heart rate (e.g., Engel and Chism, 1967; Headrick, Feather & Wells, 1971; Lang and Twentyman, 1974; Levene, Engel and Pearson, 1968; Manuck, 1976), Lang and associates' study indicated that the greater ease of speeding reported by these other researchers was largely associated with the young age of the subjects. Although the college students in the Lang et al. study were able to accelerate their heart rate successfully, the older controls did not show a significant amount of acceleration, in this respect resembling the heart disease Ss (who did not accelerate their heart rate at all) rather than the college students. The absence of a Training effect for speeding in the present study is, therefore, not at all discrepant with what has been found by other researchers, since the mean age of the Ss in the present study (52.7 years) was closer to the mean age of the older controls in Lang and associates' study (60.6 years) than to that of college-age Ss in that (or any other)

study. Both Lang and associates' study and the present study found that regardless of their cardiac status (i.e., suffering from or free from cardiac disease), middle-aged Ss were unable to increase their heart rate significantly. This finding is consistent with the normal occurrence of progressive myocardial changes in the heart of the middle-aged and elderly person, as gradual fibrosis of the conducting system and of the heart muscle causes the heart to beat more and more slowly (sometimes to the point of requiring an artificial pacemaker).

Clinical and Research Implications

Decreased heart rate of the magnitude demonstrated in this study is admittedly of little clinical value. However, the finding that the AMI Ss were not at all able to decrease their heart rate voluntarily, while the PMI Ss were able to achieve near-significant reductions in their heart rate, does have certain implications for future research and for the clinical utilization of cardiac biofeedback with patients who have had myocardial infarctions.

The first of these implications is the optimal post-infarction timing of biofeedback training. Such training would appear to be less effective if instituted very shortly after an infarction than if postponed until at least three months have passed following an infarction. The completion of the convalescent period, in fact, may be a more

important criterion for selecting candidates for biofeedback training than either the extent or the severity of cardiac disease, since it was found that post-convalescent patients with histories of multiple complicated infarctions, who were taking a variety of cardiotropic medications, responded better to training than patients who were still convalescing from an uncomplicated first infarction and required no cardiotropic medication.

The other implications are the respective contributions of psychological and social factors to the effectiveness of biofeedback training. Although it was found that the level of anxiety differentiated cardiac from non-cardiac patient Ss, there were not enough cardiac patient Ss with low levels of anxiety to test the a posteriori hypothesis that lowered anxiety among patients with cardiac disease would be associated with greater responsiveness to training. The optimization of emotional receptivity to biofeedback training among persons suffering from cardiac disease appears to be an important area for future research.

The final implication is the possible motivational importance of the perception of this study, by many of the post-convalescent cardiac patient Ss, as being part of the cardiac rehabilitation program in which they were currently participating. The considerable group support which this provided for participation in the study, as well as

the support of the program's staff for the idea of utilizing biofeedback training in the rehabilitation of cardiac patients, may have given these Ss a unique cognitive and emotional set toward the alteration of their heart rate which could have motivated them to put forth more effort during the training sessions in the belief that some benefit might be derived from the training. Both researchers and clinicians attempting to increase the responsiveness of cardiac patients to biofeedback training might do well to keep these possibilities in mind when planning future studies and/or programs utilizing biofeedback models for the modification of cardiac rate.

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APPENDIX A

Explanation of the Research

and

Informed Consent

EXPLANATION OF THE RESEARCH

You are being asked to participate in a project designed to help you learn to control the rate at which your heart beats. Self-control of heart rate has been found helpful in reducing heart rate irregularities in persons who have had heart attacks or have certain kinds of heart disease. A recording will be made of your heart rate by attaching electrodes to your chest and arm just as they do when you have an EKG taken. We will then ask you to slow down or speed up your heart rate, and will show you how your heart rate changes

There will be three sessions, each lasting about 1½ hours. We will also ask you to fill out some questionnaires and take a personality test, since we are interested in knowing about the kinds of people who can and cannot learn heart rate control. All of this information will be confidential and not a part of your VA records. You may withdraw from the study at any time without prejudicing your hospital treatment or veteran benefits. None of the procedures involve any risk or discomfort of any kind.

INFORMED CONSENT

I,_____, agree to participate in this study being conducted by Edward Friedman. I understand that (1) the study is intended to help me in the control of my heart rate; (2) previous studies have shown that people can learn to control their heart rate, and that this can be helpful in reducing irregularities in heart rate in some persons with heart disease; (3) I will receive three training sessions in which I will be provided information about how my heart rate changes while I am attempting to slow it down or speed it up; (4) none of the procedures involve any risk, and I am required only to sit quietly for about 1½ hours for each of the three training sessions; (5) no electrical shock or other discomfort is involved, and I may withdraw from the study anytime I wish.

_____ _____

Witness Subject

APPENDIX B

Autonomic Awareness Questionnaire

AUTONOMIC AWARENESS QUESTIONNAIRE

At some time all people have things happen to them which upset or worry them. Examples of such things are poor health, economic problems, loss of family members or friends, inability to achieve goals on the job or in the family, and so on. Feelings of worry usually happen not only when distressing events occur, but also when distressing events are expected whether or not they actually occur. When people feel worried and upset they tend to experience feelings in an individual way, some in one form, and some in another, and also more or less strongly. In this questionnaire we are interested in what people sense in themselves when they experience worry or anxiety.

For the purpose of answering this questionnaire, try to remember what it is like for you when you are very worried or very upset about something that has happened or which you think might happen. Use the following scale in making your response. For example, if you think you never experience the feeling described by the question, place a 1 in the blank to the left of the question. On the other hand, if you think you always experience that feeling, place a 5 in the blank to the left of the question, and so on.

You may find it difficult to answer some of these questions. That's because people differ widely in their emotional experiences. It is this variation among individual experiences which we are trying to assess. Therefore, it is extremely important that you give as much thought as possible to each of your answers. When you find it difficult to mark a particular item, make the best possible estimate.

Please read each question very carefully and come to a decision about which of the five points best describes your particular experience. There are no catch questions in this questionnaire. Its success depends entirely upon your cooperation.

Think about each question very carefully before you answer. Write by each item the number to indicate how often you have the feeling, when worried, indicated by the question.

NAME:_____ DATE:_____

These are the questions:

1. When you worry how often do you notice bodily reactions?
2. When you are worried how often do you notice that your face becomes hot?
3. When you are worried do you notice that your hands become cold?
4. When you're worried how often do you notice that you perspire even though you are not hot?
5. When you're worried how often does your mouth become dry?
6. When you're worried how often are you aware that your muscles are tense?
7. When you're worried how often do you get headaches?
8. When you're worried how often are you aware of any feelings in your chest?
9. When you're worried how often do you notice your heart beating faster?
10. When you're worried how often do you notice your heart beating harder than usual?
11. When you're worried how often are you aware of changes in your breathing?
12. When you're worried how often does your breathing speed up?
13. When you're worried how often do you breath deeply or sigh?
14. When you're worried how often do you find yourself short of breath?
15. When you're worried how often do you feel as if blood rushes to your head?
16. When you're worried how often do you get a lump in your throat or a choked up feeling?
17. When you're worried how often does your stomach get upset?

____ 18. When you're worried how often do you get a sinking, heavy feeling in your stomach?

____ 19. When you're worried how often do you have any difficulty talking?

____ 20. When you're worried how often are you bothered by the feelings in your body?

____ 21. When you're worried how often do you feel like you have to go to the bathroom?

____ 22. Do you think in general that this type of questionnaire is valuable in appraising differences in emotional experiences?

1 - Not valuable at all
2 - Somewhat valuable
3 - Fairly valuable
4 - Very valuable

____ 23. How adequately do you think the preceding questions have produced a picture of your own emotional experiences?

1 - Not at all
2 - Somewhat
3 - Quite a bit
4 - Very much

APPENDIX C

Locus of Control Scale

(Social Reaction Inventory)

SOCIAL REACTION INVENTORY

This is a questionnaire to find out the way in which certain important events in our society affect different people. Each item consists of a pair of alternatives lettered a or b. Please select the one statement of each pair (and only one) which you more strongly believe to be the case as far as you're concerned. Be sure to select the one you actually believe to be more true, rather than the one you think you should choose or the one you would like to be true. This is a measure of personal belief; obviously there are no right or wrong answers.

Please answer these items carefully but do not spend too much time on any one item. Be sure to find an answer for every choice. Each number is followed by a pair of statements lettered a and b. Draw a circle around the letter in front of the statement which you choose as most true.

In some instances you may discover that you believe both statements or neither one. In such cases, be sure to select the one you more strongly believe to be the case as far as you're concerned. Also try to respond to each item independently when making your choice; do not be influenced by your previous choices.

REMEMBER

Select that alternative which you personally believe to be more true.

NAME: _____

AGE _____ DATE _____

I more strongly believe that:

1. a. Children get into trouble because their parents punish them too much.

   b. The trouble with most children nowadays is that their parents are too easy with them.

2. a. Many of the unhappy things in people's lives are partly due to bad luck.

   b. People's misfortunes result from the mistakes they make.

3. a. One of the major reasons why we have wars is because people don't take enough interest in politics.

   b. There will always be wars, no matter how hard people try to prevent them.

4. a. In the long run, people get the respect they deserve in this world.

   b. Unfortunately, an individual's worth often passes unrecognized no matter how hard he tries.

5. a. The idea that teachers are unfair to students is nonsense.

   b. Most students don't realize the extent to which their grades are influenced by accidental happenings.

6. a. Without the right breaks one cannot be an effective leader.

   b. Capable people who fail to become leaders have not taken advantage of their opportunities.

7. a. No matter how hard you try, some people just don't like you.

   b. People who can't get others to like them don't understand how to get along with others.

8. a. Heredity plays the major role in determining one's personality.

   b. It is one's experiences in life which determine what they're like.

9. a. I have often found that what is going to happen will happen.

   b. Trusting to fate has never turned out as well for me as making a decision to take a definite course of action.

I more strongly believe that:

10. a. In the case of the well prepared student there is rarely if ever such a thing as an unfair test.

    b. Many times exam questions tend to be so unrelated to course work that studying is really useless.

11. a. Becoming a success is a matter of hard work; luck has little or nothing to do with it.

    b. Getting a good job depends mainly on being in the right place at the right time.

12. a. The average citizen can have an influence in government decisions.

    b. This world is run by the few people in power, and there is not much the little guy can do about it.

13. a. When I make plans, I am almost certain that I can make them work.

    b. It is not always wise to plan too far ahead because many things turn out to be a matter of good or bad fortune anyhow.

14. a. There are certain people who are just no good.

    b. There is some good in everybody.

15. a. In my case, getting what I want has little or nothing to do with luck.

    b. Many times we might just as well decide what to do by flipping a coin.

16. a. Who gets to be the boss often depends on who was lucky enough to be in the right place first.

    b. Getting people to do the right thing depends upon ability, luck has little or nothing to do with it.

17. a. As far as world affairs are concerned, most of us are the victims of forces we can neither understand nor control.

    b. By taking an active part in political and social affairs the people can control world events.

I more strongly believe that:

18. a. Most people don't realize the extent to which their lives are controlled by accidental happenings.

    b. There really is no such thing as "luck."

19. a. One should always be willing to admit his mistakes.

    b. It is usually best to cover up one's mistakes.

20. a. It is hard to know whether or not a person really likes you.

    b. How many friends you have depends upon how nice a person you are.

21. a. In the long run, the bad things that happen to us are balanced by the good ones.

    b. Most misfortunes are the result of lack of ability, ignorance, laziness, or all three.

22. a. With enough effort we can wipe out political corruption.

    b. It is difficult for people to have much control over the things politicians do in office.

23. a. Sometimes I can't understand how teachers arrive at the grades they give.

    b. There is a direct connection between how hard I study and the grades I get.

24. a. A good leader expects people to decide for themselves what they should do.

    b. A good leader makes it clear to everybody what their jobs are.

25. a. Many times I feel that I have little influence over the things that happen to me.

    b. It is impossible for me to believe that chance or luck plays an important role in my life.

26. a. People are lonely because they don't try to be friendly.

    b. There's not much use in trying too hard to please people; if they like you, they like you.

I more strongly believe that:

27. a. There is too much emphasis on athletics in high school.

    b. Team sports are an excellent way to build character.

28. a. What happens to me is my own doing.

    b. Sometimes I feel that I don't have enough control over the direction my life is taking.

29. a. Most of the time I can't understand why politicians behave the way they do.

    b. In the long run, the people are responsible for bad government on a national as well as on a local level.

APPENDIX D

Instructions to Subjects

INSTRUCTIONS

We want you to learn to control your heart rate. We don't know exactly how, but people are able to do this when they are shown how their heart rate changes. Different people have different ways of doing this; we want you to find a way that works for you. The important thing is to not try too hard - that will actually make it more difficult for you to learn to control your heart rate. Just sit quietly and concentrate on what you want to happen. You can't make your heart beat too fast or too slow, but if you happen to feel bad during the session, be sure to let me know at once. Don't move around or change your breathing during the session - it will interfere with our measurements. First, I want you to sit quietly for a few minutes while I get your average heart rate. (Before T-2): Now I want you to simply watch this dial and the needle for a few minutes, in order to get used to it - remember, pay close attention to it and go on paying close attention to it until I tell you to stop. (Before T-3): This dial will show you whether your heart is beating faster or slower. When the needle moves to the right, it means your heart rate is faster; when it moves to the left, it means your heart rate is slower. I will set the meter so that your average heart rate is at the middle of the dial. These lights will tell you whether to slow your heart down or to speed it up. I will start by telling you to slow down or speed up your heart rate without showing you how you are doing, and then I will turn on the meter again to show you how your heart rate is changing from beat to beat. There will also be rest periods in which you are to relax and do nothing at all. (Check to be sure that S understands procedure).