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Oklahoma City, Oklahoma

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THE EFFECT OF FEEDBACK AND INSTRUCTIONAL SET ON THE

CONTROL OF CARDIAC RATE AND VARIABILITY

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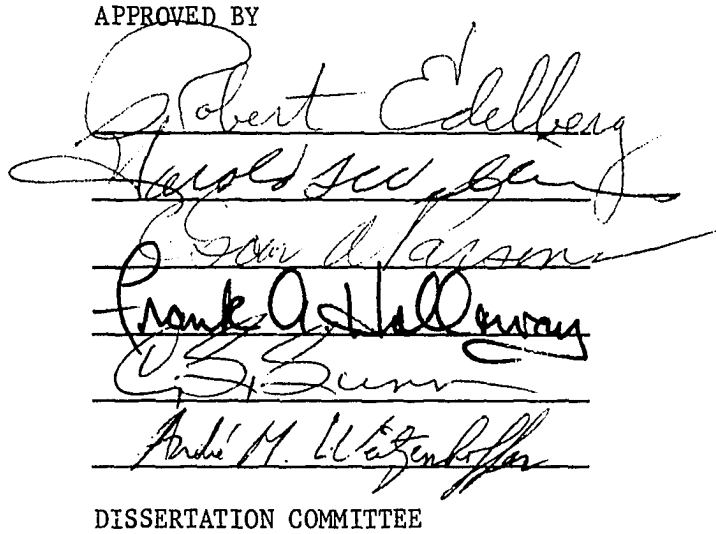

DISSERTATION COMMITTEE

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CHAPTER I

INTRODUCTION AND STATEMENT OF THE PROBLEM

Studies on cardiac control have recently been reported which demonstrate that subjects (Ss) can learn to control their own cardiac activity. These studies are an outgrowth from a larger series of studies concerned with the problem of instrumental learning of autonomically controlled visceral responses. Prior to the present interest in this area, it was widely assumed that autonomic responses were subject to modification by classical conditioning procedures only. Skinner (1953) felt that autonomic functions were not capable of providing the kinds of consequences found in operant reinforcement and that when such consequences were arranged experimentally, operant conditioning would still fail to occur. Kimble (1961) concurred with Skinner that it is impossible to condition autonomic functions by instrumental procedures.

Although Skinner (1953) may have been overly pessimistic in his belief that autonomic functions could not be conditioned instrumentally, he correctly anticipated a methodological problem with which studies in this area have had to deal. This is the problem of mediation by somatically innervated responses. Skinner (1953) cautioned that it may be

possible for autonomic activity to be shaped indirectly through inadvertent reinforcement of operant behavior which mediates the desired autonomic changes.

Studies by Rice (1966) and Van Twyver and Kimmel (1966) on instrumental conditioning of the GSR attempted to control for mediational factors by measuring respiratory and muscular changes during operant conditioning of the GSR. In the Rice study reinforcement of the emitted response was given only in the absence of EMG activity recorded from the forearm. The results suggested that instrumental conditioning of the GSR did occur in the absence of muscular changes. Van Twyver and Kimmel measured respiration and muscular activity in their study of instrumental GSR conditioning. Clear evidence for GSR instrumental conditioning was obtained in the absence of any systematic differences between experimental and control groups in respiration rate or irregularities of respiration, as well as EMG activity. These findings support the conclusions of previous investigators that electrodermal activity is directly susceptible to modification through instrumental conditioning procedures (Crider, Shapiro & Tursky, 1966; Fowler & Kimmel, 1962; Johnson & Schwartz, 1967; Kimmel & Hill, 1960; Kimmel & Kimmel, 1963; Martin, Dean & Shean, 1968; Shapiro & Crider, 1967; Shapiro, Crider & Tursky, 1964; Stern & Kaplan, 1967; Stern & Lewis, 1968).

Studies dealing with cardiac activity modification by feedback or more traditional instrumental conditioning procedures have also experienced difficulty in avoiding the problem of somatic mediation, particularly concerning respiration. For purposes of clarity, these studies will be partitioned into two major categories which will be

further divided on the basis of which responses were being conditioned. The first division consists of those studies using more traditional instrumental conditioning techniques where physical reinforcers were used. The second division consists of those studies attempting to modify cardiac function through feedback procedures where psychological rather than physical reinforcers were employed.

Studies using the more traditional instrumental procedures may be separated into three further categories. First are those studies attempting to increase cardiac rate (HR). Second are studies attempting to decrease HR. Third are several studies in which attempts have been made to both increase and decrease HR. On the basis of which response is to be modified via feedback, there are two types of studies. First are those that have attempted to both increase and decrease HR, and second are those that have attempted to stabilize the HR. The review and discussion which follows will be in the order outlined above.

Instrumental Conditioning Studies

Attempts to Increase HR

Shearn (1962) was the first to attempt to manipulate HR through response-contingent techniques. Prior to this study practically all exteroceptive control of the heart in the laboratory had involved classical conditioning. In this study a Sidman-avoidance schedule was used in which a delay of shock to the ankle in human Ss was made contingent upon HR acceleration. A variable criterion interval in which five beats had to occur to postpone shock was used in order to account for baseline changes in HR. The criterion time was arbitrarily selected as five per cent below the average time required for five beats during the preceding

period. During this avoidance procedure the Ss were given auditory feedback of their own HR. In order to control for the unconditioned HR increases produced by the shock, a yoked-control procedure was used in which each of the six Ss receiving the operant treatment had paired with them a control S who received the same number of shocks at the same time periods regardless of changes in his HR. The results showed that five of the six Ss receiving the operant treatment experienced an increase proportion of accelerations across five sessions as compared to the controls, who showed a decreased proportion of accelerations across the sessions. This difference was found to be statistically significant; but in spite of the large difference between operant and control groups, the operant group showed no reduction in the number of shocks, an atypical finding for successful treatment being found using the Sidman-avoidance schedule. Respiration records taken during the conditioning sessions showed that respiration amplitude immediately preceding and during the criterion HR accelerations was significantly greater than during periods where criterion HR accelerations failed to occur. Respiration rate was also significantly greater in the operant than in the control group. Consequently, since respiration was so closely associated with HR change, Shearn (1962) concluded that his HR results were confounded by the respiratory changes.

Frazier (1966) conducted a study in which HR increases were supposedly instrumentally conditioned using a discriminative avoidance conditioning technique such that if the HR decreased, punishment was administered. A visual discriminative stimulus was presented during the first half of each trial to signal the availability of shock. The discriminative stimulus was presented alone during the second half of the trial in

order to elicit the desired response patterns. All Ss were conditioned without awareness for ten 20-minute continuous epochs. They were given the false impression that they were serving as Ss in a signal detection study where shocks would be given for missed signals. The rationale for conditioning without awareness was twofold. First, Frazier (1966) pointed out that there was no reason to assume that S awareness of the correct contingency is necessary for successful instrumental conditioning. Second, he reported that previous exploratory work indicated that when Ss are aware of a HR increment-punishment avoidance contingency, they make every effort to increase their HR, and this leads to a great deal of artifact.

An unreported number of male undergraduate students served as Ss in the study. During the discriminative (S^d) periods a shock was given every time the HR dropped below the total of the previous minute. With removal of the visual stimulus, subsequent to a number of stimulus-shock associations, the HR decreased abruptly to a relatively stable baseline. Increases in HR soon occurred to S^d only. All Ss showed definite acquisition of the avoidance response usually within the first few trials. Maintenance of the HR increases was found to last as long as 40 minutes without further reinforcement during S^d periods.

In an attempt to deal with the somatic mediation problem, Frazier (1966) recorded skin resistance and respiration rate and amplitude along with HR. Of these three variables, skin resistance was the only one to show any changes paralleling the HR increases. The changes for skin resistance were all in the direction of increased conductance (fall in resistance) for each S^d period. Respiration

rate and amplitude showed no relationship to HR at any time during the course of the study for any of the Ss. Also, the signal detection task revealed no change in the number of correct or incorrect responses made. A general trend was noted in which baseline decreases in HR and increases in skin resistance were observed as the study time increased. This was interpreted as a decreasing level of arousal. Unfortunately, no EMG measures were recorded. As a result somatic mediation cannot be completely ruled out by this study. The finding of parallel changes in skin resistance and HR suggested to Frazier that visceral mediation may have been occurring through alterations in levels of arousal. Although Frazier claimed to have used instrumental conditioning procedures to increase the HR, it is apparent that his experiment employed a classical avoidance conditioning paradigm. With such paradigms, it is not unusual to find HR increases associated with skin resistance decreases under threat of shock, regardless of what response is intentionally being conditioned. Another difficulty with this study is that no control group was run. This becomes particularly important since it was found that the visual S^d stimulus was capable of eliciting unconditioned HR increases. These methodological problems make it extremely difficult to assess the meaningfulness of the data.

Engel and Chism (1967a) avoided some of the pitfalls encountered by the previous studies attempting to operantly increase HR by giving breathing instructions in an attempt to control for respiratory mediation. Reinforcement delivery was determined by means of the output of a cardiotachometer being fed into a Schmitt trigger. In addition, yoked-controls were also tested. The experimental Ss were informed of a cor-

rect response by the turning on of a light and a clock in view of the S. The clock, which accumulated time, served to reinforce the Ss in that they were told they would be paid ½ cent for each second accumulated. The study consisted of three phases, the first two of which served as an adaptation period and an operant baseline level on which the triggering level for reinforcement would be based. The third phase consisted of six sessions, during the first two of which the reinforcement criterion was set at 80% and for the final sessions, 3 to 6, the reinforcement criterion was set at 50%. This varying criterion procedure was adopted because it seemed analogous to shaping schedules. The yoked-controls received the same number of reinforcements as their experimental mates, the only difference being that reinforcement of the former was non-response contingent.

Three separate criteria of learning had to be met before a S could be considered as having shown definite signs of learning. These criteria were the proportion of times the light was turned on, HR average increases, and the number of beats at or above the average operant period established in phase two of the study.

The results showed that all of the experimental Ss learned, since they all showed significant changes in all three of the performance measures. However, two of the yoked-controls also satisfied all three learning criteria, and one met two of the three. The experimental Ss did increase their rates to a greater degree than did the yoked-control Ss who also increased their rates. One important observation made was that almost all experimental Ss exhibited one-trial learning reaching near maximum performance in the second or third session. This finding

was also noted in the Frazier (1966) study. Interviews with the Ss as to how they controlled the light revealed no consistent tendency. Some Ss reportedly increased their muscle tension while others decreased it. Unfortunately, the Ss' reports could not be documented through physiological recordings. Consequently, the possibility of visceral mediation cannot be ruled out. Also, a number of Ss showed some alterations in breathing rate which in some cases was related to successfully increasing their HR.

Attempts to Decrease HR

Harwood (1962) attempted to instrumentally condition HR deceleration using rewarding rather than adversive stimuli. The procedure consisted of rewarding Ss with a nickel for each HR deceleration which occurred during the presentation of a blue light which was the discriminative stimulus. The results for three Ss showed no significant change in HR during the time the discriminative stimulus was on. All three Ss at different times during the study were reported as showing some signs of conditioning. One S was reported as being able to control HR during the discrimination periods by altering his respiration. Since the Ss received up to 172 reinforcements without showing any consistent signs of instrumental HR conditioning in the absence of respiration changes, it was concluded that if conditioning was to be obtained, changes needed to be made in the procedure. Although respiration was recorded during the study, reinforcements were given in the presence of respiratory gasps and other breathing alterations. A second problem with this investigation was that the criteria of what was to be reinforced were changed several times during the course of the study in a non-systematic

manner, so that the slowest rate was reinforced during some sessions, and points of maximum deceleration were reinforced in other sessions. Also, slight decelerations were reinforced from time to time in an attempt to encourage the desired response. Furthermore, since no control group was run and the N was so small, it would be difficult to determine whether or not learning may have occurred.

In a study by Engel and Hansen (1966), many of the deficiencies of the earlier study (Harwood, 1962) were avoided in instrumental HR conditioning through the use of digital logic packages and proper controls. The purpose of this study was to determine if HR slowing could be operantly conditioned. Ten male college students served as experimental Ss while five additional students served as yoked-controls for whom responses were non-contingently reinforced. All Ss were told that they were participating in a conditioning study; however, they were not told that HR slowing was the desired response. In addition all Ss were instructed to breathe normally and that the correct response was not related to breathing. Reinforcement and experimental design details were exactly the same as those described in the Engel and Chism (1967a) study, with the exception that HR decelerations rather than accelerations were reinforced. Like the Engel and Chism study three separate learning criteria had to be met before a S could be considered as having shown definite signs of learning. These criteria were the proportion of times the light was on, HR average decreases, and the number of beats at or below the average operant period HR established in the second phase of the study.

Five of the 10 experimental Ss showed clear evidence of learning as measured by all three criteria. The yoked-controls and non-learners in the experimental group differed significantly on all three learning criteria from the learners in the experimental group. These criteria differences were not related either to the number of reinforcements received or to respiratory changes. There was a slight but non-significant tendency for the HR variability to increase in those Ss who decreased their HR. In contrast to the experimental Ss the control Ss generally increased both their rates and variability. Interviews with each of the Ss as to what procedures were used to control the light revealed nothing of a consistent nature. Some successful Ss felt that they relaxed, but others felt they tensed up. Unfortunately, the presence or absence of muscle relaxation was not verified since no EMG recordings were taken. Possible somatic mediation cannot be ruled in or out in this study. Unlike the data for HR increases reported by Frazier (1966), and Engel and Chism (1967a), evidence of learning did not occur early in the study. This observation together with the finding that only half of the Ss were successful in decreasing HR suggests that HR speeding is easier to learn than HR slowing.

Attempts to Increase and Decrease HR

Levene, Engel and Pearson (1968) attempted to replicate in a single study the results of the two previous Engel investigations (Engel and Hansen, 1966; Engel and Chism, 1967a) by alternately reinforcing Ss for increasing and decreasing HR during appropriate S^d periods. Five college females served as Ss in the study. Each S was trained in slowing and speeding separately until reinforcement was received more than

50% of the time that the appropriate S^d light cue was presented. After reaching criterion performance, each S was advanced to one continuous training session with alternating S^d light cues. Reinforcement delivery was determined by means of the output of a cardiotachometer being fed into a Schmitt trigger. The same learning criteria were used as reported in the two previous Engel studies (Engel and Hansen, 1966; Engel and Chism, 1967a), but the most important criterion was change in HR from the initial rest period.

The most apparent finding of the study was that Ss could alternately increase and decrease their HR at one-minute intervals to the appropriate S^d cues. Although HR increases were statistically significant relative to decreases and vice versa, three of the five Ss were unable to consistently change their HR in either direction relative to the initial resting level. Analysis of acquisition curves further indicated that HR speeding was easier for Ss to learn than HR slowing. There was some evidence to indicate that respiration changes were accompanying the HR alterations. Explicit instructions were then given to the Ss not to alter their breathing patterns, but respiration changes occurred anyway. The Ss were reportedly unaware of changes in their breathing. The direction of these breathing changes was not reported.

Respiration problems were successfully avoided in a series of studies initiated by Trowill (1967), who conducted a study on instrumental conditioning of the HR of rats using curare to control for mediational factors, a technique first used as a control for the instrumental conditioning of salivation in a study by Miller and Carmona (1967). The purpose of the Trowill (1967) study was to determine if

HR was susceptible to instrumental conditioning procedures while all skeletal muscles were paralyzed by curare and respiration artifically maintained at a constant level. The Ss in this study were 56 male albino rats with monopolar electrodes implanted in a pre-determined reward area of the medial forebrain bundle (MFB). Prior to HR conditioning all Ss were trained to press a bar in order to receive rewarding electrical MFB stimulation. Animals showing successful instrumental conditioning were then paralyzed, artificially respirated, and given rewarding stimulation on an FI schedule for either slow or fast HR. Half the Ss served as yoked-controls while Ss as a whole were randomly assigned to either the fast or slow condition. The results showed that a significant proportion of the Ss rewarded for fast HR did increase their rates. The mean increase for all Ss in the increase condition was 3.03 beats per 10 seconds, which was a significant change. Similarly, a significant proportion of the Ss rewarded for decreasing their rates did in fact do so with a mean decrease of 3.21 beats per 10 seconds, which was also significant. The yoked-controls unexpectedly showed trends in the opposite direction to those of their experimental partners, but this difference was not significant because of the greater variability of the yoked group. The results of this study offer fairly strong evidence for instrumental conditioning of cardiac activity in the absence of respiratory or muscular mediation.

Although the group differences were striking in the Trowill (1967) study, overall both experimental groups showed an operant produced HR change of approximately five percent. Since the overall changes were rather small, Miller and DiCara (1967) extended the

Trowill (1967) study along two lines: first, to see if larger changes could be produced in curarized rats by using shaping procedures; and second, to determine if a cardiac discrimination could be learned where the rewarded response would be more likely to occur in reward situations as opposed to non-reward situations.

Twenty-four adult albino rats were rewarded by electrical stimulation of the MFB to either increase or decrease their HR. Half of the Ss were rewarded for fast rates while the rest were rewarded for slow rates. The initial HR training procedure was the same as that described for the Trowill (1967) study. All animals were curarized and artificially respirated. Unlike the Trowill study the criterion for HR reinforcement was gradually made more difficult. As soon as the S reached criterion half of the time, S^d cues and the rewarding stimulation were shut off for 20 seconds, after which they were set to reward a HR change approximately two percent greater than in the previous trial. The results of this shaping procedure showed that the Ss rewarded for increasing rates progressively increased their HR from an initial 422 beats per minute (bpm) to 510 bpm, and those rewarded for decreased rates changed from an initial 400 to a final 316 bpm. These results were found to be highly significant for both groups and were found to be independent of the number of brain stimulations as well as muscular and respiratory changes. Since the two groups received reinforcement for HR changes in opposite directions and were a function of the type of response rewarded, the results cannot be accounted for by any unconditioned effects of brain stimulation.

Contrary to expections, most of the rats changed their overall rates in the rewarded direction during both S^d and non S^d periods, thus showing little discrimination between the two conditions. The rats were given 45 minutes of additional training in an attempt to determine if they could discriminate between the S^d and non S^d conditions. The S^d conditions, as in the first part of the study, consisted of the presentation of a pattern of tone and light. The Ss were given 53 discrimination trials. As the number of trials progressed, the rates for both the fast and slow groups changed progressively more often to the S^d period. Thus, it became clear that the Ss were able to discriminate when they would be reinforced for HR changes. This demonstration of discrimination is important in that one of the characteristics of instrumental conditioning is that a discrimination can be learned so that the operant responses are emitted in one stimulus situation where they are reinforced and not in those situations where they are not.

Although the observed HR changes seen in the Trowill (1967) and Miller and DiCara (1967) studies were independent of muscular and respiratory changes, the possibility still existed that the HR changes were the result of generalized changes in the level of arousal. With this problem in mind, Miller and Banuazizi (1968) conducted a study designed to determine the specificity of visceral learning in regard to HR and intestinal contractions. Evidence against the arousal hypothesis would be assumed if essentially no relationship was found between these two variables. Before this study was begun, an initial exploratory investigation was conducted using seven curarized respirated rats in order to determine if instrumental conditioning of intestinal contrac-

tions or relaxation could be established using rewarding MFB stimulation. Intestinal contractions were monitored by a water filled balloon inserted through the rectum into the large intestine. Four of the rats were initially rewarded for intestinal contraction and then relaxation while this order was reversed for the other three animals. The results showed that all Ss learned to increase contractions when so rewarded and decrease contractions when rewarded for intestinal relaxation. The results were highly significant.

Since the exploratory study indicated that intestinal control could in fact be established through instrumental procedures, the visceral learning specificity study was conducted. Twelve albino rats were randomly assigned among four experimental condition groups: HR increase reward; HR decrease reward; intestinal contraction increase reward; and intestinal relaxation decrease reward. All animals were curarized, respirated, reinforced via the MFB, and given 500 trials of training lasting four hours. The results showed that intestinal contractions increased when rewarded, decreased when relaxation was rewarded, and remained relatively unchanged when either fast or slow HR was rewarded. Similarly, HR increased when a fast rate was rewarded, decreased when reward was given for slow rates, and remained relatively unchanged when either intestinal contractions or relaxation was rewarded. These findings do not support an arousal hypothesis that both the intestinal and cardiac changes were mediated by the learning of some general reaction such as arousal. Thus, the degree of specificity of visceral instrumental conditioning seems to be quite strong.

DiCara and Miller (1968a) attempted to extend the previous findings of instrumentally induced HR change via MFB reward stimulation, to avoidance of mild electric shock. In addition to curarization and artificial respiration, EMG activity was monitored since the possibility existed that covert skeletal responses might still be occurring in spite of curarization and hence influence HR changes in the expected direction. The study was conducted on 12 albino rats which were randomly assigned to slow or fast HR reward groups. Within each of these groups the Ss were again randomly assigned to light-shock or tone-shock groups. A variable criterion for HR reinforcement was established for each animal thus making the HR progressively more difficult to achieve criterion in order to shape the animals' HR responses. Avoidance training consisted of 300 trials using a VI 30-second schedule. The results showed that HR increased during training for the fast group and decreased for the slow group. These results reached high statistical significance and were found to be independent of the number of shocks given for the two change groups. Furthermore, all Ss learned to escape shock by HR changes progressively more rapidly in spite of the fact that the shaping procedure involved making the criterion for avoidance progressively more difficult. Analysis of the EMG records showed that the curarization eliminated all true muscle action potentials for as long as 90 minutes after the end of training.

The Trowill (1967) study and the studies by Miller and DiCara (1967), Miller and Banuazizi (1968), and DiCara and Miller (1968a) unquestionably offer the strongest support yet established to answer the question as to whether cardiac responses are suscep-

tible to instrumental modification in the absence of somatic mediation in the form of respiratory and muscular changes. The Miller group has also reported findings similar to their HR data for instrumental modification of vasomotor activity (DiCara and Miller, 1968b), systolic blood pressure (DiCara and Miller, 1968c), and urine formation (Miller and DiCara, in press). Rats were used as Ss in each of the investigations, and in each study successful conditioning was obtained in the absence of apparent mediating influences.

Feedback Studies

Attempts to Increase and Decrease HR

In the instrumental conditioning studies, it is apparent that voluntary control of cardiac activity is not impossible. It is probable that voluntary control of cardiac activity is only possible under the special conditions offered by an effective schedule of reinforcement, the critical feature of which is that the organism must be supplied with differential feedback of its performance. This, of course, is a state of affairs which is normally nonexistent for cardiac responses under normal conditions with the possible exception of a case reported by Wenger, Bagchi and Anand (1961). They observed a number of Yoga practitioners in India and found one S who, in the absence of obvious mediational changes, was successful in slowing his HR by increasing the cardiac cycle P-R interval up to three seconds. He was also able to reduce the amplitude of the P wave, sometimes to the point of disappearance. In addition he could establish a nodal rhythm for a few seconds. It is interesting to note that Miller and DiCara (1967) examined the details of the EKGs of rats rewarded for slowing HR.

They reported that during the latter part of training the P wave showed a reduction in amplitude while the interval between it and the QRS complex increased. These effects are considered typical of the effects of vagal inhibition. It can only be assumed that this particular Yoga practitioner has somehow been able to establish an augmented sensory feedback of his cardiac activity.

The importance of feedback in the control of unconscious muscular activity has been previously demonstrated by Hefferline (1958). In this study Ss were provided feedback of jaw muscle activity at such high amplification that Ss became visually aware of moving their jaws before becoming proprioceptively aware of having actually done so. In time they were able to control their movements which were overtly imperceptible.

Brener and Hothersall (1966) adopted a version of the augmented sensory feedback technique of Hefferline (1958) in an attempt to determine if Ss could learn to decrease their HR. Five students served as Ss in the study. The cardiotachometer output for each S was fed into a bank of electromagnetic counters which analyzed the inter-beat interval (IBI) continuously. A criterion was then set for each S on the basis of his basal IBI distribution. This was then used to compare each IBI during feedback training in order to assess whether it was longer or shorter than criterion. The Ss were then presented with a low frequency tone for each long IBI (HR slower than criterion) and a high frequency tone for each short IBI (HR faster than criterion). Two lamps were present in the recording room, and these were pointed out to each S. They were instructed that when either the red or the green lamp

was on, they would hear a succession of high and low tones. When the green light was on they were asked to work for high tones (HR increase) while in the presence of the red light they were to work for low tones (HR decrease). All Ss were further instructed that the tones had something to do with their internal behavior, but they were not told that it was the heart which was being controlled. They were told not to engage in unnecessary body movements and to control the tones by mental process only. The experimental session consisted of 16 trials, eight red and eight green. The results showed that all Ss attained higher HRs in the green periods than they did in the red. For the group as a whole, these results were highly significant. Since no controls were tested, it is difficult to be sure whether Ss were actually able to both increase and decrease rate or whether they were controlling rate in only one direction. The variability of the HR during the feedback session was not reported although inspection of the individual IBI distributions presented by Brener and Hothersall (1966, p. 25) suggests a slight increase in variability for the red as compared to the green periods. Respiratory changes were noted particularly during the green periods. This finding suggests that it was primarily increases which were being controlled. This possibility agrees with the observations of Engel and Hansen (1966) and Engel and Chism (1967) that it is easier to increase HR than it is to slow it down.

In a second study, Brener and Hothersall (1967) replicated the Brener and Hothersall (1966) study under conditions of paced and unpaced respiration. In this study five Ss were run under the same

instructions and procedure as in the earlier study (Brener and Hothersall, 1966). After an initial period of training, Ss were given a brief period of instruction on paced breathing in synchrony with a white flashing signal lamp. The Ss were instructed to complete one respiratory cycle between each light flash. They were then instructed to control the tones as before while having their breathing rate paced over a period of six presentations of green and red periods. Subsequent to this, they were given three additional red and green periods with the instructions that they were no longer to be concerned with their breathing.

The results indicated that all Ss showed a significantly greater proportion of short IBIs in the green periods as compared to the red periods. This was found to be true particularly during the paced respiration period. Analysis of respiration amplitude and rate for the two periods revealed no differences. However, because of wide between-S variability in respiratory behavior, this would be expected. Visual inspection of the respiratory records indicated that the patterns during the red and green periods for paced respiration were very similar. This was considered as supporting evidence for cardiac control independent of respiratory changes. Since no control Ss were tested, it again is difficult to determine whether any cardiac control was established and, if so, whether it occurred in both the red and green periods. The technique of paced respiration without adequate experimental controls is questionable to use in a feedback or instrumental conditioning procedure since Westcott and Huttenlocher (1961) have demonstrated that controlled respiration produces unconditioned

decreases in variability within and, more importantly, among Ss. Further, if a S needed to increase his breathing rate at anytime during the course of the study but was restrained because of the pacing procedure, this would probably have the effect of decreasing the HR (Engel and Hansen, 1966, p. 183).

Donelson (1966) attempted to determine if Ss could both increase and decrease their HR when asked to match it to a standard rate which was above their base HR for four consecutive trials (fast trials) and then match their HR to a standard below their base HR for another four consecutive trials (slow trials). The Ss were divided into two separate groups: those that were to match a fast standard rate first and then match a slow one second, and those who were to match a slow standard rate first and then match a fast one second. Both the standard rate, driven by a physiological stimulator, and the Ss' HRs were displayed on a dual-beam oscilloscope. In addition there was a no-feedback control group. Subjects in this group were allowed to observe the standard rate but were not allowed to see their own, although they were given instructions similar to those of the feedback group.

Analysis of the results revealed a significant direction x order interaction which indicated that control of HR is stronger for Ss who try to increase HR first and then to decrease it, rather than vice versa. Initially, there was a slight tendency for HR to be faster in the fast first group relative to the slow first group. This difference was not reported to be significantly different, although it was reported that the HR for both groups failed to differ significantly

from the no-feedback control group. For the second four trials of the study, there was a large decrease in HR for the fast-first, slow-second group, while for the slow-first, fast-second group HR did not appear to increase much above the initial base level. No significance level was reported for any of these a posteriori comparisons. The only other consistent finding was a significant trials effect indicating that HR was tending to decrease over trials. This finding suggests the likelihood that the observed HR decreases in the fast-first, slow-second group were caused by a general decrease in HR over trials. The finding that HR was maintained at about the initial level in the slow-first, fast-second group would suggest that HR increases were probably occurring, perhaps to a greater degree than were HR decreases in the other group, but the increases were masked by the habituation effect. Since habituation was probably responsible for the observed HR decreases, and since the feedback group failed to differ significantly from the no-feedback control group, the results and conclusions of Donelson (1966) must be regarded as highly questionable.

Attempts to Decrease Variability of HR

Hnatiow and Lang (1965) were the first to provide an analogue type of cardiac feedback. Unlike the auditory binary display of Brener and Hothersall (1966; 1967) in which Ss received only information about correct or incorrect HR responses, Ss in this study were continuously provided with a visual analogue HR signal throughout the training periods. The major objective of this study was to determine if HR variability (HRV) could be decreased via feedback. Like the Brener and Hothersall (1966; 1967) studies, no physical reinforcers were used in this pro-

cedure. The only reinforcement available was the immediate feedback of success or failure. Forty students served as Ss in this study, 20 of which were assigned to an experimental group in which correct HRV feedback was available and 20 of which were assigned to a control group which was instructed to track random pointer movements while keeping their bodily processes at a steady level. The cardiac display apparatus was the output of a cardiotachometer projected by means of an opaque projector on a screen. The pointer moved on a white field, the center of which was a red stripe with a blue stripe paralleling it on both sides. The experimental Ss were instructed to keep their HR as steady as possible throughout the study by watching the HR display. They were also told that they would be more successful if they maintained the pointer on the red stripe as much as possible. The cardiotachometer output was adjusted so that each S began the study on the red stripe. During the study all Ss observed the projected display for three five-minute trials with seven minutes intervening between display trials. Prior to the initial display, all Ss were given a 16 minute adaptation period. The dependent variable, HRV, was measured two ways: time on target where HRV did not go beyond the two blue stripes and HR standard deviation.

The results for target time showed that the experimental group was on target significantly more often during the display as compared to the non-display periods. Further, the over-all curve was evaluated in an orthogonal polynomial analysis which revealed a highly significant quintic (saw-toothed) trend evident in successive high display and low non-display scores. The standard deviation scores followed a similar

trend with display standard deviations being significantly lower than non-display standard deviations. In addition the standard deviations of the experimental group for the second and third display periods were significantly lower than those of the control group. Analysis of respiration rate and amplitude failed to indicate a significant correlation with HRV. Consequently, the Hnatiow and Lang results are not subject to the interpretation that HRV changes were mediated by respiration.

No significant changes were noted for HR either for the display or non-display periods. This might be expected though, since the Ss were instructed to keep their HR on the red center line as much as possible while they kept their HR as steady as they could. These instructions concerning the center red stripe may have served to anchor the HR, thus preventing any changes from occurring.

Lang, Sroufe and Hastings (1967) attempted to extend the Hnatiow and Lang (1965) study while manipulating instructional set. Sixty students were divided into two major groups of Ss, group I being instructed that they would be given feedback of their own cardiac activity while group II was instructed that they would be given a tracking task. These two groups were further divided into experimental (E) and control (C) groups. Group I was instructed to keep their heart activity as steady and as close to the center of the feedback meter as possible. Subjects in group IE were then provided with displays of their own cardiac activity while group IC served as yoked-controls in that they received the cardiac activity of their running mate in group IE, although they thought that the cardiac activity they

were seeing was their own. Groups IIE and C were both given instructions to track pointer movements which, unknown to IIC, were being driven by the cardiac activity of each S in the IIE group.

The results were again analyzed as to time on target and HRV in standard deviations. Only the IE Ss showed a marked increment in time on target (the center of the feedback meter). Also, only IE Ss significantly decreased their HRV during the display as compared to the non-display periods, and a significant sextic trend was found. Initial differences in resting mean standard deviations between IE and IIE due to sampling error prevented a comparison of these two groups. Group IE was significantly less variable than IC.

The results of this study were interpreted as showing that feedback clearly alters HRV but that the direction and magnitude of this change depends on the task given to the S. These results apparently were not related to respiratory changes since analyses were performed and no correlations were found.

In this study HR was not so clearly anchored as in the Hnatiow and Lang (1965) study. A definite tendency is apparent in the data for HR to be greater for the IE than for the IC groups, particularly during the display periods (Lang et al., 1967, p. 428).

Statement of the Problem

Studies on cardiac control have been reviewed which attempted to demonstrate that Ss can learn to exert some degree of control over certain aspects of their cardiac activity. Research on the control of cardiac behavior through learning procedures may be divided into two major categories on the basis of the reinforcement technique which was

employed. The first category comprises those studies using more traditional instrumental procedures in that physical primary or secondary reinforcers were made contingent upon specific cardiac changes. The second category comprises those investigations where some form of cardiac feedback was employed with the only reinforcement being the immediate feedback of success or failure responses.

Those studies using physical reinforcers to increase and decrease HR have, in general, found that HR is susceptible to instrumental modification. However, there has been some question as to whether the HR was instrumentally conditioned *per se* or whether the cardiac changes were mediated by some other variable such as respiration rate. This problem has been successfully avoided only in the curarized rat studies.

Because of improper or absent controls, those studies providing feedback of cardiac activity in the absence of physical reinforcers have failed to demonstrate direction of control of HR, although it is apparent from the data that some control is occurring, but in unspecified directions. In addition the unspecified changes that have been induced through feedback have been accompanied at times by respiratory changes. On the other hand, studies attempting to stabilize the HR through feedback have been able to demonstrate direction of control both within and between groups and in the absence of obvious mediating influences other than the feedback *per se*.

Since cardiac rate has not successfully been shown to be susceptible to feedback modification in the absence of physical reinforcers, except in those studies where HR has been stabilized (Hnatiow and Lang, 1965; Lang *et al.*, 1967), it remains to be determined if feed-

back can be used to either increase or decrease the HR. Because it has been indicated that HR increases are easier to learn than decreases under conditions of physical reinforcement (Engel and Chism, 1967a; Levene et al., 1968), it appears that the best test of the hypothesis that HR can be modified by feedback would be to produce reliable HR decrements. Since the Lang experimental and feedback procedures have been successfully employed to demonstrate cardiac control in the absence of obvious mediating influences, it appears that the hypothesis that HR can be decreased through feedback can best be tested by the procedures developed by Hnatiow and Lang (1965) and Lang et al. (1967). Therefore, their basic feedback procedure and experimental design will be adopted in order to test the above hypothesis and other related problems to be outlined below. The general purpose of the present study is to extend the findings of those studies which have used feedback of cardiac activity in an attempt to decrease cardiac rate and variability and to determine the extent to which the learned cardiac changes are related to other physiological variables felt to be capable of mediating learned cardiac behavior.

Feedback Control of HRV

Hnatiow and Lang (1965) and Lang et al. (1967) have demonstrated that HRV can be decreased when appropriate instructions are given in the use of cardiac feedback. In both of these studies HR level was not allowed to change and thus in a sense was anchored, although this effect was apparently stronger in the former as compared to the latter study. To date, the finding of decreased variability following appropriate feedback instructions has not been confirmed by other investigators. As a

prelude to the following problems to be investigated, an attempt must first be made to replicate the findings of decreased HRV following appropriate feedback instructions. The present study will not be an exact replication of the Hnatiow and Lang (1965) and Lang et al. (1967) studies, primarily in that HR will not be anchored to any specific target or point on the feedback display apparatus. Preliminary studies by the present investigator have indicated that HRV can be decreased under the conditions described above. In the present study, as in the Lang investigations, HRV is implicitly regarded as the overall result of at least three contributing factors, these being sinus arrhythmia, beat-to-beat changes, and slower long-term baseline alterations. Thus, these three sources of cardiac variations are pooled together under the common term, HRV.

Feedback Control of HR

Since Hnatiow and Lang (1965) and Lang et al. (1967) did not attempt to decrease HR with their analogue feedback procedure while a number of other findings, notably those of Brener and Hothersall (1966; 1967), suggest that HR may be decreased through feedback, the question may be asked: Using the feedback procedure of Hnatiow and Lang (1965) and Lang et al. (1967), is HR susceptible to feedback decremental modification when subjects are instructed to decrease HR only?

Effectiveness of Feedback Control of HR and HRV

Related to the first problem, a second problem concerns the effectiveness of feedback in decreasing HR and HRV. Specifically, the question may be asked: Are HR and HRV equally or differentially suscep-

tible to decremental modification by feedback?

The Effect of Feedback Decreased HR on HRV and Vice Versa

A third problem is the effect on variability when rate is controlled and vice versa. A slight trend is apparent in the data of Brener and Hothersall (1966) and Engel and Hansen (1966) for variability to increase when rate is decreased. A similar but opposite trend occurs in the data of Lang et al. (1967) where rate tends to increase when variability is decreased. In consideration of these findings, the question may be asked: What is the effect on HRV when HR is decreased, and what is the effect on HR when HRV is decreased?

Simultaneous Feedback Control of HR and HRV

The fourth and final problem to be investigated in this study is the effectiveness of feedback when subjects are instructed to decrease both rate and variability together. There have been no studies to date which have attempted to determine if subjects are able to decrease both simultaneously. This problem may be partitioned into three separate but related questions. First, can subjects use feedback effectively when instructed to decrease both rate and variability together? Specifically, when rate is decreased, will variability decrease and vice versa? Second, when both rate and variability are decreased together, are these two variables equally or differentially decreased by feedback? Third, is rate more susceptible to decremental modification when controlled singly or in combination with variability? Similarly, is variability more susceptible to decremental modification when controlled singly or in combination with rate?

Statement of the Hypotheses

The following specific hypotheses were formulated relative to two of the problems and questions raised above:

1. Given appropriate instructions to decrease heart rate when correct feedback is available, heart rate will be decreased.
2. A negative relationship exists between heart rate and heart rate variability when either one is singly decreased by correct feedback.

Although a number of problems and questions are to be investigated in the present study, the direction of change, if any, could not be anticipated *a priori*, with the exception of the two problems stated as hypotheses one and two above.

CHAPTER II

METHOD

Subjects

Sixty volunteer subjects (Ss) of both sexes between 16 and 30 years of age participated in the experiment. All Ss were paid $2.40 per hour for their participation. The Ss were randomly assigned to one of the two experimental groups, either correct or incorrect cardiac feedback, so that there were 30 Ss in each group. These two groups were further divided randomly into three separate equal-N conditions according to the different instructions which were given so that there were six different groups, 10 Ss per group.

Design

This study is susceptible to a 2 x 3 x 2 x 5 factorial analysis although it was not analyzed in this manner because of a cancelling out of factors. This design includes two experimental groups (correct feedback and incorrect feedback); three levels of feedback instructions (HR single instruction condition, HRV single instruction condition and HR plus HRV combined instruction condition); two feedback display conditions (display and no-display); and five trials (5 display and 5 no-display). There are two dependent variables in this study which are HR in beats per minute and HRV in standard deviation scores. Res-

piration, EMG, and skin conductance were measured as necessary controls against the possibility of somatic mediation.

Task and Apparatus

The Ss task was to simply observe the feedback meter according to the instructions given. No other duties were to be performed.

The correct feedback (CF) display apparatus consisted of a voltage meter in parallel circuit with the pen of a Beckman Dynograph which was being driven by the amplified signal of a Beckman cardiotachometer so that beat-to-beat changes in cardiac rate were represented on the meter face. The full visual range of the meter was 80 beats per minute. For the incorrect feedback (IF) group the voltage meter was again driven by the amplified output of a Beckman cardiotachometer which was converting a tape recorded EKG into an analogue signal. The mean rate and standard deviation of the IF signal is presented in Appendix A.

Silver-silver chloride electrodes were used for all physiological recordings. The cardiac electrodes were placed, one on the left side at approximately the second or third intercostal space, and the other member of the pair on the back of the neck. Respiration, EMG, and skin conductance were monitored for all Ss. Respiration was recorded from a strain gauge strapped around the chest. Changes in chest circumference were transmitted from a constant voltage circuit to the Beckman polygraph. Muscle tension was recorded from a pair of electrodes placed on the medial surface of the right forearm. The EMG was integrated and recorded using a Beckman EMG coupler. Skin conductance was recorded from the middle palmer segments of the second and third

digits of the right hand using a constant voltage circuit. This was recorded on the Beckman polygraph.

Procedure

After approximately 20 minutes of rest, the meter display of either correct or incorrect cardiac feedback was presented for five minutes followed by five minutes of no-display in which the meter became inactive. There were five display and five no-display trials beginning with no-display. An additional no-display trial was given subsequent to the final display, making a total of 11 experimental trials.

Subsequent to electrode placement, the two major groups, CF and IF, were given identical instructions concerning the use of the meter and avoidance of somatic mediation. Instructions were given to all Ss in the presence of correct feedback for both CF and IF groups. None of the Ss were told prior to or during the experiment that they were to control heart functions. The detailed instructions for all Ss are presented in Appendix B with substitutions corresponding to the different groups. Emphasis was placed in the instructions on the use of the feedback display apparatus and how they were to respond to it. They were told only to attempt to bring under control one of their bodily functions, and that they could best do this by watching its activity on the display apparatus. They were never informed that cardiac activity was involved in any way. It was stressed that whatever control they could exert upon the physiological function should be done independent of respiratory or muscular changes. Also, they were told that there would be alternate times when they would be receiving and not receiving

feedback and that when feedback was not available, they should continue to exert control without the display apparatus informing them of how well they were doing. At the end of the study a brief post-experimental interview was given each S concerning his impressions of the experiment. The questions asked in this interview are presented in Appendix D.

The polygraph recordings were analyzed every 15 seconds although the records were continuous. A sampling of 12 absolute measures was taken from each display (D) and no-display (ND) trial, thus spanning a time period of three minutes. The two remaining minutes of unanalyzed record were used when respiration inspiration-expiration cycle deviations occurred. When a respiratory deviation did occur during a sample period, that sample was skipped for that time period and shifted forward 15 seconds. A respiratory deviation was defined as one in which the inspiration-expiration cycle changed more than approximately 33% plus or minus of its average duration in the previous D or ND trial. Also, any sudden changes in respiration amplitude greater or lesser than approximately 33% of the average amplitude in the previous D or ND trial were defined as a respiratory deviation. This type of control allowed for base-rate changes but ruled out phasic alterations such as breath-holdings, gasps, sighs, etc.

Respiration rate, skin conductance, and striate muscle activity were analyzed for the full three minutes for each D and ND trial in all experimental groups. Skin conductance and EMG records were analyzed using a planimetric integration procedure. Skin conductance and EMG changes were not used as criteria in the selection of samples for which

cardiac changes were to be analyzed, as was respiration.

The mean and standard deviation was calculated on each D and ND trial for every S based upon the 12 obtained scores for cardiac activity. The obtained mean and standard deviation was used in the analysis of group data.

In order to adjust for slight but nonsignificant initial differences in the baseline (trial ND_1) between CF and IF groups, all Ss' cardiac scores were converted to T scores. This was done for both the mean HR and the standard deviation by first finding the mean and standard deviation of the 11 trial scores (either HR $\bar{X}$ or HR SD scores) for each individual S. The derived 11 trial $\bar{X}$ and SD for each S was then used to compute a T score distribution so that a T score value was assigned to each of the S's 11 trial scores for both mean HR and standard deviation. To demonstrate this procedure, the ND_1 raw scores of S M. C., taken from Appendix E in the HR plus HRV combined instruction condition, will be converted to T scores. For HR, the mean HR equals 71.4 bpm with a standard deviation of 1.7. For the first HR score (ND_1) the raw score 73.1 bpm equals a T score of 60.0. For HRV, the mean SD equals 5.5 with a SD of 1.2. The first HRV score (ND_1) equals a raw score of 5.1 and therefore a T score of 46.7.

CHAPTER III

RESULTS

The following data will be analyzed in this section: (1) the effects of correct and incorrect feedback on the control of cardiac variability; (2) the test of the hypothesized effects of correct feedback (CF) on the control of cardiac rate; (3) the possible differential effectiveness of CF on the control of cardiac variability decreased singly and cardiac rate decreased singly; (4) the test of the hypothesized effects of CF on two aspects of cardiac activity when only one is instructed to be decreased; and (5) a four-part study of the feedback control of cardiac rate and variability when instructions are to decrease both variables together. In addition to the above analyses, two additional problems will be examined: (1) the problem of habituation versus learning over trials, and (2) the problem of response specificity.

The results of the analyses for hypotheses one and two are presented in sections two and four of the results chapter.

Cardiac Variability Single Instruction Condition

The results for the control of cardiac variability (HRV) for the single instruction condition (SIC) are presented in Figure 1. Cardiac variability was converted from standard deviation scores to T scores in order to adjust for slight but nonsignificant initial dif-

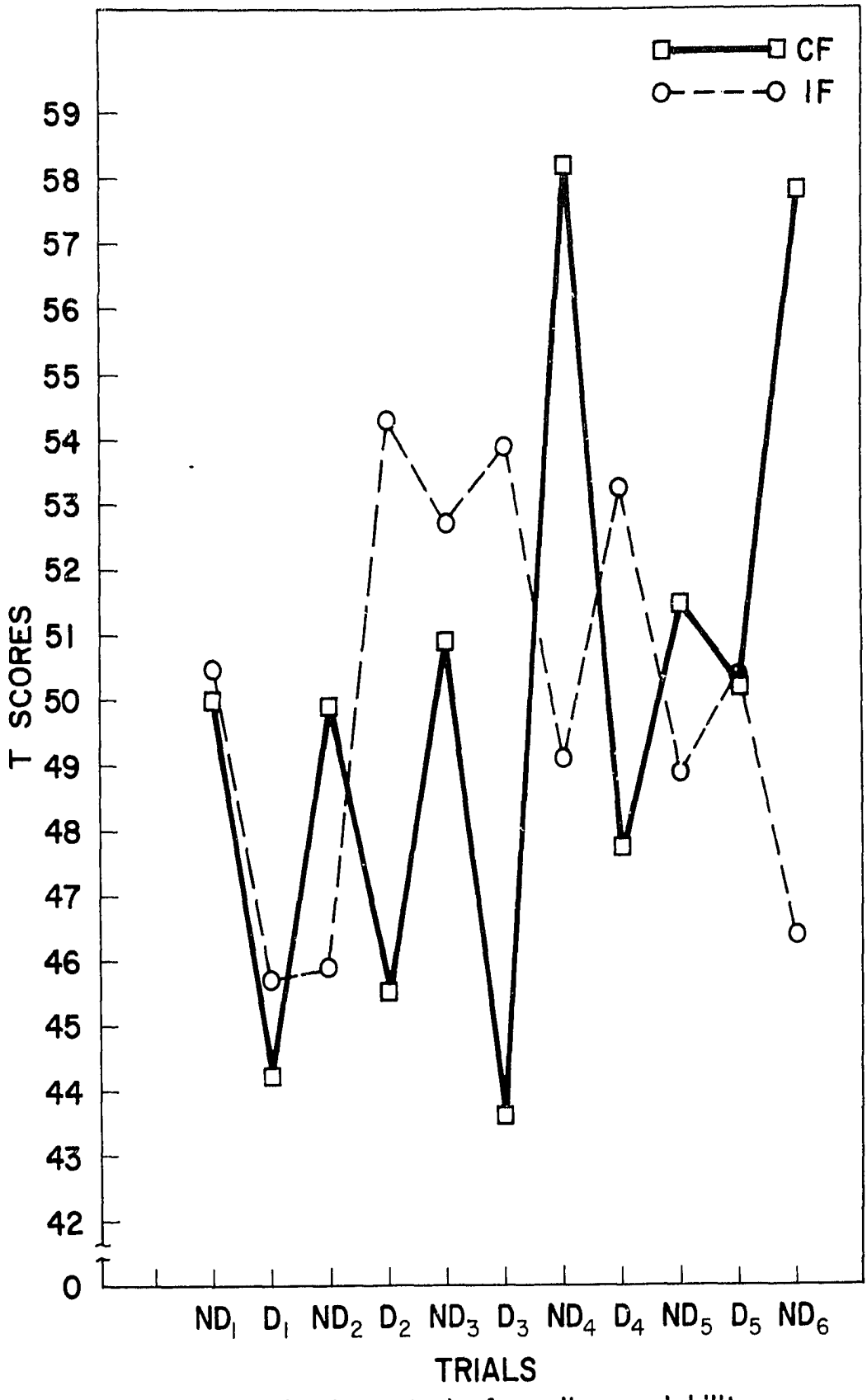

Figure I. Feedback control of cardiac variability

ferences in the baseline between CF and incorrect feedback (IF) groups. This figure shows that the CF group was able to reduce their HRV during the display (D) trials whereas the IF group was not. During the no-display (ND) trials, HRV increased, while for the IF group HRV decreased.

These results were submitted in T score form to a three-way analysis of variance for repeated measures (Winer, 1962, pp. 319-337), which gave the results contained in Table 1. Of primary importance in

TABLE 1

SUMMARY OF ANALYSIS OF VARIANCE FOR HRV (SIC)

Source	df	MS	F
Between subjects	19		
G (groups)	1	84.0	9.2 [b]
Subj w. groups	18	9.1	
Within subjects	180		
D (displays)	1	174.2	1.4
GD	1	791.0	6.2 [a]
D x subj w. groups	18	128.6	
T (trials)	4	110.6	1.2
GT	4	97.2	1.1
T x subj w. groups	72	92.2	
DT	4	85.2	<1.0
GDT	4	89.2	1.0
DT x subj w. groups	72	89.1	

[a] $p < .05$

[b] $p < .01$

this analysis is the significant group x display interaction, which indicates, as shown in Figure 1, that the CF and IF groups are responding

in opposite directions to the feedback treatment. The significant group main effect was an unexpected finding since visual inspection of Figure 1 indicates a high degree of overlap between the two groups. The meaning behind this main effect was clarified by analysis of the simple effects shown in Figure 2. Figure 2 shows that during the D trials, HRV was reduced to a greater degree in the CF group relative to the IF group, but for the ND trials the CF HRV increased and the IF HRV decreased. The analysis of variance for simple effects shown in Table 2 indicates

TABLE 2

ANALYSIS OF VARIANCE FOR HRV (SIC) SIMPLE EFFECTS

Source of Variation	df	MS	F
G for d_1 (groups for D)	1	696.0	10.2 [b]
G for d_2 (groups for ND)	1	178.0	2.6
Within cell	36	68.0	
D for g_1 (displays for CF)	1	852.0	7.9 [a]
D for g_2 (displays for IF)	1	108.0	< 1.0
Within cell	18	128.6	

[a] $p < .05$

[b] $p < .01$

that the group difference for D (G for d_1) is significant and the ND difference (G for d_2) is not. It is this strong D and weak ND difference that is responsible for the significant group main effect.

Figure 2 also shows that a large difference exists between the D and ND trials for the CF group while the difference for the IF group is smaller and in the opposite direction. Table 2 indicates that this

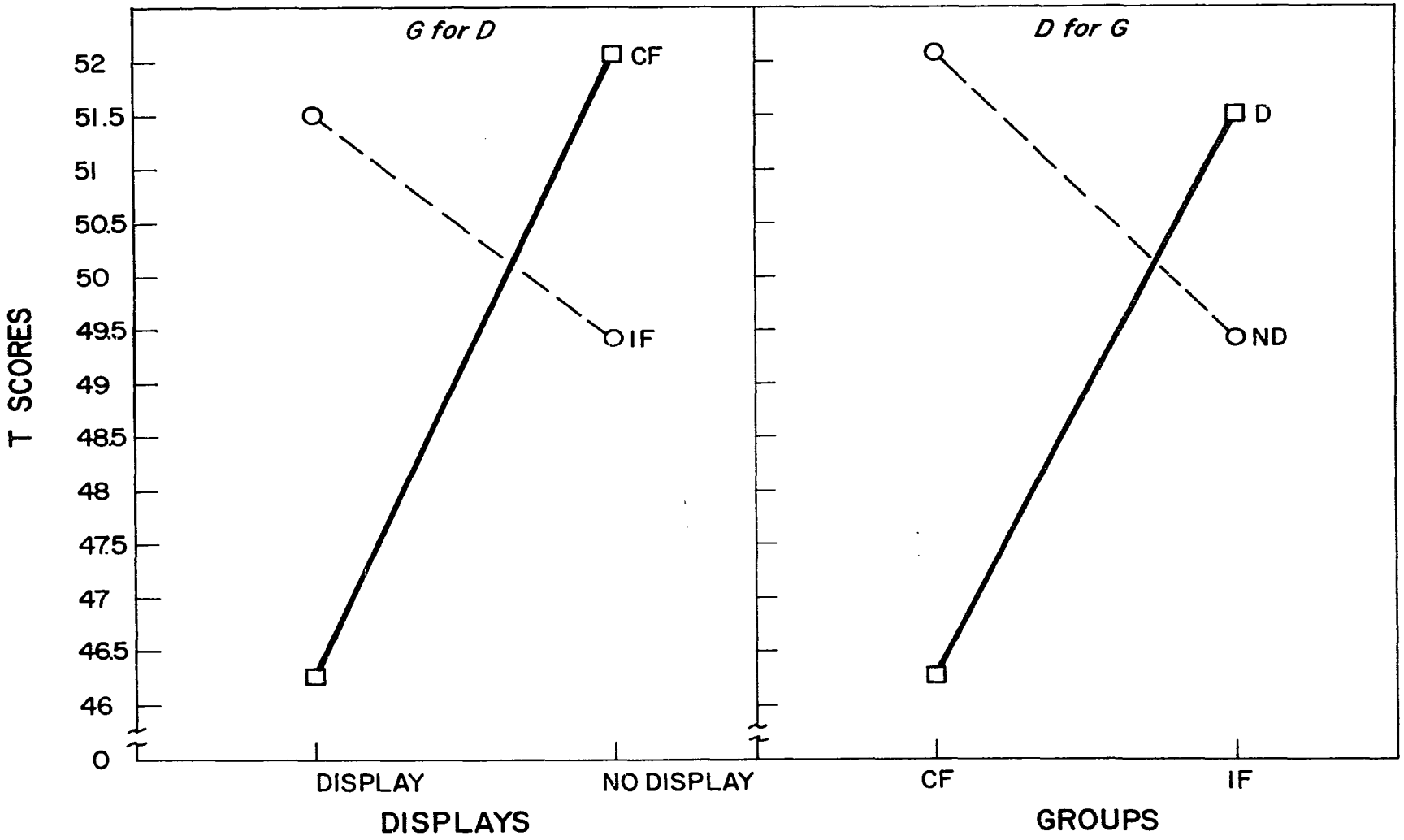

Figure 2. Profiles of simple effects for groups for HRV SIC

difference is significant for the CF group (D for g_1) only.

These findings lend additional support to previous studies which have demonstrated stabilization of cardiac activity following appropriate feedback and instructional set to control HRV.

Cardiac Rate SIC

The results for the control of cardiac rate (HR) are presented in Figure 3. Like cardiac variability, rate was converted from raw scores to T scores in order to adjust for slight but nonsignificant initial differences in the baseline between CF and IF groups. Figure 3 shows that Ss of the CF group were able to reduce their HR during the D trials whereas the IF group was not. During the ND trials HR increased in the CF group while it decreased in the IF group.

These results were submitted in T score form to a three-way analysis of variance for repeated measures, which gave the results contained in Table 3. Comparable to the HRV analysis, the group x display analysis is again statistically significant indicating that the CF and IF groups are responding in opposite directions to the feedback treatment. The significant trials main effect indicates that both groups are decreasing their HR over trials. Because of a possible absence of homogeneity of covariance found in repeated measures analyses, the significant trials main effect was reanalyzed by conservative test by reducing the degrees of freedom to the minimum. This conservative test indicates that the trials main effect is nonsignificant; consequently, the significance indicated by the usual test should be accepted with caution. Unlike the HRV instruction condition, no significant group main effect occurred. The reason for this is made apparent by analysis of the simple effects

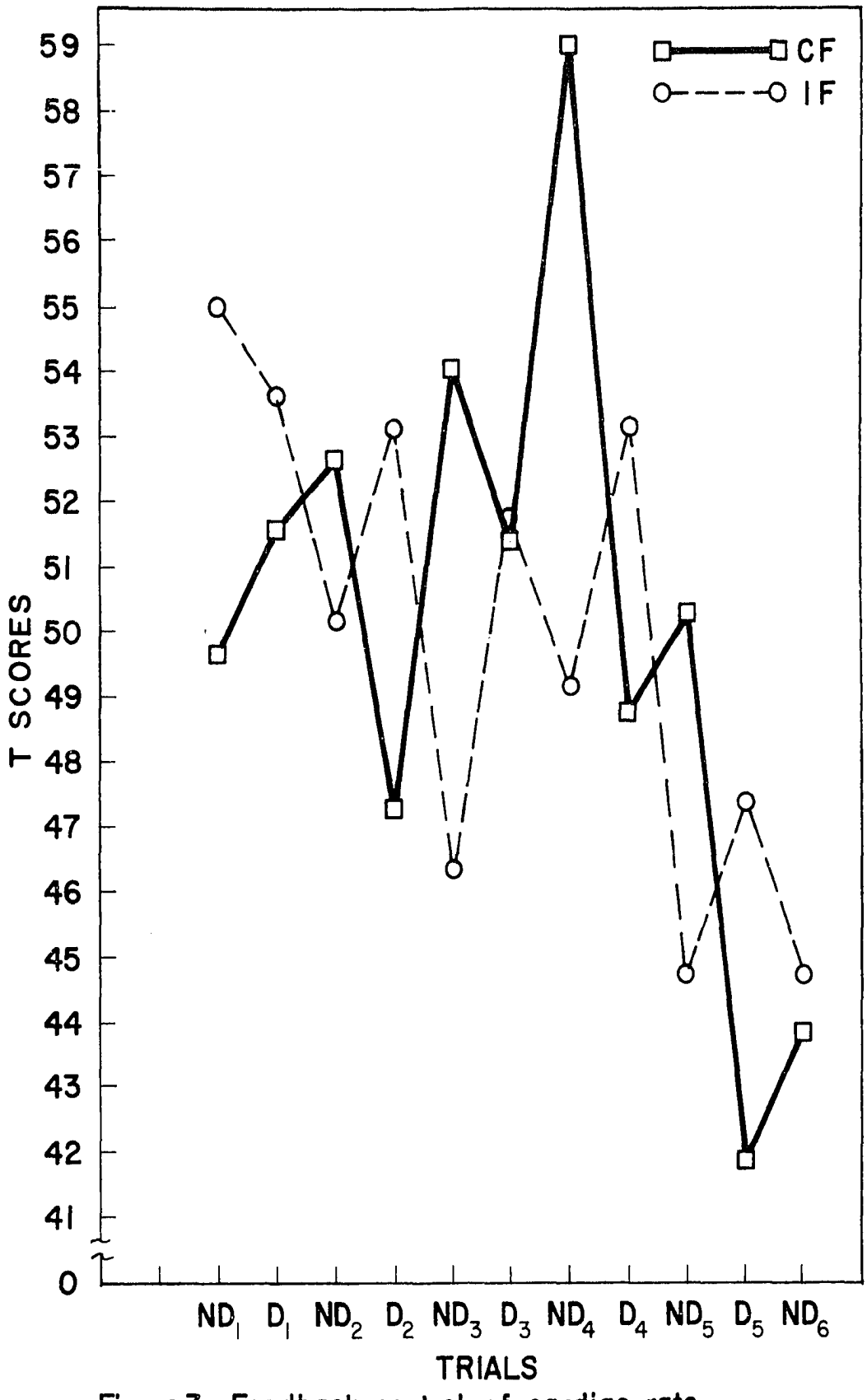

Figure 3. Feedback control of cardiac rate

TABLE 3

SUMMARY OF ANALYSIS OF VARIANCE FOR HR (SIC)

Source	df[1]	MS	F
Between subjects	19		
G	1	1.0	<1.0
Subj w. groups	18	9.9	
Within subjects	180		
D	1	68.9	<1.0
GD	1	723.0	8.0 *
D x subj w. groups	18	90.0	
T	4 (1)	281.5	2.6 *
GT	4 (1)	99.0	<1.0
T x subj w. groups	72 (18)	106.0	
DT	4	35.8	<1.0
GDT	4	115.5	1.6
DT x subj w. groups	72	72.1	

* $p < .05$

[1] conservative test df (1, 18)

shown in Figure 4.

Figure 4 shows that, similar to the HRV data, HR is reduced during the D trials to a greater degree in the CF group as compared to the IF group, and for the ND trials the CF HR increases and the IF HR decreases. The analysis of variance for simple effects shown in Table 4 indicates that both the D and ND differences for groups (G for d_1 and G for d_2) are significant. This opposite and nearly equal D and ND difference for the CF and IF groups acts to cancel the possibility of a significant group main effect such as that which occurred in the HRV instruction group analysis.

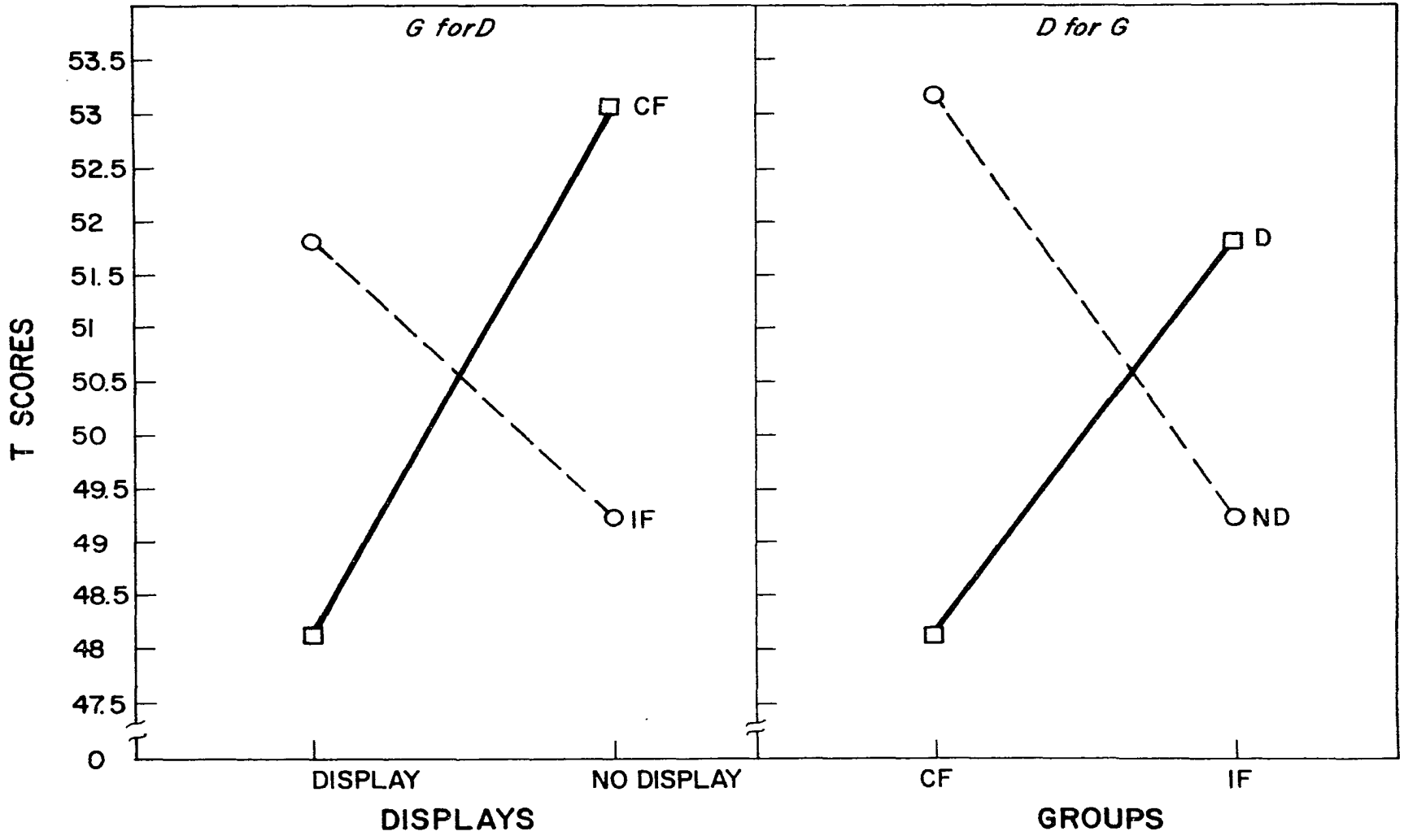

Figure 4. Profiles of simple effects for displays and groups for HR SIC

TABLE 4

ANALYSIS OF VARIANCE FOR HR (SIC) SIMPLE EFFECTS

Source of Variation	df	MS	F
G for d_1	1	336.7	6.7 [a]
G for d_2	1	386.9	7.7 [b]
Within cell	36	50.0	
D for g_1	1	619.0	6.9 [a]
D for g_2	1	172.7	1.9
Within cell	18	90.0	

[a] $p < .05$

[b] $p < .01$

Figure 4 also shows that a large difference exists between the D and ND trials for the CF group while the difference is smaller and in the opposite direction for the IF group. Table 4 indicates that this difference is significant only for the CF group. Since HR has been significantly decreased both within and between groups, it may be concluded that hypothesis 1 has been supported, therefore confirming the prediction that under conditions of correct feedback Ss are able to decrease their cardiac rate when instructed to do so.

Effectiveness of Feedback Control of HR and HRV

A two-way analysis of variance for repeated measures on one factor (Winer, 1962, pp. 303-308) was conducted on the T scores of the CF HR SIC group and the CF HRV SIC group for the D trials only. This was done after first establishing that there was no significant initial baseline difference between instruction conditions either for the raw

scores or for the T scores. The results for D and ND trials for HR and HRV instruction conditions may be seen in Figure 5, and the results for D trials only are shown in Figure 6. The results of the D trials analysis are shown in Table 5. Although there is no significant difference

TABLE 5

SUMMARY OF ANALYSIS OF VARIANCE FOR HR AND HRV INSTRUCTION CONDITIONS

Source	df	MS	F
Between subjects	19		
G (IC groups)	1	93.1	2.0
Subj w. groups	18	45.8	
Within subjects	80		
T (display trials)	4 (1)	18.0	<1.0
GT	4 (1)	212.2	3.2 *
T x subj w. groups	72 (18)	66.7	

* $p < .05$

between instruction conditions as indicated by the instruction condition (IC) group main effect, a significant IC group x trial interaction did occur which indicates that the two IC groups do respond differently across trials. Specifically, as trials increase the absolute amount of decrement decreases in the HRV IC group, while it increases in the HR IC group. The GT interaction should be accepted with caution since by conservative test it is no longer statistically significant ($p < .10$).

The Effect of Feedback Decreased HRV on HR and Vice Versa

Three methods were used in analyzing the data for this phase of the study. The first procedure consisted of finding the first-order partial

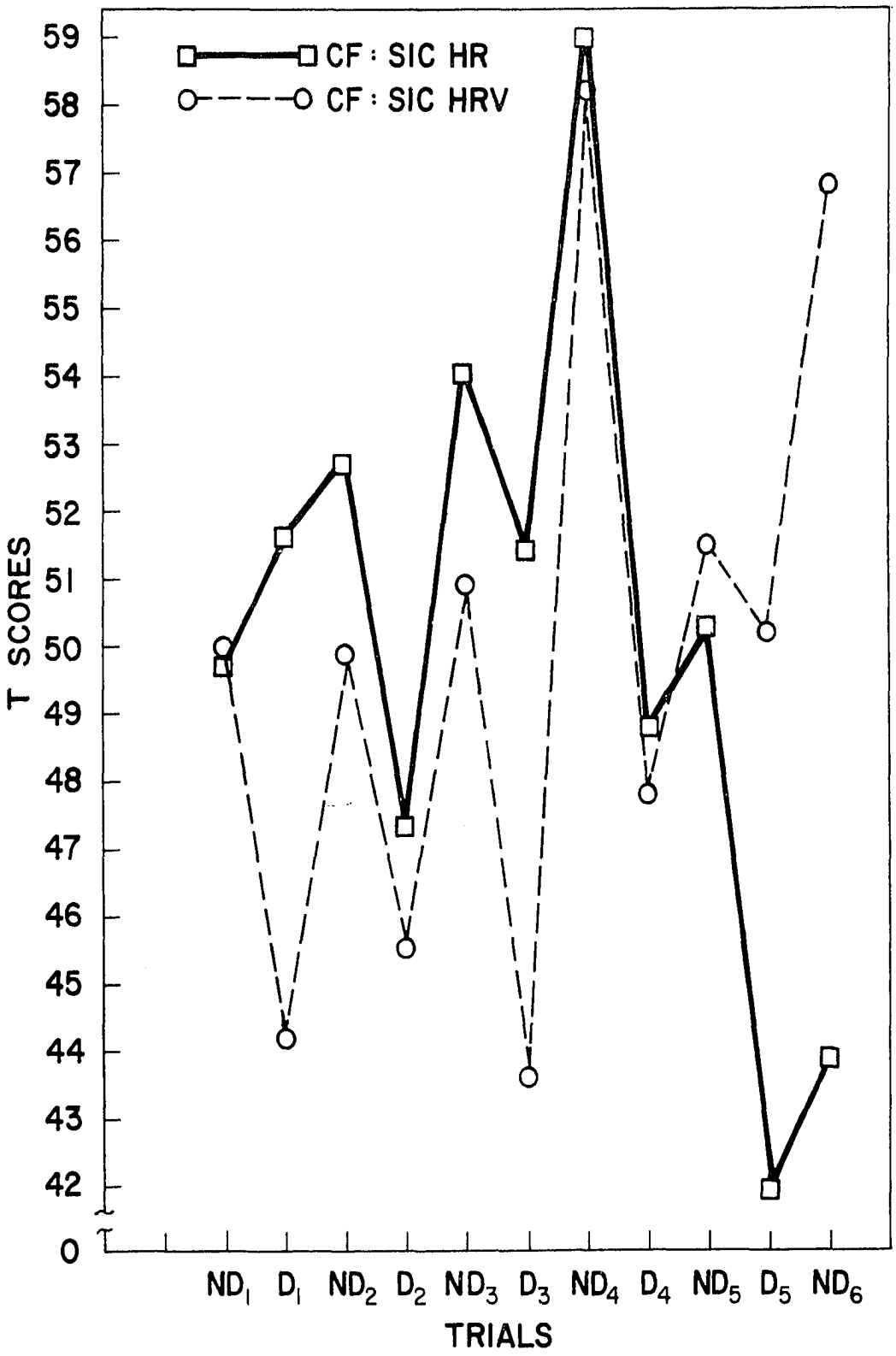

Figure 5. Effectiveness of feedback control of HR and HRV

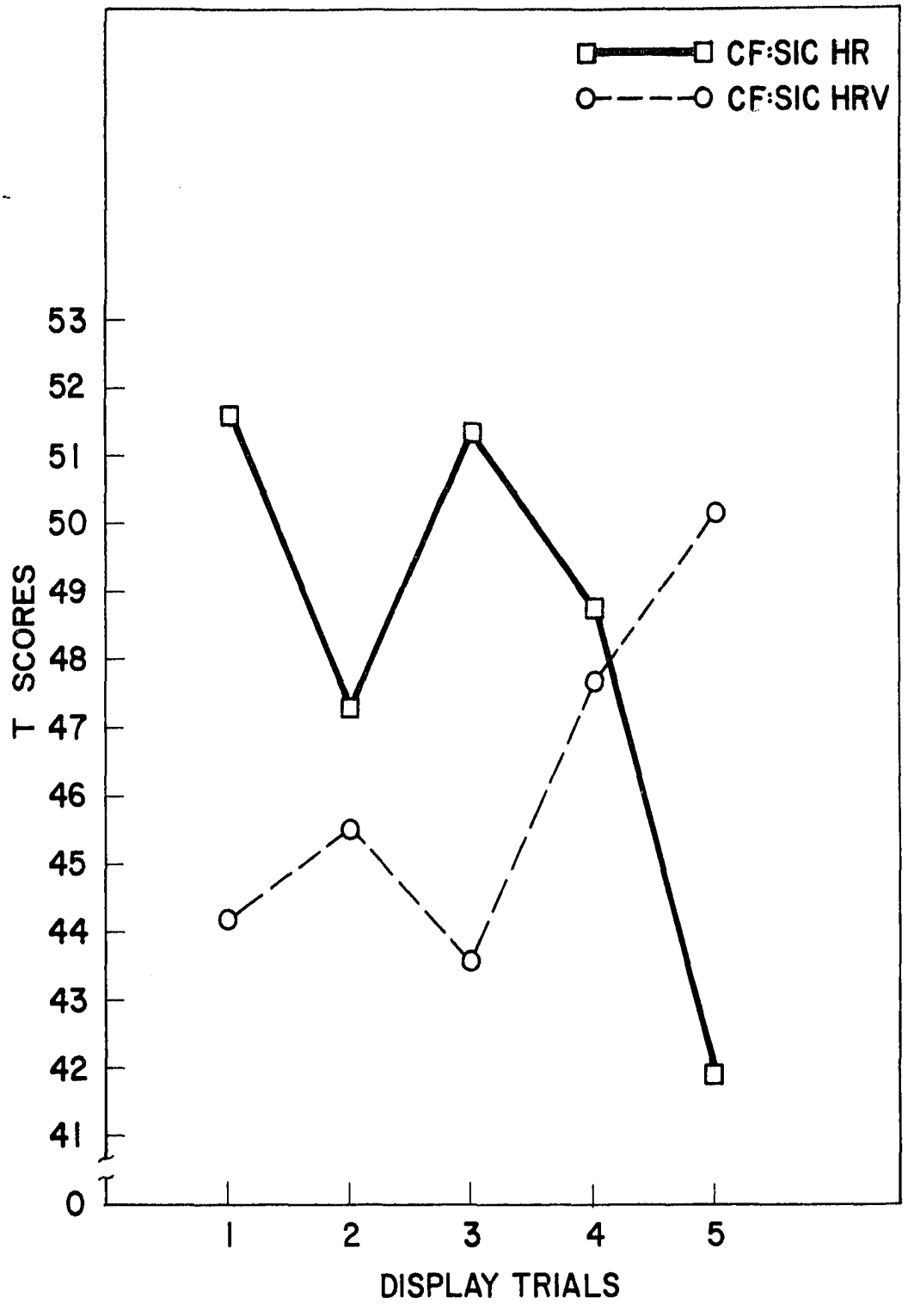

Figure 6. Effectiveness of feedback control of HR and HRV on D trials

correlation between cardiac rate and variability for each individual S in the CF and IF groups for 11 trials (D and ND combined), for five trials (D trials only), and for six trials (ND trials only). Partial correlations were computed since it was apparent that both variables were correlated with trials. The partial $r_{12.3}$ for each S was transformed to a Fisher's z-value. An estimate of the true correlation was then obtained by finding the average z-value for the CF group and for the IF group for both the HRV and HR instruction conditions. A zero-Mu t test was then computed on each distribution of z-values in order to determine if the estimated partial correlation differed significantly from zero. This procedure represents a within-groups analysis and is termed "estimated" in subsequent analyses.

The second statistical procedure, also a within-group analysis, consisted of finding the partial $r_{12.3}$ for the group mean HRV and HR on D+ND, D, and ND trials. The significance of these partial correlations from zero was then determined by a partial r t test of significance (McNemar, 1949, p. 227). The advantage of this group partial r statistical procedure over the estimated partial r was that it possessed greater statistical power when a few low partial r sign reversals were present in the group distribution. The power of the estimated partial r analysis in the first statistical procedure was much greater than that of the second procedure when the partial r distribution was more normally distributed either entirely on one side of zero or around zero. Therefore, when the group-mean partial r is greater than the estimated partial r it can be assumed that there is some degree of skewness in the distribution about zero. This would mean that the group-mean partial r is the statis-

tic of choice for that particular analysis. If the group-mean partial r is less than the estimated partial r, then the estimated partial r is the statistic which should be relied upon for that particular distribution.

The third statistical procedure may be described as a between-groups analysis. It consisted of running a t test for uncorrelated means on the z-values between the CF and IF groups. It is assumed in this analysis that the IF group is an experimental control for the experimental CF group.

The results of the above analyses for the effect of feedback decreased HRV on HR are shown in Table 6. For the CF group, none of the

TABLE 6

PARTIAL CORRELATIONS OF HRV VERSUS HR WITH TRIALS HELD CONSTANT FOR THE HRV INSTRUCTION CONDITION

Trials	CF		IF		Between groups p(2-tail)
	Group $\bar{X}$ $r_{12.3}$	Estimated $\bar{r}_{12.3}$	Group $\bar{X}$ $r_{12.3}$	Estimated $\bar{r}_{12.3}$	
D+ND	-.03	.09	-.17	.00	NS
D	.03	-.07	.48	.69 *	<.10
ND	-.09	.14	.28	.07	NS
Within group p (1-tail)					

* $p < .05$

estimates of the true partial correlation or the group mean partial

correlations between HRV and HR differed significantly from zero. For the IF group on the D trials only, the estimated partial r does differ significantly from zero. This significant positive partial r in the IF group indicates that on D trials only, HRV and HR covary in the same direction together. Thus, maximum HRV increases were associated with maximum HR increases.

The partial correlations for HR versus HRV are presented in Table 7. None of the partial correlations in the IF group are signifi-

TABLE 7

PARTIAL CORRELATIONS OF HR VERSUS HRV WITH TRIALS HELD CONSTANT FOR THE HR INSTRUCTION CONDITION

Trials	CF		IF		Between groups p(2-tail)
	Group $\bar{X}$ $r_{12.3}$	Estimated $\bar{r}_{12.3}$	Group $\bar{X}$ $r_{12.3}$	Estimated $\bar{r}_{12.3}$	
D+ND	.70 [c]	.21	.32	.19	NS
D	.40	.49 [a]	.14	.24	NS
ND	.91 [b]	.16	.20	-.20	NS

Within group p (1-tail)

[a] $p < .10$

[b] $p < .05$

[c] $p < .01$

cant, and only one of the estimated partial correlations in the CF group approaches statistical significance but not in the predicted direction.

The highly significant CF group mean partial correlations for D+ND and ND support the conclusion that HR does not vary inversely with HRV when the former is decreased through feedback. Consequently, these results fail to support the second prediction of a negative relationship between HRV and HR during correct feedback.

Simultaneous Control of Cardiac Rate and Variability Combined Instruction Condition

The results for this phase of the study have been partitioned into four separate but related analyses. The first analysis is to determine if both HR and HRV are decreased together when correct feedback is available. The second analysis is to determine the degree of correlation between HR and HRV. The third analysis will determine if HR and HRV are equally or differentially decreased by correct feedback. The fourth analysis is to determine if HR and HRV are more susceptible to decremental modification when instructions are to decrease singly or in combination with each other.

Control of Cardiac Rate plus Variability

The results for the control of cardiac rate while simultaneously controlling cardiac variability are shown in Figure 7. Cardiac rate was converted from raw scores (bpm) to T scores in order to adjust for slight but nonsignificant initial differences in the baseline between CF and IF groups. Like the results for the HR SIC group, Figure 7 shows that Ss of the CF group were able to reduce their HR on the D trials whereas the Ss of the IF group were not. During the ND trials HR increased in the CF group while it decreased in the IF group.

These findings were submitted to a three-way analysis of vari-

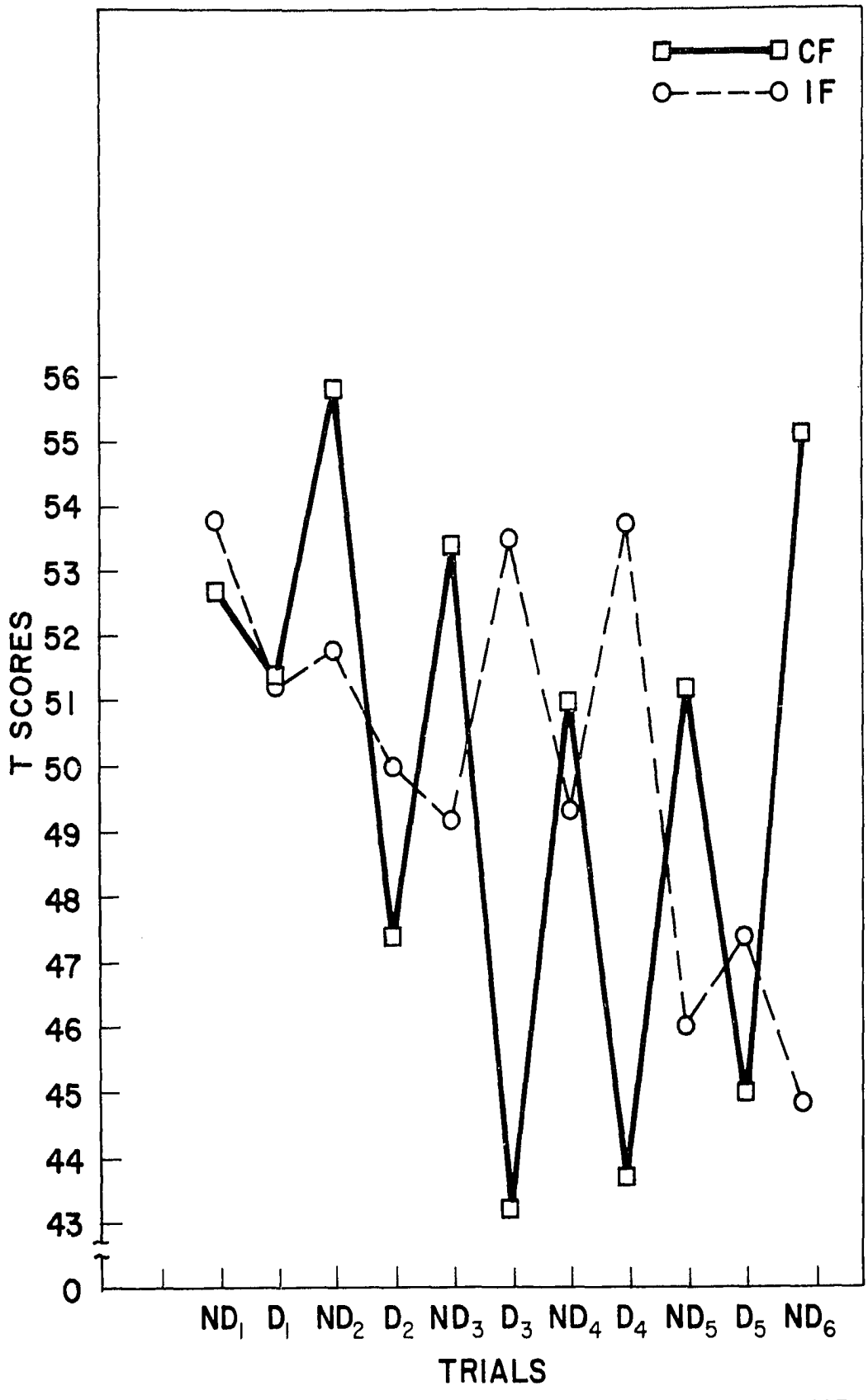

Figure 7. Simultaneous control of HR and HRV for HR

ance for repeated measures. The results of this analysis are presented in Table 8. Like the HR SIC, the group x display interaction was again

TABLE 8

SUMMARY OF ANALYSIS OF VARIANCE FOR HR (CIC)

Source	df	MS	F
Between subjects	19		
G	1	62.4	6.1 [a]
Subj w. groups	18	10.2	
Within subjects	180		
D	1	381.2	8.2 [a]
GD	1	761.0	16.5 [b]
D x subj w. groups	18	46.2	
T	4	138.7	< 1.0
GT	4	58.5	< 1.0
T x subj w. groups	72	140.4	
DT	4	20.2	< 1.0
GDT	4	89.8	1.6
DT x subj w. groups	72	57.2	

[a] $p < .05$

[b] $p < .01$

significant, thus indicating, as shown in Figure 7, that when Ss of the CF group decreased their HR, the IF group Ss increased their HR and vice versa. Also reaching statistical significance is the group main effect and the display main effect.

An analysis of the simple effects may be seen in Figure 8. In Figure 8 a large difference exists between CF and IF groups for the D trials with HR being much lower in the CF group. This relationship became reversed on the ND trials. Table 9 shows an analysis of variance

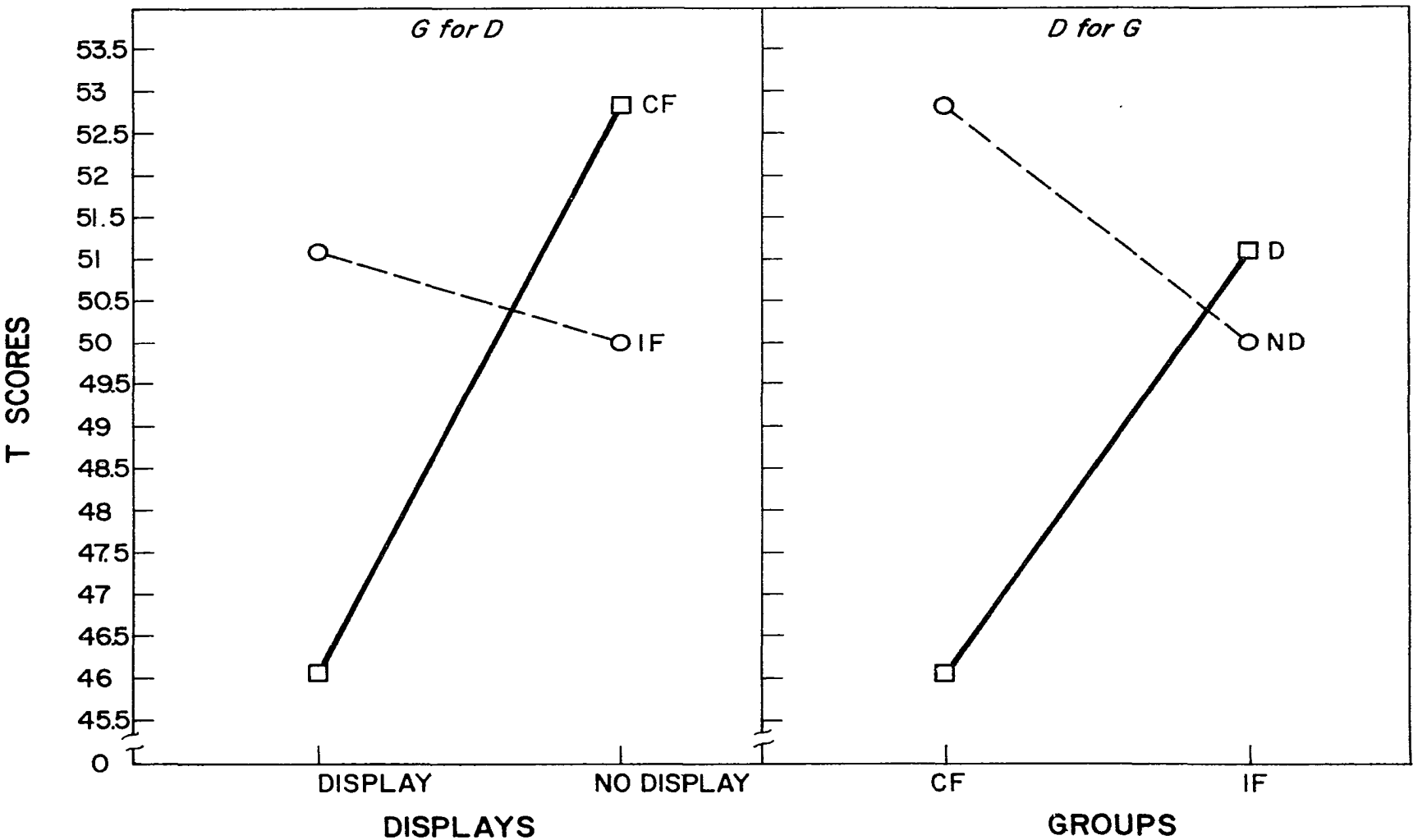

Figure 8. Profiles of simple effects for displays and groups for HR CIC

TABLE 9

ANALYSIS OF VARIANCE FOR HR (CIC) SIMPLE EFFECTS

Source of Variation	df	MS	F
G for d_1	1	629.5	22.3 [b]
G for d_2	1	193.0	6.8 [a]
Within cell	36	28.2	
D for g_1	1	1107.5	24.0 [b]
D for g_2	1	32.5	< 1.0
Within cell	18	46.2	

[a] $p < .05$

[b] $p < .01$

for simple effects of the data shown in Figure 8. The difference between the two groups for D trials (G for d_1) is highly significant, and the difference for the ND trials (G for d_2) is also significant but to a lesser degree. It is the highly significant difference between groups on the D trials which is responsible for the significant group main effect.

Figure 8 also shows a very large difference between D and ND trials for the CF group with a smaller opposite effect apparent in the IF group. Table 9 indicates that the D trials are significantly lower than the ND trials in the CF group (D for g_1), whereas the D trials are nonsignificantly greater than the ND trials (D for g_2) in the IF group. It is this highly significant difference between D and ND in the CF group and lack of significance in the IF group which accounts for the significant display main effect.

Figure 9 shows the results for the control of HRV while simultaneously controlling HR. Like the HRV SIC, HRV was converted from stan-

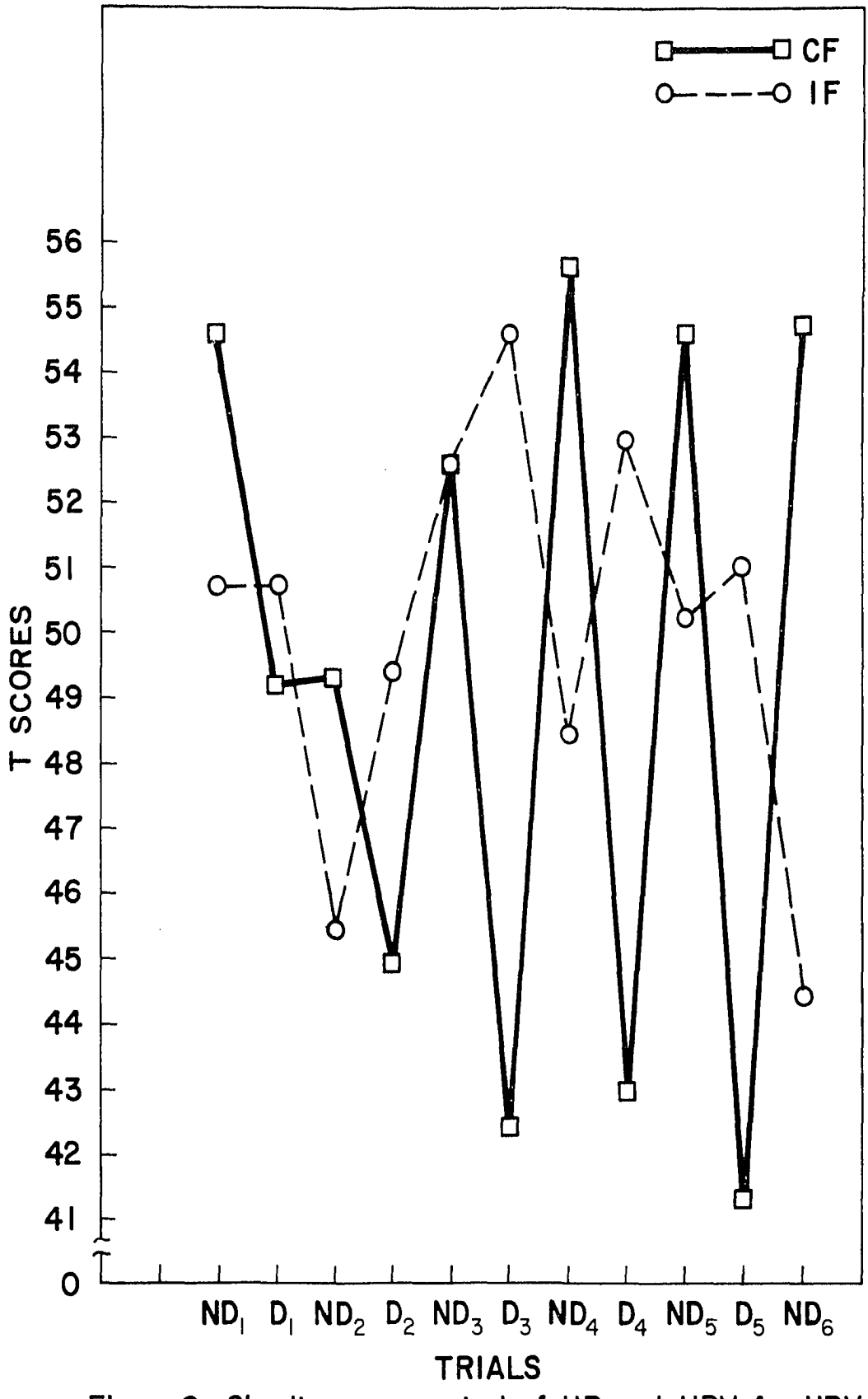

Figure.9. Simultaneous control of HR and HRV for HRV

dard deviation scores to T scores in order to adjust for slight but non-significant initial differences in the baseline between CF and IF groups. Again it is apparent that the CF group Ss were able to reduce their HRV during the D trials at the same time they were reducing their HR. The IF group Ss were not able to decrease their HRV during the D trials.

Table 10 gives the results of a three-way analysis of variance

TABLE 10

SUMMARY OF ANALYSIS OF VARIANCE FOR HRV (CIC)

Source	df	MS	F
Between subjects	19		
G	1	172.0	5.8 [a]
Subj w. groups	18	29.5	
Within subjects	180		
D	1	599.6	6.5 [a]
GD	1	1672.0	18.0 [b]
D x subj w. groups	18	92.8	
T	4	94.6	1.1
GT	4	75.5	< 1.0
T x subj w. groups	72	89.0	
DT	4	50.5	< 1.0
GDT	4	55.0	< 1.0
DT x subj w. groups	72	77.8	

[a] $p < .05$

[b] $p < .01$

for repeated measures. The results of this analysis are almost identical to those of the co-control variable HR. The simple effects are presented in Figure 10, and the analysis of variance of the simple effects for HRV in the combined instruction condition (CIC) is pre-

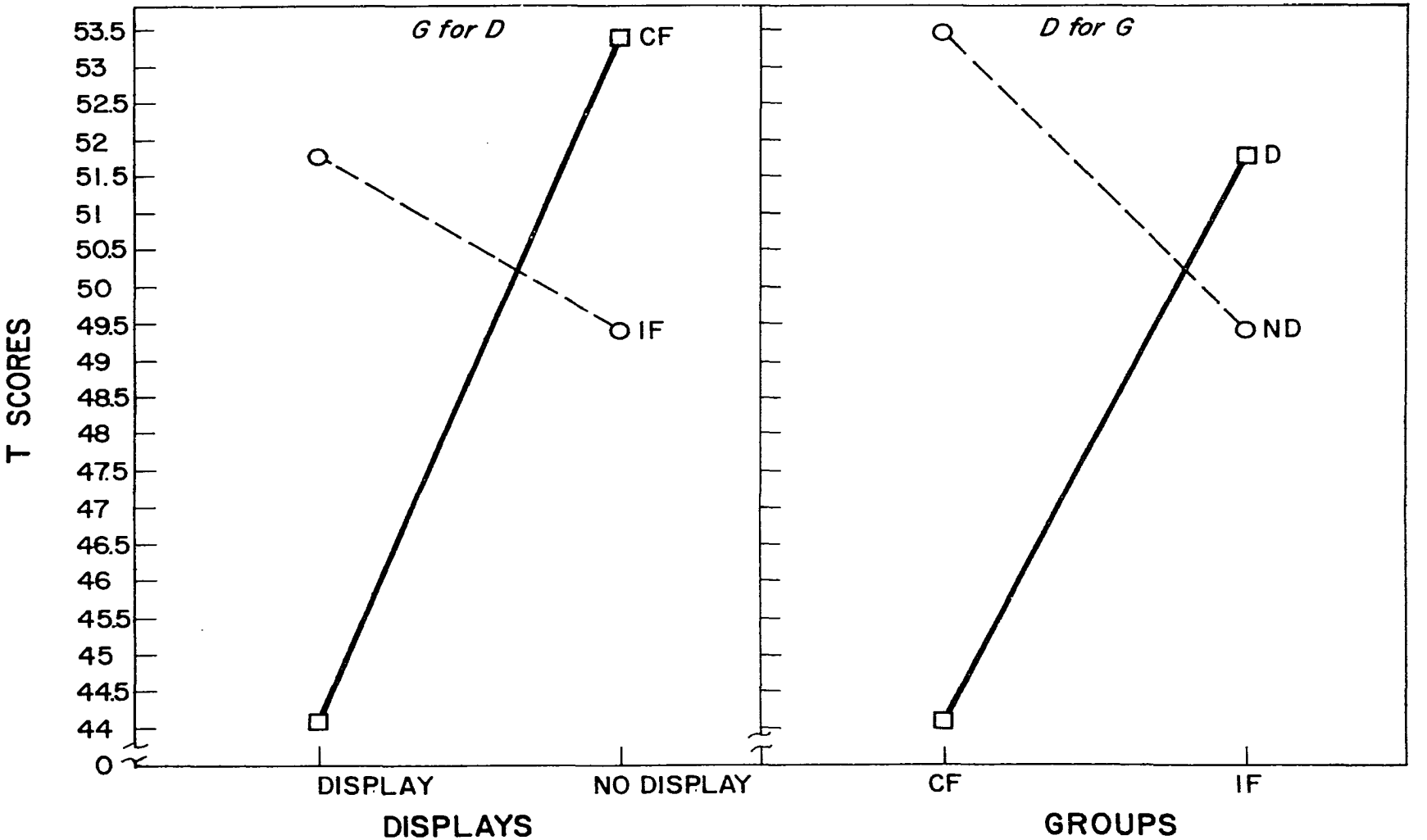

Figure 10. Profiles of simple effects for displays and groups for HRV CIC

sented in Table 11. The results of these simple effect analyses are also

TABLE 11

ANALYSIS OF VARIANCE FOR HRV (CIC) SIMPLE EFFECTS

Source of Variation	df	MS	F
G for d_1	1	1457.6	23.8 [b]
G for d_2	1	385.3	6.3 [a]
Within cell	36	61.1	
D for g_1	1	2136.3	23.0 [b]
D for g_2	1	134.3	1.4
Within cell	18	92.8	

[a] $p < .05$

[b] $p < .01$

very similar to those of the co-control variable HR.

The Relationship Between Cardiac Rate and Cardiac Variability

In order to determine the extent to which HR and HRV covary together, first-order partial correlations between HR and HRV, partialing out the trials effect, were computed using the same analysis procedure as described in the investigation of hypothesis two. The results showing the relationship between HR and HRV when the two are instructed to be decreased together are given in Table 12. Both the estimated partial correlation and the group mean partial correlation for the D+ND trials of the CF group reached statistical significance, therefore indicating that HR and HRV were increased and decreased together. There is also some indication that covariation was occurring to some degree in the IF group since IF D+ND group $\bar{X}\ r_{12.3}$ approaches significance and is in

TABLE 12

PARTIAL CORRELATIONS OF HR VERSUS HRV WITH TRIALS HELD CONSTANT FOR THE COMBINED INSTRUCTION CONDITION

	CF		IF		Between
	Group $\bar{X}$	Estimated	Group $\bar{X}$	Estimated	groups
Trials	$r_{12.3}$	$\bar{r}_{12.3}$	$r_{12.3}$	$\bar{r}_{12.3}$	p(2-tail)
D+ND	.82 [c]	.40 [b]	.60 [a]	.22	NS
D	.65	.26	.62	-.03	NS
ND	-.15	-.02	-.61	.23	NS

Within group p (2-tail)

[a] $p < .10$

[b] $p < .05$

[c] $p < .01$

the same direction as the CF group. This trend is responsible for the lack of significance between CF D+ND and IF D+ND.

Effectiveness of Feedback Control of HR and HRV in the Combined Instruction Condition

In order to determine if HR and HRV were equally or differentially decreased through feedback when both variables were instructed to be decreased together, a two-way analysis of variance for repeated measures was conducted on the data presented in Figure 11. Since both variables came from the same population (CF group, CIC), the analysis was actually performed on one group. However, since the two variables are normally not correlated (Hnatiow and Lang, 1965), they were treated

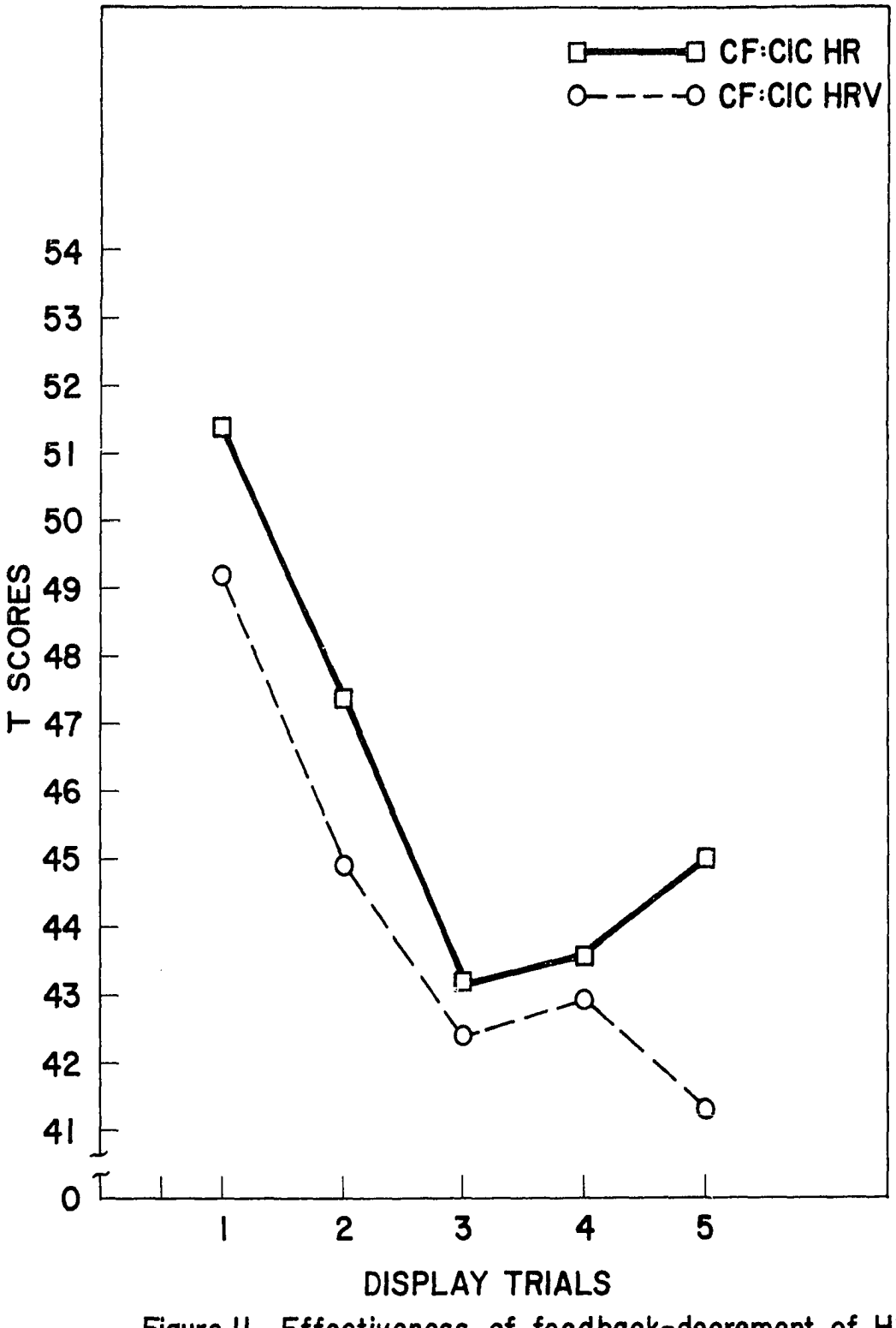

Figure 11. Effectiveness of feedback-decrement of HR and HRV when decreased together

as if they were from independent groups. The results of this analysis are shown in Table 13. In view of the finding that neither the main

TABLE 13

SUMMARY OF ANALYSIS OF VARIANCE FOR HR AND HRV (CIC)

Source	df	MS	F
Between subjects	19		
G (HR vs HRV)	1	101.4	2.6
Subj w. groups	18	39.4	
Within subjects	80		
T (display trials)	4 (1)	203.4	3.3 *
GT	4 (1)	8.0	<1.0
T x subj w. groups	72 (18)	62.2	

* $p < .05$

effect for groups or the group x trials interaction is significant, it may be concluded that the two variables were decreased to the same degree. By the usual test the trials effect is significant, but with the conservative test the significance level is decreased ($p < .10$).

Single versus Combination Instruction Conditions

In order to determine whether HR is more susceptible to decremental modification when decreased singly or in combination with HRV, a two-way analysis of variance for repeated measures was conducted on the data presented in Figure 12. The results of this analysis are presented in Table 14. It is apparent from inspection of this table that there is no significant difference between the two HR instruction condition groups since neither the group main effect nor the interaction term reached

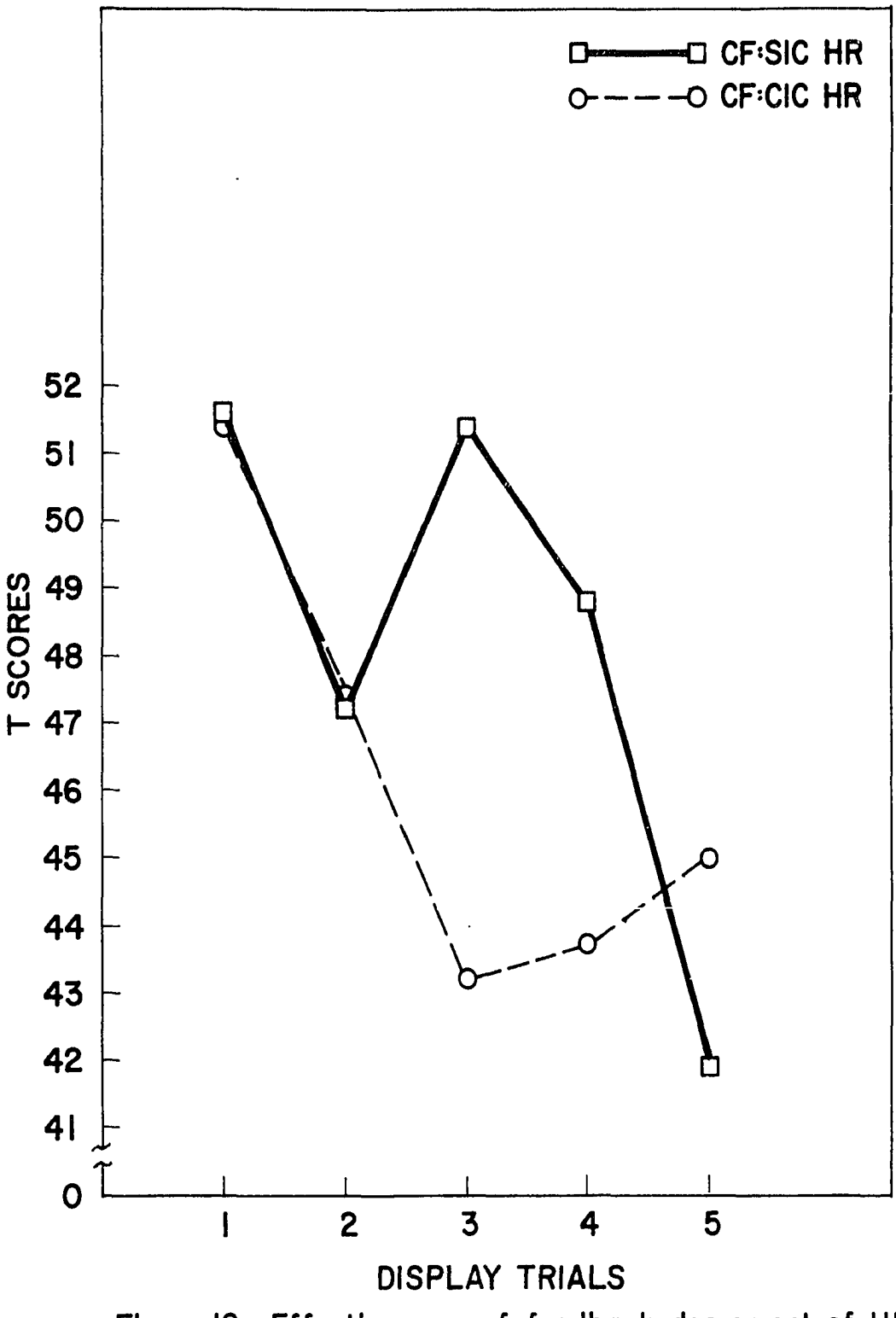

Figure 12. Effectiveness of feedback-decrement of HR decreased singly and in combination with HRV

TABLE 14

SUMMARY OF ANALYSIS OF VARIANCE FOR HR DECREASED SINGLY AND IN COMBINATION

Source	df	MS	F
Between subjects	19		
G (SIC vs CIC)	1	104.8	3.0
Subj w. groups	18	35.0	
Within subjects	80		
T (display trials)	4 (1)	168.2	2.7 *
GT	4 (1)	102.7	1.7
T x subj w. groups	72 (18)	61.7	

* $p < .05$

statistical significance. The only significant finding is the trials main effect, indicating, as shown in Figure 12, a significant decrease in HR over trials. This trials effect should be accepted with caution, since by conservative test the decrement over trials is no longer significant.

Prior to determining whether HRV is more susceptible to decremental modification when decreased singly or in combination with HR, a preliminary analysis by t test on the ND trials revealed that a significant difference existed on the first ND trial between the CF HRV SIC and the CF HRV CIC. This difference is probably due to errors of random sampling rather than, for example, instructions per se. Therefore an analysis of covariance for repeated measures (case 1) (Winer, 1962, pp. 606-615) was conducted, using HRV for ND_1 (the initial rest period baseline) as the covariate. This analysis was conducted on the data shown in Figure 13 and is presented along with an analysis of variance in Table

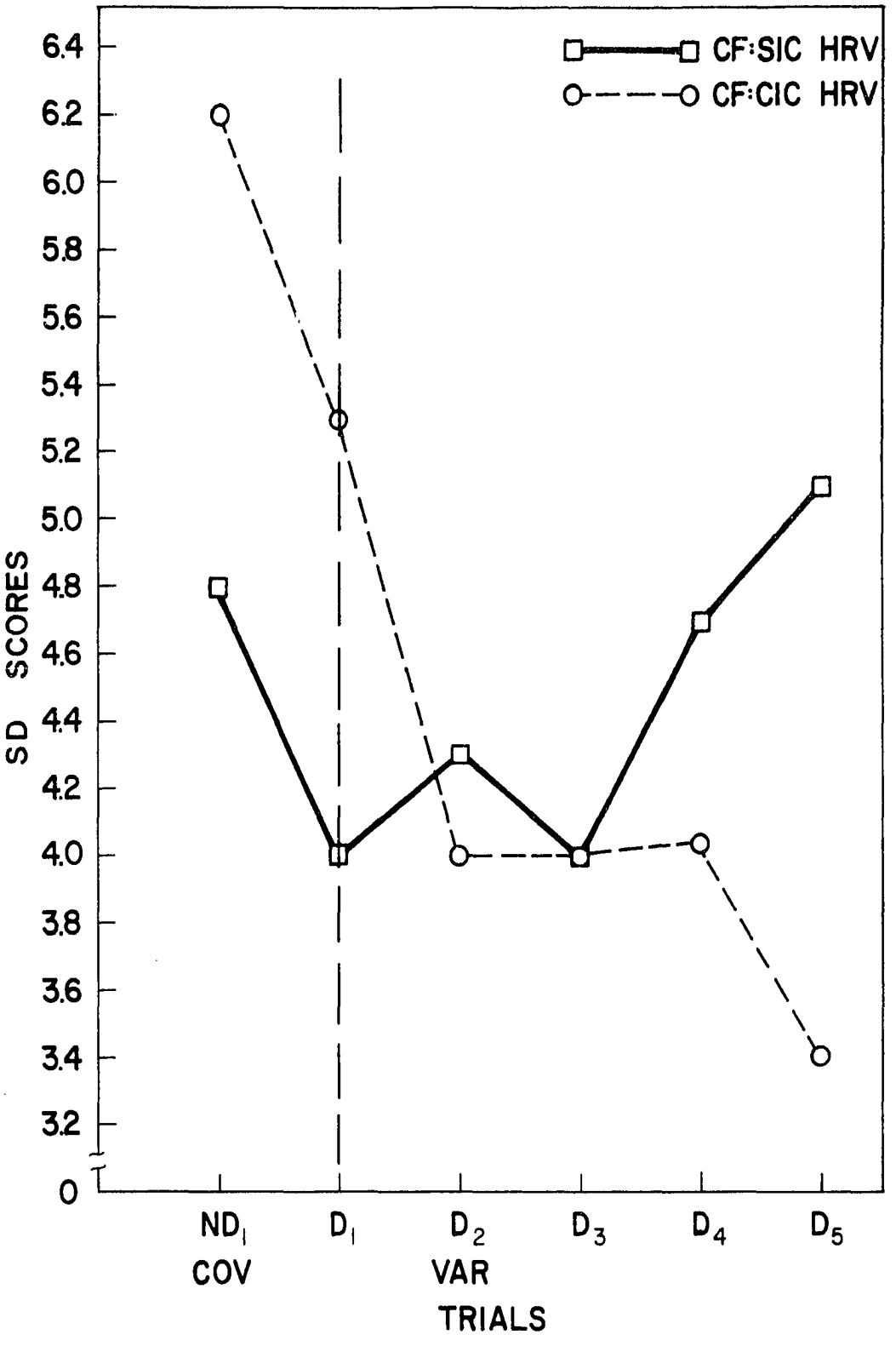

Figure 13. Effectiveness of feedback-decrement of HRV decreased singly and in combination with HR

TABLE 15

SUMMARY OF ANALYSIS OF VARIANCE AND COVARIANCE FOR HRV DECREASED SINGLY AND IN COMBINATION

Source	df	MS	F
Between subjects	19		
G	1	1.4	< 1.0
Subj w. groups	18	8.1	
Within subjects	80		
T (display trials)	4 (1)	1.1	< 1.0
GT	4 (1)	62.5	24.0 *
T x subj w. groups	72 (18)	2.6	
G (adj)	1	13.0	2.4
Subj w. groups (adj)	17	5.4	

* $p < .01$

15. Both analyses revealed no significant difference between groups during the display trials; however, a significant group x trials interaction was found which indicates that the two groups responded differently as trials increased. The high significance level remains statistically significant by conservative test ($p < .01$). Specifically, as trials increase, the amount of decrement in the HRV SIC decreases, while for the HRV CIC the amount of decrement increases.

The Problem of Habituation versus Learning over Trials

A partial correlational analysis was conducted on both cardiac variables in the two instruction conditions in order to determine the extent to which the dependent variables decreased over trials when the cardiac covariate was held constant. The results were analyzed by zero-

Mu t tests and McNemar's t (McNemar, 1949, p. 227) for the within-group effect, while the between-group partial correlations were tested by t test for uncorrelated means. A significant negative partial correlation in the within-group analysis would indicate that the dependent variable was decreasing over trials. If this effect is found in the CF group but not in the IF group, this would indicate a learning or acquisition curve in the CF group. If there is no significant difference in the between-groups analysis while the instructed cardiac variable of either one or both groups is decreasing significantly over trials, this would indicate that simple adaptation or habituation was occurring. The results of these analyses are presented in Tables 16 through 19.

TABLE 16

PARTIAL CORRELATIONS OF HRV VERSUS TRIALS WITH HR HELD CONSTANT FOR THE HRV SINGLE INSTRUCTION CONDITION

	CF		IF		Between groups p(1-tail)
Trials	Group $\bar{X}$ $r_{13.2}$	Estimated $\bar{r}_{13.2}$	Group $\bar{X}$ $r_{13.2}$	Estimated $\bar{r}_{13.2}$	
D+ND	.21	.24 [b]	-.13	.04	NS
D	.73	.42 [a]	.54	.38	NS
ND	.24	.28 [a]	.15	-.13	<.10

Within group p (1-tail)

[a] $p < .10$

[b] $p < .05$

The results for HRV versus trials in the HRV single instruction condition shown in Table 16 indicate a significant tendency for HRV to increase as trials increase as shown by the significant positive partial correlations. This positive trend approaches statistical significance in the between-groups analysis only for the ND trials. The observed relationship between HRV and trials cannot be interpreted as either habituation or learning. It is possible that fatigue or some other effect may be responsible for this reverse relationship. This problem will be taken up in the discussion section.

TABLE 17

PARTIAL CORRELATIONS OF HR VERSUS TRIALS WITH HRV HELD CONSTANT FOR THE HR SINGLE INSTRUCTION CONDITION

	CF		IF		
Trials	Group $\bar{X}$ $r_{13.2}$	Estimated $\bar{r}_{13.2}$	Group $\bar{X}$ $r_{13.2}$	Estimated $\bar{r}_{13.2}$	Between groups p(1-tail)
D+ND	-.68 [b]	-.29 [b]	-.71 [b]	-.40 [a]	NS
D	-.62	-.40 [b]	-.43	-.52 [a]	NS
ND	-.88 [b]	-.23	-.64	-.41	NS

Within group p (1-tail)

[a] $p < .10$

[b] $p < .05$

Table 17 shows a strong negative relationship between HR and trials for both CF and IF groups on the within-group analysis, with no

significant difference on the between-groups analysis for any of the trial periods. This pattern suggests habituation or adaptation of HR over trials in both groups, with no evidence of acquisition or learning in the CF group.

TABLE 18

PARTIAL CORRELATIONS OF HRV VERSUS TRIALS WITH HR HELD CONSTANT FOR THE HRV COMBINED INSTRUCTION CONDITION

	CF		IF		Between
Trials	Group $\bar{X}$ $r_{13.2}$	Estimated $\bar{r}_{13.2}$	Group $\bar{X}$ $r_{13.2}$	Estimated $\bar{r}_{13.2}$	groups p(1-tail)
D+ND	-.56 [b]	-.24 [a]	-.74 [c]	-.22	NS
D	-.78	-.34	-.39	-.19	NS
ND	-.51	-.32	-.99 [d]	-.35	NS

Within group p (1-tail)

[a] $p < .10$

[b] $p < .05$

[c] $p < .01$

[d] $p < .001$

Table 18 indicates that there is a general tendency for HRV to decrease over trials in both CF and IF HRV CIC groups, thus indicating habituation. Since the CF group does not differ significantly from the IF group, there is no evidence of a learning or acquisition curve.

The data presented in Table 19 indicate that there was little

TABLE 19

PARTIAL CORRELATIONS OF HR VERSUS TRIALS WITH HRV HELD CONSTANT FOR THE HR COMBINED INSTRUCTION CONDITION

	CF		IF		Between
Trials	Group $\bar{X}$ $r_{23.1}$	Estimated $\bar{r}_{23.1}$	Group $\bar{X}$ $r_{23.1}$	Estimated $\bar{r}_{23.1}$	groups p(1-tail)
D+ND	.39	-.01	.34	.03	NS
D	.11	-.48 *	.26	-.05	NS
ND	.13	.02	-.65	.07	NS
Within group p (1-tail)					

* $p < .10$

tendency for HR to decrease over trials in the CIC CF and IF groups. A negative relationship did occur to a slight though nonsignificant degree for the CF D trials, but this failed to differ significantly from the IF group. Consequently, this suggests that habituation rather than acquisition occurred over trials.

Because a learning or acquisition curve could not be distinguished statistically from habituation curves, further analyses were conducted in an attempt to demonstrate acquisition of cardiac control over trials. Four two-way analyses of variance for repeated measures were conducted on the ND to D trial difference scores. It was expected on an a priori basis that an acquisition curve would be seen in the CF groups, but not in the IF groups. Thus, a group x trial interaction was expected with the ND to D difference scores increasing over trials in the CF

groups only. With this analysis, none of the four instruction condition CF groups showed a significant acquisition curve. The same data was then submitted to a nonparametric trend analysis (Lubin, 1961). With this statistical procedure, the ND to D difference scores were found to increase significantly ($K = .37$, $p < .01$) over trials only in the CF HRV combined instruction condition. A significant trend was not found in the IF HRV CIC control group. Thus, evidence for a learning curve was found in only one of the four instruction conditions.

The Problem of Response Specificity

Respiration rate (RR), skin conductance (SC), and striate muscle activity (EMG) were recorded for all six feedback and instruction conditions in order to determine the degree of specificity of the feedback induced cardiac control. These findings will be presented according to the feedback instruction condition beginning with the HRV SIC, followed by the HR SIC. Next will be the HRV CIC and then the HR CIC. Because of a faulty coupler, EMG data will be presented for the CIC CF and IF groups only. In order to facilitate interpretation of the data for RR, the results of an after-the-fact analysis of the relationship between RR and respiration amplitude (RA) will also be presented.

All correlations were by first-order partial $r_{12.3}$ and all tests of significance shown in the tables below are two-tailed. As in previous analyses for partial r, significance of within-group partial correlations are determined by zero-Mu t tests, while between-group partial correlations are tested by t test for uncorrelated means. Group mean partial correlations are tested by McNemar's t (McNemar, 1949, p. 227).

HRV SIC:RR

The partial correlations of HRV versus RR with trials held constant are presented in Table 20. It is apparent that neither the within- nor the between-group partial correlations attained statistical significance. Therefore, it may be concluded that RR did not covary with HRV in the SIC CF group.

TABLE 20

PARTIAL CORRELATIONS OF HRV VERSUS RR WITH TRIALS HELD CONSTANT FOR THE HRV SINGLE INSTRUCTION CONDITION

	CF		IF		Between
	Group $\bar{X}$	Estimated	Group $\bar{X}$	Estimated	groups
Trials	$r_{12.3}$	$\bar{r}_{12.3}$	$r_{12.3}$	$\bar{r}_{12.3}$	p(2-tail)
D+ND	-.39	.02	-.18	-.02	NS
D	.58	.23	-.56	.07	NS
ND	-.54	-.29	.18	-.22	NS

HRV SIC:SC

The partial correlations of HRV versus SC are presented in Table 21. Although none of the within-group analyses are significant, the between-group analysis does approach statistical significance for D+ND and D trials. These findings suggest the possibility of a trend for SC to covary with HRV so that when HRV decreases, SC also decreases and vice versa in the CF group relative to the IF group.

TABLE 21

PARTIAL CORRELATIONS OF HRV VERSUS SC WITH TRIALS HELD CONSTANT FOR THE HRV SINGLE INSTRUCTION CONDITION

	CF		IF		Between
Trials	Group $\bar{X}$ $r_{12.3}$	Estimated $\bar{r}_{12.3}$	Group $\bar{X}$ $r_{12.3}$	Estimated $\bar{r}_{12.3}$	groups p(2-tail)
D+ND	-.12	.14	-.37	-.30	<.10
D	.68	.48	-.89	-.40	<.10
ND	-.62	.15	.03	-.20	NS

HR SIC:RR

Partial correlations for HR and RR are presented in Table 22.

TABLE 22

PARTIAL CORRELATIONS OF HR VERSUS RR WITH TRIALS HELD CONSTANT FOR THE HR SINGLE INSTRUCTION CONDITION

	CF		IF		Between
Trials	Group $\bar{X}$ $r_{12.3}$	Estimated $\bar{r}_{12.3}$	Group $\bar{X}$ $r_{12.3}$	Estimated $\bar{r}_{12.3}$	groups p(2-tail)
D+ND	-.39	-.19	.51	.13	NS
D	.57	-.24	.41	.23	NS
ND	-.76	-.25	.68	.37	<.10

None of the within-group partial correlations differed significantly from zero. The CF group did not differ significantly from the IF group for

either D+ND or D; however, significance was approached for the ND trials. Thus, a trend is apparent for RR to decrease when HR increases and vice versa on ND trials only.

HR SIC:SC

Partial correlations for HR and SC are presented in Table 23. The positive partial correlation between HR and SC for the CF group for D trials indicates that when the HR decreases, SC decreases also. The significant between-group difference for D trials indicates that the significant positive correlation is unique to the CF group.

TABLE 23

PARTIAL CORRELATIONS OF HR VERSUS SC WITH TRIALS HELD CONSTANT FOR THE HR SINGLE INSTRUCTION CONDITION

	CF		IF		Between
Trials	Group $\bar{X}$ $r_{12.3}$	Estimated $\bar{r}_{12.3}$	Group $\bar{X}$ $r_{12.3}$	Estimated $\bar{r}_{12.3}$	groups p(2-tail)
D+ND	.16	.12	.00	.18	NS
D	.96 *	.62 *	-.25	-.15	<.05
ND	-.16	-.06	.11	.23	NS
Within group p(2-tail)					

* $p < .05$

HRV CIC:RR

The results of this analysis are presented in Table 24. The

estimated partial correlation for D trials of the CF group does differ significantly from zero in the negative direction which indicates that when HRV decreased, RR increased. The significant between-groups effect for D trials indicates that the significant negative partial correlation is unique to the CF group.

TABLE 24

PARTIAL CORRELATIONS OF HRV VERSUS RR WITH TRIALS HELD CONSTANT FOR THE HRV COMBINED INSTRUCTION CONDITION

	CF		IF		Between
	Group $\bar{X}$	Estimated	Group $\bar{X}$	Estimated	groups
Trials	$r_{12.3}$	$\bar{r}_{12.3}$	$r_{12.3}$	$\bar{r}_{12.3}$	p(2-tail)
D+ND	.27	.18	-.29	-.19	NS
D	-.50	-.71 *	-.38	-.01	<.01 *
ND	.41	-.00	.16	-.24	NS
Within group p(2-tail)					

* $p < .01$

HRV CIC:SC

The results of this analysis are presented in Table 25. It is apparent that both the within- and between-group analyses fall just short of reaching an acceptable level of significance for the D+ND trials. This positive partial correlation within the CF group and relative to the IF group indicates that for the CF group, SC is decreasing

with HRV during the D trials and is increasing with the ND trials.

TABLE 25

PARTIAL CORRELATIONS OF HRV VERSUS SC WITH TRIALS HELD CONSTANT FOR THE HRV COMBINED INSTRUCTION CONDITION

	CF		IF		Between
	Group $\bar{X}$	Estimated	Group $\bar{X}$	Estimated	groups
Trials	$r_{12.3}$	$\bar{r}_{12.3}$	$r_{12.3}$	$\bar{r}_{12.3}$	p(2-tail)
D+ND	.22	.23 *	-.13	-.12	<.10
D	.38	-.02	.35	.19	NS
ND	.18	.32	-.20	-.04	NS
Within group p(2-tail)					

* $p < .10$

HRV CIC:EMG

The results of this analysis are presented in Table 26. Although none of the within-group positive partial correlations differ significantly from zero, the positive CF ND trials $\underline{\bar{r}_{12.3}}$ relative to the significant ND trials $\underline{\bar{r}_{12.3}}$ of the IF group indicate the possibility that for the CF group, EMG amplitude increases and decreases along with HRV for ND trials only.

HR CIC:RR

The results of this analysis are presented in Table 27. Examination of this table indicates that a significant negative partial

TABLE 26

PARTIAL CORRELATIONS OF HRV VERSUS EMG WITH TRIALS HELD CONSTANT FOR THE HRV COMBINED INSTRUCTION CONDITION

	CF		IF		Between groups
Trials	Group $\bar{X}$ $r_{12.3}$	Estimated $\bar{r}_{12.3}$	Group $\bar{X}$ $r_{12.3}$	Estimated $\bar{r}_{12.3}$	p(2-tail)
D+ND	.28	.13	-.28	-.12	NS
D	.47	.18	-.24	-.24	NS
ND	.46	.22	-.24	-.34 *	<.05
Within group p(2-tail)					

* $p < .05$

correlation exists for the ND trials within the CF group. Although this trend does not reach an acceptable level of significance for the CF D+ND trials, it is significant relative to the IF D+ND trials. Therefore, these partial correlations indicate that when HR increases during the ND trials, RR decreases and vice versa, while for CF D+ND trials relative to the IF D+ND trials, RR increases when HR decreases and vice versa.

HR CIC:SC

The results of this analysis are presented in Table 28. It is apparent by inspection of Table 28 that none of the within- or between-group analyses attained statistical significance. Therefore, it may be concluded that SC did not covary to a significant degree with HR for either the CF or IF groups.

TABLE 27

PARTIAL CORRELATIONS OF HR VERSUS RR WITH TRIALS HELD CONSTANT FOR THE HR COMBINED INSTRUCTION CONDITION

	CF		IF		Between
	Group $\bar{X}$	Estimated	Group $\bar{X}$	Estimated	groups
Trials	$r_{12.3}$	$\bar{r}_{12.3}$	$r_{12.3}$	$\bar{r}_{12.3}$	p(2-tail)
D+ND	.02	-.28 [a]	.12	.13	<.05
D	-.61	-.25	.45	.17	NS
ND	-.94 [b]	-.48	.53	.25	<.05

Within group p(2-tail)

[a] $p < .10$

[b] $p < .05$

TABLE 28

PARTIAL CORRELATIONS OF HR VERSUS SC WITH TRIALS HELD CONSTANT FOR THE HR COMBINED INSTRUCTION CONDITION

	CF		IF		Between
	Group $\bar{X}$	Estimated	Group $\bar{X}$	Estimated	groups
Trials	$r_{12.3}$	$\bar{r}_{12.3}$	$r_{12.3}$	$\bar{r}_{12.3}$	p(2-tail)
D+ND	.14	.30	-.07	.12	NS
D	-.33	.25	-.18	.05	NS
ND	.12	.27	.08	.12	NS

HR CIC:EMG

The results of this analysis are shown in Table 29. None of the within- or between-group analyses were statistically significant. Therefore it may be concluded that EMG did not covary to a significant degree with HR for either the CF or IF groups.

TABLE 29

PARTIAL CORRELATIONS OF HR VERSUS EMG WITH TRIALS HELD CONSTANT FOR THE HR COMBINED INSTRUCTION CONDITION

	CF		IF		Between
	Group $\bar{X}$	Estimated	Group $\bar{X}$	Estimated	groups
Trials	$r_{12.3}$	$\bar{r}_{12.3}$	$r_{12.3}$	$\bar{r}_{12.3}$	p(2-tail)
D+ND	.08	.01	.12	-.21	NS
D	-.41	.06	.42	.13	NS
ND	.69	-.18	-.02	-.49	NS

Respiration Rate versus Respiration Amplitude

The results of an after-the-fact analysis of the relationship between RR and RA for CF and IF CIC groups are presented in Table 30. Although no experimental predictions were being tested formally, one-tail significance tests were used due to the fact that under normal breathing conditions a negative relationship exists between RR and RA. Also, since RA was not anticipated as a measure in this investigation, no precautions were taken to insure accuracy in its recording. The probable effect of this oversight would be to increase the variability of the

data. Therefore, it is probable that one-tail analyses would more accurately represent the significance of the data.

TABLE 30

PARTIAL CORRELATIONS OF RR VERSUS RA WITH TRIALS HELD CONSTANT FOR THE COMBINED INSTRUCTION CONDITION

	CF		IF		Between
	Group $\bar{X}$	Estimated	Group $\bar{X}$	Estimated	groups
Trials	$r_{12.3}$	$\bar{r}_{12.3}$	$r_{12.3}$	$\bar{r}_{12.3}$	p(1-tail)
D+ND	-.01	-.21	-.01	-.17 [a]	NS
D	-.32	-.43	.25	-.54 [b]	NS
ND	-.35	-.43 [a]	-.29	-.24 [b]	NS

Within group p(1-tail)

[a] $p < .10$

[b] $p < .05$

The results shown in Table 30 indicate that there is no significant difference between CF and IF groups for any of the trial conditions, although the IF group does show a more significant negative correlation between RR and RA than the CF group. When these results for RA are considered in view of the findings for RR in the CIC CF and IF groups, it becomes apparent that although there was a general tendency for RR to vary inversely with cardiac activity in the CF group relative to the IF group, the relationship between RR and RA remained largely unaffected by the differential feedback treatment.

CHAPTER IV

DISCUSSION

The following will be discussed in this chapter: (1) the effect of feedback on the control of cardiac variability and the first hypothesis of the investigation that cardiac rate may be decreased through correct feedback; (2) the simultaneous control of cardiac rate and variability and the effectiveness of correct feedback on the control of these two variables when controlled singly and in combination; (3) post-experimental interviews; (4) the problem of habituation versus learning over trials; (5) the problem of response specificity, including within this problem the test of the second hypothesis which is concerned with the effects of feedback on two aspects of cardiac activity when only one is instructed to be decreased; and (6) theoretical and practical implications.

The following abbreviations will be used in this discussion chapter: (1) cardiac rate--HR; (2) cardiac variability--HRV; (3) correct feedback--CF; (4) incorrect feedback--IF; (5) display--D; (6) no-display--ND; (7) single instruction condition--SIC; (8) combined instruction condition--CIC; (9) respiration rate--RR; (10) respiration amplitude--RA; (11) skin conductance--SC; and (12) striate muscle activity--EMG.

The Effect of Feedback on the Control of Cardiac Variability and Cardiac Rate

The results of the present investigation clearly indicate that subjects (Ss) can reduce their cardiac variability under conditions of correct feedback, therefore confirming the findings of Hnatiow and Lang (1965) and Lang, Sroufe and Hastings (1967). The finding of no significant difference between display (D) and no-display (ND) trials for the incorrect feedback (IF) group suggests that the results cannot be fully accounted for merely by attending to the feedback.

It is also clear that Ss are able to decrease their cardiac rate (HR) when so instructed, assuming that correct feedback is available. Since Ss are able to decrease their HR just as they are able to stabilize it, support of the initial hypothesis is assured. Like the cardiac variability (HRV) results, the finding of a nonsignificant difference in HR between D and ND trials in the IF group does suggest that the results cannot be fully accounted for by the unconditioned effect of attending to an external stimulus.

It was not expected a priori that the IF group would tend to increase their instructed cardiac variable on the D trials, particularly in the HR instruction condition. It was anticipated, on an a priori basis, that HR would decrease on the D trials because of the unconditioned response of the HR to attending to a stimulus as described by Lacey, Kagan, Lacey and Moss (1963). The finding that HR, like HRV, increased during D trials in the IF group suggests, according to the Lacey et al. (1963) hypothesis, that the Ss were not attending but either pondering or rejecting the incorrect feedback. However, this possibility does not seem entirely tenable since nonsignificant HR decreases on the

D trials relative to the ND trials were observed when instructions were given to the IF group to decrease HRV only ($p > .10$). These HR decreases are presented in Appendix C. It is possible that the consistent increases for the instructed cardiac variable across D trials were caused by the assumed 50% reinforcement of HR and HRV increases. This is suspected since Engel and Chism (1967a) and Levene et al. (1968) have reported that HR increases are easier to learn than decreases. This may also be the case for HRV, although it has not been empirically determined. Engel and Chism and Levene point out the reason HR increases are easier to learn than decreases is that the most typical individual response specificity pattern to a perceived stress is HR increment. Thus, for the IF groups under a theoretical 50% reinforcement, cardiac activity increments, rather than decrements, occurred because the autonomic response system is typically biased in that direction. Had reinforcement exceeded 50%, eventually the bias would have been overcome, leading to cardiac activity decrement. It is possible that Ss were frustrated in the IF group and, consequently, responded to this frustration with cardiac increases. However, post-experimental interviews failed to reveal any tendency for the IF Ss to be more frustrated than the CF Ss. Post-experimental interview questions are given in Appendix D.

The observation of Engel and Chism (1967a) and Levene et al. (1968) that HR increases are easier to learn than decreases may cast some doubt on the authenticity of the CF cardiac activity decreases on the D trials relative to the ND trials. A case could just as easily be made, depending on how one views the data, that the Ss were actually learning to increase HR and HRV rather than to decrease it. This possi-

bility will be discussed more fully in the discussion section on response specificity. Although cardiac increments producing apparent decrements may have occurred to some degree, it is probable that cardiac activity was actively being decreased as well, since cardiac activity on the CF D trials was consistently and significantly lower than the cardiac activity of the IF group on D trials.

Hnatiow and Lang (1965) and Lang et al. (1967) also observed backlashes or increments of HRV on ND trials relative to D trials. They postulated that further trials might produce a more lasting effect leading to a reduction of the ND trial increments. In the present study, for both HRV and HR, four additional trials were given relative to the Lang investigations, two display and two no-display. It is apparent from the data presented in Figures 1 and 3 (pp. 37 and 42) that the later ND trials are still characterized by cardiac increments relative to the D trials. It is possible that mediative effects may, at least in part, account for this backlash effect on the ND trials. Discussion of this possibility will be deferred until the section on response specificity.

Control of Cardiac Activity in Combined and in Single Instruction Conditions

Simultaneous Control of HR and HRV

The finding of a significant difference between D and ND trials for both HR and HRV under the combined instruction condition, as well as the significant difference between CF and IF groups on D trials, suggests that HR and HRV can be controlled together. These observations are supported by the significant within-group D+ND estimated and group mean partial correlations existing between HR and HRV in the CF group. The

finding of a significant relationship is unusual. Normally, HR and HRV show no relationship (Hnatiow and Lang, 1965). Since the between-groups analysis for partial correlations indicated no significant difference for D+ND trials, while the positive partial correlation in the IF group is of borderline significance for D+ND trials, it is possible that the degree of relationship between HR and HRV is in part a function of not only the instructions per se but also the proportion of correct feedback available. This possibility again depends on the assumption that IF is characterized by 50% correct reinforcement or feedback, but in the absence of a display there is approximately zero feedback.

Effectiveness of Correct Feedback Under Different Instruction Conditions

In order to determine the relative effectiveness of CF under different instruction conditions, a number of logical comparisons were made within- and between-instruction conditions. Of all the logical comparisons (HRV decreased singly vs HRV decreased in combination; HR decreased singly vs HR decreased in combination; HRV decreased singly vs HR decreased singly; and HRV decreased in combination vs HR decreased in combination), CF was found to be least effective in the HRV SIC CF group. Although there was no significant difference in any of the group main effects, significant group x trial interactions were observed in the HRV SIC versus HR SIC and, more importantly, in the HRV SIC versus HRV CIC comparisons. These interactions indicated that as trials increased, the absolute amount of decrement for the HRV SIC CF group decreased, while the absolute amount of decrement for the HR SIC and HRV CIC CF groups increased. Hnatiow and Lang (1965) also reported that

overall HRV increased over trials. It was postulated that the gradual loss of cardiac stability over trials was caused by the initial rest period being too brief. However, since HRV SIC was the only group in which this occurred in the present study, it is possible that an overall HRV increase is an unconditioned effect of decreasing HRV singly. From a conceptual point of view, it is possible that HRV would be more difficult to decrease than would HR. To stabilize HRV would require the S to make correctional HR adjustments in two opposing directions while Ss instructed to decrease HR must make correctional adjustments in only one direction. If a S who was instructed to decrease HRV made a strong correction in one direction, he might be thrown in the other direction, thereby having the net effect of increasing rather than decreasing HRV. Consequently, while the Ss were apparently able to avoid this oscillation effect in the early part of the study, in the later trials, possibly due to fatigue, they became more careless, over-controlling in one direction or the other so that their performance decreased relative to the other groups. Though this interpretation successfully handles the interaction effect found in the HR SIC versus HRV SIC D trial comparison, it fails to explain the lack of difference between HRV and HR in the CIC CF group, unless it is further assumed that there is a lower physiological limit which is being infringed upon as HR is decreased. In summary, HRV could be controlled primarily in one direction, that being suppression of HR. Thus, oscillations in the direction of further decrement would be prevented by a lower physiological limit now in range by virtue of the overall HR being decreased.

Similar to the findings of previous instrumental conditioning and feedback studies of cardiac activity modification, the extent of

change in terms of raw scores is not large for either HR or HRV. For example, the largest difference between ND and D for the CF CIC group for mean HR over three-minute periods was only 3.9 bpm while for HRV on the same ND to D period there was a difference of 2.0 standard deviations (SD). In terms of T score differences based on the cardiac activity distribution of 11 trials and 10 ND to D changes, these raw score differences may take on more biological significance since both variables on the same ND to D period decreased 10.2 T scores, or approximately one-plus SDs indicating a change in the activity level of both cardiac variables of 38%.

Although sex differences were not formally examined in the present investigation, it was apparent from examination of the raw data that there was no obvious difference between the sexes as to amount of cardiac control. None of the previous studies on cardiac control have reported a sex difference.

Post-Experimental Interviews

Brief post-experimental interviews were conducted with each S as done in previous instrumental conditioning and feedback studies (see Appendix D). Perhaps the most striking finding of these interviews was that none of the 60 Ss were aware that cardiac activity was involved in any way. This finding is understandable in view of the fact that cardiac feedback was presented in an analogue rather than a digital or pulse form.

When Ss were asked whether they felt they successfully controlled their instructed bodily function, approximately 75% reported success. The remainder was nonsignificantly divided between CF and IF groups. An

examination of the individual records of those CF Ss claiming no control indicated that they were about as successful as those Ss who claimed some control in the CF group. Similarly, those Ss claiming control in the IF group performed in a manner typical of the rest of the IF group. Only one S in the entire study reported frustration in his attempts to gain control. This finding is perhaps explainable by the fact that all Ss were cautioned by instruction not to worry about how well they were doing and to avoid feelings of frustration.

Answers pertaining to how the Ss attained control were very diverse with no consistent tendency being noted. Most did say that all they did was watch the meter as they were instructed.

The most important conclusion to be drawn from the post-experimental interviews is that the IF Ss were properly deceived by the incorrect feedback signal even though they had had an opportunity to observe their own cardiac signal during the instruction period. The success of this deception is highly important to the overall findings of this investigation, since failure to deceive would imply failure of the IF group as an adequate control group.

The Problem of Habituation versus Learning Over Trials

In most instrumental conditioning and other learning situations, a learning or acquisition curve may be demonstrated over repeated trials. Surprisingly, in the present investigation little evidence of a learning curve was found. Since it is apparent that the correct feedback was effective in producing the desired cardiac responses, it must be concluded that learning did occur at some time in the study. Had cardiac activity decreases occurred in the IF groups, the observed decreases in

the CF groups would be subject to the alternative explanation that the decreases were produced by the unconditioned response of attending to the feedback. Engel and Hansen (1966), Engel and Chism (1967a), Hnatiow and Lang (1965) and Lang et al. (1967) all reported the same finding as in the present study. Since correct performance did occur as early as the first trial in each of these investigations, as well as the present one, it has been concluded by each of the cited investigators that one-trial learning must have occurred. Levene et al. (1968) is the only investigator to present evidence of an acquisition curve. Unfortunately, no control group was employed; therefore the curve which he presents may reflect habituation rather than acquisition. The significant acquisition curve found in the present study in the HRV CIC CF group represents the only learning curve in the literature which cannot be accounted for by habituation over trials.

It is also curious that successful performance failed to carry over into the ND trials. It would be expected that the Ss would be more sensitized to their own internal feedback and thus be able to carry on to some degree. The Ss were instructed to attempt to continue controlling in the absence of feedback. A problem related to the lack of transfer to ND trials was encountered by Miller and DiCara (1967) who found that curarized and artificially respirated rats changed their overall HR in the rewarded direction during both S^d and non-S^d periods. This finding was interpreted as indicating that the rats were failing to discriminate between the two conditions. With additional trials, however, discrimination was achieved. In view of Miller and DiCara's findings, it is possible that in the present study, the lack of transfer to the

ND trials represents successful discrimination between S^d (D trials) and non-S^d (ND trials) periods. The alternate possibility also exists that Ss in the present study may have been actively mediating or in some unknown manner producing cardiac activity increments as well as decrements from ND to D trials. These and other possibilities will be discussed further in the discussion section on response specificity.

In conclusion, little evidence for learning in the usual sense was found since acquisition curves could not be distinguished from habituation curves on a statistical basis, except for the HRV CIC CF group. Because successful performance did occur in all CF groups, it is probable that learning did take place within the first or even second or third trials. Failure for transfer of performance from D to ND trials to occur may be due to a number of contaminating, possibly mediational, factors. The possibility also remains that lack of transfer reflects learning superior to that in which transfer to ND trials occurred. Transfer to ND trials was, as mentioned above, not observed in the present study.

Response Specificity

The Effect of Feedback-Decreased HR on HRV and Vice Versa

The second prediction, that a negative relationship exists between HR and HRV when either is singly decreased by correct feedback, was not supported. This hypothesis was based on the presence of a noticeable trend in the results of Brener and Hothersall (1966) and Engel and Hansen (1966) for variability to increase when rate decreased, and in the results of Lang et al. (1967) where HR tended to increase when

HRV was decreased.

Covariation of Other Response Systems

RR and HRV. Although significant partial correlations were not found for every comparison between RR and cardiac activity, there was a consistent tendency for RR to be negatively correlated with both HR and HRV in all CF groups. Thus, when either cardiac variable was decreased singly or in combination, RR tended to increase. Conversely, when either cardiac variable was increased, RR tended to decrease. Judging on the basis of significance levels presented in Tables 20 (p. 73), 22 (p. 74), 24 (p. 76) and 27 (p. 79), RR was most significantly negatively correlated with HRV and HR in the CF combined instruction condition. The correlations were generally low or nonsignificant in the SIC CF groups.

Traditionally, past research in this area has interpreted any corresponding RR changes as mediating the cardiac changes. It is also known that manipulation of certain cardiac variables will affect respiration (Brener, 1967). Hence, evidence of a relationship does not imply direction of causality.

The respiration mediation hypothesis was examined by Engel and Chism (1967b) who paced RR at different speeds with a metronome. It was found that by increasing the RR, HRV decreased, while decreasing the RR increased the HRV. Engel and Chism's findings comport well with those of Westcott and Huttenlocher (1961) who observed that one major contributor to overall HRV, sinus arrhythmia, is greatest when respiration is slow and deep and least when respiration is shallow and rapid. However, Engel and Chism reported that sinus arrhythmia was maintained during the

fast-paced breathing. Therefore, other aspects of overall HRV would be decreasing such as longer-term baseline variability and beat-to-beat changes. The observation that sinus arrhythmia was persisting in the fast-paced condition suggests that the Ss' HRV may have been decreased through hyperventilation effects. Normally, in the non-exercise condition RR and respiration amplitude (RA) are inversely related, since an increase in either one without a compensatory decrease in the other would soon result in a lowering of P_{CO_2} in arterial blood. Failure of this compensatory mechanism to occur would be reflected in a greater positive relationship between RR and RA. The observation made by Engel and Chism (1967b) that sinus arrhythmia was persisting during the fast-paced breathing may indicate that these Ss were breathing not only rapidly but deeply, therefore suggesting a positive relationship for RR and RA. According to the findings of Westcott and Huttenlocher (1961), deep breathing is necessary for sinus arrhythmia to persist.

In the combined instruction condition of the present investigation, a significant negative relationship was observed for HRV (D trials only) and HR (D+ND and ND trials). Respiration amplitude was not intended as a measure in this study, and therefore no precautions were taken to insure accuracy in its recording. Consequently, this variable was not analyzed with HRV and HR under the different instruction conditions. However, in an attempt to check on the hyperventilation possibility, an after-the-fact partial correlational analysis was conducted between RR and RA holding trials constant for the CF and IF CIC groups. This instruction condition was selected for analysis, since RR was more consistently and significantly correlated with cardiac activity in this condition.

The results of this analysis suggest that the CF group, while they may have been mediating cardiac changes through RR, were not hyperventilating as the correlations did show a reasonably consistent negative trend and did not differ significantly with the IF control group. In an attempt to verify this conclusion, an examination of the individual response profiles of two CF group Ss, both showing large partial correlations between RR and RA but in opposite directions, was carried out. Subject P.S. showed a partial $r_{12.3}$ between RR and RA of +.76, while subject J.C. showed a partial $r_{12.3}$ of -.90. Both of these Ss performed poorly in cardiac activity decrement relative to the overall group performance, particularly when the direction of cardiac change between D and ND trials was analyzed. Overall for both HR and HRV, with a possible of 20 correct direction changes, P.S. made 12 correct changes while J.C. made 11. Considered on an individual basis, neither S's RR correlated significantly with HR although by one-tail test, S J.C. did show a significant negative correlation between RR and HRV. Although these individual cases cannot be considered collectively as proof that hyperventilation was not involved in the possible mediation of the desired cardiac changes, they do lend support to this thesis.

Another factor which argues against the hyperventilation-mediation possibility is the effect of such respiratory maneuvers on HR. According to Schneider (1930), Brown (1953), and Best and Taylor (1966, pp. 898-900), when hyperventilation reduces the P_{CO_2} of the arterial blood, a lowering of the tonic activity of the vasoconstrictor center is produced, which then causes arteriolar dilation resulting in a fall in blood pressure. As a compensatory adjustment resulting from the sinoaortic reflex, HR increases.

The implication of this for the present study is that, although overall HRV could be reduced through hyperventilation effects, reduction of HR would be made more difficult. The findings of the present study contraindicate this, since there was no significant difference in the amount of decrement between HR and HRV in the combined instruction condition. Engel and Chism (1967b) reported that HR increased but not to a significant level during fast-paced breathing. If their Ss were hyperventilating as suspected, HR would be expected to increase. However, results reported by Schneider (1930) indicated that HR does not increase markedly in humans until approximately 11 minutes of hyperventilation. Fast-paced breathing was terminated in the Engel and Chism study after 10 minutes; therefore, significant HR changes may not have had time to occur. A second effect of hyperventilation is compensatory peripheral vasoconstriction in the forearm and the hand (Schneider, 1930; Brown, 1953). Engel and Chism (1967b) did find that finger pulse volume and finger temperature did decrease significantly in four of their six Ss. This finding of decreased finger pulse volume and temperature does support the inference that hyperventilation may have been occurring in their Ss. Unfortunately, finger pulse volume and temperature were not measured in the present study. Therefore, it cannot be determined if these peripheral changes occurred with RR increases associated with cardiac activity decreases.

Although hyperventilation mediation can be tentatively ruled out as occurring in the present study, the possibility of RR mediation cannot. Westcott and Huttenlocher (1961) reported that rapid, shallow breathing, which yields normal ventilation, reduced HRV. It is probable that the HRV was reduced by the reduction of sinus arrhythmia caused,

at least in part, by deep breathing. The deeper the inhalation, the greater is the inhibition of the cardioinhibitory control area in the medulla oblongata, the inhibition being initiated by the activation of vagal recepters by inspiration. This disinhibition reflex causes an increase in the HR. Upon expiration there is a return of tonic vagal discharge which slows the HR.

Since HRV was decreased in the CF CIC group on D trials relative to ND trials and the D trials of the IF control group, and this decrement was associated with a significant partial correlation between HRV and RR across D trials, it is possible that these decrements were respiration mediated. The finding of a negative partial correlation between RR and HRV suggests that the greater the increase in RR, and correspondingly the greater the decrease in RA, the greater the decrease in HRV. Therefore, maximum HRV decrement for D trials only occurred in the presence of a respiration pattern known to decrease HRV. This inference is supported by eye-ball inspection of the raw data in that sinus arrhythmia does appear to be reduced to the greatest degree on those D trials where RR is most rapid and overall HRV is minimal. Had RR changes been positively associated with HRV decrements, a change not known to produce HRV decrement, an alternative interpretation would be favored such as parallel RR changes or mediation of RR changes through HRV changes. In conclusion, although these observations do not verify the possibility that HRV decrements were respiration mediated, such an interpretation is not inconsistent with the data.

Because HRV decrement was not associated with RR in the CF SIC group, it may also be tentatively concluded that RR mediation is not a necessary condition, although it may be a sufficient one for decrement

of HRV.

It should be pointed out that although a negative relationship does exist between HRV and RR for D trials, indicating that maximum HRV decreases are associated with maximum RR increases, the relationship is not maintained beyond the D trials. Therefore, it does not follow that RR would be slower on ND trials than on D trials, even though HRV increases. Consequently, it appears that one effect of correct feedback is to couple RR and HRV. This coupling effect is not maintained beyond the D trials.

RR and HR. Though much of the discussion concerning RR and HRV involves HR, it appears, on the basis of Engel and Chism's (1967b) findings and from the analyses of the combined instruction condition data in the present study, that RR mediation of HR, if it is occurring, is doing so in a manner different from that described for HRV. Engel and Chism reported that although average HR across Ss was not affected by either fast or slow paced respiration, a minute-by-minute analysis of HR during paced breathing indicated that there was a definite tendency for slow breathing to increase HR while fast breathing did not. In the present study under the CF CIC, HR increases for ND trials only were significantly correlated with decreases in RR. This trend was also apparent on the ND trials in the CF HR SIC group. Although maximum HR for ND trials in the CF CIC group was associated with minimal RR and maximal RA, HRV was not significantly correlated with RR primarily because HRV was not significantly correlated with HR on ND trials, and secondarily because the RA increments were probably not of sufficient magnitude to produce increased HRV via sinus arrhythmia. However, on

the ND trials in the HR CF SIC group, there was a trend falling just short of statistical significance for HR to be correlated with RR while HRV in the same group was correlated positively and significantly with HR. It is probable, therefore, that increased HRV was caused by increased sinus arrhythmia.

The finding of a significant negative partial correlation between HR and RR for ND trials in the CF CIC group suggests that although the Ss were given instructions designed to decrease HR, they may have been increasing it on ND trials, possibly by respiratory mediation. The findings of Brener and Hothersall (1966; 1967), Donelson (1966), and Levene et al. (1968) support the suspicion that the HR increased actively on the ND trials, since in each of these studies it was difficult to determine whether Ss were either increasing or decreasing their HR or both. Two of the Ss in the Levene study did show evidence of being able to alternately increase and decrease their HR at one-minute intervals, but these results may have been biased in favor of decreases through habituation over trials. Such a bias could occur, yet HR increases would still be able to reach statistical significance when compared to the initial baseline. This contention is supported by the observation of Engel and Chism (1967a) and Levene et al. (1968) that HR speeding is easier to learn than HR slowing. This final point is particularly relevant to the interpretation of the findings of the present study, since active HR increases may have been mediated by RR during ND trials.

Results reported by Donelson (1966) on the order effect of attempts to either increase or decrease the HR first, followed by the

reverse order a short time later, indicated that control is best for Ss who try to increase HR first and then try to decrease it, rather than vice versa. These results are comparable to those obtained on the first few trials in the CF HR SIC group where HR failed to decrease on the first D trial, but increased further on the second ND trial. On the second D trial a reliable decrease occurred for the first time. This should be contrasted to the CF HRV SIC where a reliable decrease occurred on the first D trial. These findings, along with those of Donelson (1966), offer additional support to the interpretation that not only are HR increases being actively produced in the present study, but they may in fact be necessary for HR decreases to occur. It should be pointed out that regardless of how or when HR decreases were effected, whether they were active or passive, the decreases were more than just apparent as contrasted to the ND trials. This is supported by the observation that a significant difference did exist between CF and IF HR single and combined instruction groups for D trials, while there was no significant difference between D and ND trials in the IF groups.

Levene et al. (1968) reported results on one particular S who showed evidence of RR mediation of HR increases. When this particular S, who had marked sinus arrhythmia in the resting state, managed to slow her HR, sinus arrhythmia became greatly reduced. On the other hand, her HR increases were associated with increases in sinus arrhythmia. This suggests that this S was breathing slowly and deeply. It is possible that the HR increases were produced, at least in part, by the peaks of the sinus arrhythmia. Although the evidence in the present study failed to indicate that increased HRV during the ND trials in the CF CIC group was caused by increased sinus arrhythmia, support was found for this

possibility in the CF HR SIC group. As pointed out earlier in this discussion, another factor contributing to the HR increases is the tendency for most Ss to react to experimental conditions with increased HR. The reason HR decreases are apparently more difficult to effect is because most Ss' individual response specificity profiles include a pattern of HR increment to any perceived stress situation.

As a tentative conclusion to this discussion, it appears that RR mediation on the ND trials could have been partly responsible for the D trial HR decreases. This conclusion is based not only on the results of the present study but also on the findings of previous investigations in this area. There is no definitive evidence to support the respiration mediation hypothesis. However, the evidence presented in this discussion is not inconsistent with the hypothesis, since the respiratory patterns found in this and other studies have been demonstrated by Engel and Chism (1967b) to be capable of producing the types of cardiac activity observed in the present study.

Future research dealing with the problem of respiration mediation of cardiac activity should approach the problem in reverse order to the present study, by providing feedback and instructions to control respiration while observing the concomitant cardiac changes. The effect of cardiac changes on respiration induced by methods other than feedback should also be examined. If it can be demonstrated that these induced cardiac changes do not produce the type of respiration activity seen in the present study and if it is apparent that respiration feedback can produce cardiac changes similar to those produced by cardiac feedback, then the respiration mediation hypothesis can be accepted with less reservation than in the present study.

Skin conductance and cardiac activity. Similar to the findings for respiration, skin conductance (SC) did not correlate significantly with cardiac changes on every comparison. There was, however, a consistent tendency for SC to be correlated positively with both HRV and HR, particularly in the single instruction conditions for D trials only. Thus, when either cardiac variable was decreased, SC tended to decrease as well. Conversely, when either cardiac variable increased, SC also tended to increase.

Only two previous studies (Donelson, 1966; Frazier, 1966) have recorded electrodermal activity while attempting to modify cardiac activity through instrumental conditioning or feedback procedures. The only significant finding of the Donelson study was an increase in skin resistance over trials. Frazier did report SC changes accompanying HR changes. However, these parallel changes only occurred under threat of shock, a finding which is not at all unusual.

Traditionally, past studies and theoretical papers in this area have chosen to interpret any additional autonomic changes accompanying instrumentally conditioned or feedback-induced cardiac changes as evidence for lack of response specificity. Further, the co-variable is interpreted as being either a mediational variable as is respiration, or the covariation is interpreted as reflecting a general change in activation level or arousal, especially when there is more than one covariate.

In the present study, the finding of a significant correlation between SC and HR may be interpreted as not inconsistent with the possibility that arousal mediation or mediation by relaxation occurred in the CF HR SIC group for D trials only. It is possible, of course, that the feedback-induced HR changes mediated the SC changes either through vas-

cular changes or by changing levels of arousal. In consideration of the latter possibility, if the SC changes were secondary to arousal used in control, it would be expected that SC would increase on ND trials. This did not occur. Therefore, it is doubtful that arousal changes were secondary to the HR changes.

As arousal mediation is more typically thought of, the case for activation mediation of HR activity would have been more defensible had RR also covaried with HR during the D trials, not because RR might be a good measure of arousal, but because it would be expected on the basis of an arousal hypothesis that more than one response system should covary with HR. If it is assumed that any trend toward a significant correlation between cardiac activity and RR or SC reflects some form of mediation of the cardiac changes during correct feedback, the results of the present study suggest that there may be more than one type of mediation. However, since RR and SC failed to approach correlating significantly with the instructed cardiac variable simultaneously, it appears that these two types of mediation are mutually exclusive. This very tenuous interpretation makes the assumption that it is proper to deal with continuous variables as if they were discrete.

Regardless of whether or not the interpretation of arousal mediation of HR activity during D trials is warranted on the basis of the available data, the finding that SC did correlate with HR does indicate lack of response specificity. This is also true for HRV in the CF CIC group during D+ND trials and to a lesser extent in the HRV SIC group for D+ND and D trials, since both HRV groups showed partial correlations falling just short of acceptable significance levels ($p < .10$).

Striate muscle and cardiac activity. Very little evidence was found to support the possibility that the observed cardiac changes were associated with changes in striate muscle control. In general, the EMG data for the CIC groups indicated a slight but nonsignificant tendency for EMG activity to decrease when cardiac activity was decreased. This relationship reached significance in the between-groups analysis for the ND trials when HRV was decreased in the CF CIC group. This relationship was reversed and differed significantly from zero on the ND trials in the IF group, so that when cardiac variability increased, EMG activity decreased. This relationship is unique only to the ND trials of the IF group. It does not follow that this relationship would be maintained across all trials as it was observed that both HRV and EMG decreased together. It is important to note that the significant negative relationship was apparent only when the trials effect was partialed out.

The observation of a tendency for HR and EMG to be positively related is supported in a loose sense by the findings of Obrist (1968) who observed an inhibition of EMG activity which was concomitant with anticipatory deceleration of HR. Conversely, the HR accelerated when a burst of EMG activity occurred. Though the differences between the Obrist study and the present are obvious in terms of conditioning procedure and type of response measured, the concordance between the two investigations suggests that the direction of the relationship between cardiac activity and EMG is fairly consistent.

Because the trends examined in this discussion were largely nonsignificant, it cannot be concluded with any degree of certainty that

the observed cardiac changes were related to muscular changes. It is possible that striate muscle activity elsewhere in the body may have been covarying to a significant degree with cardiac changes. Future research on this problem should attempt to record EMG from a number of different sites. Since the Ss in this investigation were instructed to remain as still as possible throughout the 55 minutes recording time, the low partial correlations which were observed may have been due to the difficulty of detecting further probable decreases in flexor muscle amplitude because the muscles were already in a voluntarily induced state of minimal activity.

Theoretical and Practical Implications

The evidence in this study points to the conclusion that Ss can gain considerable control over their own cardiac rate and/or variability when correct feedback is available. This study provides the first demonstration of HR decrement induced by an instrumental conditioning technique where psychological rather than physical reinforcement was used. Three other feedback studies have attempted to decrease HR but were unsuccessful due to the lack of adequate experimental controls, therefore making it impossible to distinguish HR increment from decrement (Brener and Hothersall, 1966; 1967) and decrement from habituation (Donelson, 1966). The necessity for having proper experimental controls, especially when attempting to decrease HR, was made particularly clear by the findings of Engel and Chism (1967a) and Levene et al. (1968) who found that with physical reinforcers HR increments were easier to learn than HR decrements.

Beyond demonstrating that feedback of cardiac activity can be

used to decrease and/or stabilize the HR in the absence of physical reinforcers, the present investigation was designed to distinguish whether feedback-induced changes in cardiac activity show response specificity. Though it is recognized that evidence against response specificity could be due to changes induced in other response variables by the cardiac changes or by other variables responding in parallel with the cardiac changes, lack of response specificity is damaging to the hypothesis that instrumental conditioning of cardiac activity occurred directly. The reason for this is twofold. First, it was originally believed by Skinner (1953) that autonomic functions could not be conditioned instrumentally, and second, it is theoretically possible for autonomic activity to be shaped indirectly through inadvertent reinforcement of operant behavior which mediates the desired autonomic changes. Thus, any substantial evidence of a lack of response specificity accompanying instrumentally induced cardiac changes has automatically been interpreted by most in the literature as indicating mediation. If respiratory or striate musculature changes occur, then the autonomic activity has been mediated by instrumentally conditioned somatically innervated responses. If skin conductance changes occur or a number of other response systems become more active simultaneously, then autonomic activity has been mediated by instrumentally conditioned changes in arousal. The validity of these mediation interpretations has never been determined, although it has been challenged by Crider, Schwartz and Shnidman (in press). Until the validity of these interpretations is established, evidence of a lack of response specificity raises the alternative interpretation that mediation of the dependent variable has occurred rather than instrumental conditioning *per se*.

The mediation problem has led Katkin and Murray (1968) to state that instrumental ANS conditioning has not been demonstrated definitively in humans, although it has in the curarized rat studies. Katkin and Murray have forced the issue in a sense by their statement that "...at a practical level a distinction must be drawn between conditioning the ANS and controlling it." (Katkin and Murray, 1968, p. 66). They also added, "In fact, it is probably fruitless to pursue further any attempts at providing such demonstrations in humans...." (Katkin and Murray, 1968, p. 66).

To return to the findings of the present study, it appears that Katkin and Murray may have been overly pessimistic about "...such demonstrations in humans...." since there was no significant evidence in the HRV single instruction condition to indicate a lack of response specificity. This instruction condition is, for all practical purposes, a replication of the Hnatiow and Lang (1965) and Lang et al. (1967) studies on feedback-induced stabilization of HR. In both of the Lang studies, respiration activity was found to be unrelated to reduction of HRV. The results of the present investigation may be interpreted as supporting the findings of the Lang studies. In addition, there was no evidence to indicate that SC and HRV were covarying to any significant degree. Therefore, the results of the Lang studies are both confirmed and extended to include electrodermal activity. From a theoretical viewpoint, these combined findings may be taken as evidence for instrumental conditioning of the ANS, specifically cardiac variability. This conclusion should be accepted with caution, however, since it is possible that response systems other than those selected for observation could have been responding in unison with the cardiac variability.

Because there was evidence of a lack of response specificity in the HR and combined instruction condition groups, the possibility does exist that the Ss' cardiac activity was not instrumentally conditioned directly but controlled either through arousal changes, as in the HR single instruction condition group, or by respiratory maneuvers, as in the combined instruction group, where RR decreased when cardiac activity increased and vice versa. The conclusion of control rather than instrumental conditioning per se is further supported by the finding of an absence of a learning curve for the dependent cardiac variables. If some form of mediation was occurring, one trial learning with little or no further improvement might be expected, since the desired cardiac changes would actually be unconditioned responses which would by nature be subject to habituation to the unconditioned physiological stimuli producing them.

Although lack of response specificity in an ANS instrumental conditioning or feedback study may presently imply that it is possible to attain control through mediation of one's own involuntary behavior, but not instrumental conditioning per se, at a practical level there are many obvious implications which are not dependent upon theoretical distinctions. The finding that autonomic responses may be modified through instrumental procedures, as in the HRV SIC CF group, indicates that the reinforcement of such changes is not restricted to unconditioned stimuli which produce as an unconditioned response the specific desired change. The finding that autonomic responses, such as cardiac activity, can be modified either directly or indirectly through instrumental conditioning using either physical or psychological reinforcers clearly in-

dicates that it is possible for the visceral responses to be altered by instrumental processes. This may lead to the formation of individual response specificity hierarchies which under extreme reinforcing conditions may lead to psychosomatic symptoms. The degree to which such visceral responses occur probably depends upon the degree to which particular reinforcement conditions are present in the life of each individual.

Finally, from a very practical viewpoint, the instrumental control of autonomic responses has great therapeutic potential for both clinical psychology and medicine. By providing feedback to a patient, it should be possible to replace undesirable responses with more desirable ones. It is apparent from the results of the present study that response specificity can be altered by instructions resulting in the possible modification of either one or, if desired, two variables at a time, as shown by the single and combined instruction condition groups. As an example of the possible therapeutic implications of the control of two variables simultaneously, Levene et al. (1968) reported that some of the Ss in the study, in the course of learning to control their HR, acquired control over their cardiac arrhythmias as well.

Any possible applications of feedback treatment should be used with caution, particularly when attempting to modify vital functions, since the findings of the present study concerning the ND trial backlash effect imply that when cardiac activity is decreased through feedback, the physiological pendulum seems to swing too far in the undesirable direction when feedback is removed abruptly. While the backlash effect may be necessary for the control of HR, the effect may also indicate

that the homeostatic mechanisms are being altered too greatly or perhaps too rapidly. Future research should observe the presence of the effect after extended periods of feedback treatment.

CHAPTER V

SUMMARY AND CONCLUSIONS

Studies on cardiac control have recently been reported which demonstrate that Ss can learn to control their own cardiac activity. Prior to the present interest in this area, it was widely assumed that autonomic responses could not be instrumentally conditioned, since autonomic functions were not capable of providing the kinds of consequences found in operant reinforcement.

Studies claiming successful instrumental conditioning of ANS functions have been challenged to demonstrate response specificity. Otherwise, results were open to the alternative interpretation that inadvertent reinforcement of operant behavior had occurred which served to mediate the desired autonomic changes. Studies attempting to modify cardiac rate through feedback procedures where psychological rather than physical reinforcers were used have also experienced difficulty in avoiding the problem of mediation. In addition, results demonstrating control over cardiac activity have been ambiguous as to whether cardiac rate was being increased or decreased. Since cardiac rate had never been shown to be susceptible to feedback-induced decrement, regardless of the presence of possible mediating variables, a feedback procedure was adopted which has been used successfully by

Hnatiow and Lang (1965) and Lang et al. (1967) to stabilize cardiac rate in the absence of obvious mediating influences.

The present investigation attempted to replicate and extend the findings of the Lang studies. Thus, it was predicted that feedback not only could be used to stabilize the HR but also could be used to decrease it. Although no prediction was made, it was anticipated that feedback could be used to both stabilize and decrease the HR at the same time. As a control for this demonstration, it was necessary to determine the mutual effect of the two variables when each was singly decreased through feedback. On the basis of previous findings, it was predicted that the two variables would be inversely related. It was anticipated that this analysis would be useful in the interpretation of the data in the combined instruction condition. Comparisons were then made within and among instruction conditions in order to detect possible differences in the stabilization and/or decrement of HR using amount of absolute decrement as the dependent variable.

The results supported the first hypothesis that feedback could be used to decrease the HR just as it could be used to stabilize the HR. The results were found to be significant for both HR and HRV instruction conditions both within (display versus no-display trials) and between (correct feedback versus incorrect feedback) groups. It was also found that HR and HRV could be decreased together. The results for the second hypothesis were not supported, since the correlation for HRV and HR did not differ significantly from zero when HRV was decreased singly. In addition, HR was found to be positively correlated with HRV when HR was decreased through feedback. This in-

dicates that simultaneous HR decrement and stabilization may have been achieved through concentration upon the decrement of HR. The results for the comparison analyses indicated that when HRV was decreased singly, the absolute amount of decrement decreased as trials increased, relative to HR decreased singly and HRV decreased in combination with HR. Cardiac rate was implicated as being partly responsible for this interaction effect. It was further suggested that HRV is more difficult to control than HR.

Partial correlation analyses indicated a general lack of response specificity occurring in all but the HRV singly instruction condition, where no evidence was found to be inconsistent with the interpretation that instrumental conditioning rather than control of HRV had occurred. Evidence was found in the combined instruction condition that respiration rate was varying inversely to a significant degree with HRV during display trials and with HR during the no-display trials. This finding allows the alternative interpretation to be made that cardiac activity, rather than being directly instrumentally conditioned, is controlled by the instrumentally conditioned respiratory rate, which in turn mediates the desired cardiac decrements. Analysis of respiration amplitude in the combined instruction condition failed to indicate that hyperventilation was occurring, since RR and RA were negatively related in both correct and incorrect feedback groups. Therefore, it is unlikely that Ss were mediating cardiac changes through hyperventilation.

On the basis of the finding that RR was inversely related with HR on no-display trials, it was inferred that HR may have been actively

mediated by RR to produce HR increases rather than decreases as instructed. Since RR was inversely related with HRV on the display trials, it was inferred that if mediation of HRV was occurring, it was taking place during HRV decreases. Respiration rate did not change significantly from display to no-display trials for any of the instruction or feedback conditions.

There was very little evidence to indicate that skin conductance was covarying with cardiac activity, although there was a general, but nonsignificant, trend for SC to correlate positively with HR and HRV in all correct feedback instruction conditions. The relationship was strongest in the HR single instruction condition, where SC and HR were significantly correlated on the display trials both within and between groups. Until more is known about the problem of mediation, the observed absence of response specificity for SC and HR may indicate mediation through changes in arousal.

Striate muscle activity was analyzed only in the combined instruction condition and was found in general to be nonsignificantly, but positively correlated with both HR and HRV.

Of the four instruction condition CF groups, only the HRV combined instruction condition group showed a significant acquisition curve over trials. In those cases where no acquisition curve for cardiac changes was found, it is probable that one-trial learning occurred, perhaps through mediation.

The theoretical and practical implications of this investigation were discussed with special emphasis on the issue of whether instrumental conditioning or control of cardiac changes occurred. Suggestions were made for future research using feedback procedures.

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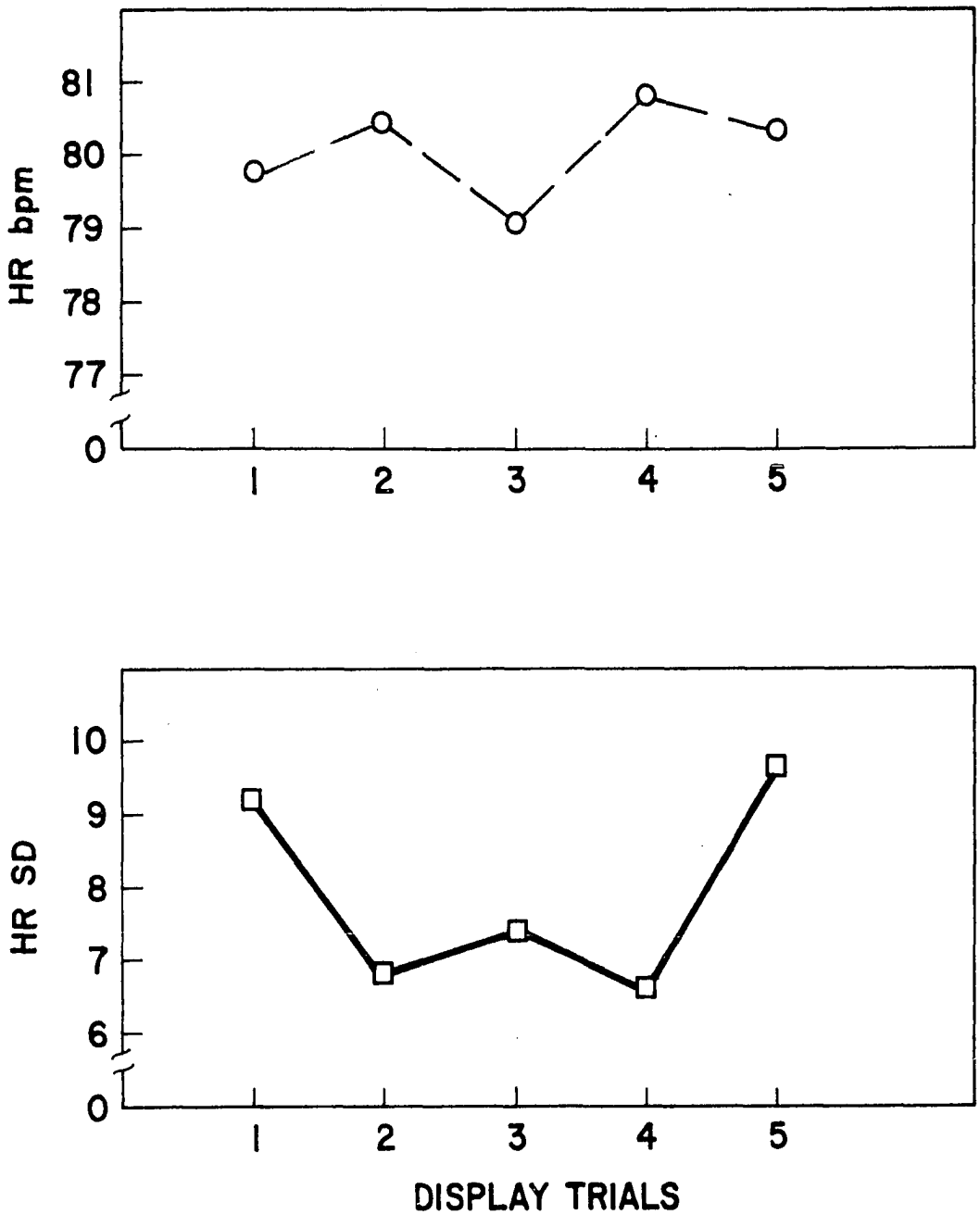
APPENDIXES

APPENDIX A

RATE & VARIABILITY FOR IF SIGNAL



APPENDIX B

INSTRUCTIONS TO SUBJECTS

You have probably guessed by now that we are recording a number of your bodily functions. One of these is being displayed to you on the meter you see in front of you. (During instructions all Ss were allowed to see their own cardiac activity.) This bodily function we are showing you is an extremely sensitive one. It is sensitive to your breathing, your movements, and even your thoughts. The reason you are being shown the meter is that your task today is to learn to control this, your own bodily function. Normally, it is not under your conscious control, but with the aid of the meter you may learn to be able to control it. Specifically, what we want you to learn to do is to keep the pointer, which reflects the amount of activity of your bodily function, (for HRV instruction group) as steady as you possibly can so that the pointer does not wiggle back and forth; (for HR instruction group) as low as you possibly can--a low level of activity is represented on the left side of the face of the meter, to your left; (for HRV plus HR instruction group) at as low and steady a level as you possibly can--low level being represented on the left side of the face of the meter, to your left. Steady means to keep the pointer from wiggling back and forth.

Now the task you have been given is an extremely difficult one, so I do not expect you to be absolutely perfect. Please don't become frustrated because you do not feel you are doing well. Just do the best that you can. That is all I expect from you. (At this point they were asked if they understood the instructions so far and if they had any questions.)

You probably remember that I told you how sensitive this bodily function is to breathing and moving. To be very successful in this task, you must not try to manipulate this bodily function by breathing in unusual ways such as holding your breath, yawning, or things of this nature. Also, you must remain very still in your chair because even the very slightest of movements will hinder your performance. Of course, you may feel a strong urge to yawn or to move around and if you do, go ahead and do so, but remember to minimize these things as much as possible for they will defeat your purpose.

Now in regards to your breathing, I want to give you some instructions as to how you should breathe in this study. Actually, there is really nothing special about this. I just want you to breathe regularly in an out, in an out and so on, just as you probably are doing now anyway. Do you have any questions now about this?

Many Ss sometimes begin to breathe in unusual ways such as holding their breath for a short period of time or gasping, and they don't realize that they are doing this. I realize that it is very easy to slip into unusual modes of breathing in an attempt to control this bodily function, but I don't want you to do this. Watch your breathing very closely so that this does not happen with you. If, how-

ever, you do begin to breathe irregularly I will tell you of this and caution you to breathe more regularly. If this happens, do not feel that you are doing poorly in the task I have given you. This is simply a precaution I must take for all Ss in the study. Do you have any questions?

Since I have told you two do-nots, do not move around unnecessarily and do not breathe in unusual ways, I will now tell you a little more about what you can do. The meter is providing you with continuous information about yourself. All you have to learn to do is use it. This will require a continuous mental effort on your part, so you must remain alert throughout the study. I have already told you how to read the meter, but now I will tell you a little more about how to use the information the meter is giving you. If the pointer (for HRV instruction group) remains very steady so that it does not wiggle back and forth; (for HR instruction group) goes to the low side of the scale to your left; (for HRV plus HR instruction group) goes to the low side of the scale and remains very steady, then the meter is in a sense telling you that whatever you are doing, whatever you are thinking, whatever bodily state you perceive yourself to be in for the moment, that you should continue that thought, thing, or state that you are doing or that you are in, for it is helping you attain the goal I have given you.

On the other hand, if the pointer is (for HRV instruction group) not steady but is wiggling back and forth a great deal; (for HR instruction group) not low but is high; (for HR plus HRV instruction group) not steady and is wiggling back and forth a great deal and also is not low

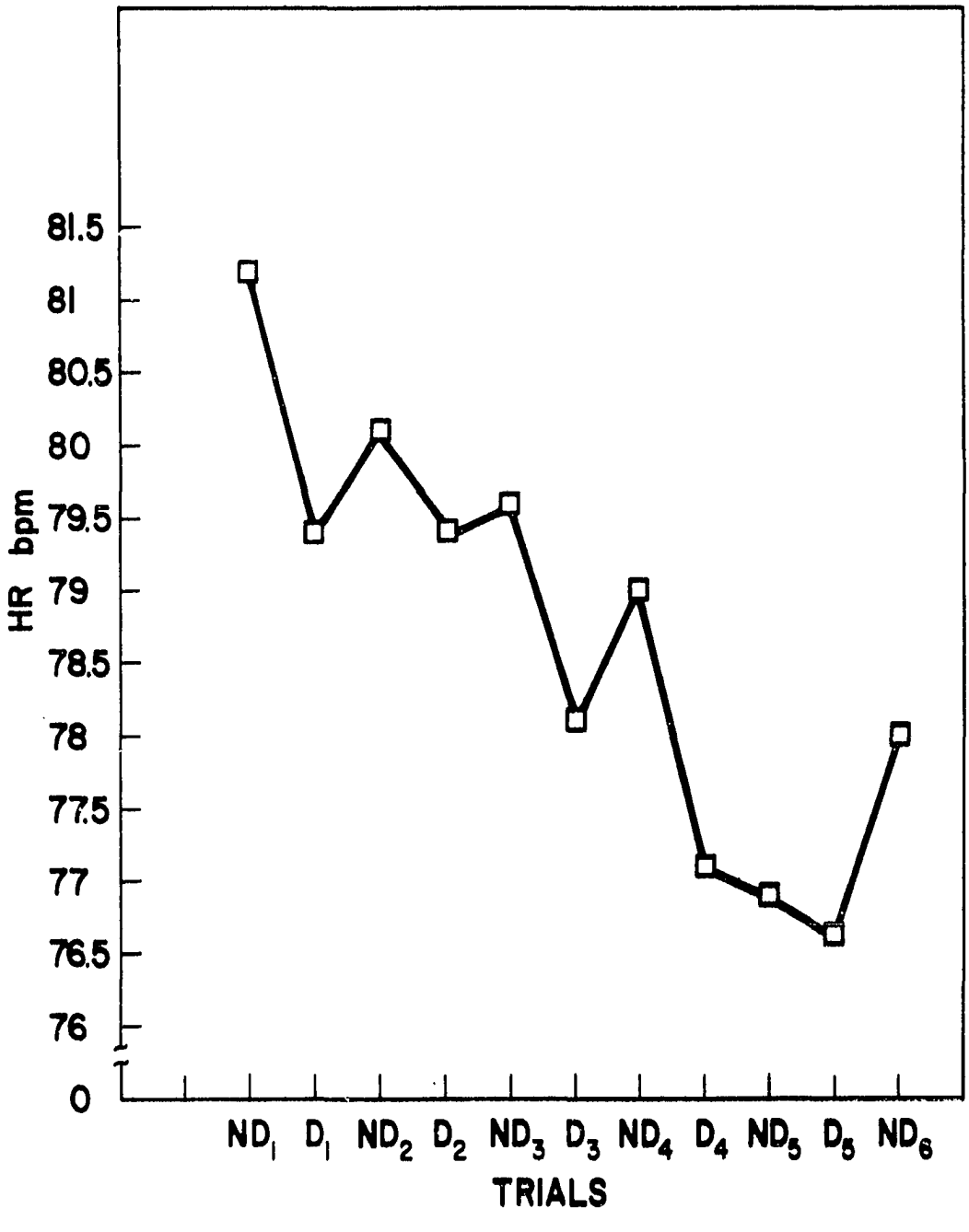
but is high, then the meter is in a sense telling you that whatever you are doing, whatever you are thinking, whatever bodily state you perceive yourself to be in for the moment, that you should drop that thought, thing or state that you are doing or that you are in, for it is not helping you attain the goal I have given you. Do you have any questions?

There are two other things I wish to tell you before we begin the experiment. First, there will be periods during the course of the study when you are "on the meter" as you are now, meaning that you are connected with it so that you may gain information about yourself. There will be other times during the study when you will be "off the meter" in that the meter will go dead, and you will not be able to get any information about yourself by watching it. You will be disconnected from it. Now during these periods where you are off the meter, your task is still the same as it was before, the only difference being that you will not have the meter to assist you. Do you understand the difference between on and off the meter? Do not interpret the off the meter period as a rest period because it is not, so you must still remain alert. I will tell you each time you are to be put on the meter or taken off it in order to bring it to your attention. Are there any more questions?

The second thing I wished to tell you is that from this moment on until the end of the experiment, which will be about one hour from now, there will be no talking between us because as you have probably noticed, talking causes your bodily function to become more active. If there are no more questions, we will begin.

APPENDIX C

HR DECREASES FOR HRV SIC IF GROUP



APPENDIX D

POST-EXPERIMENTAL INTERVIEW QUESTIONS

1. Which alternative would you choose as most accurate concerning your experience in this study: one, you feel that you were able at times to exert some control over the physiological function, or two, you feel that absolutely and at no time did you have any control at all over the physiological function? In other words, you feel you had either some control, or you feel you had no control. Which alternative do you feel is most accurate?

2. Do you have any idea which physiological function was being displayed to you on the meter? Could you identify it?

3. Did you feel frustrated in your attempts to gain control over the physiological function?

4. If you feel you did have some control over the physiological function, could you describe how you were able to do this?

APPENDIX E

Raw Data for Cardiac Rate and Variability

STANDARD DEVIATION SCORES

Subject	ND_1	D_1	ND_2	D_2	ND_3	D_3	ND_4	D_4	ND_5	D_5	ND_6
					Raw Data for HRV SIC CF Group						
J.H.	6.1	3.1	2.5	2.1	3.1	2.8	4.2	3.2	3.4	3.3	5.4
H.K.	4.4	5.4	4.8	4.8	3.6	3.5	5.8	5.6	5.5	2.7	5.2
B.B.	3.5	4.4	3.5	3.3	9.0	0.8	9.5	7.1	5.6	2.1	7.2
D.P.	5.9	2.7	3.2	5.1	6.1	3.7	3.6	5.9	4.1	5.7	5.7
K.C.	1.8	1.8	11.9	3.3	4.2	2.8	13.3	3.4	10.7	4.0	5.5
L.C.	2.4	2.3	3.2	4.3	6.4	7.4	7.0	3.4	6.1	5.8	4.3
D.H.	4.9	4.7	4.2	4.1	3.2	4.3	6.7	3.4	5.3	6.1	6.8
J.L.	3.7	3.4	6.2	3.4	4.4	1.6	2.7	3.4	2.2	4.8	4.2
W.H.	8.8	6.9	9.3	6.8	11.0	5.4	9.9	5.8	10.4	6.8	10.4
D.B.	6.2	4.9	6.9	5.4	4.0	8.0	9.7	5.4	4.4	10.0	10.1
					Raw Data for HRV SIC IF Group						
C.D.	5.6	5.8	4.0	6.5	4.9	6.8	4.1	4.8	4.7	5.2	4.6
S.T.	3.6	3.0	2.9	3.9	3.8	4.2	5.3	4.5	4.3	5.9	3.2
L.F.	5.6	4.6	6.1	6.6	9.0	6.4	4.9	6.0	4.7	4.7	6.8
M.R.	2.5	3.9	2.2	5.7	3.5	4.9	3.0	4.2	2.7	3.7	6.6
L.E.	5.2	4.8	5.7	5.9	6.2	6.9	4.0	4.4	4.0	3.7	3.6
E.F.	4.9	3.7	4.2	3.6	3.6	4.4	5.5	4.6	5.3	3.3	2.8
A.H.	5.2	4.8	4.5	3.5	4.1	3.7	2.4	7.9	3.8	5.7	4.8
E.C.	2.8	1.3	1.7	1.3	1.4	1.2	4.2	1.4	2.0	2.7	1.5
R.S.	2.7	2.8	2.3	3.8	3.2	3.2	2.0	2.3	2.5	2.9	2.1
D.T.	2.0	1.2	2.3	2.8	2.9	2.0	2.5	3.8	3.4	2.2	2.2

HR BPM SCORES

Subject	ND_1	D_1	ND_2	D_2	ND_3	D_3	ND_4	D_4	ND_5	D_5	ND_6
					Raw Data for HR SIC CF Group						
R.W.	62.6	66.8	66.4	69.1	67.8	65.8	67.4	67.2	65.2	65.6	65.3
D.S.	76.1	76.3	68.5	72.2	73.2	74.1	81.1	77.1	77.9	71.0	77.0
M.D.	84.1	82.8	78.4	81.2	69.9	73.2	74.5	67.9	69.3	74.6	68.9
F.G.	61.5	62.8	63.8	59.8	66.8	63.0	64.2	61.4	59.8	58.2	55.8
C.H.	69.9	69.8	70.2	66.6	73.0	66.7	75.7	70.2	67.4	69.0	71.2
W.A.	68.5	69.2	70.5	66.8	68.9	71.6	72.9	68.5	70.1	66.7	66.9
P.F.	67.2	68.8	68.8	67.6	68.4	68.8	68.6	68.5	72.0	68.7	69.7
V.J.	88.4	88.4	91.3	86.3	93.0	85.2	92.5	89.0	89.5	82.1	85.1
G.F.	88.8	81.3	91.8	85.4	91.5	87.2	80.2	78.2	83.3	78.6	75.8
J.M.	74.2	66.3	69.3	66.1	64.8	83.4	79.5	70.4	63.1	61.2	62.0
					Raw Data for HR SIC IF Group						
B.G.	100.0	97.1	96.9	97.8	95.4	93.4	93.2	95.7	91.6	97.0	93.3
N.H.	86.5	85.8	86.2	85.7	85.7	85.5	86.9	82.2	83.1	82.2	84.4
P.V.	85.6	87.0	83.8	81.8	78.3	77.8	74.0	80.4	71.6	71.9	68.6
L.F.	59.1	66.2	62.5	66.9	64.4	67.6	63.4	68.8	60.8	62.2	64.5
J.G.	68.1	67.4	70.3	64.3	63.9	65.8	63.8	68.9	67.6	65.8	64.9
J.N.	71.5	73.2	70.3	71.0	67.8	72.8	73.3	73.9	74.1	76.5	74.3
B.B.	75.2	78.2	76.6	78.5	76.9	72.5	73.2	70.8	67.8	65.8	72.0
H.L.	67.3	63.7	61.8	67.4	63.7	69.2	65.8	63.2	63.1	62.9	57.8
P.L.	71.3	65.6	66.2	65.5	67.0	69.3	67.0	71.2	68.9	74.6	73.6
J.R.	64.0	60.8	56.8	64.2	58.6	60.8	64.6	65.8	61.9	58.8	56.4

STANDARD DEVIATION SCORES

Subject	ND_1	D_1	ND_2	D_2	ND_3	D_3	ND_4	D_4	ND_5	D_5	ND_6
					Raw Data for HRV CIC CF Group						
M.C.	5.1	6.3	7.2	5.0	7.1	3.6	5.2	5.3	5.9	3.6	6.2
L.K.	4.8	4.2	7.3	2.2	7.2	7.9	8.1	6.7	3.3	2.7	9.4
O.P.	6.1	5.0	8.5	4.8	5.4	3.4	11.2	4.8	6.9	8.3	6.9
B.K.	4.8	4.2	5.4	3.2	8.1	5.2	8.2	5.1	7.9	2.1	6.7
P.S.	5.5	3.2	2.7	3.9	3.8	2.9	5.1	3.1	8.4	3.8	4.5
F.M.	6.1	2.6	3.8	2.6	5.5	2.4	4.0	1.9	5.2	0.9	4.0
P.G.	8.2	6.4	5.5	3.8	8.5	4.2	8.3	5.0	5.7	2.9	3.8
J.C.	9.9	12.8	5.3	4.3	4.9	4.7	3.5	3.9	3.5	5.0	4.9
B.C.	8.0	6.0	5.6	6.6	7.3	3.5	9.7	4.0	8.1	2.2	9.4
P.M.	3.2	2.5	2.0	3.7	2.6	2.6	3.3	1.0	3.2	2.4	6.0
					Raw Data for HRV CIC IF Group						
E.S.	8.9	10.4	6.8	3.9	8.2	11.0	3.4	7.1	5.0	4.5	2.5
M.R.	3.6	5.4	6.5	8.4	9.1	12.6	5.4	7.6	7.0	5.7	5.8
S.M.	4.5	4.2	3.1	3.2	4.2	3.5	3.7	5.2	5.8	3.8	2.6
H.P.	4.2	2.7	4.0	4.4	4.6	4.5	4.1	4.2	4.2	3.4	4.9
B.H.	7.2	6.7	4.6	5.0	4.2	4.6	4.5	4.0	3.0	6.4	2.9
D.S.	8.2	3.0	3.1	2.9	3.6	7.4	4.9	4.5	6.2	3.0	4.5
J.H.	4.9	12.3	4.2	6.1	7.3	11.0	6.8	8.9	6.4	13.1	6.2
L.J.	6.7	8.6	7.6	6.5	6.6	5.9	6.4	10.5	8.0	7.6	6.9
K.A.	2.6	3.3	2.6	4.4	5.3	2.7	4.3	2.9	3.6	4.6	4.7
J.N.	4.2	4.2	4.4	6.2	4.6	5.2	5.7	4.8	3.6	5.1	1.8

HR BPM SCORES

Subject	ND_1	D_1	ND_2	D_2	ND_3	D_3	ND_4	D_4	ND_5	D_5	ND_6
					Raw Data for HR CIC CF Group						
M.C.	73.1	72.1	71.0	70.2	70.9	70.2	71.4	68.1	71.6	71.7	75.0
L.K.	82.2	82.4	81.3	74.7	84.3	82.9	86.5	80.9	72.8	72.8	88.2
O.P.	69.8	69.4	78.2	72.7	78.4	73.8	80.9	66.9	72.7	72.9	76.1
D.K.	95.3	78.9	85.1	77.8	88.1	82.4	88.1	75.8	82.2	73.8	81.6
P.S.	60.1	54.4	54.6	55.4	54.3	55.0	54.6	56.8	62.7	59.0	61.7
F.M.	93.4	104.2	102.8	101.2	100.2	96.0	103.7	101.1	102.9	98.8	98.0
P.G.	83.2	82.5	85.6	77.6	78.2	70.6	74.9	78.9	79.2	79.9	77.6
J.C.	93.8	83.5	99.7	94.6	90.2	87.2	86.8	85.3	86.6	81.2	86.8
B.C.	58.3	61.7	65.2	63.7	68.2	61.8	63.8	60.6	63.2	63.7	66.7
P.M.	73.0	71.9	70.2	68.6	69.8	64.5	64.8	69.0	71.7	70.2	69.3
					Raw Data for HR CIC IF Group						
E.S.	60.6	62.2	55.9	52.3	58.1	60.8	55.8	56.2	54.8	53.0	51.1
M.R.	56.6	58.8	57.6	59.1	62.1	64.8	61.6	67.2	61.2	68.3	59.7
S.M.	86.5	87.2	86.3	89.8	85.7	80.2	78.0	80.3	78.4	77.8	79.1
H.P.	79.9	75.8	81.1	77.6	75.3	77.7	78.0	74.7	74.9	77.6	79.4
B.H.	72.3	71.8	68.9	72.8	73.4	71.9	70.2	72.7	68.7	68.0	66.4
D.S.	77.2	74.6	73.8	72.5	67.9	72.2	71.9	71.1	67.5	66.4	69.0
J.H.	85.0	84.8	88.2	88.9	83.0	86.2	80.7	82.2	82.5	75.3	82.0
L.J.	71.3	69.9	72.8	61.8	64.2	60.3	65.0	73.6	68.2	74.3	62.0
K.A.	82.2	80.0	81.6	81.9	84.5	85.3	85.8	87.9	83.8	83.4	81.1
J.N.	93.0	94.8	93.6	94.2	94.4	100.9	98.9	99.3	99.9	101.3	103.2