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Cardiac-Respiratory-Somatic Relationships and Feedback Effects in a Multiple Session Heart Rate Control Experiment

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ABSTRACT

An experiment addressing several unresolved issues in operant conditioning of heart rate (HR) in human subjects was performed. Cardiac-respiratory relationships, cardiac-somatic relationships, effects of biofeedback, and individual differences were examined in terms of their stability across multiple training sessions.

Thirty subjects participated in 3 training sessions. Each session began with 4 trials of attempted HR change without feedback followed by 8 trials with "proportional" feedback of HR. On half the trials HR decrease was attempted while HR increase was attempted on the other half. Subjects were instructed to keep their respiration rate (RR) constant and not to engage in undue movement or muscle activity.

Results indicated that subjects were able to produce significant HR increases and decreases from baseline levels, but these changes were accompanied by parallel changes in respiratory and somatic variables which persisted across sessions. Analysis of data from individual subjects was performed to explore the nature of individual differences in cardiac-respiratory-somatic patterns. The effects of biofeedback were unimpressive, suggesting at best a minor improvement in cardiac control with increased respiratory concomitance. Cardiac control, feedback effects, and cardiac-somatic patterns were stable over sessions. There was evidence of some reduction in cardiac-respiratory parallelism across sessions.

DESCRIPTORS: Operant conditioning, Heart rate, Respiration, Cardiac-somatic relationship, Biofeedback, Individual differences.

In spite of difficulties which have surfaced with research on operant conditioning of cardiac responses in animals (Miller & Dworkin, 1974), research of this nature with human subjects has continued. Human research, although unlikely to answer definitively the question of whether a "pure" cardiac response can be operantly conditioned, has potential for answering a number of questions concerning the relationship between autonomic and central processes. Although a large body of human research exists, many basic questions remain unanswered. In a recent review of operant conditioning of cardiac functions, McCanne and Sandman (1976) identified several areas in need of further research including: the precise nature of the relationship of respiration to operant heart rate (HR), the role of somatic-muscular functions during cardiac conditioning, and the nature of individual differences. Issues such as different patterns of cardiac-respiratory and cardiac-somatic interactions in different stages of cardiac conditioning (Engel, 1972), efficacy of various methods for constraining respiratory and somatic activity, and differences among various operant procedures (e.g., avoidance conditioning, biofeedback) are also legitimate concerns. Research pertinent to several of these issues and to the present investigation will be briefly reviewed.

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Cardiac-Respiratory Relationships

In general, when a cardiac-respiratory relationship has been investigated, subjects have only been instructed not to change their respiratory patterns. From these studies a considerable amount of evi-

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dence exists that HR changes are accompanied by parallel changes in respiration (e.g., Shearn, 1962; Brener & Hothersall, 1966; Brener, Kleinman, & Goesling, 1969; Brener, 1974; Lang & Twentyman, 1976; Levenson, 1976). Cardiac-respiratory data derived from these studies are essentially correlational and as such do not allow determination of the necessity of respiratory maneuvers for producing HR change.

In two studies in my laboratory we have attempted to improve on instructional control of respiration by using more active control through respiration rate (RR) feedback (Levenson, 1976) and pattern feedback of HR and RR (Newlin & Levenson, in press). Although in the latter study we had a small degree of success, taken together the results of these two studies indicate the considerable difficulty in dissociating HR and RR using feedback. Another strategy for controlling respiration has been the use of paced respiration (Brener & Hothersall, 1967; Wells, 1973; Obrist, Galosy, Lawler, Gaebelein, Howard, & Shanks, 1975; Manuck, 1976). Although potentially capable of going beyond the correlational approach in terms of specifying the nature of cardiac-respiratory relationships, studies using active control of respiration are plagued by at least two problems. First, subjects may alter a given respiratory parameter despite feedback or pacing (Wells, 1973; Levenson, 1976); and second, subjects may compensate for pacing of one parameter (e.g., rate) by altering a second parameter such as depth (Brener, 1974). Only when subjects are passively respirated can the importance of respiratory changes for operant conditioning of HR be specified. In the sole instance where human subjects were passively respirated, previously learned cardiac control was greatly attenuated (Vandercar, Feldstein, & Solomon, 1977).

In the present study the use of instructional control of respiration has been adopted. This decision reflects converging evidence from the research cited that respiratory change is a natural concomitant of HR control. Further attempts to refute this conclusion would be best accomplished using passive respiration (Vandercar et al., 1977), a procedure which is not within the capabilities of our laboratory. Thus, the cardiac-respiratory questions of concern in the present study are those of concomitance rather than causation, with an emphasis on issues of stability and individual differences which have not been addressed systematically in previous research.

Cardiac-Somatic Relationships

Despite basic relationships between the cardiac and somatic-muscular systems, somatic variables have rarely been investigated during operant conditioning of HR in humans. Results of studies monitoring these variables have been mixed. Cohen (1973) was able to demonstrate conditioning of HR without chin EMG changes, but Brener (1974), Obrist et al. (1975), Manuck (1976), and Vandercar et al. (1977) found muscular concomitants of HR change.

Other Issues

While most human research has used conditioning via positive reinforcement (i.e., biofeedback), several investigators have used avoidance procedures (Shearn, 1962; Cohen, 1973; Obrist et al., 1975). Certain characteristics of avoidance studies (e.g., large HR increases on early trials, difficulties obtaining HR decreases) suggest that the two procedures are substantially different and may by implication reveal different aspects of the cardiac-respiratory-somatic interaction.

The present study is an attempt to examine cardiac, respiratory, and somatic variables during a cardiac conditioning procedure in which biofeedback is used as positive reinforcement, respiration is under instructional control, and a multiple session design is utilized. Additions of a measure of general somatic activity, of multiple sessions, and analysis of individual differences are viewed as extensions of the methodology used in a previous study (Levenson, 1976). As noted earlier, the nature of somatic activity during cardiac control has not been well documented in the literature. Multiple session designs have been used fairly frequently (e.g., Headrick, Feather, & Wells, 1971; Wells, 1973), but their results have not been reported in terms of the stability of cardiac-respiratory-somatic relationships over sessions.

Method

Subjects

Seventeen female and 13 male undergraduate students were recruited from the introductory psychology classes at Indiana University. Their participation in the experiment fulfilled a course requirement. In addition they were given a $3.00 bonus for completing the 3 experimental sessions.

Apparatus

Physiological Data. Heart rate data were recorded bipolarly, using Beckman surface electrodes attached to the chest, and were amplified using a Grass Model 7 polygraph. Respiration rate (RR) data were obtained using a mercury-filled strain gage stretched across the subject's chest. A Parks model 270 Plethysmograph, operating as a strain gage transducer, provided the analog respiratory signal to the polygraph. General motor activity (GA) was recorded using a locally designed system.[1]

[1]The author wishes to thank John Waltke for his help in designing this system.

An electromagnetic sensor located under the subject's chair produced an analog signal which was filtered to remove exceedingly slow activity (such as respiration effects) and then integrated at a fixed rate using a Grass Polygraph Integrator. After a fixed amount of activity the integrator saturated and reset. Counting the number of resets and the remaining signal following the last reset over a fixed time interval yielded a measure of GA which was directly comparable between subjects and between sessions.

Computer Functions. A PDP-11 digital computer with an AR-11 analog interface was used to process and quantify all physiological data on-line. In addition, the computer ran the experiment and controlled the HR feedback display.

Feedback and Task Information. A LED digital display device was used. The leftmost digit was used to signal the subject as to whether to attempt HR increase, attempt HR decrease, or "rest" (while a pre-trial baseline was being calculated). The rightmost digit was used to present the HR feedback. A digit was illuminated after each interbeat interval (IBI). The digit "5" was equated with the baseline mean IBI plus or minus 30 msec. Successive 60-msec bands were established for digits below and above "5" such that HR increases (shorter IBIs) were associated with higher digits and HR decreases (longer IBIs) were associated with lower digits. Digits "1" to "9" were used, thus covering an IBI range of 540 msec around the baseline mean.

Procedure

Three experimental sessions were scheduled for each subject at an interval of 2-3 days between sessions. The procedure for each session was identical. Following attachment of electrodes, subjects were instructed as to the nature of the experiment and operation of the feedback display. They were explicitly informed in each session that their task was to attempt to change their HR in the instructed direction while keeping their RR constant and without engaging in undue movement or muscle activity. After the first session the explanation of the operation of the feedback display was dropped if the subject indicated it was unnecessary. The other instructions were always repeated.

Each session was structured into 12 trials. A trial consisted of a 50 heart beat baseline, followed by a signal to either increase or decrease HR, and then 120 beats of attempted HR control followed by a 1-min rest period. During trials 1-4 no feedback was given. During trials 5-12 HR feedback was given. Each block of 4 trials consisted of 2 trials of attempted HR increase and 2 trials of attempted HR decrease; however, counterbalanced orderings of the increase-decrease orders within each block of 4 trials were used so that the subject would not be aware of any pattern of increase and decrease trials.

Results[2]

Data for the following dependent measures were obtained: 1) IBI, 2) ICI, 3) Correct IBIs—number of HR interbeat intervals which met criterion (i.e., an IBI change in the instructed direction of at least 30 msec from the baseline mean IBI), 4) Correct ICIs—number of RR intercycle intervals which met criterion (i.e., an ICI within 700 msec[3] from the baseline mean ICI), 5) Correct IBI-ICIs—number of IBIs which met the HR criterion and occurred during respiration cycles which met the ICI criterion, and 6) GA—number of integrator resets per minute.

The primary analysis of the dependent measures was by analysis of variance (ANOVA). IBI, ICI, and GA data were submitted to a $3 \times 3 \times 2 \times 2 \times 2$ (session $\times$ trial block[4] $\times$ trial pair $\times$ direction of HR change $\times$ baseline or HR control) repeated measures ANOVA. Correct IBI, Correct ICI, and Correct IBI-ICI data were submitted to a similar ANOVA with the "baseline or HR control" factor omitted. To test specific hypotheses concerning physiological changes from baseline *a priori* t-tests were used. Selection of the appropriate error term and degrees of freedom for these comparisons followed Winer's (1971) procedure for completely within-subject effects.

To enable assessment of individual differences in response patterns, correlations were computed between IBI and ICI and between IBI and GA using trial means during attempted HR control. These correlations were computed both on a session by session basis and for all 3 sessions combined.

The presentation of results will be organized in terms of: 1) cardiac-respiratory-somatic relationships, and 2) feedback effects.

Cardiac-Respiratory-Somatic Relationships

HR Control. Subjects were able to produce significant changes in IBI from pre-trial baseline values (Table 1). This was indicated by a significant interaction of direction of HR control $\times$ baseline or HR control, $F(1/29) = 32.53$. These changes were bidirectional with significant HR increases, $t(29) = 4.28$, and HR decreases, $t(29) = 3.78$, from pre-trial baselines[5]. Subjects evidenced no preference

[2]The .05 level is used as the rejection level unless otherwise noted.

[3]The value of 700 msec was used in an earlier study in which some subjects received feedback of ICI (Levenson, 1976). The purpose of the correct IBI-ICI variable is to provide a convenient summary variable of the extent of concomitance between HR and RR. More detailed analysis can be done using the raw IBI and ICI data, but for examination of feedback effects and correlational work, a single summary variable such as correct IBI-ICI has proven useful.

[4]A trial block consisted of 2 pairs of HR increase and decrease trials. The first trial block corresponded to the "no feedback" portion of each session.

[5]Since many studies have used a pre-training baseline rather than a pre-trial baseline, these analyses were repeated using the

TABLE 1

Mean IBI, ICI, and GA during HR decrease and increase trials

Measures	Means			
	Baseline	HR Decrease	Baseline	HR Increase
IBI (msec)	825	852*	823	792*
ICI (msec)	4012	4486*	4008	3549*
GA (resets/min)	1.88	1.33*	1.88	1.90

*Significantly different from baseline.

TABLE 2

Correct IBIs and correct IBI-ICIs during sessions 1, 2, and 3

Measures	Means		
	Session 1	Session 2	Session 3
Correct IBIs	56.2	56.7	57.0
Correct IBI-ICIs	23.3*	26.0	28.5*

*Significantly different.

for either HR increase or HR decrease as indicated by a nonsignificant main effect for direction of HR control in the Correct IBI measure (Table 2), $F(1/29) < 1$.

There was no improvement in ability to control HR across the 3 sessions. This was indicated by a nonsignificant interaction of session × direction of HR control × baseline or HR control for IBI data, $F(2/58) = 2.35$, and a nonsignificant sessions effect for Correct IBIs, $F(2/58) < 1$.

A correlational analysis of the relationship between baseline variability of IBI and correct IBIs produced a low ($r = .09$) nonsignificant correlation. Thus, ability to control HR was not greater in subjects who had higher resting HR variability.

HR-RR Interaction. Changes in RR paralleled changes in HR throughout the experiment. This was indicated by a significant interaction of direction of HR control × baseline or HR control for ICI data, $F(1/29) = 48.64$. Analysis of changes in RR from pre-trial baseline values (Table 1) revealed significant RR increase during attempted HR increase, $t(29) = 4.85$, and significant RR decrease during attempted HR decrease, $t(29) = 5.01$.

Analyzing the HR-RR interaction across sessions revealed no changes in the magnitude of ICI change across sessions. This was indicated by a nonsignificant interaction of session × direction of HR control × baseline or HR control for ICI data, $F(2/58) = 2.75$. There was, however, some evidence of improvement in RR control in the analysis of Correct IBI-ICI data, indicated by a significant session effect, $F(2/58) = 4.44$. Analysis of session means (Table 2) revealed that the number of Correct IBI-ICIs in session 3 was greater than in session 1, $t(58) = 2.98$. Changes in the number of Correct IBI-ICIs between sessions 1 and 2, $t(58) = 1.55$, and between sessions 2 and 3, $t(58) = 1.43$, were not significant.

Correlational analysis revealed significant correlations between IBI and ICI for 17 of 30 subjects over all sessions, range[6] of $r(36) = .28$–$.80$. Results of session by session analysis indicated significant IBI-ICI correlations for 16 subjects in session 1, range of $r(12) = .52$–$.89$; for 15 subjects in session 2, range of $r(12) = .55-.91$; and for 14 subjects in session 3, range of $r(12) = .52-.90$.

Reasoning that a significant *negative* IBI-ICI correlation would be evidence of cardiac-respiratory dissociation, the data from individual subjects were examined in search of such correlations. In the third session, one subject did have a negative IBI-ICI correlation; however this subject also had below average HR control and a significant correlation between HR and GA indicating parallel somatic activity during attempted HR control.

HR-GA Interaction. Changes in GA generally paralleled changes in HR in all sessions. This was indicated by a significant interaction of direction of HR control × baseline or HR control for GA data, $F(1/29) = 12.31$. Analysis of changes in GA from pre-trial baselines (Table 1) revealed nonsignificant increases in GA from pre-trial baselines during attempted HR increase, $t(29) = .17$, and significant decreases in GA during attempted HR decrease, $t(29) = 4.79$. A nonsignificant interaction of session × direction of HR control × baseline or HR control, $F(2/58) = .15$, indicated no differences in the magnitude of GA change across sessions.

Correlational analysis revealed significant negative correlations between IBI and GA (i.e., greater activity during HR increase) for 24 of 30 subjects over all sessions, range of $r(36) = -.28$ to $-.89$. Session by session analysis indicated significant correlations for 17 subjects in session 1, range of $r(12) = -.58$ to $-.96$; for 15 subjects in session 2, range of $r(12) = -.54$ to $-.96$; and for 19 subjects in session 3, range of $r(12) = -.50$ to $-.94$.

Reasoning that a significant *positive* IBI-GA correlation would suggest cardiac-somatic dissociation, data from individual subjects were examined

pre-training baseline. Using this alternative baseline, all reported changes would still be significant.

[6]All reported ranges are those of significant r statistics, one-tailed.

in search of such a correlation. Three positive IBI-GA correlations were found. In two of these cases, there was a significant correlation between IBI and ICI (indicating parallel respiratory activity). The third case was a subject who in session 2 exhibited average HR control and did not have a significant IBI-ICI correlation. If the stated rationale for examining these correlational data is accepted, this latter subject's performance in session 2 could be viewed as an example of cardiac-respiratory-somatic dissociation, although other explanations (e.g. chance occurrence at .05 rejection level) are certainly viable.

Sex Differences. Male and female subjects showed no differences in the ability to control HR, nor were there sex differences in patterns of cardiac-respiratory-somatic relationships. These findings are similar to those reported in an earlier study (Levenson, 1976).

Feedback Effects

HR Control. Results concerning the effects of feedback on ability to control HR were complicated by differences between the analyses of IBI and Correct IBI data. The analysis of IBI data suggested no effects of feedback on the magnitude of IBI change from baseline (nonsignificant interaction of trial block × direction of HR control × baseline or HR control, $F(2/58) = 2.22$). The analysis of Correct IBI data (Table 3), however, indicated a larger number of Correct IBIs on feedback trials compared to no feedback trials (significant main effect for trial block, $F(2/58)=5.01$, and a significant difference between feedback trials and no feedback trials, $t\ (58)=2.66$).

To attempt to resolve these differences an additional ANOVA was performed on Correct IBI data in which trials were examined pairwise rather than in blocks of 4 trials. The resultant analysis was a 3 × 6 × 2 (session × trial pair × direction of HR control) ANOVA. The main effect for trial pair was significant, $F(5/145) = 2.76$. *A posteriori* comparisons among trial pair means (Table 4) were performed using Duncan's Multiple Range test revealing significantly more correct IBIs on trial pairs 5–6, 7–8, and 9–10 in comparison with the *first 2* no feedback trials, but not the second 2 no feedback trials. A proposed resolution of these results will be presented in the discussion section of this paper.

An analysis of the stability of feedback effects revealed no changes across sessions in IBI data (session × trial block × direction of HR control × baseline or HR control, $F(4/116)<1$), or in the Correct IBI data (session × trial block, $F(4/116)<1$).

RR Control. Respiratory concomitance during HR control increased on trials where feedback was given. This finding was indicated in both the ICI and Correct ICI data. For ICI there was a significant interaction of trial block × direction of HR control × baseline or HR control, $F(2/58)=5.39$, with larger ICI changes from baseline occurring during feedback trials. For Correct ICIs there was a significant effect for trial block, $F(2/58)=11.14$, with fewer Correct ICIs on feedback trials as compared to no feedback trials, $t(58)=4.08$ (Table 3). Expressed as a percentage of total ICIs per trial, subjects had 63% Correct ICIs on no feedback trials and 53% Correct ICIs on feedback trials.

HR-RR Control. Heart rate feedback had no effect on the number of Correct IBI-ICIs (Table 3). This was indicated by a nonsignificant effect for trial block, $F(2/58)=1.16$. Regarding this lack of change in the number of Correct IBI-ICIs, it should be noted that this measure is affected by both the number of Correct IBIs and the number of Correct ICIs. Thus, in the present study, the numerical increase in the number of Correct IBIs on feedback trials and the

TABLE 3

Correct IBIs, correct ICIs, and correct IBI-ICIs on no feedback and HR feedback trials

Measures	Means	
	No Feedback	HR Feedback
Correct IBIs	52.7	58.6*
Correct ICIs	15.3	12.9*
Correct IBI-ICIs	27.3	25.3

*Significantly different from no feedback trials.

TABLE 4

Correct IBIs for trial pairs

Measure	Means					
	No Feedback Trials 1–2	No Feedback Trials 3–4	Feedback Trials 5–6	Feedback Trials 7–8	Feedback Trials 9–10	Feedback Trials 11–12
Correct IBIs	51.2	54.1	59.1*	59.8*	59.1*	56.6

*Significantly greater than no feedback trials 1–2.

significant decrease in the number of Correct ICIs on feedback trials combined to yield no change in the number of Correct IBI-ICIs.

GA Control. Heart rate feedback had no effect on the amount of general activity evidenced by subjects. This was indicated by a nonsignificant interaction of trial block × direction of HR control × baseline or HR control for GA data, $F(2/58)<1$.

Discussion

The results of this experiment can most readily be discussed in terms of three aspects of operant conditioning of heart rate: 1) cardiac-respiratory-somatic relationships, 2) feedback effects, and 3) multiple session effects.

Cardiac-Respiratory-Somatic Relationships

Patterns of HR and RR activity in the present study were essentially equivalent to those found in an earlier single session study (Levenson, 1976). Subjects in both studies were able to produce significant HR increases and decreases which were accompanied by parallel RR changes. Correlational analysis in the present study extended this finding by allowing individual differences to be determined. Considering that these studies involved different subject populations, different laboratories, and different experimental procedures, it seems safe to say that these patterns of cardiac and respiratory activity are reliable. Since similar results have been reported by other investigators and since mechanisms for HR-RR parallelism are biologically represented in the sinus arrhythmia and elsewhere, these results are not surprising. More unique to these two studies is the ability of subjects to consistently produce HR decreases. In the case of avoidance conditioning, difficulties obtaining HR decreases may be related to the state of arousal associated with the onset of the warning signal—a state antagonistic to HR decrease. For other biofeedback studies, procedural differences such as the length of time allowed for subjects to adapt to the laboratory, and statistical differences such as use of pre-training vs pre-trial baselines may affect the "baseline" value against which HR decreases are evaluated. Beyond these simple procedural and statistical explanations are more complex explanations involving differences in training, feedback, and motivation which go beyond the scope of the present investigation.

A strong cardiac-somatic relationship was revealed in the analysis of GA data in the present study. Parallels between HR and GA data were reflected in significant correlations between these variables for a large proportion of subjects. Changes in GA from baseline were only significant, however, during HR decrease. It is possible that failure to find bidirectional GA change from baseline was due to a "baseline" artifact of the kind described earlier. Subjects may have used the baseline period to engage in random motor activity (e.g., looking around the room, stretching) which would have elevated baseline GA, making increases from that level less likely. A worthwhile procedural modification might be to have subjects' movement restrained during the actual baseline measurement period to determine its effect on baseline values of both GA and HR and on the subsequent bidirectionality of changes in these variables during HR control.

A pattern of strong cardiac-respiratory-somatic relationships emerges from the 1976 and present studies, with RR and GA paralleling HR change. Additional indication of a cardiac-respiratory relationship can be seen in the increases in respiratory depth during HR increase found in the 1976 study as well as by several other investigators (Wells, 1973; Cohen, 1973). The weight of the evidence clearly leads to the expectation of a relationship in minimally restrained subjects along the lines suggested by Obrist, Webb, Sutterer, and Howard (1970). There is also evidence that the parameters of this relationship are not invariant, ranging from the individual differences in HR-RR-GA patterns demonstrated in the present study, to the demonstration of some cardiac specificity during HR increase in acutely stressful situations (Obrist, Lawler, Howard, Smithson, Martin, & Manning, 1974).

Feedback Effects

The present study does not make a strong case for the necessity or efficacy of biofeedback for producing HR change. An improvement in one measure of HR change (i.e., correct IBIs) as a function of feedback must be weighed against no improvement in the magnitude of IBI change. Further, closer examination of correct IBI data revealed improvement with feedback only in relationship to the first 2 no feedback trials of the experimental session. Although the present study's within-subject design confounds practice effects with feedback effects, an earlier study (Levenson, 1976) without this confound revealed no improvement in HR control attributable to feedback. Further, since the earlier study included a period of practice with the feedback display preceding the experiment proper, it may be that the first 2 trials served a similar purpose in the present experiment. Thus, the lack of significant improvement with feedback compared to the last 2 no feedback trials may be a more relevant comparison. Although it is apparent that subjects can produce significant HR increases and decreases without an assist from biofeedback, it is unlikely that feedback is totally irrelevant to HR control. Its role in maintaining motivation (especially over mul-

tiple sessions) and in producing more complex patterns of physiological activity is yet to be fully determined.

Multiple Session Effects

There were no improvements in cardiac control, no changes in the magnitude of HR-GA concomitance, and no differences in feedback effects found across sessions. There was some indication of alteration in the cardiac-respiratory relationship. An increase in correct IBI-ICIs between sessions 1 and 3 and a decrease in the number of subjects having significant HR-RR correlations across sessions suggested a diminishing of cardiac-respiratory parallelism. However, the fact that the magnitude of ICI change from baseline was not altered across sessions makes any strong conclusion as to changes in the cardiac-respiratory relationship tenuous. Aside from this finding, there were no effects attributable to multiple sessions. Improved cardiac control and large magnitude HR change would seem to be the most likely benefits of extended training. Yet, as I indicated in an earlier paper (Levenson, 1976), these seem to be more attributable to individual differences than to multiple sessions, with some subjects manifesting excellent HR control with large magnitude changes early in training. At this juncture the evidence would seem to support the conclusion that the prominent aspects of cardiac control, cardiac-somatic-respiratory interactions, and feedback effects are well established in the initial session for most subjects.

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