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Feedback Effects and Respiratory Involvement in Voluntary Control of Heart Rate

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ABSTRACT

One within-subject single session experiment involving 30 subjects and one between-subject single session experiment involving 42 subjects were performed to evaluate the effects of augmented sensory feedback and to specify the nature of cardiac-respiratory relationships during attempted bidirectional control of heart rate (HR). Subjects were instructed to attempt to control HR while maintaining a constant respiration rate (RR), and were provided with either no feedback, HR feedback, or HR and RR feedback of the "digital" proportional type. In addition to measures of heart and respiratory period, a measure of respiratory volume was included in the second experiment. Results of both experiments indicate that (1) subjects can significantly increase and decrease HR; (2) feedback does not affect the magnitude or consistency of HR control; (3) significant parallel changes in RR accompany changes in HR regardless of the amount of feedback provided; (4) significant increases in respiratory volume accompany HR increases; and (5) large magnitude HR changes are produced by some subjects within the single session. Implications of these findings for the concomitance model of autonomic and central nervous system interactions; for single vs multiple training sessions; for the use of paced respiration; and for a learning model of HR control are discussed.

DESCRIPTORS: Heart rate, Respiration rate, Respiration depth, Augmented sensory feedback, Voluntary control.

The ability of human subjects to produce reliable changes in heart rate (HR) has been well documented over the past decade. Almost all of this research has been carried out using normal populations in laboratory settings and using some form of augmented sensory feedback. The robust nature of the phenomenon is attested to by its replication by a host of investigators using a range of methodologies (e.g., different feedback modalities, different lengths of training, subjects aware and unaware of the response which was being "reinforced"). Nonetheless, in spite of this extensive replication many fundamental questions remain, including the clinical significance and utility of the obtained changes; the degree of central nervous system (CNS) involvement; the precise role of feedback; and the extent to which HR control is "learned." The two experiments to be presented here attempt to address several of these questions within the framework of one of the most common methodologies, namely, the single session, multi-trial paradigm in which subjects attempt to produce directional HR change. Special emphasis is given to the importance of feedback and the extent to which HR changes are accompanied by respiratory changes.

Feedback

With a handful of exceptions (Brener, Kleinman, & Goesling, 1969; Bergman & Johnson, 1972; Blanchard, Young, & McLeod, 1972) investigators have not explicitly compared subjects receiving feedback with subjects receiving no feedback. This comparison seems especially important in light of Bergman and Johnson's (1971) finding that subjects could increase and decrease HR without feedback. Brener et al. (1969) found that subjects who received feedback evidenced greater HR control; Bergman and Johnson (1972) found no differences between their feedback and no feedback groups; and Blanchard et al. (1972) found superiority for feedback in HR increase but no differences for HR decrease. Thus on the basis of these studies no definitive conclusion can be drawn as to the role of feedback in HR control.

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Respiration

The question of the role of respiration in HR control is a subset of the general issues of mediation and of concomitance of CNS and autonomic nervous system (ANS) changes. The mediation issue has been raised by many authors and is well summarized by Katkin and Murray (1968). A range of procedures has been used to determine the extent of respiratory involvement (reviewed by Blanchard & Young, 1973). In studies of directional HR control which have actually monitored respiration these procedures have fallen into two categories:

1) Statistical control—subjects were instructed not to use respiratory changes to effect HR changes. Respiration (usually rate) was monitored and analyzed post facto.
2) Pacing—subjects were instructed to breathe in cadence with a mechanical device (a tone, light, or simulation of breathing sounds).[1]

Although the bulk of the evidence derived from investigations which have looked at respiratory involvement (especially those using pacing) seems to support the notion of HR control free from respiratory involvement, the question remains open in view of some findings of respiratory change (e.g., Brener et al., 1969) and the failure of most investigators to include an adequate measure of respiratory volume (V) in addition to respiration rate (RR).

EXPERIMENT I

Method

Subjects

Twenty male and 10 female undergraduate students in the introductory psychology classes at Vanderbilt University were recruited in fulfillment of a course requirement.

Apparatus

(1) Physiological Input. HR data were recorded bipolarly using Beckman surface electrodes and a Grass Model 7 polygraph. The polygraph output was connected to an analog conversion channel of a Hewlett Packard 2114A digital computer.

RR data were obtained using series-connected mercury strain gages stretched across the subject's chest and stomach. A Park's model 270 Plethysmograph, operating as a strain gage transducer, provided the analog signal to the polygraph which then was routed to a second analog input on the computer.

(2) Computer Functions. The computer was used on-line to calculate the dependent measures used in the experiment and to control the HR and RR feedback display.

(3) Feedback. Feedback was presented via a readout device containing two chambers. HR feedback was presented in the right hand chamber and RR feedback was presented in the left. The feedback took the form of illuminated digits which were updated following each IBI or ICI. Feedback criteria were contingent on mean HR and RR determined during the baseline period which preceded each control trial. For HR, the baseline mean IBI plus or minus 30 msec was equated with the digit "4" in the right chamber. Successive 60 msec intervals were established for digits below and above "4" such that HR increases (shorter IBIs) were associated with higher digits. Digits "1" to "7" were used, thus covering an IBI range of 420 msec around the baseline mean. A similar scheme was used for RR feedback with the baseline RR plus or minus 350 msec being equated with the digit "4" in the left chamber and successive 700 msec bands established for the other digits. RR increases (shorter ICIs) were associated with higher digits.

Procedure

Following attachment of electrodes subjects were instructed as to the nature of the experiment. They were informed that they were to attempt to either increase or decrease their HR without changing their RR and without engaging in unusual movement or muscular activity. To familiarize subjects with the apparatus and to assure them that their cardiac and respiratory activities were being monitored, they were then given 10 min of practice with the feedback display which provided both HR and RR feedback. The experiment itself consisted of 12 baseline periods (50 heart beats) and 12 periods (75 heart beats) in which HR control was attempted. The 12 periods of HR control were divided along factors of "direction" (i.e., 6 periods of attempted HR increase and 6 periods of attempted HR decrease) and "feedback level" (i.e., 4 periods of no feedback, 4 periods of HR feedback only, and 4 periods of HR and RR feedback). Orders of "direction" were counterbalanced as were orders of "feedback level" with the exception that all subjects started with no feedback for their first 4 periods. Subjects were signalled on the feedback display before each HR control period as to which direction of HR change they were to attempt and were signalled at the end of each HR control period so that they could rest while the data were printed out and a new baseline was being computed.

Data on the following dependent measures were obtained: HR; RR; 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); Correct ICIs—number of RR intercycle intervals which met criterion (i.e., an ICI within 350 msec from the baseline mean ICI); and Correct IBI-ICIs—number of IBIs which met the HR criterion *and* occurred during respiration cycles which met the ICI criterion.

Results[2]

Analysis of the dependent measures was by analysis of variance (ANOVA). IBI and ICI data were submitted to a $3\times2\times2\times2$ (feedback level $\times$ repetition $\times$ direction of HR change $\times$ baseline or HR control) ANOVA while correct IBI and correct IBI-ICI data were submited to a $3\times2\times2$ ANOVA with the "baseline or HR control" factor omitted.

[1]The technique is not ideal insofar as it imposes an unnatural regularity on subjects' breathing patterns. Further, pacing does not guarantee subjects will not alter their breathing. Wells (1973) reports significant changes in RR in spite of pacing.

[2]The .05 level is used as the rejection level. All reported values of F were obtained from analysis of variance while values of t were obtained using the technique of "a priori orthogonal tests using t ratio" (Kirk, 1968).

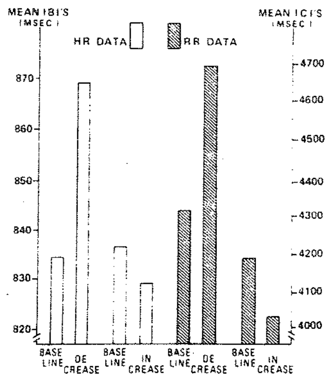

Fig. 1. Mean heart rate interbeat intervals and respiratory intercycle intervals during heart rate decrease and increase trials (Experiment I).

HR Control

In Fig. 1 IBI data for baseline periods and HR control periods are displayed. A significant interaction of direction of HR change × baseline or HR control was obtained ($F(1/29)=47.23$). Planned comparisons by t-test revealed significant HR increases from baseline ($t(29)=1.82$) and significant HR decreases from baseline ($t(29)=7.93$) indicating subjects were able to produce reliable changes in HR from resting levels. Feedback level (i.e., no feedback, HR feedback, or HR and RR feedback) did not affect the magnitude of HR change ($F(2/58)<1$) nor did it affect the number of correct IBIs ($F(2/58)=1.21$, see Table 1).

RR Changes

In Fig. 1 ICI data for baseline periods and HR control periods are also displayed. A significant interaction of direction of HR change × baseline or HR control was found ($F(1/29)=13.80$). Planned comparisons by t-test revealed that RR increases from baseline during periods of attempted HR increase were not significant ($t(29)=1.56$), while RR decreases from baseline during periods of attempted HR decrease were significant ($t(29)=3.69$). The nature of the relationship between HR and RR is further illustrated by comparing the correct IBI and correct IBI-ICI data presented in Table 1. Overall subjects were able to produce 34.8 heart beats per trial (75 beats) which met criterion, but only 7.6 of these occurred during respiratory cycles which met criterion. This comparison considered in combination with the ICI data suggests that very little cardiac control was obtained without accompanying respiratory changes.

Feedback level did not affect the magnitude of RR change ($F(2/58)<1$) but it did significantly affect the number of correct IBI-ICIs ($F(2/58)=3.15$).

TABLE 1

Correct IBIs and correct IBI-ICIs for HR control trials (75 beats) (Experiment I)

Trials	Correct IBIs	Correct IBI-ICIs
Overall	34.8	7.6
No Feedback Trials	34.1	7.8
HR Feedback Trials	36.7	6.2*
HR and RR feedback Trials	33.7	8.8

*Significantly fewer than no feedback and HR and RR feedback trials.

This latter effect was further analyzed using t-tests which revealed that the no feedback and HR and RR feedback conditions did not differ in terms of the number of correct IBI-ICIs ($t(58)=.94$) but the HR feedback condition was productive of significantly fewer correct IBI-ICIs ($t(58)=2.02$).

Sex Differences

A separate analysis of variance was determined for each dependent measure comparing the male and female subjects who participated in the study. No sex differences were found in any of the main effects or interactions in which sex was a factor.[3]

Discussion

The data obtained from Experiment I confirmed the already well documented fact that human subjects can alter their HR when provided with biofeedback. Somewhat more surprising were the findings that 1) subjects produced equal amounts of HR change regardless of amount of feedback they received, and 2) changes in RR seemed to parallel changes in HR. Both of these findings will be discussed in more detail following the description of Experiment II which was designed to both replicate and extend the findings of Experiment I.

EXPERIMENT II

The design utilized in Experiment II attempted to correct several methodological problems which existed with the first study. First, the three feedback conditions were converted to between-subjects factors removing a possible confound in Experiment I where subjects' experience with one feedback level might have affected performance with other feedback levels. Second, the total length of time in which subjects were attempting to control

[3]Young and Blanchard (1972) have reported finding male subjects to be better at HR increase than females. In the present study, male subjects were slightly better at HR increase but the difference was not significant.

their HR was extended from the relatively brief 9 min utilized in Experiment I to a full 28 min. Third, an appropriate measure of respiratory depth (RD) was added to the measure of RR to allow a more comprehensive analysis of respiratory behavior during HR control.[4] Finally, in hopes of increasing their level of motivation, it was decided to pay subjects for participating in the experiment and to provide them with additional payment based on how well they performed (Engel & Hansen (1966) have used a similar procedure).

Method

Subjects

Forty-two undergraduate students (24 males, 18 females) attending the summer session at Vanderbilt University were recruited. Subjects were advised that they would be paid $4.00 for participating and would have an opportunity to earn additional remuneration based on their performance.

Apparatus

Apparatus were identical to that of Experiment I with the addition of a calibrated pneumotachograph used to obtain a measure of tidal air flow. The electrical output of the pneumotachograph was integrated using a Grass 7P10 integrator to provide a measure of tidal volume. As all subjects breathed through a mouthpiece device with clamped nostrils, an accurate measure of RD in liters of air was obtained on every breath via the computer and this measure was directly comparable between subjects. The electrical signal from the pneumotachograph also provided a convenient indicator for the determination of the ICI since the polarity of the signal changed at the start of each inspiration and expiration.

Procedure

Following attachment of electrodes, subjects were instructed to begin breathing through the pneumotachograph mouthpiece. A 20-min adaptation period ensued during which time the subject alternately breathed through the mouthpiece and breathed normally for 5-min sub-periods.

After the adaptation period subjects were given written instructions appropriate to their assigned feedback conditions. In all conditions they were informed that they would be attempting to either increase or decrease their HR without changing their RR and without engaging in unusual movement or muscular activity. Subjects in the feedback conditions were additionally instructed as to the nature of the feedback they would be receiving. Subjects in all conditions were told they would earn ½¢ for each heart beat which occurred in the instructed direction during a period of unaltered breathing and thus could earn up to $10.00 in addition to the $4.00 they earned for participating in the experiment.

The experiment proper consisted of 20 trials. Each trial consisted of a 40-beat baseline followed by 100 beats of attempted HR control. Before each control period the subject was signalled as to whether to attempt HR increase or decrease. Random orders of HR increase and decrease trials were used. At the end of each control trial subjects were signalled again.

Results

Analysis of the dependent measures was by ANOVA. IBI, ICI, and RD data were subjected to a 3×2×10×2 (feedback group × direction of HR change × trial × baseline or HR control) ANOVA. In addition, change scores were computed for each trial by subtracting the baseline value for each measure from the value obtained during attempted HR control. These change scores and the data for correct IBI and correct IBI-ICI were subjected to a 3×2×10 ANOVA with the "baseline or HR control" factor omitted.[5]

Adaptation Period

No differences were found between subjects assigned to the three feedback conditions in terms of their mean IBI or mean ICI averaged over the entire adaptation period.

HR Control

In Fig. 2 IBI data for baseline periods and HR control periods are displayed. A significant interaction of direction of HR change × baseline or HR control was obtained ($F(1/39)=42.970$). Planned comparisons by t-test revealed significant HR increases from baseline ($t(39)=5.89$) and significant HR decreases from baseline ($t(39)=3.39$) indicating subjects were able to reliably produce directional HR change. Feedback group (i.e., no feedback, HR feedback, or HR and RR feedback) did not affect the magnitude of HR change ($F(2/39)<1$) nor did it affect the number of correct IBIs ($F(2/39)<1$, see Table 2). Thus no evidence was found to support the importance of feedback in the control of HR.

RR Changes

In Fig. 2 ICI data for baseline periods and HR control periods are presented.[6] A significant in-

[4]Sroufe (1971) has manipulated RD and RR directly and found RD to have a more profound effect on HR.

[5]An additional ANOVA was calculated to determine whether HR control improved or deteriorated within trials. Each 100 beat control trial was subdivided into 20 beat subperiods for this analysis. No differences were found ($F(4/156)=1.116$).

[6]Comparison of Figs. 1 and 2 indicates that subjects in Experiment II breathed slower than subjects in Experiment I (mean ICI=4955 msec vs 4259 msec) but had similar HR (mean IBI=830 msec vs 836 msec). Data obtained during the adaptation period in Experiment II revealed that clamping subject's nostrils and having them breathe through the respiratory depth apparatus reliably caused slower breathing. However, the HR slowing which accompanied this slower breathing did not continue into the experiment proper. A possible explanation may be that homeostatic controls eventually returned HR to resting levels in spite of chronically altered RR. This would be in line with findings of studies which have paced RR and found little effect on HR (Engel & Chism, 1967; Sroufe, 1971; Sroufe &

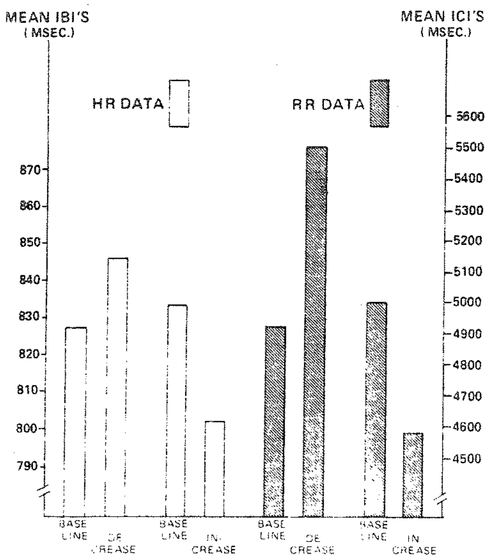

Fig. 2. Mean heart rate interbeat intervals and respiratory intercycle intervals during heart rate decrease and increase trials (Experiment II).

teraction of direction of HR change × baseline or HR control was found ($F(1/39)=63.24$). Planned comparisons by t-test revealed significant RR increases from baseline during periods of attempted HR increase ($t(39)=4.66$) and significant RR decreases during periods of attempted HR decrease ($t(39)=6.59$). Comparison of correct IBI and correct IBI-ICI data (Table 2) further illustrates the relationship between HR and RR changes. Overall subjects produced an average of 45.2 heart beats per 100 beat trial which met criterion. Only 10.6 of these occurred during respiratory cycles which met the criterion for RR.

Feedback level did not affect the magnitude of RR change ($F(2/39)=1.086$) nor did it affect the number of correct IBI-ICIs ($F(2/39)<1$). Thus no evidence was found to support the notion that feedback affected the amount of RR change during HR control.

RD Changes

RD (Table 3) increased significantly from baseline levels during HR increases ($t(36)=2.98$) but did not significantly change during HR decreases ($t(36)=1.56$).

Morris, 1973). The cardiac-respiratory concomitance seen in the present experiments may be more relevant to the acute changes in HR, RR, and RD which occur when subjects attempt short-term HR control.

TABLE 2

Correct IBIs and correct IBI-ICIs for HR control trials (100 beats) (Experiment II)

Groups	Correct IBIs	Correct IBI-ICIs
Overall	45.2	10.6
No Feedback Group[a]	45.8	10.1
HR Feedback Group	47.9	11.5
HR and RR Feedback Group	42.0	10.1

[a]No significant differences between feedback groups were obtained for either Correct IBIs or Correct IBI-ICIs.

Changes in Control Across Trials

To provide some indication of the extent to which control ability improved during the experimental session, change scores for IBI, correct IBI, and correct IBI-ICI data from trials 1–5 and 6–10 were averaged separately for HR increase and decrease trials. Planned comparisons by t-test revealed significant performance improvements on trials 6–10 for IBI change during HR increase ($t(338)=2.93$), IBI change during HR decrease ($t(388)=2.39$), correct IBIs ($t(351)=4.06$), and correct IBI-ICIs ($t(351)=1.69$). This performance improvement did not differ as a function of feedback level.

Sex Differences

No evidence was found for sex differences in the ability to control HR and RR. Female subjects did evidence faster HR during the adaptation period (79 bpm vs 72 bpm for male subjects; $F(1/30)=4.767$).

DISCUSSION

Experiment II provided a needed replication and extension of the findings of Experiment I. In this discussion I will comment on the major findings of the two experiments.

HR Control

Subjects in both studies produced statistically significant HR increases and decreases. The

TABLE 3

Respiratory depth means for HR control trials (Experiment II)

RD (liters)			
Baseline	HR Increase	Baseline	HR Decrease
.640	.729*	.642	.689

*Significant change from baseline.

magnitude of these changes averaged across all subjects and trials was not large but was consistent with the changes generally reported in single session HR control studies. This is not to say that no large magnitude changes were produced. In Experiment II HR increases of up to 34 bpm and decreases of up to 17 bpm were produced by certain subjects on certain trials. In reporting group data these individual heroics were obscured.

These findings of reliable HR change are not in and of themselves of great importance except to the extent that they indicate that the methodology used in the present investigations is capable of replicating the phenomena reported by other investigators using slightly different methods. The large magnitude changes produced by several subjects are of anecdotal interest because they are of similar magnitude to those produced by subjects in the multi-session studies of Headrick, Feather, and Wells (1971) and Stephens, Harris, and Brady (1972). Multiple sessions, however, may not be necessary to produce large magnitude HR changes. Stephens et al. reported that several of their subjects were able to produce large magnitude HR changes as early as their first training trial and Headrick et al. reported a subject in their preliminary study who was able to produce large magnitude HR increases after only 6 min of training. Taken together with the large magnitude changes produced in the present single-session investigation, these reports do not support a "learning" model of HR control. Rather, it would seem that subjects vary on their ability to control HR before they enter the HR control experiment. Thus, rather than "teaching" subjects to control HR, the HR control experiment may actually only be assessing subjects' pre-experimental ability to control HR.

Respiration Changes

A general pattern of faster RR during HR increase and slower RR during HR decrease was obtained in both experiments. As with HR data, the magnitude of the RR changes was not large but was statistically significant and lends support to a concomitance model of CNS-ANS interaction (as in Obrist, Webb, Sutterer, & Howard, 1970). RD data did not exhibit the bidirectionality of RR data with significant RD changes (i.e., increased RD) ocurring only during HR increase. Cohen (1973) has reported a similar finding in an escape-avoidance paradigm and Wells (1973), using a measure of RD equivalent to that used in the present study, reported consistent large magnitude RD increases during HR increase.[7]

[7]See Wells (1973), Table 1, page 267.

The question of whether the respiratory changes observed in the present investigations "caused" the HR changes cannot be answered within the design utilized. However, based on the present findings it may be justified to expect to find small magnitude respiratory changes as concomitants of small magnitude HR changes under the following conditions:

1) the measurement of respiratory activity is of the same level of sensitivity and accuracy as the measure of cardiac activity;
2) respiration is not "paced."

Feedback Effects

The present investigations produced virtually no support for the generally held notion that feedback is the sine qua non of HR control. This supports the findings of Bergman and Johnson (1971, 1972) that 1) subjects can control HR without feedback, and 2) the addition of feedback produces no improvement in performance. This finding does not contradict a great body of empirical evidence because, as indicated earlier, few experiments have included a test of the null hypothesis (i.e., HR control is independent of feedback). It also does not directly comment on the possibility that feedback effects might only become significant in multi-session experiments.

A comment is in order as to the nature of the feedback used in these studies. The original intention in designing a system of "digital" proportional feedback (Blanchard & Young, 1973) was to provide more information to subjects than simple binary (right-wrong) feedback while avoiding some of the ambiguities of proportional feedback schemes which use a meter scale or the like. Since subjects in the present studies did as well without feedback as subjects in other studies have done with feedback (and as well as subjects in the present study did with feedback) there is no reason to suspect that digital proportional feedback is any better or worse than any other kind of feedback. It was also hoped that providing subjects with feedback of RR would help them control that variable. The fact that no support was found for the utility of RR feedback may indicate that subjects cannot effectively process and utilize two simultaneous channels of biofeedback. Thus it seems that the strategy of pacing respiration may be a better choice for the single-session experiment.

Changes in HR Control Ability Across Trials

Results from Experiment II do indicate an improvement in subjects' abilities to control HR over the course of the session. More importantly, a similar trend was found for correct IBI-ICI data suggesting less concomitance of HR and RR on later trials. The implications of this finding are unclear. An optimistic view might conclude that subjects were learning to efficiently control HR without using mediators. Alternately one could conclude that subjects were learning to use non-respiratory mediators (e.g., muscle tension, recollection of past events, mental imagery) more efficiently to effect changes in HR. A final selection between these two conclusions awaits a multi-session experiment in which all of these potential mediators are monitored.

REFERENCES

Bergman, J. S., & Johnson, H. J. The effects of instructional set and autonomic perception on cardiac control. *Psychophysiology*, 1971, *8*, 180–190.

Bergman, J. S., & Johnson, H. J. Sources of information with affect training and raising of heart rate. *Psychophysiology*, 1972, *9*, 30–39.

Blanchard, E. B., Young, L. D., & McLeod, P. G. Awareness of heart activity and self-control of heart rate. *Psychophysiology*, 1972, *9*, 63–68.

Blanchard, E. B., & Young, L. D. Self-control of cardiac functioning: A promise as yet unfulfilled. *Psychological Bulletin*, 1973, *79*, 145–163.

Brener, J., Kleinman, R. A., & Goesling, W. J. The effects of different exposures to augmented sensory feedback on the control of heart rate. *Psychophysiology*, 1969, *5*, 510–516.

Cohen, M. J. The relation between heart rate and electromyographic activity in a discriminated escape-avoidance paradigm. *Psychophysiology*, 1973, *10*, 8–20.

Engel, B. T., & Chism, R. A. Effect of increases and decreases in breathing rate on heart rate and finger pulse volume. *Psychophysiology*, 1967, *4*, 83–89.

Engel, B. T., & Hansen, S. P. Operant conditioning of heart rate slowing. *Psychophysiology*, 1966, *3*, 176–187.

Headrick, M. W., Feather, B. W., & Wells, D. T. Unidirectional and large magnitude heart rate changes with augmented sensory feedback. *Psychophysiology*, 1971, *8*, 132–142.

Katkin, E., & Murray, N. Instrumental conditioning of autonomically mediated behaviors: Theoretical and methodological issues. *Psychological Bulletin*, 1968, *70*, 52–69.

Kirk, R. E. *Experimental design: Procedures for the behavioral sciences*. California: Brooks/Cole, 1968.

Obrist, P. A., Webb, R. A., Sutterer, J. R., & Howard, J. L. The cardiac-somatic relationship: Some reformulations. *Psychophysiology*, 1970, *6*, 569–587.

Sroufe, L. A. Effects of depth and rate of breathing on heart rate and heart rate variability. *Psychophysiology*, 1971, *8*, 648–655.

Sroufe, L. A., & Morris, D. Respiratory-cardiac relationships in children. *Psychophysiology*, 1973, *10*, 377–382.

Stephens, J. H., Harris, A. H., & Brady, J. V. Large magnitude heart rate changes in subjects instructed to change their heart rates and given exteroceptive feedback. *Psychophysiology*, 1972, *9*, 283–285.

Wells, D. T. Large magnitude voluntary heart rate changes. *Psychophysiology*, 1973, *10*, 260–269.

Young, L. D., & Blanchard, E. B. Sex differences in the ability to control heart rate. *Psychophysiology*, 1972, *9*, 667–668.

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