Patterns of autonomic and central nervous system responding during human operant heart-rate conditioning

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Automatic and central nervous system responding was assessed during human operant heart-rate acceleration and deceleration conditioning. The results indicated that the decelerative changes in heart rate that developed were relatively independent of responses in other physiological modalities. For subjects who were informed of the response-reinforcement contingencies, operant heart-rate acceleration was associated with an increase in respiratory rate. Both heart-rate acceleration and deceleration were superimposed upon a background of increased sympathetic-like skin responding. No significant changes in chin muscle tension and bi-temporal electroencephalographic alpha activity were noted during either operant heart-rate acceleration or deceleration. Individual subjects exhibited response stereotypy over sessions, although there were considerable differences between individuals in physiological response patterns. The results of the study and their relation to current theories concerning the mediation of operant heart-rate changes are discussed.

In the last decade there has been considerable inquiry into the degree to which the functioning of the autonomic nervous system (ANS) can be modified by operant conditioning. In particular, several teams of investigators have used operant techniques to produce heart-rate accelerations (e.g., Engel & Chism, 1967; Headrick, Feather, & Wells, 1971; Wells, 1973) and decelerations (e.g., Brener & Hothersall, 1966, 1967; Engel & Hansen, 1966; Schwartz, 1972) in human subjects. While there is some controversy concerning the clinical significance of such changes (e.g., Blanchard & Young, 1973), it is nevertheless well established that operant methods can be used to alter human heart rate.

One of the questions raised in the early work on operant heart-rate conditioning in animals concerned the interrelationships among ANS modalities during such conditioning. In a series of studies with animals, Miller and his colleagues examined the relationships between operant heart-rate changes and changes in other autonomic responses in animals (e.g., DiCara & Miller, 1969; Miller & Banuazizi, 1968; Miller & DiCara, 1968). In general, the results of these studies suggested that changes in operant heart rate were relatively independent of changes in other ANS responses. More specifically, Miller and Banuazizi (1968) demonstrated that autonomic

responding could be conditioned in one direction in one modality but in the opposite direction in another modality (e.g., simultaneous heart-rate increases and decreases in gastrointestinal contractions could be conditioned). Similarly, Schwartz (1972) and Shapiro, Tursky, and Schwartz (1970) have demonstrated that simultaneous increases in operant heart rate and decreases in operant systolic or diastolic blood pressure can be conditioned in human subjects, and vice versa.

Although the work cited above seems to indicate that changes in operant heart rate are relatively unrelated to other autonomic events, theorists have continued to speculate about the physiological events that mediate such changes. Changes in respiratory rate and depth, in somatic-muscular activity, and in central neural activity have all been suggested as events associated with, and perhaps important in the mediation of, operant heart-rate changes. There has been a considerable amount of experimentation conducted in order to elucidate more specifically the events related to the mediation of operant heart-rate changes. However, much of this research has produced equivocal results (see McCanne & Sandman, 1976, for a review of the literature). For example, Obrist and his colleagues have noted that changes in heart rate during classical conditioning and reaction time tasks are associated with changes in other somatic-muscular variables such as chin muscle activity, eyeblinks, and respiratory rate and depth (Obrist, Webb, Sutterer, & Howard, 1970a, b). They have suggested that changes in heart rate are "coupled" to changes in these other somatic variables at a central level. However, the correlations among these somatic-muscular events, while

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statistically significant, are nevertheless quite modest, indicating a fair amount of independence of the responses from one another. Moreover, changes in somatic-muscular variables have not been examined during human operant heart-rate conditioning. In addition, Cohen (1973), utilizing an avoidance conditioning paradigm rather than the positive reinforcement procedures more commonly employed, found that he could produce changes in heart rate in human subjects without concomitant changes in chin muscle tension, and vice versa. Thus, there is some question about the tenability of the "coupling" hypothesis during human heart-rate conditioning.

More recently, Schwartz (1975) has reported that changes in heart rate are associated with operant changes in occipital electroencephalographic (EEG) activity. In particular, he has noted that increased alpha wave activity is associated with a decrease in the ability of subjects to produce operant heart-rate acceleration. These results suggest that changes in occipital EEG activity may be important elements in the production of changes in operant heart rate. However, Schwartz (1975) has not examined other sites of EEG activity. The relationships between EEG activity and operant heart-rate changes may be specific to the occipital regions and not characteristic of other regions in the brain.

Despite the intriguing results obtained by Cohen (1973) and Schwartz (1975), relatively few experimenters have examined relationships between heart-rate changes and changes in other automatic or central nervous system responses during human operant heart-rate conditioning. Such investigations would seem critical in evaluating the mediating schemas that have been proposed. Moreover, individual differences in human physiological responding have been noted during a variety of experimental procedures (e.g., Davis, 1957; Lacey & Lacey, 1958; McCanne & Sandman, 1976). It seems quite likely that individual differences in physiological responding during operant heart-rate conditioning may account for some of the equivocal data on the mediation of such heart-rate changes. That is, there may be fundamental differences between individuals in neurobiological organization and in the autonomic and central events associated with operant heart-rate changes.

The present experiment was undertaken in order to examine the autonomic and central nervous system changes which occur in humans during operant heart-rate conditioning. The heart-rate data from the experiment have been presented in a previous report (McCanne & Sandman, 1975), but the data from the other autonomic and central nervous system modalities have not been reported. An examination of the correlates of changes in operant heart rate would appear to be a necessary first

step towards establishing what autonomic and central events may be involved in the mediation of such changes and in assessing the extent to which there may be individual differences in the mediation of those changes.

METHOD

Procedure

Twelve male students enrolled in introductory psychology courses at Ohio State University served as subjects in the experiment. Each subject was tested every day for five successive sessions. All 12 subjects were conditioned to accelerate their hearts to one stimulus (either a red or a green slide) and to decelerate their hearts to a second stimulus. The subjects were reinforced for producing changes in heart rate by means of a small real-time clock which ran whenever they produced changes exceeding criterion magnitude. All subjects were paid $\frac{1}{2}\text{\$}$ per second for keeping the clock on. In addition, half of the subjects were informed that heart-rate changes were correct responses, while the other half of the subjects were uninformed of the specific responses to be produced. This design thus explored heart-rate acceleration and deceleration within subjects over a number of operant conditioning sessions.

During each session, the subjects were given 10 acceleration and 10 deceleration trials, each of which was 50 heart interbeat intervals (IBIs) in length. Each trial was followed by a variable intertrial interval of 10 to 30 sec. The order in which the acceleration and deceleration trials were presented was randomized, with the restriction that there could not be more than two successive trials with the same color stimulus. In addition to heart rate, the following autonomic and central nervous system responses were recorded: (1) galvanic skin response (GSR), (2) endosomatic skin potential (SP), (3) digital plethysmograph (PL), (4) bitemporal electroencephalographic activity (EEG), (5) respiratory rate (RR), and (6) chin electromyographic activity (EMG).¹

An operant shaping procedure was employed throughout the experiment. Each subject received feedback contingent on each heart IBI during conditioning trials. The initial criterion for reinforcement was based on a 1-min estimate of the subject's resting heart rate at the beginning of each session. If a subject received more than 70% reinforcement during a trial (i.e., received reinforcement on 36 or more of 50 IBIs during a trial), the criterion for the next trial was made more difficult by 2 beats/min. If a subject received less than 30% reinforcement during a trial (i.e., received reinforcement on less than 15 IBIs), the criterion for the next trial was made easier by 2 beats/min.

Apparatus

Respiration. The respirometer consisted of a sliding piston mounted on an elastic band and placed around the subject's chest. A photocystal in one end of the piston served as an arm of the resistance bridge. The resistance of the photocystal was modulated by a small light in the opposite end of the piston and was determined by the distance between the light and the photocystal.

Heart rate. Ag cup electrodes filled with electrode paste were placed on the lower left rib cage and the right collar bone. The signal was amplified by a wide-band ac preamplifier and averaged by a cardi tachometer which provided a beat-by-beat record of the heart rate.

Digital plethysmograph amplitude. A photoelectric plethysmograph mounted in a Plexiglas ring was placed over the moon of the fourth finger on the subject's nonpreferred hand. The plethysmograph signal was amplified by an ac-coupled preamplifier.

Exosomatic GSR. A curved Ag-AgCl electrode of 3 cm² placed on the volar surface of the second phalange of the subject's pre-

ferred hand served as the active electrode, and a curved armband of 58 cm² placed on the upper arm served as the inactive electrode of a monopolar placement. A constant current of 20 μ A supplied by a Con-Sol GSR preamplifier was impressed at the active site making the portion of the subject's epidermis lying between the active and inactive electrodes an arm of a resistance bridge. A bandage with a 2-cm² hole in it restricted the current density to 10 μ A/cm² at the active electrode, whereas the current density at the reference electrode was .314 μ A/cm². A .05-M NaCl solution suspended in an inert plastic medium served as electrode paste. Surface body oils were removed by swabbing the electrode sites with acetone.

Bitemporal EEG. Ag-AgCl cup electrodes were employed in a bipolar temporal placement. The electrode sites were areas T₃ and T₅, according to the ten-twenty electrode placement system (Jasper, 1958). Body oils were removed by swabbing the electrode sites with acetone. Grass EEG paste served as the electrode medium. The signal was amplified by a wide-band EEG preamplifier.

Skin potential. Beckman low-bias potential (less than 50 μ V), low drift Ag-AgCl electrodes were employed. The volar surface of the second finger of the subject's nonpreferred hand served as the active electrode site, and the inner surface of the nonpreferred forearm served as the reference site. The electrode sites were swabbed with acetone to remove oils, and activity at the reference site was abolished by brisk rubbing with a gauze pad. The electrode medium was .05 M NaCl in an inert plastic medium. The difference in the standing potential at the two sites was amplified by a 1-megohm input impedance dc preamplifier. A bucking voltage of opposite polarity supplied by the preamplifier balanced the dc potential and kept the oscillograph on channel.

Chin EMG. Low-standing-potential (less than 100 μ V), low-drift Ag-AgCl cup electrodes were employed. The electrode sites were those described by Lippold in Venables and Martin (1967): $\frac{3}{4}$ in. above and below the midline of the chin. The electrode sites were swabbed with acetone to remove body oils. The electrode medium was .05 M NaCl in an inert plastic medium. The signal was amplified by a wide-band ac EMG preamplifier.

Physiological Data Reduction

Heart rate. The length of each trial was measured to the nearest millimeter. Since each trial was 50 IBIs long, the length of the trial was determined by the subject's average heart rate during the trial. The trial length was transformed into a measure of average heart rate. A similar procedure was employed to determine the subject's average heart rate during the 10 IBIs immediately preceding each trial.

Exosomatic GSR. The number of decreases in resistance of 100 ohms magnitude was tallied for each trial and for 10 IBIs preceding each trial.

Digital plethysmograph amplitude. The peak-to-trough height (in millimeters) of each wave form was calculated for the 1st, 10th, 20th, 30th, 40th, and 50th pulse during each trial. These six scores were averaged to yield an average height per trial. The same procedures were employed for tabulating the height of the 1st, 5th, and 10th pulses during the 10 IBIs preceding each trial. The average height of these three pulses was then computed for each pretrial.

Respiration. Frequency of respiration was determined by peak-to-peak count to the nearest 1/10th of a cycle for each trial and for

the pretrial. The peak-to-peak count in each interval was divided by the number of seconds in the interval, yielding a measure of respiratory cycles per second.

Bitemporal EEG. The number of seconds of slow-wave activity (5-12 cycles/sec) was tabulated and converted to a percentage of the total trial or pretrial time.

Skin potential. Total negative baseline shifts (tonic responses; Kaiser & Sandman, 1975; Miller, 1967) were expressed as millivolt change for each trial and each 10 IBI pretrial. Frequency of a-wave response (Forbes, 1964) or phasic response (Kaiser & Sandman, 1975; Miller, 1967) was tallied for each trial and the 10 IBI pretrials.

Chin EMG. The number of separate "bursts" of 50 μ V or more in magnitude was tabulated for each trial and for the 10 IBI pretrial.

Data Transformations

Data were reduced from each conditioning trial and from the 10 IBIs preceding each trial. It was thus possible to examine pretrial to trial changes in each physiological modality. However, since the physiological parameters were measured in different units, it was difficult to compare and contrast changes across response modalities.

All data were transformed to standard score units (z scores) prior to any analyses. For the heart-rate variable, the heart rate computed for every pretrial and trial during each session was transformed into z-score units for each subject. The transformations utilized the mean and standard deviation computed for all 12 subjects during trials and pretrials over all five experimental sessions. Identical transformations were applied to all the other physiological variables.

Design

The statistical model for the study was a 2 (pretrial vs. trial) $\times$ 5 (sessions) $\times$ 10 (trials) repeated measures factorial design. For each physiological variable, separate 2 $\times$ 5 $\times$ 10 analyses of variance were computed for informed and uninformed subjects and for acceleration and deceleration training.

RESULTS

The untransformed means and standard deviations for each of the physiological variables are presented in Table 1. Pretrial-to-trial changes in response were computed (in z-score units) for every physiological variable for each session. Separate change scores were computed for acceleration and deceleration training trials. Profiles of the average change scores for informed and uninformed subjects over all five conditioning sessions are presented in Figures 1 and 2.

In addition to the overall profiles illustrated in Figures 1 and 2, pretrial-to-trial changes in responding were computed for each of the five sessions for informed and uninformed subjects. For each group of subjects, the changes in physiological variables were rank ordered during each session. The

Table 1
Means and Standard Deviations of Untransformed Physiological Variables

	HR (beats/min)	GSR (number)	PL (in mm)	EEG (percentage)	RR (cycles/min)	SP Tonic (in mV)	SP Phasics (number)	EMG (number)
Mean	69.85	1.72	13.68	10.67	13.20	.16	.12	8.59
SD	8.88	2.57	11.50	12.64	5.40	.41	.47	26.62

Figure 1. Pretrial-to-trial changes in physiological responding for informed subjects during acceleration and deceleration training. The values for physiological variables have been transformed into standard scores and can be converted into raw scores by reference to Table 1. HR = average heart rate; GSR = number of decreases in GSR; PL = digital plethysmograph amplitude; EEG = percent of EEG alpha activity; RR = respiratory rate; SPT = skin potential tonic height; SPP = number of skin potential phasics; EMG = chin EMG.

Figure 2. Pretrial-to-trial changes in physiological responding for uninformed subjects during acceleration and deceleration training. The values for physiological variables have been transformed into standard scores and be converted into raw scores by reference by Table 1. Legend is the same as Figure 1.

variable that displayed the most positive change was assigned a rank of 1, the variable that displayed the next most positive change was assigned a rank of 2, etc. Coefficients of concordance (Siegel, 1956) were computed using the rank orders of the changes in the variables for each of the five sessions. Separate coefficients were computed for acceleration and deceleration training. These coefficients indicated that the response patterns displayed in Figures 1 and 2 were quite similar to the response patterns during any single session ($W = +.61$ for informed subjects, acceleration training; $W = +.76$ for uninformed subjects, acceleration training; $W = +.68$ for informed subjects, deceleration training; $W = +.80$ for uninformed subjects, deceleration training; $p < .01$ for all coefficients).

One potential difficulty in analyzing the change scores presented in Figures 1 and 2 seemed to be that the changes portrayed in Figures 1 and 2 may have resulted from differences in pretrial responding during acceleration or deceleration training rather than from changes in responding during conditioning trials. For example, it was conceivable that the pretrial to trial heart-rate acceleration noted for informed subjects (Figure 1) resulted because the pretrial heart rate preceding acceleration trials was relatively low, thus facilitating heart-rate acceleration. To examine such possible fluctuations, separate repeated measures analyses of variance comparing pretrial responding during acceleration training to pretrial values during deceleration training across sessions and trials were computed for each physiological variable for informed and uninformed subjects. The results of these analyses indicated that the pretrial values for acceleration trials did not differ significantly from pretrial values for deceleration trials for any of the physiological responses. Thus, it did not appear that the changes illustrated in Figures 1 and 2 were attributable to fluctuations in pretrial responding.

Examination of the acceleration and deceleration training profiles presented in Figures 1 and 2 suggested that the patterns of response were somewhat similar during both types of training. The significance of the pretrial-to-trial changes illustrated in Figures 1 and 2 was reflected by the pretrial/trial main effects for the repeated measures analyses of variance computed for each physiological variable. Significant pretrial/trial main effects are reported below.

During acceleration training, informed subjects displayed increases in heart rate which were of borderline statistical significance [$F(1,5) = 5.44$, $p < .10$]. Significant increases in GSR [$F(1,5) = 24.48$, $p < .01$], RR [$F(1,5) = 6.80$, $p < .05$], SP tonic [$F(1,2) = 20.55$, $p < .05$], and SP phasic [$F(1,2) = 18.99$, $p < .05$] responding were also noted during acceleration training. During deceleration

training, informed subjects also displayed significant increases in GSR [$F(1,5) = 22.16$, $p < .01$], SP tonic [$F(1,2) = 20.11$, $p < .05$], and SP phasic responding [$F(1,2) = 19.03$, $p < .05$]. However, in contrast to the acceleration training results, informed subjects displayed highly significant decreases in heart rate during deceleration training [$F(1,5) = 30.28$, $p < .01$], and no significant change in RR responding was noted. Thus, both operant heart-rate acceleration and deceleration were accompanied by sympathetic-like increases in skin responding, but heart-rate acceleration was also accompanied by faster respiratory frequencies while no change in respiration was noted during heart-rate deceleration.

For uninformed subjects, significant increases in GSR [$F(1,5) = 26.19$, $p < .01$], SP tonic [$F(1,2) = 20.81$, $p < .05$], and SP phasic [$F(1,2) = 19.92$, $p < .05$] responding were detected during acceleration training. The pretrial/trial main effect for changes in heart rate was not significant for uninformed subjects during acceleration training, indicating that for these subjects the acceleration conditioning procedures did not result in discriminative increases in heart rate. During deceleration training, uninformed subjects produced significant increases in GSR [$F(1,5) = 23.18$, $p < .05$] and SP phasic [$F(1,2) = 18.50$, $p < .05$] responding, and a significant decrease in heart rate [$F(1,5) = 8.80$, $p < .05$]. The increases in skin responding detected during both types of training were similar to the response patterns noted for informed subjects.

It is interesting to note that the significant decreases in heart rate noted for informed and uninformed subjects were *not* accompanied by significant decreases in respiratory rate. Nor were they paralleled by significant changes in any physiological variables other than the increases in responding for the skin response variables. Thus, it appeared that the operant heart-rate deceleration that occurred during conditioning was relatively specific and unrelated to changes in other modalities. The differences between the acceleration and deceleration patterns of responding raise the possibility that different neurophysiological response mechanisms may be involved in the mediation of the two responses.

Individual Differences in Patterning

Individual patterns of changes in responding were computed for each subject over the five conditioning sessions. These individual profiles are illustrated in Figures 3 and 4.

As Figures 3 and 4 indicate, individual subjects displayed somewhat idiosyncratic response profiles. To determine whether each individual's overall profile was representative of the individual's response profiles during each session, intraindividual coefficients of concordance were computed across

INFORMED SUBJECTS

Figure 3. Pretrial-to-trial changes in physiological responding for individual informed subjects during acceleration and deceleration training. The values for physiological variables have been transformed into standard scores and can be converted into raw scores by reference to Table 1. Legend is the same as Figure 1.

the rank orders of the variables during each of the five conditioned sessions. Separate coefficients were computed for acceleration and deceleration training. These intraindividual coefficients are presented in Table 2.

As Table 2 indicates, every subject in the study displayed significant patterning of responses over the five conditioning sessions. Thus, the individual patterns of responding displayed in Figures 3 and 4 were highly consistent from session to session, although there were marked differences between subjects in the shapes of the response patterns. Within the literature on physiological responses to stress, the Laceys (Lacey, 1950; Lacey, Bateman, & VanLehn,

1953; Lacey & Lacey, 1958) have demonstrated that subjects display highly idiosyncratic patterns of response, but have also noted that the patterns are consistent over time and under a variety of stressors ("response stereotypy"; Lacey & Lacey, 1958). It appears that operant heart-rate conditioning also produces "response stereotypy" similar to that noted during stressful experiences.

Inspection of Figures 3 and 4 indicated that the magnitude of the operant heart-rate changes varied from subject to subject. In addition, there were a number of striking discrepancies between the individual response patterns illustrated in Figures 3 and 4 and the group response profiles displayed in

Table 2
Coefficients of Concordance Across Sessions
for Individual Subjects

Subject Number	Acceleration Training		Deceleration Training	
	W	p	W	p
Informed Subjects				
1	+.93	< .01	+.85	< .01
2	+.51	< .05	+.69	< .01
3	+.79	< .01	+.47	< .05
7	+.72	< .01	+.55	< .01
8	+.55	< .01	+.78	< .01
9	+.58	< .01	+.77	< .01
Uninformed Subjects				
4	+.54	< .05	+.60	< .01
5	+.52	< .05	+.68	< .01
6	+.62	< .01	+.68	< .01
10	+.61	< .01	+.69	< .01
11	+.54	< .05	+.78	< .01
12	+.55	< .01	+.40	< .05

Figures 1 and 2. For most subjects, there was a pervasive increase in GSR responding during both acceleration and deceleration conditioning. There were, however, several subjects who did not display such increases (Nos. 4, 7, and 11). Thus, it appeared that there were notable individual differences in responding even for the one response modality which exhibited the largest changes for most subjects.

There were also individual differences in responding which were somewhat contradictory to those noted for the group response profiles. Thus, while five of the six informed subjects exhibited increased respiratory rates during acceleration training and decreased rates during deceleration training, one subject (No. 8) displayed the opposite pattern of respiratory changes. Yet subject 8 also produced the second largest heart-rate increase and the largest heart-rate deceleration of all 12 subjects. These results suggest that increased respiratory rates were generally associated with increased operant heart rate, but also indicate that changes in respiration were not necessary concomitants of operant heart-rate acceleration for all subjects.

Correlational Analyses

The analyses presented above examined the extent to which pretrial to trial changes in heart rate were accompanied by changes in other response modalities. It was also possible to examine the relationships between the magnitudes of heart-rate responses during training trials and the magnitudes of other physiological responses. Such an analysis should provide information concerning the extent to which heart-rate responses were related to other physiological responses.

Intraindividual correlations were computed between heart-rate responses and the other physiological variables. All correlations were computed utilizing

the z-score units described above. The correlations were computed using data obtained during training trials; that is, data obtained during pretrial periods were not utilized. Separate intraindividual correlations were computed for acceleration and deceleration training. The individual correlations and the mean correlations for informed and uninformed subjects (computed by means of Fisher's z-score transformation) are presented in Tables 3 and 4.²

Since the values of the physiological variables used in computing the correlations were not independent events (i.e., responding during each trial was related to responding during preceding and subsequent trials), the correlations illustrated in Tables 3 and 4 should be interpreted with caution. As the tables indicate, the averages of the individual coefficients were relatively small for both acceleration and deceleration training for informed and uninformed subjects. In addition, there were considerable differences between individuals in both the magnitude and directions of the correlations. These results provide additional confirmation of the individual idiosyncracies in response patterning noted above.

DISCUSSION

In general, the results of the present experiment indicate that increases in operant heart rate are paralleled by increases in respiratory rate for informed subjects. The respiratory data suggest that informed subjects may have accomplished the changes in heart rate by producing changes in breathing patterns. Significant changes in operant heart rate and respiratory rate were not detected for uninformed subjects during acceleration conditioning. These results indicate that the type of instructions given to subjects may influence the strategies subjects employ in attempting to produce increases in heart rate. The changes in respiration noted for informed subjects were in accord with the observations of several other investigators (e.g., Brener & Hothersall, 1966, 1967; Shearn, 1962).

Operant heart-rate acceleration training was also accompanied by sympathetic-like increases in skin responding. However, similar increases in skin responding were also noted during deceleration. While the heart-rate increases noted during acceleration training may have been part of a generalized increase in sympathetic-like responding, the heart-rate decreases detected during deceleration training were relatively independent of the increases in skin response. Thus, it appears that the significant decelerative changes in heart rate were highly specific.

It seems possible that the relative independence of decelerative changes in heart rate and changes in other autonomic and central responses may be due to the dual innervation of the heart by the sympathetic and parasympathetic nervous systems

UNINFORMED SUBJECTS

Figure 4. Pretrial-to-trial changes in physiological responding for individual uninformed subjects during acceleration and deceleration training. The values for physiological variables have been transformed into standard scores and can be converted into raw scores by reference to Table 1. Legend is the same as Figure 1.

(Neil & Heymans, 1962). The parasympathetic nervous system, with its long, myelinated preganglionic fibers, seems much more capable of mediating the specific operant heart-rate deceleration noted than the sympathetic system, with its long, unmyelinated postganglionic fibers. Moreover, this formulation is consistent with data from reaction time and classical conditioning paradigms, which indicate that the heart-rate deceleration noted during those tasks is vagal in origin and is blocked by the administration of atropine (e.g., Hein, 1969; Obrist, Wood, & Perez-Reyes, 1965; Obrist et al., 1970b). In summary, it appears that *different* neural processes may be involved in the production of operant heart-

rate acceleration and deceleration, rather than a single process at work during both.

It is interesting to note that significant changes were not detected for either of the CNS variables (EEG and EMG) during either acceleration or deceleration training. These results indicated that the operant heart-rate changes that occurred were not paralleled by changes in these particular indices of electrical activity initiated at a central level. This lack of parallel CNS change appears to contradict Schwartz's (1975) data indicating relationships between operant heart-rate changes and changes in EEG occipital alpha activity. However, the EEG placement sites in the current experiment were

Table 3
Correlations Between Heart Rate and Other Dependent Variables During Acceleration Training Trials

	GSR	PL	EEG	RR	SP Tonic	SP Phasics	Chin EMG
Informed Subjects							
1	.19	.13	.06	.38	-.28	.16	
2	-.18	.13	.03	-.51	.03	-.03	
3	.31	.29	.32	.44	.23	.10	
7	-.14	-.47	-.44	.04			.15
8	.70	-.68	-.37	-.23			.32
9	.19	.40	.80	-.31			.28
Mean r	.18	-.03	.07	-.03	.01	.08	.25
Uninformed Subjects							
4	.85	-.27	-.25	-.07	-.01	.00	
5	-.22	-.57	-.43	-.73	.18	-.18	
6	.82	-.59	-.01	-.64	.52	.23	
10	-.06	-.06	-.19	-.05			.27
11	-.32	.57	.26	.35			.10
12	.21	.46	-.48	-.23			.15
Mean r	.21	-.08	-.18	-.23	.23	.02	.17

Note—SP was monitored only for Subjects 1-6. Chin EMG was monitored only for Subjects 7-12. Correlations were computed using data that had been averaged into blocks of two trials per block. There were five trial blocks per session, for a total of 25 trial blocks.

temporal rather than occipital. The differences between the present data and those presented by Schwartz (1975) suggest that changes in operant heart rate may be paralleled by changes in brain functioning in relatively discrete locations in the brain.

In addition, the lack of significant changes in chin EMG activity during operant heart-rate changes apparently contradicts data presented by Obrist and his colleagues which indicate parallel relationships between heart-rate and EMG activity (Obrist et al., 1970a b). However, as pointed out above, most of Obrist's data has come from reaction time and classical and conditioning paradigms, rather than operant conditioning approaches. Moreover, the present results are in accord with those noted by

Cohen (1973) during avoidance conditioning of changes in heart rate.

Finally, and perhaps most importantly, analysis of the individual response patterns noted during the study indicated considerable differences in patterning between subjects. The idiosyncratic response patterns suggested that different physiological events may be associated with changes in operant heart rate for different subjects. That is, for some subjects changes in heart rate may be associated with respiratory changes, while for other subjects heart-rate increases and decreases may be associated with changes in muscle tension, etc. Such idiosyncratic response patterns have been noted by a number of other researchers during a variety of tasks (McCanne & Sandman, 1976). In the context

Table 4
Correlations Between Heart Rate and Other Dependent Variables During Deceleration Training Trials

	GSR	PL	EEG	RR	SP Tonic	SP Phasics	Chin EMG
Informed Subjects							
1	-.46	.52	.14	.54	.44	.00	
2	-.01	.57	.06	-.62	-.30	-.05	
3	.25	.44	.14	.37	.24	.02	
7	-.17	-.60	-.26	.06			.23
8	.39	-.20	-.02	.06			-.01
9	.02	.33	.72	-.22			.12
Mean r	.00	.18	.13	.03	.13	-.01	.11
Uninformed Subjects							
4	-.22	-.25	-.23	.10	.51	.37	
5	-.26	-.41	-.40	.21	.02	-.14	
6	.31	-.32	-.30	-.10	.17	.10	
10	-.33	-.02	.08	-.39			.24
11	-.12	.62	.09	.20			.18
12	-.28	.78	-.65	.36			-.01
Mean r	-.15	.07	-.24	.07	.23	.11	.14

Note—SP was monitored only for Subjects 1-6. Chin EMG was monitored only for Subjects 7-12. Correlations were computed using data that had been averaged into blocks of two trials per block. There were five trial blocks per session, for a total of 25 trial blocks.

of the present study, the individual differences in response patterning suggest that there may be fundamental differences between subjects in neurobiological organization. Experimenters increased in elucidating the mechanisms involved in producing changes in operant heart rate need to consider such potential differences between subjects in future research.

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NOTES

1. Endosomatic skin potential and chin EMG were recorded for three uninformed and three informed subjects, rather than six subjects from each group.
2. Between-subject correlations were also computed using each subject's average response over sessions and trials. These between-subject correlations were similar to the mean intra-individual correlations reported in Tables 3 and 4.

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