RESPIRATORY AND HEART RATE INDICES OF REACTION TIME

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ABSTRACT

RESPIRATORY AND HEART RATE INDICES OF REACTION TIME

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The respiratory and heart rate (HR) indices of attention to reaction time (RT) signal and control nonsignal stimuli were investigated in male college students. The two conditions produced differential respiratory and HR responses. Heart rate accelerated in response to the warning and respond signals. The HR acceleration to the respond signal was concordant with increases in HR variability, respiratory frequency and amplitude. In constrast to the mean HR, which did not change in anticipation of the respond signal, respiratory frequency increased and respiratory amplitude and HR variability decreased. In response to the nonsignal stimuli HR decelerated. Mean HR variability prior to the trial onset and mean magnitude of the reduction of HR variability in anticipation of the respond signal were highly significant correlates of RT when the preparatory interval (PI) was of variable duration. When the PI was of fixed duration HR variability was not correlated with performance. Although HR decelerated prior to the respond signal, the magnitude of deceleration was not related to performance. The data support a two component hypothesis of attention: the first, a phasic reflexive response dependent upon the specific stimulus change and characterized by a directional HR response; the second, a tonic instrumental response related to attentional performance and characterized by a decrease in HR variability.

RESPIRATORY AND HEART RATE INDICES OF REACTION TIME

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To a girl named Sue

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INTRODUCTION

Considerable research has been involved in the identification of heart rate (HR) components of attention (Lacey, 1959, 1967; Graham & Clifton, 1966; Porges & Raskin, 1969). These investigations of the HR indices of attention have lead to attempts to relate HR responses, most commonly HR deceleration, with sensorimotor performance (Obrist, Webb, & Sutterer, 1969; Webb & Obrist, 1970; Lewis & Wilson, 1970). Although HR deceleration is a reliable response during specific aspects of attention (Lacey, 1967; Porges & Raskin, 1969), the studies relating magnitude of HR deceleration with simple reaction time (RT) performance have indicated weak relationships. This repeated finding, must, therefore, question the use of HR deceleration as an index of attentional performance.

Lacey (1959) proposed that attentive observation of the external environment is accompanied by cardiac deceleration, whereas, situations that require rejection of the external environment (cognitive tasks or tasks requiring attentive observation of internal stimuli) produce HR acceleration. Lacey interpreted this directional HR response as a neurophysiological link in an afferent feedback mechanism responsive to the efferent processes of the CNS, and, therefore, capable of influencing behavioral events. Porges and Raskin (1969) substantiated the directional HR response by demonstrating that the direction of the HR response was dependent upon the experimental situation; HR accelerated while Ss attended internally to their own HR, and HR decelerated while Ss attended to intermittent tones or lights.

The recording of HR responses during classical conditioning has demonstrated HR deceleration in anticipation of the unconditioned stimulus (Obrist, Sutterer, & Howard, 1969). Zeaman and Smith (1965) confirmed the finding that anticipatory HR deceleration occurred independent of direction and magnitude of the cardiac component of the unconditioned response. They reported HR deceleration not only in anticipation of shock, but also in anticipation of both pleasant and unpleasant stimuli. Obrist (1969) reported anticipatory HR deceleration to the unconditioned stimulus and during subject preparation to respond in a simple RT task. Chase, Graham, and Graham (1968) used a simple RT task and reported two deceleratory HR responses within the PI. The first deceleration followed the onset of the warning signal and the second preceded the onset of the respond signal. They interpreted the first deceleration as an OR to the warning signal and the second deceleration as a conditioned "attention" response enhancing stimulus reception. Other investigators (Lacey, Kagan, Lacey, & Moss, 1963; Obrist, 1963) have also reported deceleratory HR responses in anticipation of various motivationally significant stimuli.

Contiguous with the research designating HR deceleration as an autonomic component of attention is the research attempting to forge a relationship between HR deceleration and performance on attention demanding tasks. Obrist, Webb, and Sutterer (1969) using a 5 sec. fixed PI in a simple RT task, found a weak, but significant, correlation between the magnitude of HR deceleration and RT ($r = .32$, $p < .05$). Lewis and Wison (1970) reported that HR deceleration accompanied visual scanning in 44-month old children. The deceleration was related to the accuracy of the behavioral response, but not to the response latency, a measure of RT.

The RT studies reviewed above have utilized fixed PIs. By using a fixed PI, the RT task may be explained in classical conditioning terminology; the warning and the fixed PI may be interpreted as a compound CS and the respond signal as the UCS. Since the conditioned HR response is decelerative, HR deceleration prior to the respond signal would indicate a conditioned anticipatory response, temporally bound to the fixed PI.

Performance during simple RT tasks is a function of the PI. Faster RT occurs during conditions in which the temporal relationship between the warning and respond signals is fixed. Thus, when a simple RT task with fixed PIs is used to investigate the relationship between autonomic components of attention and attentional performance, the results may be confounded by the temporally conditioned autonomic responses. The possibility, therefore, exists that the HR deceleration prior to the respond signal is not related to perceptual enhancement of the respond signal but a conditioned anticipatory response. If this is the case, it is not surprising to find a weak relationship between HR deceleration and RT.

In addition to the confounding effect of temporal conditioning, the orienting response to the warning signal may influence autonomic responses during the PI. If the PI is of short and constant duration, the conditioned and orienting responses may mask the HR response related to performance. To solve these problems and to enhance the probability of finding a HR response related to attentional performance, the PI may be extended to partition the orienting components and presented for random variable durations to reduce the possibility of temporally conditioned anticipatory responses. Thus, the use of an extended variable PI increases the probability of finding a HR response which would reflect an instrumental act by the organism to lower selected perceptual thresholds.

The present experiment was designed to investigate the respiratory and HR indices of attention to RT signal and control nonsignal stimuli and to relate these indices to RT performance. A second purpose was to investigate the differences between respiratory and HR responses to temporally bound stimuli (fixed interstimulus intervals) and responses to stimuli not temporally bound to the stimuli (variable interstimulus intervals). Thus, changes in HR and respiration could be compared for *Ss* attending to signal or nonsignal stimuli during schedules of fixed or variable interstimulus intervals.

Method

<u>Subjects</u>. Forty-eight male volunteers from introductory psychology courses at Michigan State University received extra course credit for serving as <u>S</u>s. As they appeared at the laboratory, they were assigned to experimental conditions according to a predetermined random schedule.

<u>Apparatus</u>. Stimuli were programmed by means of magnetic tape and were presented automatically (Porges & Fitzgerald, in press). Reaction time was measured by a Standard electronic timer. The ambient noise level of the experimental room was 51 db. (re .0002 dynes/cm^2). Room temperature was maintained at approximately 70° F.

The physiological responses were continuously recorded on a Beckman Type RS Dynograph at a paper speed of 5mm/sec. The EKG recording sites were cleaned with 70% ethanol prior to the application of the electrodes. Zinc cup electrodes with a surface area of 3.14 sq. cm. and filled with cotton soaked in a 1% $ZnSO_4$ solution were used to record HR from EKG lead II. The HR was measured using a Beckman 9857 cardiotachometer. Respiration was monitored with a Parks Electronics 4-in. mercury strain gage attached attached around the chest by a Velcro fastener. Changes in chest circumference were measured by a Beckman 9875B mercury strain gage coupler.

<u>Procedure</u>. Subjects were randomly assigned to one of four groups of 12 <u>S</u>s each. A two-by-two factorial design was used with the factors being task (RT and control) and schedule of PI (fixed and variable). The RT groups were instructed to respond as rapidly as possible following the termination of the warning signal (green light) that remained illuminated

for the duration of the PI. Simultaneous with the termination of the warning signal, the respond signal (red light) was illuminated. The respond signal was terminated when S pressed a button. The control groups were instructed to merely watch the signal lights. The respond signal was presented for 60 msec. following the termination of the warning signal in the control, since it was only necessary to elicit a response to change in stimulus conditions.

The S was seated in a comfortable armchair in a sound-attenuated room. After the pickups were attached, E read the instructions which informed S of the task and then calibrated the recording equipment. The experimental room was dimmed and the stimuli were presented by the automatic equipment. Each S then received ten trials. The four groups had the same schedule of intertrial intervals, which varied among 45, 60, and 75 sec. according to a predetermined random schedule. The PI duration was 16 sec. for the fixed PI groups and varied among 16, 22, and 28 sec. according to a predetermined random schedule for the variable PI groups. The RT groups were instructed to press a button on the arm rest at the cessation of the warning signal. The latency between the termination of the warning signal and the button press was recorded as S's RT.

Quantification of the data. The responses during each of the 10 trials were scored for HR and respiation. Each trial was divided into four periods: a "pre" period consisting of the 8 sec. prior to trial onset, an OR period consisting of the first 8 sec. of the PI, an attend period consisting of the last 8 sec. of the PI, and a respond period consisting of the first 8 sec. following the onset of the respond signal.

Respiration rate was analyzed by counting the frequency of initiations and terminations of inspirations in each period. The mean amplitude of complete inspirations in each period was measured in millimeters of pen deflection. The HR was obtained from a beat-by-beat read out of the cardiotachometer which transformed the R-R intervals into HR. The HR reading for each measurement interval of 1 sec. consisted of the HR in beats/min calculated from the R-R interval which was completed during that second. If more than one R-R cycle was completed during a given second, only the later cycle was scored. The HR response was evaluated by a comparison of mean HR and HR variance during the four periods and by a second-by-second analysis of HR during the OR and attend periods.

Results

<u>Respiration frequency</u>. There was a significant increase in respiration frequency from 3.98 during the "pre" period to 4.49 during the respond period [$F(3, 132) = 27.7$, $p < .0005$]. However, as illustrated in Fig. 1, the respiration frequency during the four periods differed under the two task conditions [$F(3, 132) = 12.4$, $p < .0005$]. Although both curves show monotonic increases, the curve depicting the RT groups exhibits a pronounced increase compared to the controls that maintained an approximately constant rate. A test for simple effects showed significant differences among the means of the four periods only for the RT task [$F(3, 132) = 38.1$, $p < .0005$]. A Newman-Keuls test showed that the respiration frequency for the RT task during the respond period was significantly greater than during the other periods ($p < .01$) and that the frequency was greater during the attend period than during the "pre" period ($p < .05$). There was no over all trial effect, but the two tasks differed across trials [$F(9, 396) = 3.8$, $p < .0005$]. The mean of 4.81 for the RT groups on Trial 1 decreased to 4.18 on Trial 10, while the mean of 3.96 on Trial 1 for the control groups increased slightly to 4.07 on Trial 10.

<u>Respiration amplitude</u>. The respiration response exhibited a significant trend of decreasing amplitude from the "pre" (11.93 mm) through the attend (10.77 mm) periods, followed by a return to approximately "pre" level during the respond period (11.55 mm), [$F(3, 132) = 4.4$, $p < .005$]. Figure 2 illustrates the significant differences between the two tasks for respiration amplitude during the four periods [$F(3, 132) = 6.1$, $p < .001$]. The RT task resulted in a pronounced decrease in amplitude during the attend period followed by an increase during the respond

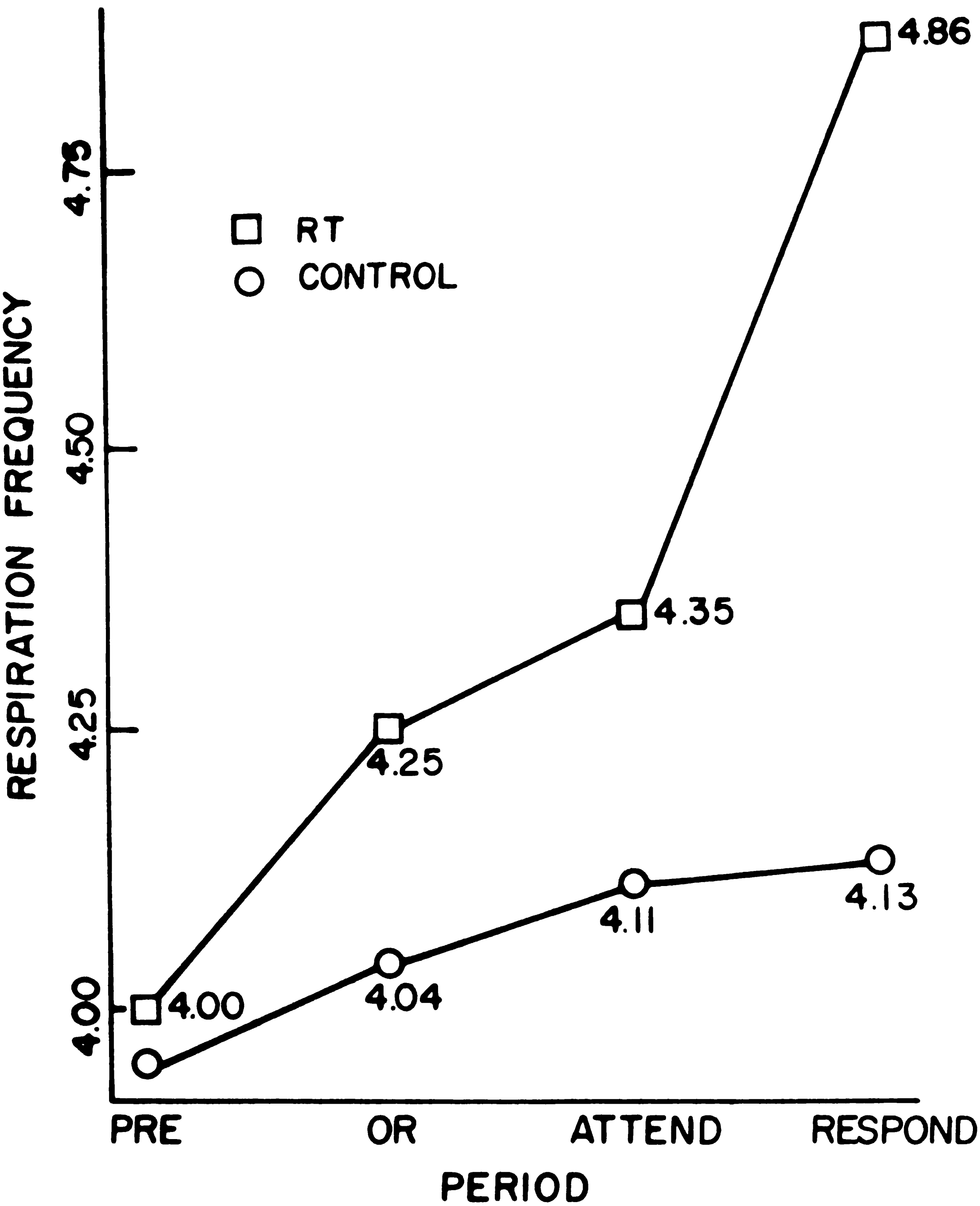

Fig. 1. Mean respiration frequency as a function of periods for the two tasks.

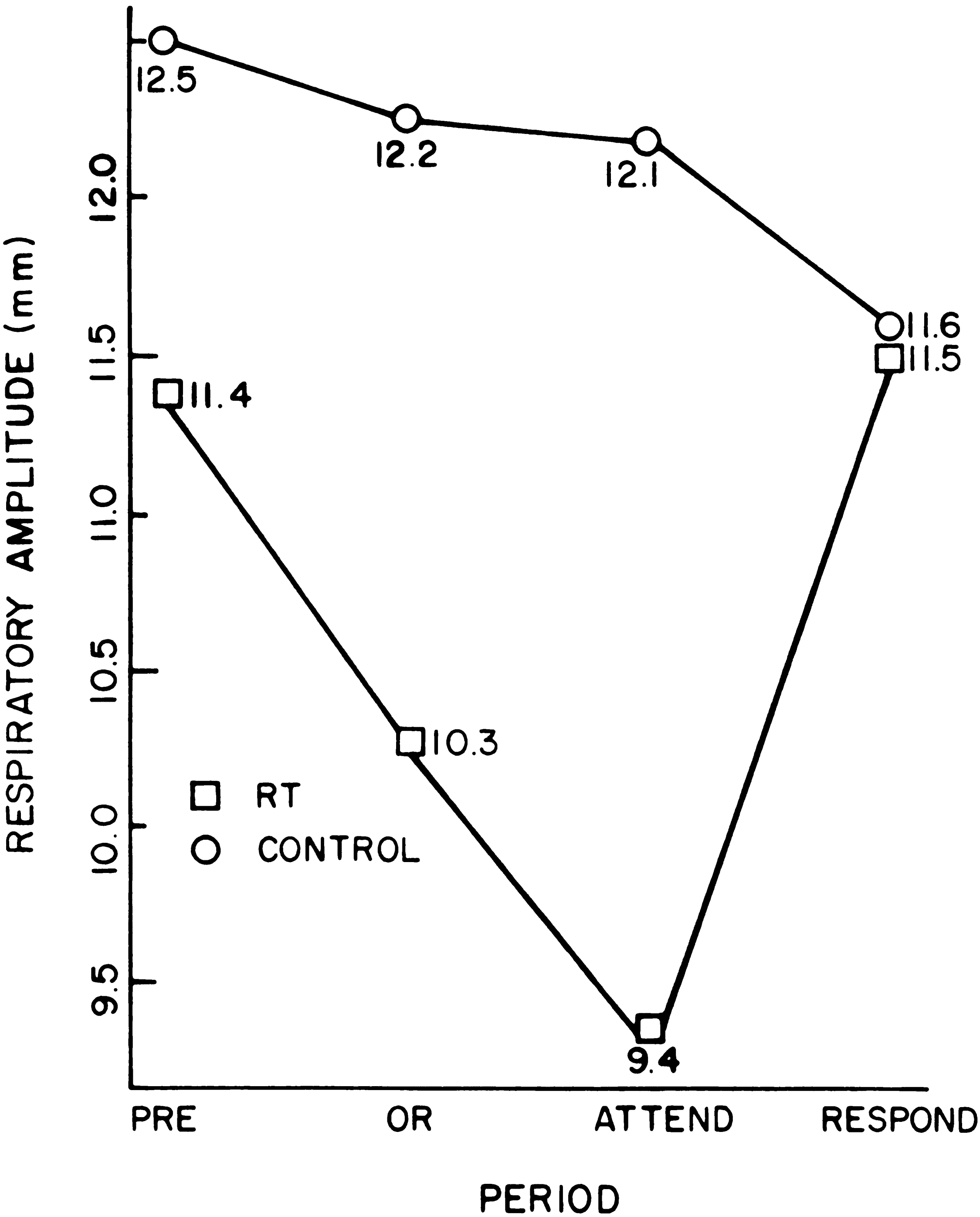

Fig. 2. Mean respiration amplitude as a function of periods for the two tasks.

period. The control task resulted in a gradual decrease in amplitude from the "pre" through the respond periods. A test for simple effects showed that significant differences among the four periods existed only for the RT task [$F(3, 132) = 9.25$, $p < .001$]. A Newman-Keuls test showed that the respiration amplitude during the attend period differed significantly from the "pre" and respond periods ($p < .05$). There was an over all decrease in mean amplitude from 12.7 on Trial 1 to 10.7 on Trial 10 [$F(9, 396) = 4.6$, $p < .0005$]. Confounding the trial effect were significant Task x Trial [$F(9, 396) = 2.7$, $p < .005$] and Trial x Period x Task [$F(27, 1188) = 1.7$, $p < .01$] interactions which statistically emphasized task specific adaptation of respiration amplitude across trials.

Mean heart rate. The data revealed that mean HR varied significantly among the four periods [$F(3, 132) = 18.8$, $p < .0005$]. The mean HR decelerated from the "pre" (76.05 beats/min) through the attend (75.13 beats/min) periods. This was followed by a substantial acceleration during the respond period (77.33 beats/min). Figure 3 illustrates the significant HR differences for the two tasks during the four periods [$F(3, 132) = 23.01$, $p < .0005$]. The RT groups showed two pronounced HR responses, deceleration during the attend period followed by acceleration during the respond period. The control groups, following a slight deceleration from the "pre" period, maintained approximately the same HR from the OR through the respond periods. A test for simple effects indicated that the mean HR differed significantly among the four periods only for the RT task [$F(3, 132) = 40.15$, $p < .0005$]. A Newman-Keuls test showed that the mean HR was significantly greater during the respond

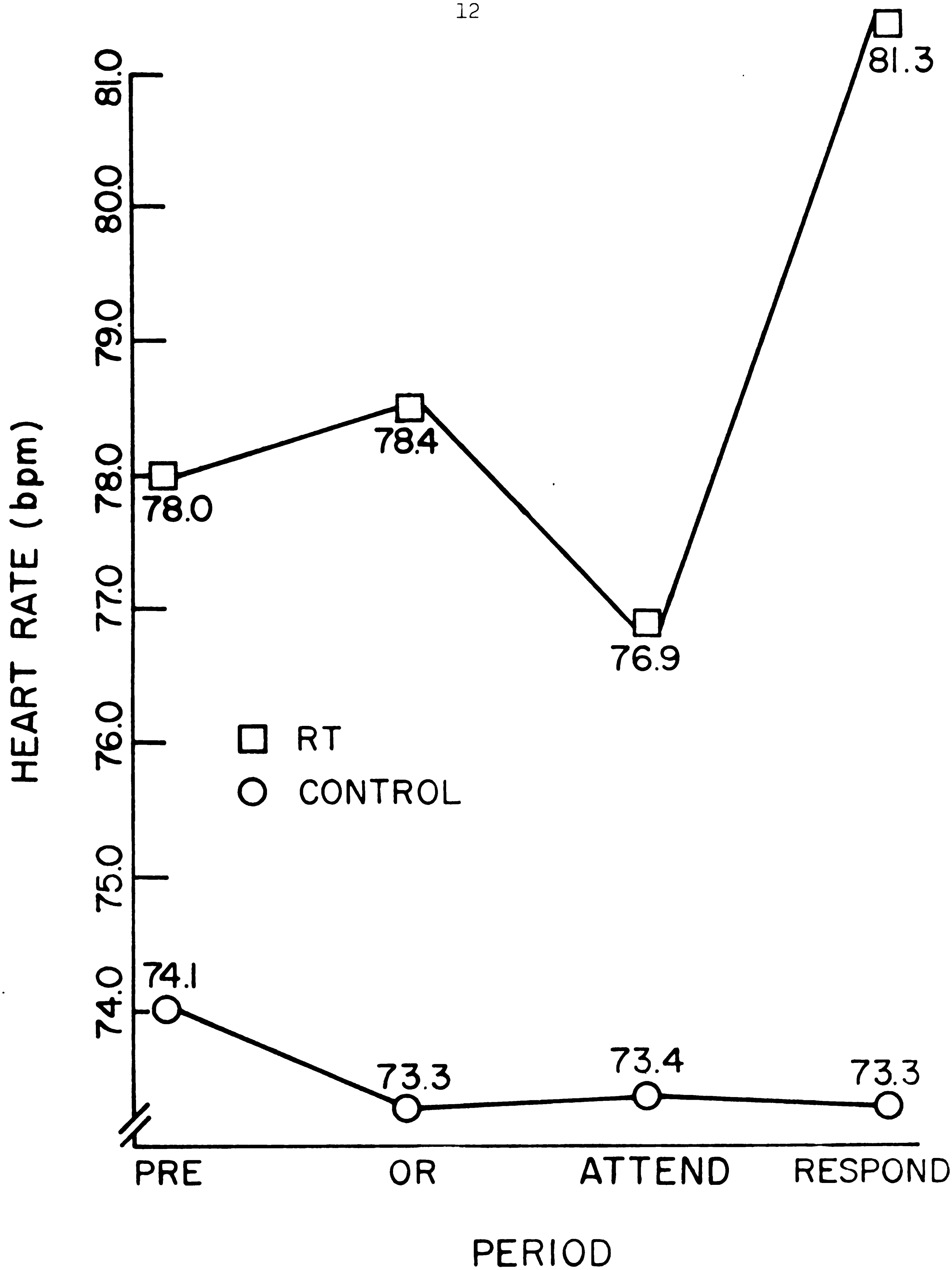

Fig. 3. Mean heart rate as a function of periods for the two tasks.

period than during the other periods, which did not differ ($p < .01$). There was an over all decrease in HR from 80.01 beats/min on Trial 1 to 75.12 beats/min on Trial 10. The controls exhibited only a very minute deceleration from 74.91 beats/min on Trial 1 to 73.12 beats/min on Trial 10. The reliability of this task difference was supported by a significant Task x Trial interaction [$F(9, 396) = 3.84$, $p < .0005$]. Across trials, the RT groups exhibited a greater reduction in HR during the respond period than during the OR period. This may be demonstrated by a comparison of Trial 1 means for the OR (83.11 beats/min) and the respond (91.09 beats/min) periods and Trial 10 means for the OR (78.27 beats/min) and the respond (78.42 beats/min) periods. This selective HR reduction during the respond period as a function of trials is substantiated by two significant interactions, Trial x Period [$F(27, 1188) = 2.61$, $p < .0005$] and Task x Trial x Period [$F(27, 1188) = 3.73$, $p < .0005$].

Heart rate variance. Heart rate variance significantly differentiates between the two tasks [$F(1, 44) = 8.84$, $p < .005$]. Heart rate was more variable for RT groups (13.29) than for controls (7.76). The data revealed significant differences in HR variance among the four periods [$F(3, 132) = 6.04$, $p < .001$]. However, the changes in HR variance among the periods differed significantly for the two tasks as illustrated in Fig. 4. For the RT task increases in HR variability followed the warning and respond signals, contrasting with a prominant decrease during the attend period. The controls also exhibited the least variability during the attend period, although there were no variability changes following the warning and respond signals. The reliability of these task differences was supported by a significant Task x Period interaction [$F(3, 132) =$

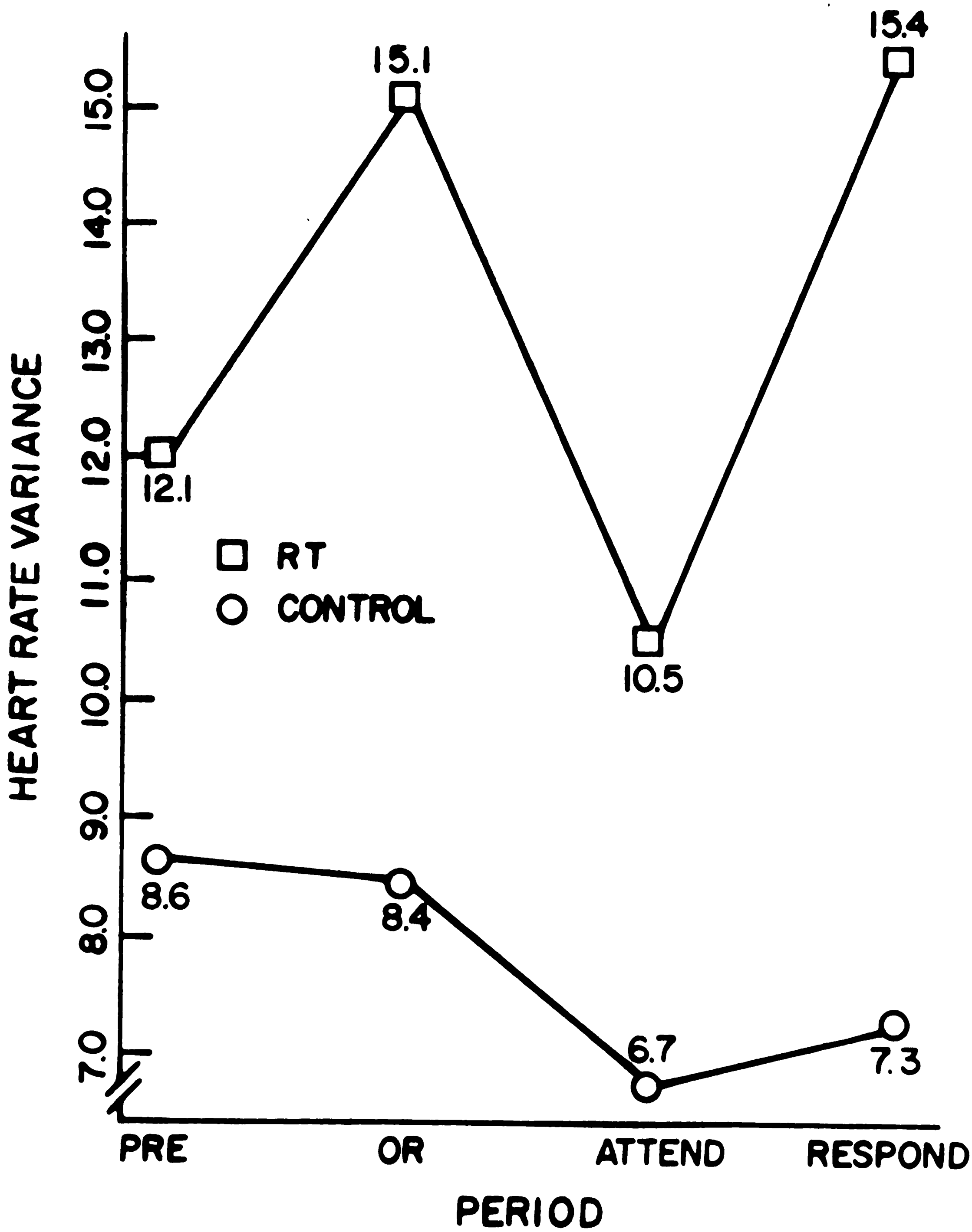

Fig. 4. Mean heart rate variance as a function of periods for the two tasks.

4.04, $p < .01$]. A test for simple effects showed that HR variance differed significantly among the four periods only for the RT task [$F(3, 132) = 8.81$, $p < .001$]. A Newman-Keuls test showed that the attend period differed significantly from the OR and respond periods ($p < .05$).

Second-by-second heart rate. The HR response during the PI was evaluated by a second-by-second analysis. For each S, HR during each second of the OR and attend periods was calculated as the magnitude of change in beats/min from the last second of the "pre" period. The second-by-second response pattern differed significantly between the two periods [$F(7, 308) = 10.09$, $p < .0005$]. There were, however, reliable differences in the shapes of the HR response during the PI for the four groups, reflecting differential task and schedule effects. This is statistically substantiated by a Schedule x Task x Period x Second interaction [$F(7, 308) = 2.62$, $p < .01$]. Figure 5 illustrates that the second-by-second HR response pattern for the groups with the fixed interstimulus interval exhibited more pronounced directional responses during the OR period. The fixed PI RT group showed the largest acceleration, while the fixed PI controls exhibited the largest deceleration. During the attend period the most prominant response occurred for the fixed PI RT group which exhibited an acceleration followed by a deceleration prior to the onset of the respond signal. The development of this biphasic response may be investigated by inspecting Fig. 6. Fig. 6 depicts the development of the second-by-second HR response during the attend period as a function of trials for the fixed PI RT group. The figure illustrates the temporal conditioning of HR deceleration prior to the respond signal. This conditioned deceleration contrasts with the relative stability over trials

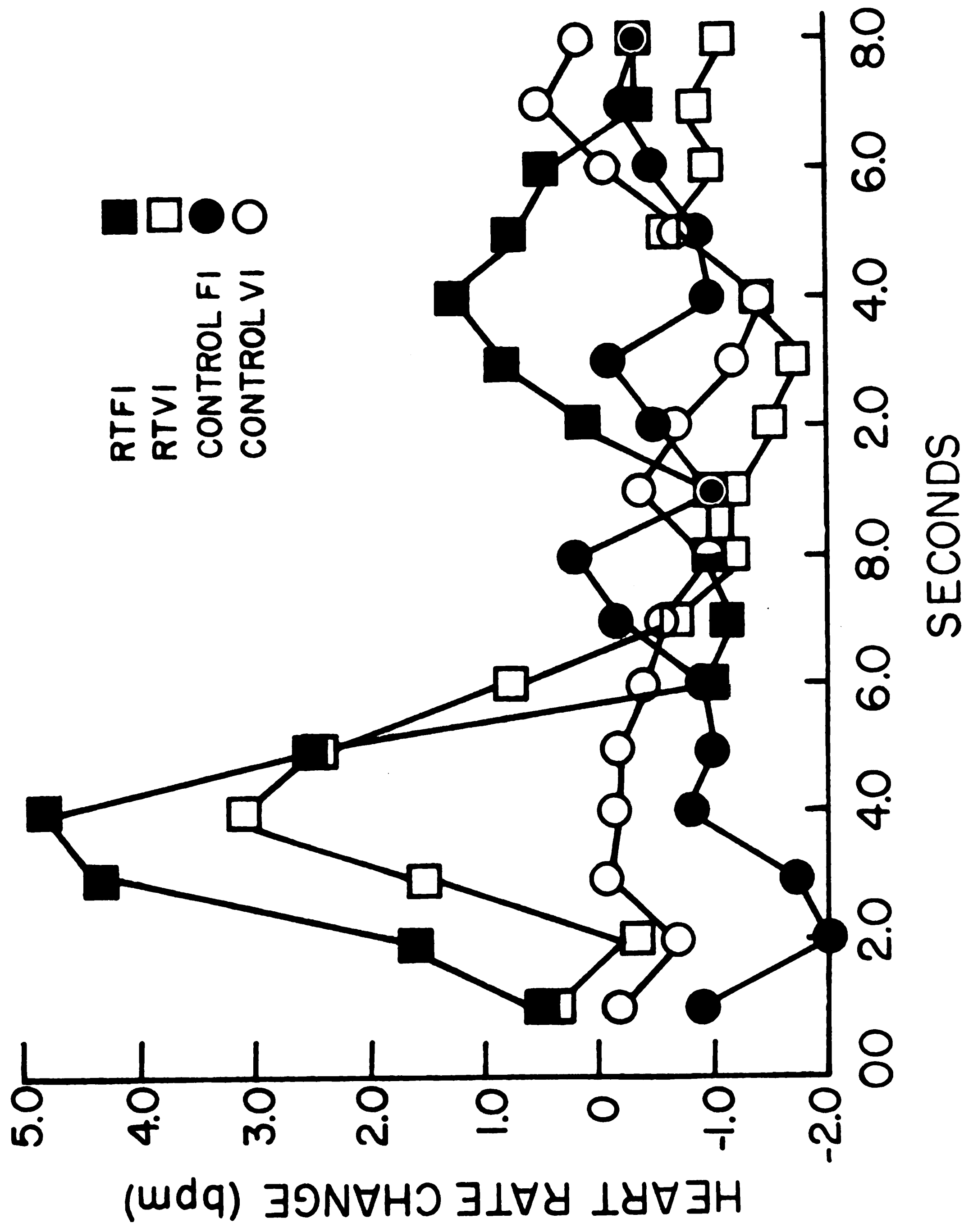

Fig. 5. Mean heart rate change during the preparatory interval as a function of seconds for each group.

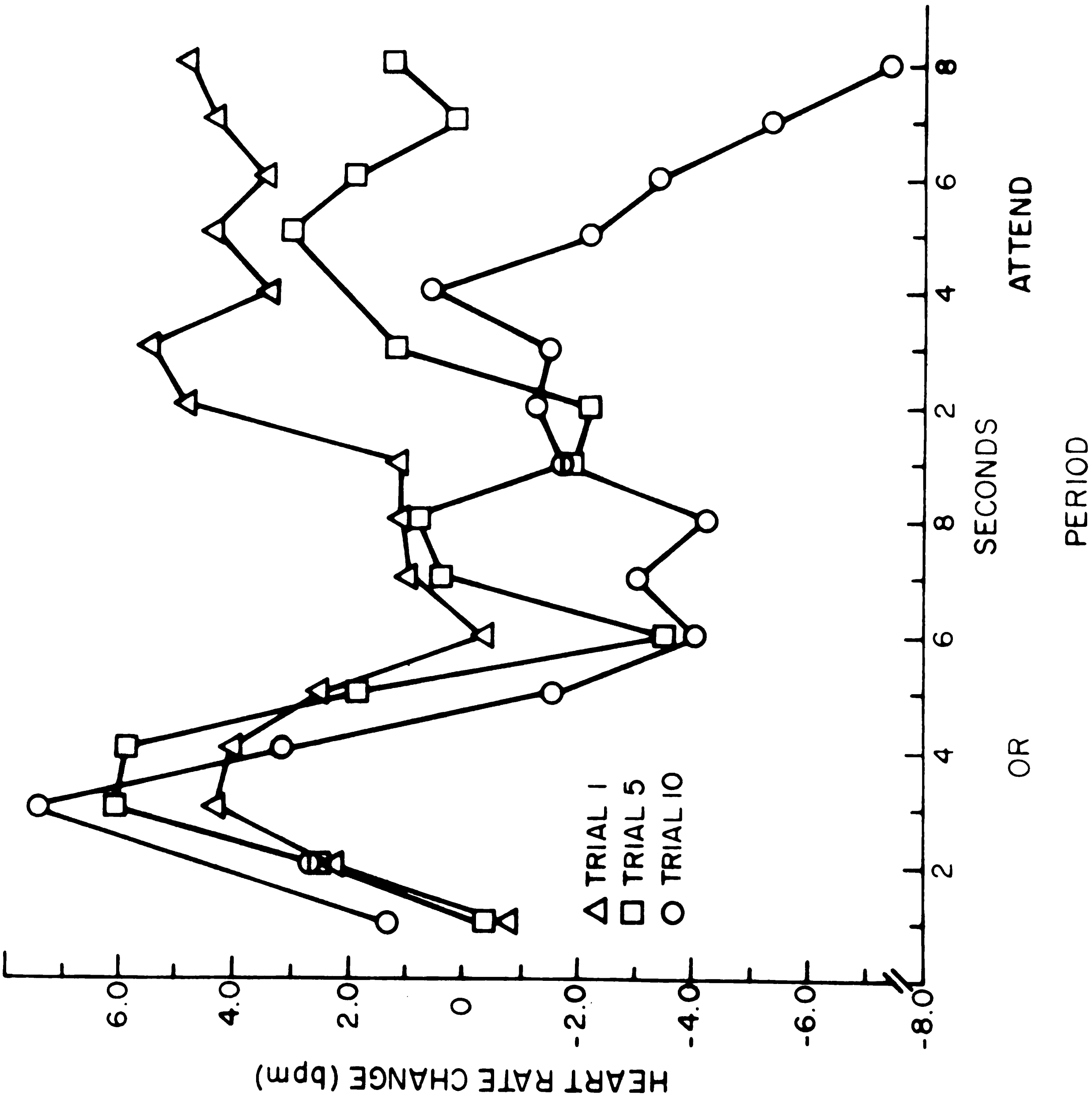

Fig. 6. Mean heart rate change during the preparatory interval during Trial 1, 5, and 10 for the fixed PI RT group.

of the biphasic response during the OR period. During the attend period on Trial 1 the HR accelerates, on Trial 5 the response appears biphasic, and on the last trial there is evidence of substantial deceleration. These observations suggest that the use of a fixed PI results in a temporally conditioned anticipatory HR deceleration. This notion is supported by a significant Schedule x Task x Trial x Period x Second interaction [$F(63, 2772) = 1.44$, $p < .025$]. When the data were analyzed as a function of task, significant Task x Period x Second [$F(7, 308) = 5.06$, $p < .0005$] and Task x Second [$F(7, 308) = 11.78$, $p < .0005$] interactions demonstrated that the second-by-second HR patterns differed in response to the two tasks. When the data were analyzed as a function of interstimulus schedule, significant Schedule x Second [F97, 308) = 2.35, $p < .025$] and Schedule x Task x Second [$F(7, 308) = 2.10$, $p < .05$] interactions emphasized the effect of the interstimulus interval on the second-by-second HR response pattern in contrast to the lack of differentiation on the previously presented HR and respiration measures.

Reaction time performance. Mean RT did not differ significantly between the fixed PI and the variable PI RT groups. Reaction time performance did improve as a function of trials [$F(9, 198) = 4.83$, $p < .001$]. Confounding this effect, as illustrated in Fig. 7, is the differential improvement of the two groups. The fixed PI group, although initially responding slower, exhibits consistently faster responses following Trial 3. This is supported statistically by a significant Schedule x Trial interaction [$F(9, 198) = 1.98$, $p < .05$].

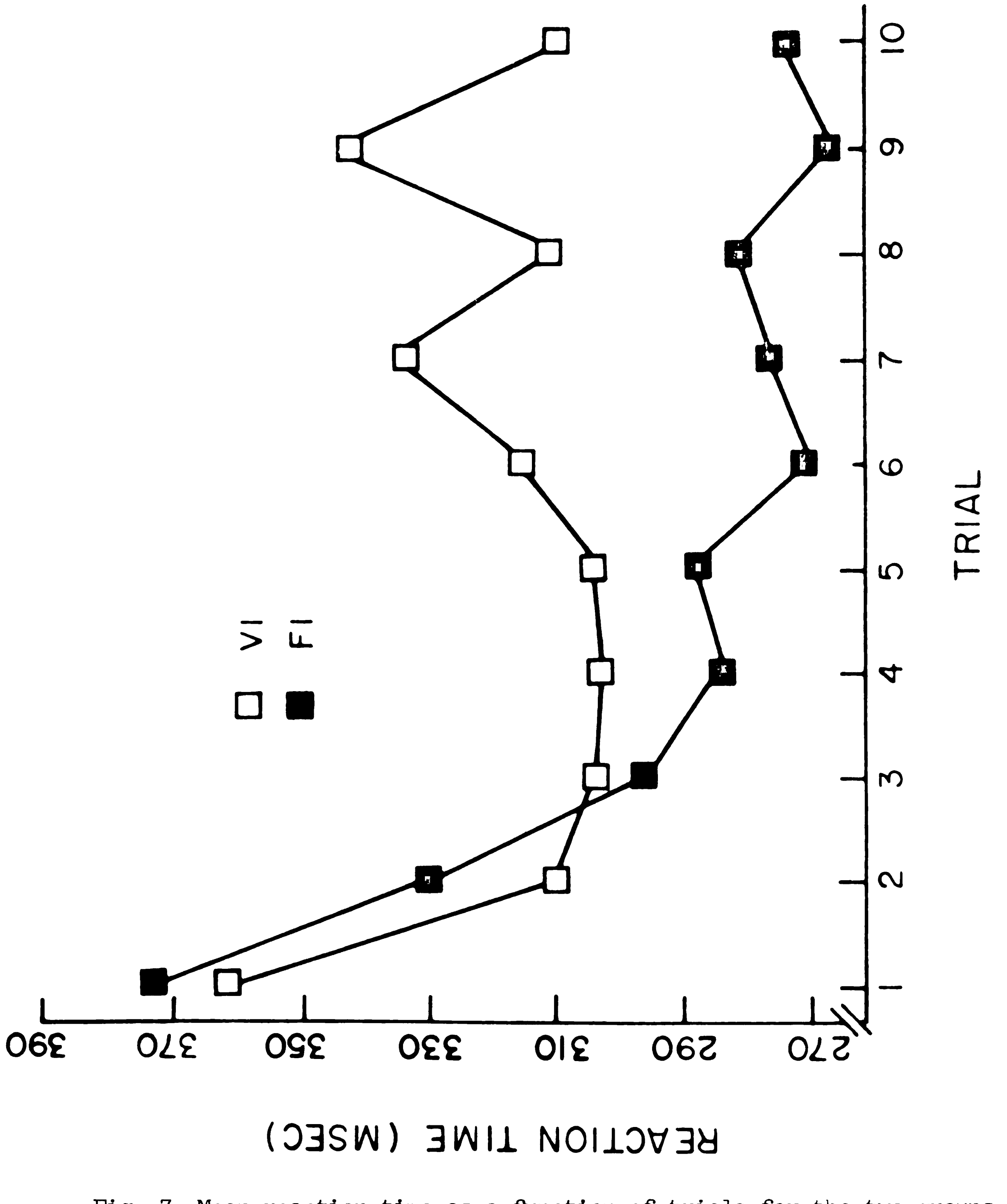

Fig. 7. Mean reaction time as a function of trials for the two groups.

Physiological indices of RT performance. The mean HR variance during the "pre" period was subtracted from the mean HR variance during the attend period for each S in order to generate an index of change in HR variability. When this index was correlated with RT, a significant relationship ($r = .417$, $n = 24$, $p < .05$) was demonstrated. This indicated that Ss exhibiting the greatest decrease in HR variance during the attend period tended to have faster RT. The mean respiration amplitude of the last complete inspiration during the attend period was subtracted from the first complete inspiration during the respond period to generate an index of change in respiratory amplitude in response to the respond signal. This index was used to relate the effect of changes in the depth of the respiratory cycle on RT performance. There was a significant correlation ($r = -.452$, $n = 24$, $p < .025$) between the magnitude of this respiratory change and RT. This indicated that there was an inverse relationship between the amplitude changes during the respond period and RT; the greater the increase in respiration amplitude following the respond signal, the faster the RT.

Since the previously described Analyses of Variance indicated that the schedule of the PI affected both RT performance and second-by-second HR responses, correlational analyses were conducted separately for each RT group between RT performance and the recorded physiological variables. Only one statistically significant relationship existed for the fixed PI group. This relationship, upon which the previously reported combined groups relationship is dependent, indicated that Ss who exhibited increased respiratory amplitude to the respond signal had faster RTs ($r = -.579$, $n = 12$, $p < .05$).

The relationships among the physiological variables and performance in the variable PI group were far more substantial. There was a highly significant correlation between the change in HR variance from the "pre" to attend periods and RT ($r = .717$, $n = 12$, $p < .01$). This strong relationship significantly contributed to the previously reported combined groups correlation. This concomitance indicates that the greater the decrease in HR variability from the "pre" to attend periods, the faster the RT.

There was a significant relationship between respiration frequency during the OR period and RT ($r = .574$, $n=12$, $p < .05$). This correlation indicated that the greater the frequency during the OR period the slower the RT. When the respiration amplitude during the "pre" period was subtracted from the OR period to correct for base level amplitude, there was a correlation between this amplitude difference and RT ($r = .572$, $n = 12$, $p = .052$).

A prominant relationship was uncovered between the level of HR variance during the "pre" period and RT ($r= -.774$, $n = 12$, $p < .005$). This highly significant correlation indicated that, independent of the HR variance reduction during the PI, the larger the mean HR variance prior to trial onset, the shorter the response latency or the faster the RT. A coefficient of multiple determination using the "pre" level HR variance and the HR variance change from the "pre" to attend periods as predictor variables indicated that 75% of the variance of RT was accounted for by these two HR variability measures.

Physiological intercorrelations. The mean HR, HR variance, respiration frequency, and respiration amplitude were calculated for each of the 48 Ss and mutually correlated. Mean HR and HR variance were statistically independent. There was a significant relationship between HR variance and respiratory frequency ($r = -.32$, $n = 48$, $p < .05$), indicating that the greater the respiration frequency the smaller the HR variance. Respiration frequency and amplitude were related ($r = -.34$, $n = 48$, $p < .05$), which indicated that an increase in frequency was generally accompanied by a decrease in amplitude.

Discussion

The findings may be summarized with respect to the respiratory and HR components of attention to RT signal and control nonsignal stimuli. The two stimulus conditions produced differential respiratory and HR responses. Heart rate accelerated in response to the warning and respond signals. The HR acceleration to the respond signal was concordant with increases in HR variability, respiratory frequency and amplitude. In contrast to the mean HR, which did not change in anticipation of the respond signal, respiratory frequency increased and respiratory amplitude and HR variability decreased. In response to the nonsignal stimuli HR decelerated.

In contrast to previous research (Porges & Raskin, 1969) respiratory responses were task dependent. In the present study, the RT task emphasized the importance of perceiving the onset and termination of the stimuli, juxtaposed to the passive attentional requirements of the control task during which the same stimuli were merely presented without necessitating a behavioral response. The experimental situation of the control task was similar to the conditions in the Porges and Raskin (1969) study in which Ss were not cognizant of a grading criteria for their performance and in which response speed, inherent in any RT task, was not emphasized. Thus, it is not surprising. that the responses during the control task replicated the patterns reported by Porges and Raskin. The respiratory response pattern during the RT task, therefore, is in conflict with previous reports (Lynn, 1966; Porges & Raskin, 1969). Lynn based his report on an experiment that used high intensity stimuli, Porges and Raskin used mild auditory and visual stimuli, and the present study used signal and nonsignal visual stimuli. The variety of results indicates that specific respiratory responses are dependent upon the signal or eliciting value of the stimulus.

In the present study, the HR variance decreased during the attend period for the RT groups. This replicated Porges and Raskin (1969) who demonstrated that the magnitude of HR variance reduction during attention was dependent upon the subject involvement necessitated by the task. The decrease in HR variance during the attend period coincided with increased respiration frequency and decreased respiration amplitude.

The decelerative second-by-second HR response during the OR period for the control groups was consistent with the findings of other investigators (Chase & Graham, 1967; Lacey, 1959; Porges & Raskin, 1969). The accelerative HR response during the OR period for the RT groups was supported by Obrist, Webb, and Sutterer (1969) who observed that the direction of the HR response was dependent upon the motivational significance of the signal. The HR decelerated during the attend period for the fixed PI RT group as a result of temporal conditioning. The temporally conditioned HR deceleration has been reported by numerous other investigators (Obrist, Webb, & Sutterer, 1969; Fitzgerald & Porges, 1970; Chase, Graham, & Graham, 1968).

The data support a two component hypothesis of attention: the first, a phasic reflexive response dependent upon the specific stimulus change and characterized by a directional HR response; the second, a tonic instrumental response related to attentional performance and characterized by a decrease in HR variability. These responses may be mediated by different neurophysiological mechanisms. Possibly, the phasic response is initiated at the brain stem level and dominated by diencephalic-limbic mechanisms, while the tonic response with its instrumental aspects may be initiated at the cortical level having the ability to inhibit the reflexive or instinctive responses.

The data in this study indicated that the tonic attention response was related to attentional performance. From these results, it has been clearly demonstrated that when the masking effect of temporal conditioning is avoided and the phasic reflexive response is removed by utilizing an extended variable PI, there is an extremely significant relationship between RT and the reduction of HR variability.

Obrist et al. (1969, 1970) have attempted to demonstrate that the greater the HR deceleration prior to the respond signal the faster the RT. In the present experiment, the HR during the last second of the "pre" period was subtracted from HR during the last second of the attend period to generate an index of HR deceleration to test Obrist's hypothesis. Contrary to Obrist's hypothesis, there was no significant relationship between the degree of deceleration prior to the respond signal and RT in either of the RT groups. Chase et al. (1968) also investigated the deceleration prior to the respond signal. They demonstrated that the HR deceleration prior to the respond signal was temporally conditioned. However, they imply that the the conditioned "attention" response is related to perceptual enhancement, inferring that faster RT would occur following conditioning. An inspection of the data in the present experiment reveals that this is not the case. There was no difference between the RT on Trial 5 and Trial 10, although the anticipatory deceleration took the full 10 trials to be conditioned. From these results and the data previously presented, the deceleratory response prior to the termination of a fixed PI must be interpreted merely as a temporally conditioned anticipatory response unrelated to performance. Consistent with this interpretation, it would not seem logical if conditioned HR deceleration were related to perceptual enhancement to report its occurrence in anticipation of noxious stimuli.

The intercorrelation of physiological responses demonstrated statistical independence between HR and mean HR variance. However, HR variance was influenced by respiration frequency. Respiration frequency was negatively correlated with HR variance. Moreover, respiration amplitude, although not correlated with HR variance, was negatively correlated with respiration frequency.

There was a pronounced negative correlation between "pre" trial HR variance and RT. Subsequent research (Porges, 1970) has indicated that HR variance may be used to predict future attentional performance. These findings are supported by observation by Lacey (1958) that autonomic variability was positively correlated with the length of time an individual could maintain maximal readiness to respond.

In summary, the results have: (a) distinguished phasic and tonic HR components of attention; (b) demonstrated respiratory responses related to attentional task demands; (c) demonstrated the relationship between the HR component of tonic attention and attentional performance; (d) distinguished between two decelerative HR responses, the orienting response and the conditioned anticipatory response; (e) substantiated the independence of HR and HR variance; and (f) identified base level HR variability as an extremely sensitive correlate of RT performance.

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APPENDICES

APPENDIX A

Instructions

Reaction time groups. The pickups I have attached will record changes in HR and in respiration. They are very sensitive to movement, so try not to make any unnecessary movements. Your task will be to watch the green light on the left of the panel appear and to press the button on the arm rest when the green light disappears. The speed at which you press the button will determine the length of time the red light will remain illuminated. This period of illumination will be your reaction time. Your task is to press the button as rapidly as possible when the green light goes off. Remember not to make any unnecessary movements and to respond as rapidly as possible when the green light disappears. You will receive one series of trials. When the white light goes off the experiment will begin. When the white light comes on again, it will designate the end of the experiment. At that time please remain seated until I come back and remove the electrodes. Do you have any questions? I am now going into the other room to calibrate the equipment, when I return I will close the chamber door. Remember to relax and to make as few body movements as possibile, especially of the arm and leg to which the electrodes are attached.

Control groups. The pickups I have attached will record changes in HR and in respiration. They are very sensitive to movement, so try not to make any unnecessary movements. Your task is simply to watch the lights on the panel in front of you, the green light on the left of the panel and the red light on the right of the panel. The red light will be illuminated momentarily when the green light disappears. A trial will consist of one presentation of the pair of lights. You will receive one series of trials. When the white light goes off the experiment will begin. When the white light comes on again, it will designate the end of the experiment. At that time please remain seated until I come back and remove the electrodes. Do you have any questions? I am now going into the other room to calibrate the equipment, when I return I will close the chamber door. Remember to relax and to make as few body movements as possible, especially of the arm and leg to which the electrodes are attached.

APPENDIX B

Analyses of Variance Summary Tables

Respiration frequency.

Source	df	MS	F
Schedule (S)	1	34.13	
Task (T)	1	45.63	
S x T	1	13.00	
Error	44	42.34	
Trial (Tr)	9	1.45	
S x Tr	9	1.12	
T x Tr	9	2.93	3.78****
S x T x Tr	9	0.82	
Error	396	0.77	
Period (P)	3	22.57	27.68****
S x P	3	0.89	
T x P	3	10.08	12.37****
S x T x P	3	0.26	
Error	132	0.82	
Tr x P	27	0.47	
S x Tr x P	27	0.65	
T x Tr x P	27	0.64	
S x T x Tr x P	27	0.66	
Error	1188	0.59	

**** $p < .0005$

Respiration Amplitude.

Source	df	MS	F
Schedule (S)	1	6.88	
Task (T)	1	1032.97	
S x T	1	76.12	
Error	44	730.06	
Trial (Tr)	9	125.95	4.59****
S x Tr	9	31.80	
T x Tr	9	75.30	2.75**
S x T x Tr	9	13.23	
Error	396	27.41	
Period (P)	3	114.99	4.42**
S x P	3	9.98	
T x P	3	159.20	6.12***
S x T x P	3	6.03	
Error	132	26.02	
Tr x P	27	36.00	
S x Tr x P	27	21.42	
T x Tr x P	27	44.60	1.75*
S x T x Tr x P	27	33.35	
Error	1188	25.53	

**** $p < .0005$

*** $p < .001$

** $p < .005$

* $p < .01$

Mean heart rate.

Source	df	MS	F
Schedule (S)	1	193.31	
Task (T)	1	12821.15	
S x T	1	540.04	
Error	44	5762.30	
Period (P)	3	403.52	18.76****
S x P	3	9.02	
T x P	3	494.83	23.01****
S x T x P	3	44.96	
Error	132	21.51	
Trial (Tr)	9	418.07	9.04****
S x Tr	9	43.80	
T x Tr	9	177.84	3.84****
S x T x Tr	9	62.00	
Error	396	46.26	
P x Tr	27	22.53	2.61****
S x P x Tr	27	12.77	
T x P x Tr	27	32.26	3.74****
S x T x P x Tr	27	8.63	
Error	1188	8.63	

**** $p < .0005$

<u>Heart rate variance.</u>

Source	df	MS	F
Schedule (S)	1	744.96	
Task (T)	1	14682.09	8.83**
S x T	1	275.74	
Error	44	1661.55	
Period (P)	3	933.99	6.04***
S x P	3	53.02	
T x P	3	625.17	4.04*
S x T x P	3	109.69	
Error	132	154.60	
Trial (Tr)	9	58.31	
S x Tr	9	140.05	
T x Tr	9	148.21	
S x T x Tr	9	128.02	
Error	396	157.30	
P x Tr	27	92.74	
S x P x Tr	27	113.93	
T x P x Tr	27	100.31	
S x T x P x Tr	27	84.88	
Error	1188	101.00	

*** $p < .005$

** $p < .001$

* $p < .01$

Second-by-second heart rate.

Source	df	MS	F
Schedule (S)	1	96.23	
Task (T)	1	667.82	
S x T	1	33.54	
Error	44	377.47	
Period (P)	1	438.97	7.62**
S x P	1	73.42	
T x P	1	597.47	10.38***
T x S x P	1	7.61	
Error	44	57.58	
Second (Se)	7	82.84	5.71****
S x Se	7	33.99	2.35*
T x Se	7	170.75	11.78*****
S x T x Se	7	30.39	2.10*
Error	308	14.50	
Trial (Tr)	9	434.17	
S x Tr	9	387.92	
T x Tr	9	358.01	
S x T x Tr	9	255.95	
Error	396	255.00	
P x Se	7	104.42	10.09*****
S x P x Se	7	15.55	
T x P x Se	7	52.39	5.06*****
S x T x P x Se	7	27.08	2.62**
Error	308	10.34	

P x Tr	9	113.60	4.26****
S x P x Tr	9	25.07	
T x P x Tr	9	42.57	
S x T x P x Tr	9	12.25	
Error	396	26.66	
Tr x Se	63	10.08	
S x Tr x Se	63	12.02	
T x Tr x Se	63	11.99	
S x T x Tr x Se	63	10.74	
Error	2772	11.70	
P x Tr x Se	63	8.42	
S x P x Tr x Se	63	9.86	
T x P x Tr x Se	63	11.46	
S x T x P x Tr x Se	63	15.08	1.44*
Error	2772	10.46	

***** $p < .0005$

**** $p < .001$

*** $p < .005$

** $p < .01$

* $p < .05$

Reaction time.

Source	df	MS	F
Schedule (S)	1	41003.20	
Error	22	14873.96	
Trials (Tr)	9	13144.91	4.83****
S x Tr	9	5382.44	1.98*
Error	198	2720.19	