Cardiovascular effects of paced respiration and selective attention¹

HIRAM E. FITZGERALD and STEPHEN W. PORGES, *Michigan State University, East Lansing, Mich. 48823*

The effects of paced respiration (PR) and attentive observation (ATT) on heart rate (HR) and finger-pulse amplitude (FPA) were investigated in adult female Ss. Although HR responses to trial onset were task dependent, accelerating for Group PR and decelerating for Group ATT, temporally conditioned anticipatory HR deceleration was obtained across tasks. Across trials, HR decelerated and FPA dilated, indicating autonomic habituation to the experimental conditions. Methodological implications for paced respiration research and FPA analysis were discussed.

Paced respiration is a technique used to investigate the relationship between respiratory and cardiovascular responses. During paced respiration, Ss pace their breathing rate to that of an external stimulus that is typically presented in the visual or auditory modality. Using this technique, Brener & Hothersall (1966) showed that high heart rates (HR) were associated with quick erratic breathing, while low HRs were associated with normal breathing. Hnatow & Lang (1965) observed a "weak relationship" between respiratory cycle time and HR variability. Porges & Raskin (1969) found that, although the direction of the HR response appears to be independent of respiratory responses, HR variability decreases concomitantly with increases in respiratory frequency and decreases in respiratory amplitude.

Previous research has suggested that the direction of the HR response is task dependent: HR acceleration accompanying cognitive tasks and HR deceleration accompanying perceptual tasks (Lacey, 1959; Porges & Raskin, 1969). Most studies that have used the paced-respiration technique have not controlled for the effects of selective attention to the pacing stimulus. Thus, it is not clear whether the effects of paced respiration are due to S's attending to the pacing cue or to the requirements of the pacing task, i.e., are these effects task dependent? The first purpose of the present study was to investigate the effects of paced respiration and selective attention on two cardiovascular responses that are strongly influenced by psychological processes: HR and finger-pulse amplitude (FPA).

The use of a fixed intertrial interval (ITI) represents the Pavlovian procedure for demonstrating temporal conditioning, the fixed ITI serving as a temporal conditional stimulus (CS). Fitzgerald, Lintz, Brackbill, & Adams (1967) reported successful temporal conditioning of an autonomic response (pupillary reflex) in human infants, while Lipsitt & Ambrose (1967) obtained temporal conditioning of an autonomic (HR) and a somatic response (body activity). Lockhart (1966) temporally conditioned GSR with adult Ss, and Gantt (1946) reported successful temporal conditioning of adult HR. It would appear that both somatically mediated and autonomically mediated responses are subject to temporal conditioning. But the picture is not that clear. For example, Brackbill, Lintz, & Fitzgerald (1968) failed to obtain temporally conditioned eyeblinking in infants, and Abrahamson, Brackbill, Carpenter, & Fitzgerald (in press) were equally unsuccessful in their attempt to temporally condition another somatic response, sucking. The second purpose of the present study was, therefore, to investigate the effects on HR and FPA of presenting stimuli in a fixed temporal sequence using the methods of paced respiration and selective attention.

SUBJECTS

Sixteen female volunteers from introductory psychology courses at Michigan State University received extra course credit for serving as Ss. As they appeared at the laboratory, they were assigned to experimental conditions according to a predetermined random schedule.

APPARATUS

Stimuli were produced by a modified Crystal Lab Metronoma that presented a neon light at the rate of 32 flashes/min. The neon light from the Metronoma was mounted on a panel that was positioned in front of S. The duration of each trial and the ITI were programmed and presented by means of magnetic tape (Porges & Fitzgerald, in press). Ambient noise level of the experimental room was 40 dB, room temperature was 70°F. To mask background noise, 60-dB white noise was played through earphones placed on each S. Physiological responses were continuously recorded on a Beckman Type RS Dynograph at a paper speed of 5 mm/sec. EKG recording sites were cleaned with 70% ethanol prior to application of the electrodes. HR was

measured from EKG Lead II using a Beckman 9857 cardiometer. Finger vasomotor activity was measured with a photoelectric plethysmograph. The pickup consisted of a block of black phenolic plastic in which were mounted a Clairex CL 704 photocell and a General Electric 683 miniature lamp powered by a 3-V battery. The pickup was mounted flush to the tip of the finger by means of a plastic clamp. This arrangement held the surface of the pickup securely against S's fingertip with relatively light, constant pressure, which prevented discomfort to S and occlusion of the local blood vessels. The signal from the plethysmograph bridge was recorded ac.

PROCEDURE

The Ss were assigned randomly to one of two groups, eight Ss per group, Ss in the paced-respiration group (PR) were instructed to pace their breathing rate at 16 respiration cycles per minute during each trial by inhaling and exhaling in rhythm with the flashing light. Ss in the attentive-observation group (ATT) were instructed to count the number of light flashes during each trial. Each S received 10 trials. The ITI for each experimental group was a constant 20 sec. The duration of each trial presentation ranged from 10 to 20 sec, with a mean of 15 sec.

DATA QUANTIFICATION

The responses during each of the 10 trials were scored for HR and FPA. Each trial was divided into two periods: a preperiod, consisting of the first 10 beats prior to stimulus onset, and an evoked period, consisting of the first 10 beats following stimulus onset. HR was obtained from the beat-by-beat readout of the cardiometer, which transformed the R-R intervals into HR. HR and FPA responses were evaluated by a comparison of means, variances, and beat-by-beat analysis during both periods. FPA was measured beat by beat in terms of millimeters of pen deflection for each pulse and corrected for amplifier gain.

RESULTS

Heart Rate

Combining the 20 beats of both periods, there was a significant decrease in mean HR as a function of trials [$F(9,126) = 3.7, p < .005$]. Although the two tasks produced no differential effects on mean HR ($F < 1$), Group PR exhibited mean HR acceleration, while Group ATT exhibited mean HR deceleration to stimulus onset. The reliability of this group difference was supported by a significant Group by Period interaction [$F(1,14) = 4.9, p < .045$]. The HR response during the two periods was evaluated by a beat-by-beat analysis. Figure 1 illustrates the mean HR of each beat of both periods as the magnitude of change in beats per minute from the last

Fig. 1. Mean heart-rate change during the preperiod and evoked period as a function of beats for Group PR and Group ATT.

beat of the preperiod. Although the two groups did not show significant differences in the beat-by-beat patterns, there was a significant difference between the shapes of the HR pattern for the two periods [$F(9,126) = 2.2$, $p < .025$], indicating significant anticipatory HR deceleration. Moreover, as illustrated in Fig. 1, HR variability was significantly greater in Group PR [$F(1,14) = 5.37$, $p < .05$].

Finger Pulse Amplitude

There was a significant increase in FPA as a function of trials [$F(9,126) = 3.1$, $p < .005$]. The tasks produced no differential effects on mean FPA ($F < 1$). However, Group PR exhibited vasoconstriction to stimulus onset, while Group ATT exhibited vasodilation to stimulus onset. Although not statistically significant, this was demonstrated by the direction of the Group by Period interaction [$F(1,14) = 3.6$, $p < .08$].

DISCUSSION

Assuming that sequential counting of light flashes required Ss in Group ATT to attend to the external stimulus, and that the task requirements were indeed different for the two groups, the results of the present study support the research cited above by indicating that the direction of the HR response is task dependent, i.e., Ss in the paced respiration group evidenced cardiac acceleration to stimulus onset, while those in the selective-attention group evidenced cardiac deceleration. Although the response to trial onset was task dependent, the anticipatory HR deceleration more specifically appears to be a function of the fixed ITI (or more explicitly, the temporal CS), with the resulting responses reflecting temporal conditioning. This is congruent with the decelerative HR response preceding CS presentation found by Hastings & Obrist (1967) in their study of interstimulus-interval effects on conditioned heart rate.

There was no evidence for temporally conditioned FPA. Nevertheless, considering the qualitative nature of photoplethysmographically recorded FPA, one cannot conclude that temporal conditioning of FPA is not possible. Since summated data characteristic of between-groups designs tend to mask individual differences, a nonparametric design emphasizing the direction of individual differences may be more appropriate for evaluating this response.

In agreement with physiological evidence, psychophysiological research has shown the finger vasomotor response to be

sympathetically innervated nonspecific constriction, i.e., vasomotor control of cutaneous blood flow is solely constrictor (Gaskell, 1956; Roddie, Shepherd, & Whelan, 1957). Since this peripheral vasomotor constriction is independent of type of stimulation, the increase in FPA following stimulus onset observed in Group ATT might easily be inappropriately interpreted as vasodilation. An alternative interpretation would suggest that for Group ATT, stimulus onset marked the point at which FPA anticipatory constriction terminated. Thus, FPA vasodilation observed at stimulus onset more accurately represents inhibition of preperiod sympathetic anticipatory vasoconstriction, i.e., a release of sympathetic tone.

Group differences in HR variability can be attributed to task requirements, paced respiration increasing sinus arrhythmia and its effect on HR fluctuation. The significant decrease in HR and increases in FPA may be attributed to habituation to the experimental situation and are consistent with previous research (Porges & Raskin, 1969).

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NOTE

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