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Does the Heart Know What the Eye Sees? A Cardiac/Pupillometric Analysis of Motor Preparation and Response Execution

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ABSTRACT

Autonomic response measures are well suited for the study of preparation because they allow the analysis of covert aspects of performance. This is illustrated by an experiment in which task-evoked cardiac and pupillary responses were compared during a disjunctive (Go/No Go) reaction task. The motoric demands of the task were varied by manipulating foreperiod length (4 and 8 s) and probability of response (25%, 50%, and 75%). Reaction time increased with foreperiod length and decreased with probability of response. The depth of anticipatory heart rate deceleration was affected only by foreperiod length. Analysis of the beats during, and directly preceding and following the imperative stimulus revealed that interbeat intervals increased with probability of responding and foreperiod duration. The effect of stimulus timing relative to the R-wave of the ECG was also analyzed. Early occurring stimuli prolonged the cycle of their occurrence more than late occurring stimuli. The cycle time effect was somewhat more pronounced for No Go stimuli than for Go stimuli. The subsequent cycle was shorter for early occurring stimuli compared to late stimuli. This effect was stronger for Go compared to No Go trials. Both Go and No Go reactions elicited significant pupil dilations. The No Go dilation peaked earlier than the Go dilation and its amplitude was smaller. Probability of responding affected the latency of the No Go dilation but not that of the Go dilation. The current results justify an interpretation of preparation in terms of a timing mechanism (indexed by heart rate deceleration during the foreperiod) and a mechanism allocating processing resources to stimulus encoding (indexed by cardiac slowing just prior to stimulus occurrence) and response preparation (indexed by continued cardiac deceleration and pupillary dilation).

DESCRIPTORS: Cardiac deceleration, Pupil dilation, Reaction time, Motoric activation, Processing demands.

Heart rate/reaction time studies using a two-stimulus paradigm (i.e., warning and reaction stimulus) typically obtain a heart rate deceleration that reaches its nadir at some point in time near the stimulus and motor response (Bohlin & Kjellberg, 1979). Anticipatory heart rate deceleration is usu-

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ally considered to index preparatory processes related to stimulus detection and response execution (Lacey & Lacey, 1974; Coles & Strayer, 1985). This interpretation is supported by the finding of a weak but consistent association between deeper decelerations and faster motor responses (Jennings & Hall, 1980). More recently, Lacey and Lacey (1977, 1980) introduced a procedure that focused on a single cardiac cycle. They sorted trials, post hoc, on the basis of the R-wave-to-stimulus interval, and observed that early occurring stimuli (i.e., short R-wave-to-stimulus intervals) are associated with longer cycles compared to late occurring stimuli (i.e., long R-wave-to-stimulus intervals). The latter prolong the cycle following the cycle in which the stimulus is presented. Lacey and Lacey called this cardiac cycle time effect "primary bradycardia." Most authors assume that primary bradycardia reflects the encoding of the stimulus. The more resources are allocated to encoding, the stronger the postulated cardiac cycle time effect (Coles & Strayer, 1985).

In previous studies we investigated the effects of foreperiod manipulations on anticipatory deceleration and primary bradycardia (Jennings, van der Molen, & Terezis, 1988; van der Molen, Somsen, Jennings, Orlebeke, & Nieuwboer, 1987; van der Molen, Somsen, & Orlebeke, 1983). In one of those studies the reaction stimulus followed the warning stimulus either by a fixed 6 or 12 s foreperiod or by a variable 6, 9, or 12 s foreperiod (van der Molen et al., 1987). The results showed that when the subject knows in advance that the stimulus will be presented after 12 s (i.e., in the fixed foreperiod condition) heart rate deceleration is maximal at some point in time near the stimulus. In contrast, when the subject is kept uncertain about the time of stimulus occurrence (i.e., in the variable foreperiod condition), heart rate deceleration reaches its nadir after about 9 s and then levels off until the stimulus is presented. These results clearly indicate that subjects do not passively await the stimulus. If they did so, anticipatory deceleration would reach its maximal amplitude at the time of the stimulus both in the fixed and the variable foreperiod conditions. The levelling off of anticipatory deceleration at about 9 s in the variable foreperiod condition strongly suggests that subjects actively target their responses at the expected time of stimulus occurrence (i.e., at about the medium length of the variable foreperiod interval).

The view that anticipatory deceleration is associated with temporal prediction was supported by data obtained in another study in which subjects saw a LED display count down from either 5 or 1 s to a reaction time signal (Jennings et al., 1988). In both conditions, heart rate did not decelerate until just prior to the reaction stimulus suggesting that when there is no need for active prediction of the time of stimulus occurrence sustained anticipatory heart rate deceleration disappears. Even more importantly, the results of these studies suggest that heart rate deceleration during the time-interval of the foreperiod may actually consist of two components—sustained anticipatory deceleration associated with event timing, and brief, phasic, deceleration associated with the preparation to detect the stimulus and execute a motor response.

Turning to phasic, event-related changes, phase-dependent deceleration (i.e., primary bradycardia) has been studied by manipulating the significance of the imperative stimulus using a signalled choice reaction time task (van der Molen et al., 1983). Stimulus significance was manipulated by the information given at the warning signal. On some trials the warning stimulus indicated to the subject to withhold the motor response to the upcoming imperative stimulus (signaled No Go trials). Informing the subject not to respond to the imperative stimulus should deprive this stimulus of its significance. On other trials subjects were kept uncertain whether a response to the next imperative stimulus was required or not (Go and unsignalled No Go trials, respectively). Thus, although signalled and unsignalled No Go trials differ with respect to stimulus significance, unsignalled No Go and Go trials differ with respect to the motor requirements at the imperative stimulus. The results showed primary bradycardia for Go and unsignalled No Go trials but not for signalled No Go trials, suggesting that only significant stimuli induce phase-dependent cardiac deceleration.

Recently, Richer, Silverman, and Beatty (1983) recorded the pupillary response in a similar type of task and reported that both Go and (unsignalled) No Go signals elicit pupillary dilations. More important, however, the probability of Go stimuli in a block of trials modifies the pupillary response on No Go trials but not on Go trials. When stimulus frequency was biased toward Go reactions, No Go dilations increased. In contrast, Go dilations remained very similar for probabilities varying from 33% to 66%. The authors suggested that this pattern of results indicates that the task-evoked pupillary response is associated with motoric preparation processes. More specifically, the absence of a probability effect in Go trials may indicate that the dilation to Go reactions represents a maximum level for the motoric processing demands of the task. No Go reactions could be conceived as evoking a fraction of the maximal response—the proportion elicited depending on the completeness of the prepa-

ratory motor process performed (Richer et al., 1983, p. 369).

The current study relates the van der Molen et al. (1983) study investigating cardiac responses in a signalled Go/No Go task with the Richer et al. (1983) study investigating the pupillary response in a similar task. Basically, we wanted to integrate the previous findings suggesting that the task-evoked cardiac response consists of at least three distinct components—anticipatory deceleration associated with temporal prediction, added deceleration just prior to the stimulus associated with preparation for stimulus detection and response execution, and cycle time-dependent deceleration elicited by significant stimuli. Subjects performed a signalled choice reaction time task under different foreperiod and Go probability conditions. Task-evoked pupillary and cardiac responses were measured during task performance. Anticipatory heart rate deceleration associated with temporal prediction was expected to increase as a function of foreperiod duration (van der Molen et al., 1983) whereas pupil dilation was not expected to be affected by longer foreperiods (Richer et al., 1983). The pupillary response on No Go trials but not on Go trials should increase when a Go response is more likely (Richer et al., 1983).

The main purpose of the current research is to investigate whether cardiac deceleration just prior to the stimulus parallels the pupil response; that is, to assess whether this response is affected by Go probability but not by foreperiod duration and whether this dependency, as for the pupil response, is seen only for No Go trials and not for Go trials (Richer et al., 1983). Van der Molen et al. observed a somewhat stronger deceleration and more pronounced primary bradycardia for No Go trials compared to Go trials. The current research should establish whether this difference is altered as a function of Go probability. In that case primary bradycardia would also reflect processes that occur later than stimulus encoding—most probably motoric activation (e.g., Richer et al., 1983).

Method

Subjects

The subjects were 15 male students aged 21–27 years from the department of dental surgery at a local university. All had good eyesight and had already participated at least once in a similar kind of experiment. They were paid Dfl 60.00 (approximately $30.00) for their participation.

Apparatus

Testing took place in a sound attenuating room with normal incandescent lighting. The subject sat at a table with his head positioned in a chin rest and fixated a central location on a television screen at a distance of 3.5 m. In this situation, the pupillary accommodation reflexes were generally relaxed, establishing a stable baseline from which task-evoked changes in pupillary diameter could be measured. Visual stimuli were presented at the central location on the television screen. Auditory stimuli were binaurally presented through padded earphones with rise and decay times of 5 ms or less. The subject operated two conventional reaction time keys with the index fingers of his left and right hands. The keys were connected to microswitches that required a pressure of 150 g to traverse 2 mm. Presentation and duration of the stimuli was controlled by an Apple II microcomputer. This computer also sent the reaction time and pupillary data to a DEC PDP11/44 computer. Reaction times were measured in ms and stored on disk.

Pupillary diameter was recorded using a Whittaker pupillometer. The output of the pupillometer was digitized at 8 bits with a sampling frequency of 50 Hz. Pupillary diameter was recorded during each trial. Data from individual trials were stored on disk. The pupillometer was turned off between trials so that the exposure of the subject's eye to the infrared light was not longer than necessary.

The ECG electrodes were placed on the sternum and lateral margin of the chest. The ECG signal was led through a Beckman polygraph. The ECG, trial onset, and subject's motor responses were recorded on magnetic tape using a Bell and Howell recorder. Time between successive R-waves and between R-wave and stimulus onset were read off-line by an LSI 11/23 computer to 1-ms accuracy. For the heart rate analysis, the R–R intervals were converted into beats per minute using a moving weighted average (e.g., Cheung & Porges, 1977). For each successive 500-ms window, the weighted heart period was calculated as the sum of each heart period occupying that window, multiplied by the proportion of the window that it occupied. To study cardiac cycle time effects, the R–R intervals were sorted into thirds of the cycle in which the imperative signal happened to fall. Respiration was recorded to ensure that changes in interbeat interval were not due to major respiratory maneuvers.

Task and Procedure

A signalled choice reaction time task with varying foreperiod and probability of responding was used. The beginning of each trial was marked by an "attend" signal telling the subject to fixate the central location on the television screen without eye blinking. The attend stimulus was the appearance of a visual display on a television screen. The attend stimulus was followed 3 s later by a warning signal (Ws) which was a change in the visual display as shown in Figure 1. The light intensity of the two stimuli was the same and both stimuli were constructed in such a way that when the subject fixated the central location on the television screen all information fell on the fovea. The imperative stimulus (Is) consisted of a change in the visual

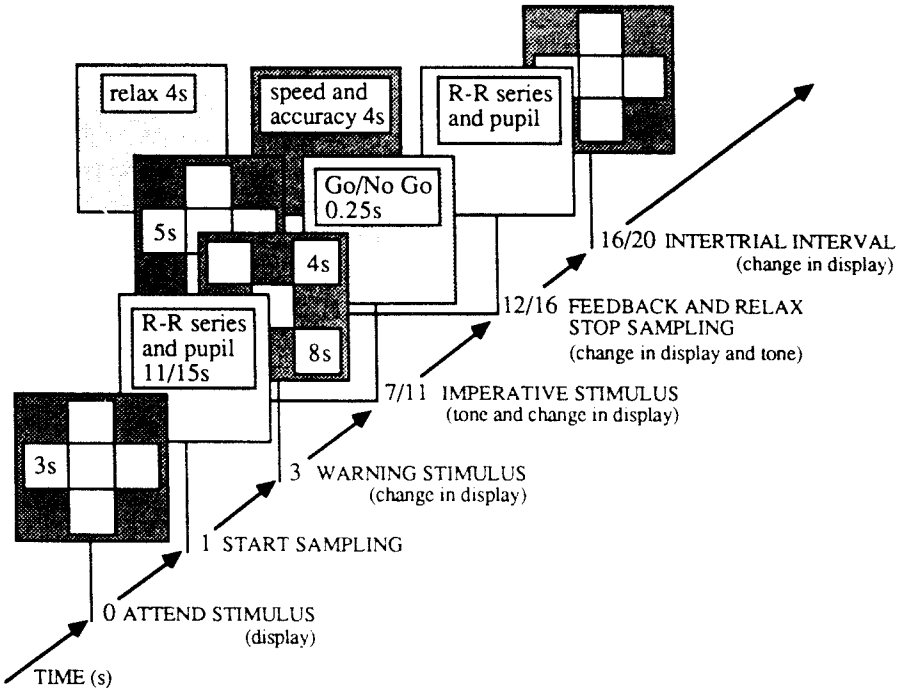

Figure 1. Schematic representation of the sequence of events during one trial. The dark, middle, and light grey squares represent the visual input channel, the auditory input channel, and the physiological output channel, respectively. The duration of the events is presented in the small white squares and their timing is given along the arrow axis. See text for further details.

display and the presentation of a tone of 60dB(A) with a duration of 250 ms. On Go trials a 1000 Hz tone signalled a left hand button press and a 3000 Hz tone signalled a right hand button press (or vice versa). On No Go trials, a 2000 Hz tone indicated that the motor response was to be withheld. Five seconds after the onset of the imperative stimulus, a "relax" signal (500 Hz tone) indicated that the subject could move his head and blink his eyes. This tone was heard for 4 s. During this time the subject was informed whether his response was correct and, in case the response was correct, also the reaction time. The intertrial interval (i.e., the time interval between successive attend signals) was either 16 or 20 s. The sequence of events for one trial is presented in Figure 1.

Foreperiod length (i.e., the Ws–Is stimulus interval) was either 4 or 8 s. Go probability (i.e., the percentage of Go trials in a block of trials) was either 25%, 50%, or 75%. Foreperiod length (2) and Go probability (3) were presented in six separate blocks of 80 trials. Within blocks the order of Go and No Go trials was randomized with the constraint that no more than 4 trials of the same kind would follow each other. Before each block the subject was informed about foreperiod length and Go probability. The order of blocks was randomized with no blocks of equal Foreperiod or Go probability following each other. The subject completed the task on 3 consecutive days of testing. He was asked not to consume alcohol or use drugs on the evening before the experiment and not to drink coffee or tea before coming to the laboratory. On the first day subjects underwent a training session consisting of approximately 300 trials. On the second and the third days subjects received 3 blocks of 80 trials each day. Each block of trials was preceded by 4 warm-up trials which were discarded in the data analysis. Instructions emphasized both speed and accuracy.

Results

Performance

Mean reaction time was 478 ms in the short foreperiod condition and 501 ms in the long foreperiod condition. This effect was significant at the $p<.05$ level, $F(1/14)=6.2$. All other effects reported below were significant at the .05 level or better, unless stated otherwise. The degrees of freedom of repeated-measures factors were adjusted by the Greenhouse and Geisser (1959) correction. The effect of Go probability was also significant, $F(2/28)=6.7$. Mean reaction time values for the 25%, 50%, and 75% Go probability conditions were 491, 504, and 473 ms, respectively. On No Go trials, there was an increase in errors of commission with increasing Go probability from 6.8% to 14% to 26.2%, $F(2/28)=36.7$. Go probability did not systematically affect the number of errors on Go trials, $F(2/28)<1$.

Heart Rate

In the analysis of the physiological data only those trials were used in which the subject pressed the correct button within acceptable speed limits (within two standard deviations). Heart rate data were obtained for each of the 480 experimental trials from 2 s before warning signal (Ws) onset until 5 s after imperative stimulus (Is) onset. Figure 2 depicts the average task-evoked heart rate response for all Probability × Foreperiod × Go/No Go combinations. All waveforms are characterized by a marked deceleration during the Ws–Is foreperiod. Neither Go probability nor the Go versus No Go conditions seem to affect the waveforms to a great extent. The only apparent difference might be a somewhat slower shift from anticipatory deceleration to acceleratory recovery on No Go trials compared to Go trials. The amplitudes of anticipatory deceleration (i.e., the heart rate value at the 0.5 s just prior to the imperative stimulus) and acceleratory recovery (i.e., the maximal heart rate value after the imperative stimulus) were subjected to ANOVA. The effects of experimental conditions were analyzed relative to a prewarning baseline; i.e., the mean of the four data points preceding the warning signal (Bohlin & Kjellberg, 1979). The prewarning baseline did not differ between conditions. Foreperiod length was the only significant effect. Anticipatory deceleration increased from −4.79 to −5.45 bpm with the increase in foreperiod, $F(1/14)=32.3$. Acceleratory recovery decreased with increasing foreperiods from −0.67 to −1.47 bpm, $F(1/14)=13.2$.

Interbeat Intervals

In order to examine more fully the time course of events around the imperative stimulus, analyses were performed on the interbeat interval (IBI) dur-

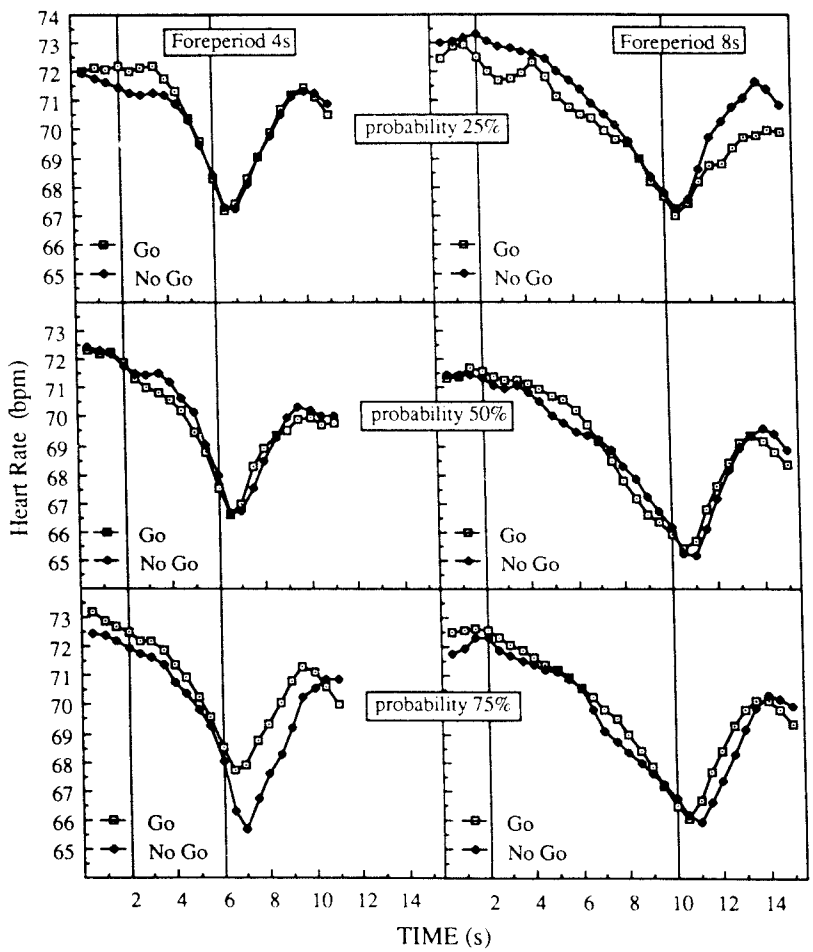

Figure 2. Task-evoked heart rate responses in Go and No Go trials as a function of Go probability and foreperiod length (left panel: Foreperiod = 4 s, right panel: Foreperiod = 8 s). Please note that in each panel of this figure Go probability for Go trials and No Go probability for No Go trials are the same.

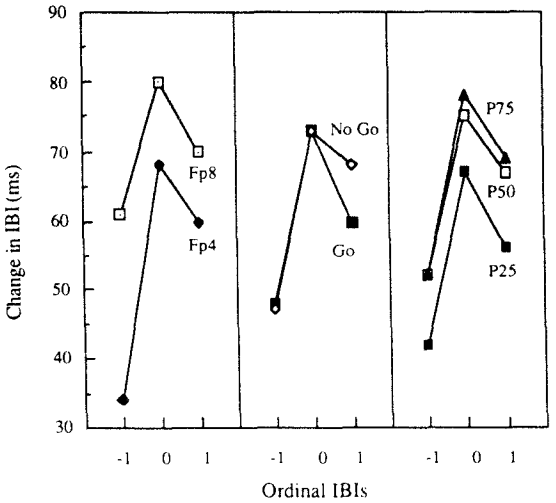

Figure 3. Interbeat interval (IBI) response proximal to the stimulus. Changes in IBIs −1, 0, and 1 are presented relative to a prewarning baseline. IBI −1 and IBI 1 are arbitrarily displayed along the abscissa over an equal distance in either direction from IBI 0.

Positive values indicate a lengthening of interbeat interval (decrease in heart rate). The left panel illustrates the foreperiod effect upon the IBI response. The middle panel shows the IBI response on Go and No Go trials. The right panel presents the IBI response as a function of Go probability.

ing which the imperative stimulus happened to fall (IBI 0), the preceding IBI (IBI −1), and the subsequent IBI (IBI 1). The effects of experimental conditions were analyzed relative to a prewarning baseline, i.e., the last full interbeat interval preceding the warning signal. Prewarning baseline did not differ between conditions.

Figure 3 illustrates the effects of Foreperiod, Go/No Go, and Go probability on the IBI response. The left panel shows that the lengthening of the stimulus IBI relative to the preceding IBI is more pronounced for the short foreperiod. The middle panel shows that the shift from deceleration to acceleration during the poststimulus IBI is stronger on Go trials compared to No Go trials. Actually, the cardiac shift on No Go trials occurred during the next IBI rather than during the poststimulus IBI. (Post-hoc analyses indicated that on No Go trials the IBIs 0 and 1 did not differ significantly.) This result supports the impression created by the heart rate data—on Go trials there is an earlier shift from anticipatory deceleration to acceleratory recovery than on No Go trials. The right panel shows the effect of Go probability on the IBI response. It can be seen that with increasing Go probability, there is an increase in the duration of all three interbeat intervals. This effect is due mainly to the lowest Go probability. Finally, it is important to note that the effects of Go probability and Go/No Go trials were additive for all three interbeat intervals.

These visual impressions were statistically verified by an ANOVA performed on the IBI response, yielding significant main effects of sequential IBI, $F(2/28)=13.5$, Foreperiod, $F(1/14)=73.3$, and Go probability, $F(2/28)=6.1$. The effect of IBI interacted with the effects of Foreperiod, $F(2/28)=30.8$, and Go/No Go, $F(2/28)=8.7$. There were no other interactions.

The nature of the significant interactions was further assessed with post-hoc analyses on separate interbeat intervals. The analysis performed on IBI −1 showed significant main effects of Foreperiod, $F(1/14)=266.5$, and Go probability, $F(2/28)=4.0$, but not Go/No Go, $F(1/14)<1$. The same pattern of results was obtained for IBI 0: Significant main effects of Foreperiod, $F(1/14)=19.6$, and Go probability, $F(2/28)=4.4$, but not of Go/No Go, $F(1/14)<1$. The analysis performed on IBI 1, however, showed a significant main effect of Go/No Go, $F(1/14)=7.1$. In addition, there were significant effects of Foreperiod, $F(1/14)=9.3$, and Go probability, $F(2/28)=7.7$. Again, there were no interactions between the effects of these factors.

Cardiac Cycle Time

The time of stimulus occurrence within the cardiac cycle was expressed as the ratio of the R-wave-to-tone-onset interval to the R–R interval. These ratios were then divided into three equal bins (thirds). The cardiac cycle time effects, relative to the prewarning interbeat interval, are depicted in Figure 4. The left panel illustrates the effect of stimulus occurrence in the cardiac cycle on the stimulus interbeat interval (IBI 0) for Go and No Go trials. It can be seen that early occurring signals prolong the cycle of their occurrence more than later signals. This primary bradycardia effect is more pronounced for No Go than for Go trials. The effects of later occurring signals (thirds 2 and 3) do not differ between Go and No Go conditions. The middle panel shows the effect of stimulus occurrence on the subsequent interbeat interval (IBI 1). It can be seen that early stimuli are associated with shorter cycles than later stimuli. This effect is stronger for Go compared to No Go signals. The right panel shows the effect of foreperiod length on primary bradycardia. Although the level of deceleration during the stimulus interbeat interval is higher for the long foreperiod compared to the short foreperiod, primary bradycardia is more pronounced for the short foreperiod.

The effects of stimulus occurrence in the cardiac cycle were assessed with separate ANOVAs—Prob-

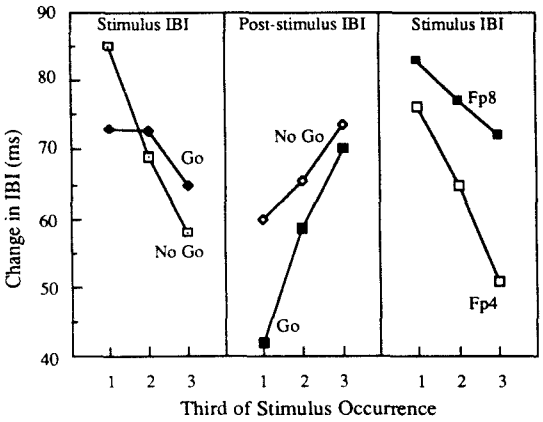

Figure 4. Interbeat interval (IBI) change as a function of time from the R-wave of the ECG to the imperative stimulus. The difference between IBI 0 or IBI 1 and a prewarning baseline is presented for each third of the heart period of IBI 0. Positive values indicate a lengthening of interbeat interval (a decrease in heart rate). The left panel illustrates the slowing of IBI 0 as a function of stimulus occurrence on Go and No Go trials. The middle panel shows cardiac acceleration during the subsequent IBI (IBI 1) on Go and No Go trials as a function of stimulus occurrence in the preceding IBI (IBI 0). The right panel presents the interaction between the effect of stimulus occurrence on IBI 0 and the effect of foreperiod length.

ability(3) × Foreperiod(2) × Go/No Go(2) × Third(3)—performed on the IBIs 0 and 1. The analysis carried out on the stimulus interbeat interval (IBI 0) showed significant main effects of Third, $F(2/28)=17.7$, and the factors already mentioned in the previous section. Significant interactions emerged for Third × Go/No Go, $F(2/28)=10.1$, and Third × Foreperiod, $F(2/28)=2.5$. The effect of Third did not interact with the effect of Go probability, $F(4/56)=1.7$, $p=.16$.

The analysis performed on IBI 1 showed a slightly different pattern of results. Again, there was a significant main effect of Third, $F(2/28)=25.8$. Also significant was the Third × Go/No Go interaction, $F(2/28)=4.4$. In contrast to the results for IBI 0, however, there was no significant Third × Foreperiod interaction, $F(2/28)<1$.

Pupil Dilation

For ease of comparison, this section directly follows the sequence of presentation used in the Richer et al. study. Pupillary records were analyzed for an interval extending from 2 s before the warning signal to 5 s after the imperative stimulus. The individual records were inspected using an interactive program. Traces containing large eyeblinks or movement artifacts at the moment of the response were rejected. These traces were identified by the presence of sharp discontinuities or large constrictions in the pupillary record, which were not related to psychosensory variations in pupil diameter. For each subject stimulus-locked averages of the pupil response were computed from all artifact-free records for each of the 12 trial types (Go/No Go(2) × Go-probability(3) × Foreperiod length(2)). An average percentage of 66% of the total number of traces were included in each of the average waveforms computed (this percentage is comparable with Richer et al.). The conditions did not differ in percentage of acceptable data.

Figure 5 contrasts Go and No Go waveforms obtained by conventional averaging, in the three probability conditions for short and long foreperiods, separately. When Go probability increased from 25% to 75%, the peak amplitude of the dilation in No Go trials rose from 5.03 to 5.07 to 5.16 mm. However, dilations on Go trials slightly decreased from 5.35 to 5.29 to 5.28 mm. Additionally, the No Go dilation peaked earlier than the Go dilation (1122 and 1381 ms, respectively).

The results of ANOVAs performed on the peak amplitudes of pupil dilation and the latencies of these peaks supported the impressions created by the visual inspection of the data. Peak amplitudes were significantly larger for Go reactions than for No Go reactions, $F(1/14)=84.1$. Also significant was the Go probability × Go/No Go interaction, $F(2/28)=46.0$. The main effect of Go probability was not significant, $F(2/28)<1$. There were no foreperiod effects. A similar ANOVA carried out on the latencies of the peak amplitudes of the dilations indicated that latency was significantly shorter for No Go reactions compared to Go reactions, $F(1/14)=9.0$. There was a significant interaction between the effects of Go/No Go and Go probability on the latency of the pupil dilation, $F(2/28)=7.9$. There was no main effect of Go probability on the latency of pupil dilation, $F(2/28)<1$. Again, there were no foreperiod effects.

Relationships Among Reaction Time, Interbeat Interval, and Pupil Dilation

Relationships among reaction time, interbeat interval, and pupil dilation were studied using correlational analysis. Pearson correlations were computed for reaction time and the interbeat interval of stimulus occurrence for each subject and each combination of Foreperiod duration and Go probability. Similarly, correlations were computed between reaction time and pupil latency and between reaction time and pupil amplitude. Finally, correlations were computed between interbeat interval and pupil latency and between interbeat interval and pupil amplitude, for Go and No Go trials sep-

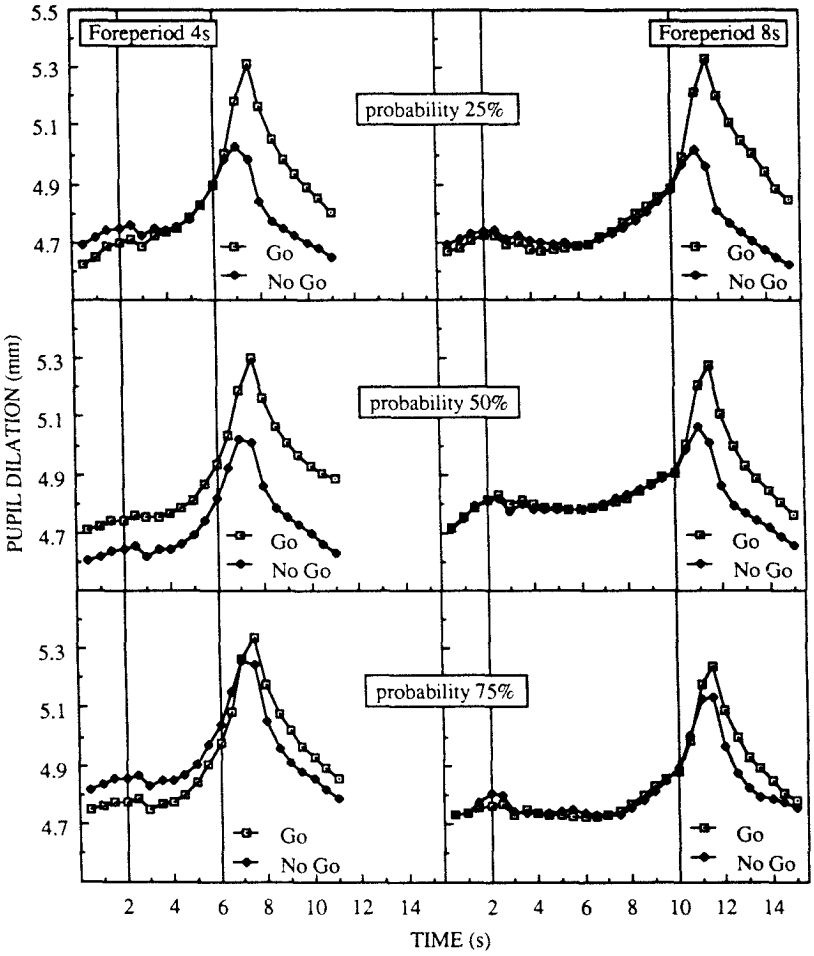

Figure 5. Task-evoked pupillary responses in Go and No Go trials as a function of Go probability and foreperiod length (left panel: Foreperiod = 4 s, right panel: Foreperiod = 8 s). Please note that in each panel of this figure Go probability for Go trials and No Go probability for No Go trials are the same.

arately. In general correlations were low, in different directions, and nonsignificant. There were also no systematic trends for the experimental conditions.

Discussion

Reaction time increased on Go trials with the increase of time and event uncertainty. The number of response anticipations on No Go trials increased when stimulus frequency was biased toward Go reactions. However, since the processes elicited by the warning stimulus are, by and large, not manifest in overt behavior, a description of preparation and response execution would be incomplete should the analysis be restricted to reaction time. The first manifestation of the processes elicited by the warning stimulus is shown in the task-evoked cardiac response. The stimulus elicits a heart rate deceleration that reaches its nadir at some point in time near the imperative stimulus. The current finding of a foreperiod effect on sustained anticipatory deceleration is consistent with the view that associates anticipatory heart rate deceleration with a need for temporal prediction (see Bohlin & Kjellberg, 1979). Jennings previously suggested that heart rate deceleration may be related to temporal prediction as part of the preparation for an attentional shift to active processing after holding processing capacity available in anticipation of subsequent activation (e.g., Jennings, Lawrence, & Kasper, 1978).

The current foreperiod results suggest, however, that just prior to the stimulus preparatory processes induce added cardiac deceleration. In the short foreperiod condition there was a stronger increase in the interbeat interval of stimulus occurrence than in the long foreperiod condition. Further, the cycle time analysis indicated that primary bradycardia

(i.e., cardiac slowing during the IBI of the stimulus IBI as a function of stimulus occurrence—see Figure 4, right panel) was stronger in the short foreperiod than in the long foreperiod condition. Assuming a perceptual locus of the primary bradycardia effect (e.g., Lacey & Lacey, 1980), the current data suggest that when the timing of a stimulus is predictable, processing resources can be allocated to encoding just prior to stimulus presentation (see also Coles & Strayer, 1985).

Response as well as perceptual preparation, appeared to alter prestimulus changes. When stimulus frequency was biased toward Go reactions, all three interbeat intervals selected around the reaction stimulus progressively lengthened. In the reaction time literature the Go probability effect is generally considered to have a motoric locus. Most authors assume that motor preparation is directly related to the likelihood that a particular signal will be presented (see reviews by Gaillard, 1978; Sanders, 1980). This finding then is well in keeping with Lacey's (1967) hypothesis suggesting a relationship between the autonomic nervous system and motor preparation.

In suggesting that autonomic response measures may be used to index motor preparation, the cardiac and pupil data provide converging evidence. The pupillary response shows a preparatory dilation that begins about .5 s before and peaks about .5 s after the imperative stimulus. We, like Richer et al. (1983), found a No Go dilation that could be made comparable in amplitude to the Go dilation by increasing Go probability. Second, as in the Richer et al. study, the amplitude of dilation was not affected by foreperiod length (see also Bradshaw, 1969). Richer et al. interpreted the effect of Go probability on No Go dilation as reflecting a process of motor preparation that is common to both Go and No Go responses until the imperative signal is presented. They obtained additional support for the hypothesis that pupillary dilation reflects motor preparation in a follow-up study showing that preparatory dilation is sensitive to force and complexity parameters of the motor response (Richer & Beatty, 1985). Thus, both pupillary and heart rate responses suggest that response preparation processes may antedate stimulus occurrence. The current results justify an interpretation of preparation in terms of a timing mechanism (indexed by heart rate deceleration during the foreperiod) and a mechanism allocating processing resources to stimulus encoding (indexed by cardiac slowing just prior to stimulus occurrence) and response preparation (indexed by continued cardiac deceleration and pupillary dilation).

At the time of stimulus occurrence, autonomic changes occur which again are relevant to the inference of psychological process. The task-evoked cardiac response was different for Go and No Go trials, replicating van der Molen et al. (1983). First, primary bradycardia (i.e., cardiac slowing during the stimulus interbeat interval as a function of stimulus occurrence; see Figure 4, left panel) was stronger on No Go trials compared to Go trials. The task of evaluating No Go signals seems to take longer than that of evaluating Go signals, hence the transition of deceleration into acceleration occurs later. Similar findings have been obtained at a behavioral level—the processing of negative statements takes longer than the processing of positive statements (Carpenter & Just, 1975; Whitaker, 1982). Second, cardiac acceleration during the subsequent interbeat interval as a function of stimulus occurrence (see Figure 4, middle panel) was more pronounced on Go trials than on No Go trials. This finding is well in keeping with data obtained by Hollander and co-workers (see Hollander, 1975; Gelsema, Hollander, Karemaker, & Bouman, 1985). They observed that short muscle contractions give rise to cardiac acceleration almost without delay. The same cardiac cycle during which the contraction starts is shortened if the contraction begins in the first half of the cycle; the cycle is not influenced when the contraction is later. Previously, Jennings (1985) suggested that the precise timing of the cardiac shift may be useful in timing the regulation of attention during information processing. When information processing enters response initiation, either overt or covert (Schwartz & Higgins, 1971), cardiac deceleration shifts to acceleratory recovery. The use of the timing of the cardiac shift may be particularly valid in conjunction with other psychophysiological measures such as event-related brain potentials (Jennings, 1985).

The relationships among reaction time, interbeat interval, and pupil dilation, all being influenced by Go probability, were assessed by correlational analyses. The results showed that the relation among these measures was effectively zero. Low or absent correlations between response speed and cardiac deceleration have been reported previously (see review in van der Molen, Somsen, & Orlebeke, 1985). One of the possible explanations of uncorrelated behavioral and cardiac measures refers to the level of their relative sensitivity to computational and energetic processing demands, but the data do not permit a detailed analysis of this issue. It could be that, whereas reaction time predominantly reflects the processing durations of computational mechanisms, cardiac deceleration is primarily associated with the allocation of energetic

support to the computational mechanisms (cf. van der Molen et al., 1987). A similar explanation cannot be entertained for the nonsignificant correlation between interbeat interval and the pupil response, in that pupil dilation, like cardiac deceleration, is commonly interpreted in terms of processing capacity (e.g., Beatty, 1982; Kahneman, 1973). In this case, a possible explanation might refer to a difference in physiological mechanism. Although phasic cardiac deceleration in reaction time tasks is regulated by the parasympathetic branch of the autonomic nervous system (Obrist, Webb, Sutterer, & Howard, 1970), pupil dilation is predominantly controlled by sympathetic influences (Hess, 1972). The current data then seem to suggest that reaction time, cardiac deceleration, and pupil dilation provide different windows on motoric activation. This conclusion emphasizes the need for a multi-measure analysis of motoric activation including variables related to both computational and energetical processing demands and derived from psychophysiological systems ranging from central to more peripheral levels.

Before closing, a final question needs to be addressed. Go probability and foreperiod length produced statistically independent effects on all measures (reaction time, preparatory dilation, IBI lengthening, and primary bradycardia). Both variables, however, are commonly associated with motor preparation. Why, then, did they not interact in their contributions to the behavioral and autonomic response measures? The most likely explanation is that the preparatory processes indexed by both IBI lengthening and pupillary dilation, influenced by Go probability but not foreperiod length, can only be maintained for a short period of time (Gottsdanker, 1975). Possibly, they are initiated just prior to the imperative stimulus, rather than being a gradual process which is optimal toward the end of the foreperiod. For example, Sanders (1980), in measuring the electromyogram (EMG), observed that the EMG response did not differentiate between a short and a long foreperiod condition. According to this interpretation, the preparatory processes reflected in pupillary dilation and IBI lengthening should be affected when the time of stimulus occurrence is much more difficult to predict (e.g. under mixed foreperiod conditions when duration is varied across a greater range).

In conclusion, the cardiac and pupillary responses associated with motoric activation show strong similarities with other physiological measures. At the electrocortical level, a readiness potential, recorded on the scalp, can be observed prior to the execution of a motor response. The readiness potential has been shown to vary in amplitude with the probability that the imperative stimulus occurs (Gaillard, 1978). Moreover, single cell activity recorded in the precentral motor cortical structures during the last 250 ms of the foreperiod identified "presetting" cells whose firing rate was related to response probability (Requin, Lecas, & Bonnel, 1984). Finally, at a spinal level, it has been demonstrated that the excitability of the Achilles tendon (T) reflex is systematically smaller when evoked in the muscles involved in the prepared response than in uninvolved muscles. This differential effect varies with response probability. Under conditions of low response probability the effect is absent (Brunia, Haagh, & Scheirs, 1985). All these measures converge to indicate that the presetting of the nervous system is an important part of response preparation (cf. Richer et al., 1983).

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