Beat-by-Beat Cardiac Responses in Normals and Schizophrenics to Events Varying in Conditional Probability

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ABSTRACT

Anticipatory cardiac deceleration and poststimulus acceleration were studied in schizophrenic inpatients and controls during performance of a counting task. Reduced cardiac responding has been reported for schizophrenic patients for paradigms using relatively long intertrial intervals. During a relatively fast rate of stimulus presentation (3-s interstimulus interval), changes in cardiac interbeat interval were measured in 20 inpatient male chronic schizophrenics and 18 control volunteers. Subjects counted an infrequent tone which was always followed by at least one frequent tone. Control subjects showed significant anticipatory cardiac deceleration preceding the unpredictable tones, whereas patients did not show a differential cardiac deceleration. Control subjects showed poststimulus acceleration that was inversely proportional to the conditional probability of events, whereas patients exhibited greatly reduced poststimulus acceleration; patterns for both groups resembled findings previously observed for event-related potential and pupillary dilation data. Analysis of cardiac cycle time indicated significant variation in primary bradycardia associated with the delay between stimuli and immediately preceding R-waves in controls (replicating Lacey & Lacey, 1980), with only an immediate bradycardia at stimulus reception for patients regardless of cardiac cycle time. The data reinforce the notion that the manner in which information is used by schizophrenics, as reflected by cardiac responsivity, differs both quantitatively and qualitatively from that of controls.

DESCRIPTORS: Heart rate, Cardiac cycle time, Schizophrenia, Information processing, Primary bradycardia.

Cardiac responsiveness provides a major source of information on autonomic nervous system activity in schizophrenics and normal controls (Zahn, 1986). Considerable attention has been paid to tonic heart rate levels in schizophrenics, with elevated heart rates typically reported among patients compared to controls (Spohn & Patterson, 1979; Zahn, 1986). Without questioning the value of these differences, it is also informative to examine phasic differences in cardiac activity during stimulus presentation. Phasic responses appear sensitive to information processing (Coles & Duncan-Johnson, 1975; van der Molen, Somsen, & Orlebeke, 1985), and the information processing of schizophrenics is known to differ from that of normals (Kietzman, Spring, & Zubin, 1980). Phasic cardiac changes, therefore, provide a noninvasive probe of physiological activities that reflect more centrally mediated processing. Cardiac responses during stimulus presentation have been recorded both during active task performance in schizophrenics and controls, and during the presentation of simple stimuli with no explicit task required.

In those studies dealing with cardiac responses to orienting stimuli (a nontask situation), in which deceleration is normally expected (Graham, 1979), unmedicated schizophrenics have been reported to exhibit cardiac acceleration (Zahn, Rosenthal, &
Lawlor, 1968). Gruzelier (1975) reported acceleration in schizophrenics who also showed a substantial skin conductance response (responders), but not in those patients who were nonresponders. In contrast, Bartfai, Levander, Edman, Schalling, and Sedvall (1983) observed no cardiac acceleration among schizophrenics, but identified poststimulus deceleration to at least the first tone in 92% of patients and 92% of controls.

These only partially consistent results from non-task situations can be compared to those in which patients are asked to perform a simple task. When a signal stimulus can be anticipated in time, cardiac deceleration is typically observed beginning prior to stimulus onset (anticipatory deceleration); after stimulus delivery, cardiac acceleration tends to occur over the next several beats (Lacey & Lacey, 1974). These phenomena can be readily observed during reaction time tasks, and have been conceptually related to the development and dissipation of anticipatory attention (Coles & Duncan-Johnson, 1975; van der Molen et al., 1985).

Several studies have examined cardiac activity during active task performance by schizophrenics. Schizophrenics did not show typical cardiac deceleration between a warning signal and an aversive tone, which could be terminated by a response following the aversive tone (Waddington, Maccolloch, Schalken, & Sambrooks, 1978). Similarly, unmedicated schizophrenics have been reported to show decreased anticipatory deceleration during simple reaction time tasks (Gray, 1975; Zahn, Carpenter, & McGlashan, 1981a, 1981b). Gruzelier (1975), employing an unwarned tone discrimination task, also failed to find deceleration; he reported acceleration to signals by all patients, with schizophrenic responders (defined by skin conductance response) exhibiting cardiac acceleration to nonsignals as well. In contrast, during performance of a span of apprehension task, Spohn, Thetford, and Woodham (1970) found that both patients and controls exhibited pre- and poststimulus deceleration.

The poststimulus cardiac response has also been studied in size estimation tasks, with varying results depending on the classification of patients based on additional diagnostic criteria. Warner (1973) reported cardiac deceleration for schizophrenics with good premorbid histories, but cardiac acceleration among those with poor premorbid histories. McCormick and Broekema (1978) reported that cardiac deceleration was greater in both paranoid and nonparanoid patients than in controls.

This brief review suggests that different tasks may alter pre- and poststimulus cardiac responding in schizophrenics and normals. The findings suggest that it may be of value to vary task characteristics within a single study of schizophrenics and normal controls. For example, signal stimuli could be varied in informational complexity and predictability.

Finally, changes of the heartbeat just following stimulus presentation may be a sensitive index of stimulus registration. The phenomenon of primary bradycardia (Lacey & Lacey, 1980) involves a relatively early deceleratory response, which has been attributed to stimulus reception, rather than anticipation. According to the Laceys, the time at which bradycardia can be observed is dependent on the latency of the stimulus from the previous R-wave. When this latency is short, an immediate bradycardia can be seen, with a lengthening of interbeat interval during the very interval in which the stimulus occurred (Interbeat Interval 0). If the stimulus occurs late in the interbeat interval, then deceleration is delayed until the succeeding interval (Interbeat Interval +1) rather than the stimulus interval. The presence of primary bradycardia among schizophrenics, even in the absence of other major cardiac changes, would suggest that there has been registration of stimulus reception. To date, this phenomenon has not been examined in schizophrenic patients.

The present study involved the adoption of a paradigm that was originally employed for investigation of event-related potential and pupillary responses (Clark, Niemi, & Balogh, 1985; Steinhauer & Zubin, 1982), in which probability was varied to produce both predictable and unpredictable events. In the typical oddball paradigm, one of two stimuli occurs less frequently than the other, with the infrequent stimulus producing larger amplitude psychophysiological responses. A modification of the paradigm restricts the occurrence of infrequent events to no more than one in a row. By informing the subject that this contingency is in effect, the occurrence of the infrequent stimulus permits the attentive subject to expect that the next stimulus will always be the frequent stimulus. In contrast, when a frequent stimulus occurs, it is not possible to predict accurately whether a frequent or infrequent stimulus event will occur next. The design enables the evaluation of differential anticipatory deceleration to predictable and unpredictable events, poststimulus primary bradycardia, and poststimulus acceleratory activity. In addition, it allows comparison with data previously collected on pupillary and event-related potential responding, which indicated atypical responsivity among schizophrenic patients related to stimulus change, rather than to overall or conditional probability effects (Steinhauer & Zubin, 1982).
Method

Subjects

Twenty male patients from the Highland Drive VA Medical Center, meeting Research Diagnostic Criteria for chronic schizophrenia, were recruited for the study. Patient subjects were recently hospitalized. Mean age ± SD of the patients was 33.6 ± 8.0 years. All but 2 patients were receiving neuroleptic medication. Eleven patients were receiving haloperidol, 4 received thiothixene, and 1 each were receiving thioridazine, trifluoperazine, or fluphenazine decanoate. This provided a daily median dose of 875 chlorpromazine equivalents (CPZeq), with a range of 300-2000 CPZeq for 17 subjects, and 1 patient on haloperidol receiving 3500 CPZeq. Seven patients were receiving anticholinergics: 1 patient on haloperidol received 30 mg/day of amantadine, while 3 patients on haloperidol, 2 on thiothixene, and the patient on trifluoperazine each received 2-4 mg benztropine.

Male control volunteers, recruited primarily through local employment agencies, were screened for psychiatric disorder using the Renard Diagnostic Interview (Helzer et al., 1977). Eighteen control subjects meeting RDC criteria for no psychiatric disorder were tested. Mean age of these subjects was 28.4 ± 5.1 years, significantly younger than the patient group (p = .025); subsequent analyses were performed using age as a covariate. Controls were drug-free. Controls received $5/hour, in addition to a small bonus associated with task performance. Patients received only the bonus associated with the task. Informed consent was obtained from all patient and control subjects prior to testing.

Psychophysiological Recording

The electrocardiogram was derived from large Beckman Ag/AgCl electrodes placed under the left rib cage and on the right shoulder, with a small ground electrode on the forehead. The signal was amplified by a Grass Model 78 polygraph, and a peak detection circuit (Shimizu, 1978) was used to detect the R-wave. The time of each R-wave, to the nearest ms, was stored in a PDP-11/03 (MINC) laboratory minicomputer. Interbeat interval in ms and heart rate in bpm were computed off-line.

Procedure

Each subject was presented with high-pitched tones (1500 Hz) and low-pitched tones (800 Hz) at 65dB, 40 ms in duration (with rise/fall times of 4 ms), through a speaker placed in front him. One tone was presented every 3 s for a total of 80 tones (trials) per block. At least 4 blocks of tones were presented to each subject.

Subjects were first asked to identify sample tones as "high" or "low" in pitch. The subject was instructed to count silently the number of high tones (targets), and report the number of high tones (but not low tones) at the end of each block. Subjects were told that: 1) there would be fewer high than low tones, and 2) there would never be two high tones in succession. Prior to recording, subjects were asked what would occur next if a high tone was heard; all subjects could correctly answer that a low tone would occur next. The sequence of tones was generated randomly by computer to produce high tones at an overall probability of .25, restricted only by the requirement that two high tones not occur in succession.

Subjects sat in a separate testing room and communicated with the experimenter by intercom. The room was maintained in darkness (because of simultaneous measurement of pupillary diameter); subjects used a head rest and focused on an array of four red LEDs to maintain fixation and minimize head movements.

Subjects were rewarded with a bonus of 25 cents when they counted accurately, with 10 cents if correct to within one or two counts, but no bonus for three or more errors. The bonus and number of errors were told to the subject after his report on each block.

Data Reduction and Analysis

To ensure at least minimal attention to the task, data were included for analysis only when the subject reported with no more than three errors on a block; in some cases, additional blocks were run to provide four blocks of usable data per subject. However, most subjects made relatively few errors throughout the task. Trials on which the subject moved or blinked excessively were coded and excluded from the data analysis. The detection of multiple apparent R-waves within 400 ms, or large changes in interbeat interval (>200 ms) from one interval to the next, were interpreted as artifacts by the data analysis program.

To examine cardiac effects that both preceded and followed stimulus events, a trial was included for analysis only when that trial, as well as the preceding and subsequent trials, were all free of artifacts, providing an overall time window of 9 s for examining cardiac changes.

Although the overall probability was .25 for targets and .75 for nontargets, stimulus events were further characterized by their second-order sequence (conditional probability). Those nontargets preceded by a target, symbolized by Tt, were completely predictable, thus having a conditional probability of 1.00. All other stimulus events were not perfectly predictable. Following any nontarget, the next stimulus would be a target on one-third of all trials (T, conditional probability = .33), and conversely, a nontarget on the remaining two-thirds of such trials (Nt, conditional probability = .67; note that this represents the repetition of a nontarget following the preceding nontarget). Thus, data were analyzed for each of the three conditional events labeled as T(.33), Nt(.67), and Nt(1.0). Note that the labels employed, based on conditional probabilities, do not necessarily reflect the subjective probabilities of each event for the controls and patients.

The initial series of analyses was based on the average interbeat intervals across trials for each of the
three probability conditions, beginning at two intervals prior to stimulus onset (Interbeat Interval –2), through the interval of stimulus delivery (Interbeat Interval 0), and continuing for four intervals following stimulus onset (Interbeat Interval +4).

Analysis of variance was employed using a repeated measures design, with Diagnostic Group (control vs. schizophrenic) × Fixed Factors of Probability (3 stimulus events) × Interbeat Interval (7 intervals). Where appropriate, degrees of freedom were reduced according to the Greenhouse-Geisser correction (Jennings & Wood, 1976).

The data were also analyzed for each subject by converting interbeat intervals on individuals trials to heart rate in bpm. The average heart rate was calculated for 14 periods of .5 s each, beginning 2.5 s prior to stimulus onset. Each interval bridging two separate interbeat intervals was interpolated so that the rate measure was weighted by the proportion of time spent in each interbeat interval. Because these analyses resulted in similar findings, details of the heart rate analyses have been omitted.

Results

Beat-by-Beat Analysis

Interbeat interval data are plotted as a function of event probability for controls (Figure 1) and patients (Figure 2). Increasing interbeat interval—that is, cardiac deceleration—is indicated by upward deflections in these figures. Several trends can be seen in the data for the controls (Figure 1). Prior to the two stimulus events that were not predictable (the T(.33) and Nn(.67) conditions), cardiac deceleration was observed. Following stimulus presentation, greater cardiac acceleration occurred following the T(.33) condition than the Nn(.67) condition. The response in the Nt(1.0) condition showed little

Figure 1. Interbeat Interval data for control subjects (n=18) for each conditional probability event. Increasing interbeat interval indicates cardiac deceleration prior to stimulus delivery in the “T” and “Nn” conditions, followed by cardiac acceleration, which is greatest for the least probable event (“T”).

prestimulus change, with acceleration rather than anticipatory deceleration prior to stimulus delivery, and some deceleration following the stimulus event.

The average interbeat interval for schizophrenics (718.53 ms, equal to a heart rate of 83.50 bpm) was significantly shorter than for controls (835.19 ms; 71.84 bpm) (F(1,36)=10.54, p=.0025). The faster heart rate of the patients is consistent with the majority of reports in the literature.

Significant main effects were also obtained for probability (F(2,64)=3.39, p=.045) and over successive interbeat intervals (F(3,102)=3.95, p=.012), with a Probability × Interbeat Interval interaction (F(4,132)=4.17, p=.0042) also observed. A three-way Probability × Interbeat Interval × Diagnosis interaction (F(4,132)=2.96, p=.026) reflects significantly greater changes among the control subjects across both probability and interbeat interval as compared to patients.

Given the multiple interactions of interbeat interval with probability in the control data set, further analyses were carried out in which prestimulus, poststimulus, and immediate stimulus reception could be focused upon. For this purpose, scores were developed to capture the prestimulus related changes (i.e., anticipatory deceleration), poststimulus changes (i.e., poststimulus acceleration), and changes occurring at the time of stimulus delivery. The analyses focusing on the time of stimulus reception took into account stimulus timing at different intervals with respect to the preceding R-wave in the investigation of primary bradycardia.

Anticipatory Deceleration

The extent of anticipatory deceleration prior to stimulus onset was calculated as the difference between Interbeat Interval –2 and Interbeat Interval
Poststimulus Acceleration

Poststimulus acceleratory activity was calculated as the difference between Interbeat Interval +3 and Interbeat Interval 0 (Figure 3). In parallel with the above findings, there was greater acceleration among controls than patients (diagnosis: $F(1,36)=5.97$, $p=.02$), increasing acceleration for more rare events (probability: $F(2,54)=7.15$, $p=.0041$), with a Probability X Diagnosis interaction ($F(2,54)=7.80$, $p=.0027$) also observed. Simple effects indicated significantly greater acceleration in the infrequent T(.33) condition than in both of the frequent conditions for controls only. Covarying prestimulus level eliminated only the main effect for diagnosis. In separate analyses by subject group, control subjects showed a significant overall increase in rate from Interbeat Interval 0 to +3 ($F(1,17)=16.19$, $p=.0009$). For patients, a trend for acceleration ($F(1,19)=3.62$, $p=.072$) seemed related to acceleration only in the T(.33) and Nt(1.0) conditions. This paralleled findings reported previously for other psychophysiological measures. Planned analyses to evaluate acceleration between Interbeat Interval 0 and Interbeat Interval +3 only for the T and Nt conditions in the patients indicated a significant acceleration ($F(1,19)=4.68$, $p=.043$), which was not reduced by covarying age or prestimulus level.

Anticholinergic Medication Effects

To evaluate the contribution of anti-Parkinsonian agents, comparisons between the 7 patients receiving anticholinergics and the other 13 patients were carried out. There were no significant differences in prestimulus level or acceleration, but a marginal trend ($F(1,18)=3.17$, $p=.092$) was observed for greater average decelerations in the patients receiving anticholinergics than for those without anticholinergics. Thus, use of anticholinergics is unlikely to account for the smaller decelerations seen in general among patients as compared to controls.

Primary Bradycardia

To test for the occurrence of primary bradycardia in relation to cardiac cycle time, individual trials at each probability were first sorted according to the delay between stimulus onset and the preceding R-wave, using increments of 250-ms intervals (e.g., 0–250 ms, 251–500 ms, etc.). Because the number of trials in each condition is reduced by this sorting, only three intervals provided sufficient numbers of trials for cardiac cycle analysis (0–250, 251–500, and 501–750). Data for one patient and
two control subjects were excluded due to fewer than five trials in at least one cell for each subject. In this analysis, changes were of interest only across the three interbeat intervals proximal to stimulus onset—Interbeat Interval $-1$, Interbeat Interval 0, and Interbeat Interval $+1$. These intervals, separated by probability and by beat-to-stimulus delay, are plotted in Figure 4 (controls) and Figure 5 (schizophrenics). In addition to findings reflecting the analyses described previously, there were significant interactions with cardiac cycle time (Interbeat Interval $\times$ Cardiac Cycle: $F(3,98) = 3.38, p = .022$, and Probability $\times$ Interbeat Interval $\times$ Cardiac Cycle: $F(5,150) = 3.38, p = .0087$).

![Controls](image)

**Figure 4.** Cardiac cycle time effects (Beat-to-Stimulus delay) for control subjects ($n=16$). For each event, the interbeat intervals preceding, during, and following stimulus onset are plotted as a function of the latency between stimulus onset and the preceding R-wave, grouped by 250-ms intervals.

![Schizophrenics](image)

**Figure 5.** Cardiac cycle time effects for schizophrenic patients ($n=19$), scaled to same range as in Figure 4.

Although there was no significant interaction between cardiac cycle time and diagnosis, it was of interest to examine effects during the period of stimulus reception separately for each of the two groups. Controls exhibited the phenomena of primary bradycardia as described by the Laceys (Lacey & Lacey, 1980), with differences due both to changes across interbeat interval as well as among probabilities. The effect for primary bradycardia occurred in the T(33) and Nn(67) conditions: greater deceleration was observed for the interbeat interval of stimulus occurrence when there was less delay between the stimulus and the preceding R-wave.

For the schizophrenic patients, there were no significant differences related to the delay between the previous beat and the time of stimulus onset—that is, no differential effect of primary bradycardia occurred in relation to the interval between the R-wave and stimulus. However, a significant main effect among the three interbeat intervals was found ($F(2,35) = 3.48, p = .043$), reflecting deceleration from Interbeat Interval $-1$ to Interbeat Interval 0.

**Discussion**

The greatest difference observed between schizophrenics and controls was in average heart rate: patients had an average rate that was approximately 12 bpm faster than the rate for controls. This replicates the major findings on overall heart rate level in schizophrenia (Zahn, 1986). In addition, patients exhibited an absence of anticipatory deceleration and little poststimulus acceleration. In general, there was a pattern of less cardiac responsivity during this task for the patients.

Reduced phasic cardiac responsivity has been reported in several other paradigms employing both drug-free and medicated patients. Zahn, Schooler, and van Kammen (1985) reported less anticipatory deceleration among patients during performance of a recognition task. These effects were present even though all patients were medication free. Increases in heart rate have been seen for patients receiving phenothiazines, with a correlation between dosage and increased rates for those patients with the lowest heart rates (Spohn, Thetford, & Cancro, 1971). For patients administered chlorpromazine, decreased decelerative responses were seen over an eight-week period as compared to patients receiving placebo (Spohn, Lacoursiere, Thompson, & Coyne, 1977). However, other data strongly indicate that neuroleptics by themselves are not likely to be primarily responsible for either the greatly elevated heart rates or the reduced cardiac responsivity in schizophrenics during tasks. Patients tested first while drug-free and later after either haloperidol or chlorpromazine treatment showed higher rates than
controls regardless of medication status (Castellani et al., 1982). In addition, going from a supine to standing position resulted in a minimum mean acceleration of at least 15 bpm, even for patients with extremely high initial heart rates (Castellani et al., 1982).

In the present data, control subjects showed a clear anticipatory deceleration when the succeeding stimulus could not be predicted—i.e., in the T(.33) and Nn(.67) conditions. For these conditions, stimulus delivery eventually resulted in increased acceleration, with greater acceleration associated with the T(.33) condition. Given that the rare T(.33) tone was also the relevant event associated with counting behavior, these data cannot dissociate the effects of the rareness of the event from the relevance of the target on acceleratory activity. Prestimulus acceleration in the Nt(1.0) condition appears to be a continuation of the large acceleration generated by the immediately preceding target stimulus. These results replicate the primary cardiac findings of Lahey and Lacey (1980), who employed a similar paradigm differing primarily in the difficulty of the auditory discrimination (target and nontarget tones were, respectively, 1000 and 1050 Hz), and who did not inform their subjects that targets never occurred in succession.

Schizophrenic subjects differed from controls in both anticipatory deceleration and poststimulus acceleration. Several possibilities are suggested by these findings. That medications were responsible for floor and ceiling effects, restricting possible variations in heart rate, is unlikely given the report of Castellani et al. (1982), although it would have been useful to have obtained similar data resulting from changes in bodily position for the current subjects. As indicated by the present findings, administration of anticholinergic agents also are not sufficient to explain the reduced reactivity of the patients.

A second alternative is that patients were not aware of the fact that some stimuli were predictable and others were not. The instructions to subjects only required counting of the infrequent target once it had occurred, so patients may have ignored the fact that only single targets occurred. We note, however, that patients were able to report, even at follow-up discussions several weeks after testing, the contingency of no more than one target occurring in a row. If patients were actively treating all events as unpredictable, then clear decelerations should have been generated prior to all stimuli, which was not clearly demonstrated.

Two other alternatives are quite possible. It is conceivable that patients were not actively trying to employ the information regarding predictability of subsequent stimuli after a target or nontarget had been presented. At the other extreme, they may have been attempting to perform the task as well as possible (as patients often report), but could not effectively focus their attention. Zahn (1970) observed that schizophrenics were unable to overcome effects of “minor set” during a reaction time task even when preparatory intervals were indicated to the patients.

Other data suggest that differences in autonomic responsivity may be associated with an ability to utilize information. In an initial conditioning task, schizophrenics as a group showed less skin resistance and cardiac (deceleration) change than controls, and only controls benefitted from information on the conditioning contingency (Rist, Baumann, & Cohen, 1981). Rist and colleagues then selected only patients who were electrodermal responders. Cardiac responding was observed prior to and during the conditioning phase in all groups. During conditioning, greater deceleration to the CS+ preceding a noxious loud tone, and greater acceleration to an innocuous imperative stimulus as UCS, were seen among those patients who were informed about the CS-UCS contingencies than among those patients who were uninformed. Thus, utilization of information corresponded with cardiac changes in these autonomically responsive patients.

The pattern of poststimulus acceleration in the schizophrenic patient data (Figure 3) is of particular interest. In the two extreme probability conditions—T(.33) and Nt(1.0)—there was mild though significant acceleration, but little change in the Nn(.67) condition. In our earlier evaluation of this paradigm (Steinhauer & Zubin, 1982), involving different samples of schizophrenics and controls, we reported patterns for both pupillary dilation and the P300 component of the event-related potential that are similar to those observed in the present cardiac acceleration data. The patients were similarly characterized by greater amplitudes in the T(.33) and Nt(1.0) conditions, in which the stimulus was physically changed from the previous trial, as compared to the Nn(.67) condition, which was associated with stimulus repetition. Controls showed increasing amplitudes of pupillary dilation and P300 with decreasing event probability. Thus, the trends observed in these cardiac data are similar to those obtained with other psychophysiological measures in schizophrenics. The status of such cardiac changes as either indicators of episodes or persistent markers of vulnerability to schizophrenia (Zubin & Steinhauser, 1981) remains a further question to be clarified.

There are several ways in which the paradigm utilized in this study differs from previous evaluations of cardiac responses during task performance.
in schizophrenics. In the present study, testing was prolonged over at least 320 trials, with a short 3-s interval between trials. In previous studies, the number of experimental trials has ranged from as few as 5 stimuli plus 2 catch trials (Waddington et al., 1978) to 20 warned reaction time trials (Zahn et al., 1981a), with intervals between trials of no less than 24 s before a warning signal (Gray, 1975) to as much as 60 s (Gruzelier, 1975). Thus, in the present study, more consistent attention was required to a considerably greater number of trials, with a faster rate of presentation, and over longer periods of time, than for previous experiments.

Although the studies of Gray (1975), Gruzelier (1975), Waddington et al. (1978), and Zahn and colleagues (1981a; Zahn, Rumsey, & van Kammen, 1987) all involved an overt motor response by the subjects, in the present task, a covert behavior (counting) was employed. However, the counting task was sufficient to elicit vigorous anticipatory deceleration and poststimulus acceleratory activity in control subjects. A separate group of 19 control subjects has also been tested using the same paradigm involving the counting task on some blocks, and a discriminative button press to different tones on other blocks. The same pattern of cardiac changes reported here is seen across the two tasks, with the exception that deceleration is also seen prior to the 1.0 condition when a response must be executed. Thus, the nature of the task itself does not seem to preclude the development of cardiac activity. In current studies, both the counting and discrimination tasks are being employed with patients.

Time-dependent primary bradycardia was observed across different probabilities among control subjects, as previously described by Lacey and Lacey (1980). Schizophrenics did not exhibit a differential response on the basis of the delay between the preceding R-wave and the stimulus. However, the analysis indicated a significant deceleration between Interbeat Intervals — 1 and 0 for the patients.

This immediate deceleration in the schizophrenic sample is surprising, because it was not observed in the initial analyses across all seven interbeat intervals (presumably because of greater variability when all intervals were considered at once) or even in the anticipatory deceleration response. It suggests that there is indeed some cardiac response in the patient group, which may be related to effects of stimulus reception. However, while stimulus reception occurs, the effects of further evaluation, normally exhibited by cardiac acceleration in relation to the information provided (Lacey & Lacey, 1974), are not observed in the patients. This situation has an analog in the event-related potential literature: brainstem auditory potentials, which are related primarily to sensory processing, are generally reported to be intact in schizophrenics (Brecher & Begleiter, 1985; Pfefferbaum, Horvath, Roth, Tinklenberg, & Kopell, 1980), whereas the longer-latency P300 component, associated with cognitive processing, is typically attenuated in these patients compared to controls (Roth, Tecce, Pfefferbaum, Rosenbloom, & Callaway, 1984; Zubin, Kietzman, & Steinhauster, 1985).

The increased average heart rate for the schizophrenic patients compared with controls is consistent with the notion of maintained high levels of sympathetic arousal (Zahn, 1986) in schizophrenia. What is less clear is why there should be so little change in heart rate, especially the lack of slowing of an already quickly beating heart. Slowing of heart rate is primarily due to vagal inhibition (cardiac deceleration following increased vagal activity). This would suggest decreased responsiveness of central parasympathetic mechanisms, but could also be due to central inhibition of parasympathetic activity by the ongoing high levels of sympathetically mediated processes.

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