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Heart Rate Variability During Attention Phases in Young Infants

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ABSTRACT

Heart rate variability during visual attention was studied in infants who were tested cross-sectionally at 14, 20, or 26 weeks of age. They were presented with a recording of a *Sesame Street* program on a TV screen. After heart rate had decelerated below the prestimulus level and then returned to prestimulus level, a computer-generated pattern replaced the *Sesame Street* display. Heart rate variability changed throughout attention. The change consisted of a decrease in variability during attention and a return to prestimulus levels approximately five seconds following attention termination. The heart rate and variability responses are consistent with a model of parasympathetic vagal influence on the heart in which vagal firing is increased during sustained attention and is inhibited during attention termination.

DESCRIPTORS: Heart rate variability, Visual attention, Infants, Vagal influence.

A close relation between attention and the variability in heart rate has been demonstrated in adults (e.g., Coles, 1972; Porges & Raskin, 1969) and older children (e.g., Porges & Humphrey, 1977), in which heart rate slows and variability decreases during intensive attending. In infants, change in mean heart rate during attention has been studied extensively; however, studies of change in heart rate *variability* are rare. Porges and his coworkers studied the variance of heart rate in newborn subjects during visual (Porges, Stamps, & Walter, 1974) and auditory (Porges, Arnold, & Forbes, 1973) stimuli. Porges et al. (1973) reported that there was an increase in heart rate variance in the first five seconds of the auditory stimulus. Presumably this increase was a result of the changing mean levels associated with the accelerating heart rate (4 bpm increase). Heart rate variance was not significantly different from prestimulus levels for seconds 6–10 following stimulus onset, or for a period following stimulus offset. Porges et al. (1974) reported similar changes for heart rate variance of newborns in response to visual stimuli.

Although heart rate variability during attention has not been studied extensively in infants, baseline heart rate variability has been used frequently in the prediction of individual differences in sustained attention in infants. Newborns with high heart rate variability have larger mean heart rate responses to visual and auditory stimuli than newborns with low heart rate variability (Porges, 1974; Porges et al., 1973, 1974; Vranekovic, Hock, Isaac, & Cordero, 1974; Williams, Schacter, & Tobin, 1967). The specific type of baseline heart rate variability that is related to infant attentional responses is respiratory sinus arrhythmia (RSA) in heart rate. Compared to infants with low baseline RSA, infants from 14 to 26 weeks of age with high baseline RSA show larger and more sustained heart rate responses during sustained attention, and are not as easily distracted by a secondary stimulus (Casey & Richards, 1988; Richards, 1985b, 1987, 1989a). Infant baseline RSA magnitude is positively correlated with visual recognition memory scores (Linnemeyer & Porges, 1986) and heart rate responses during visual habituation (Richards, 1985a). These studies suggest that RSA may be indexing a general attentional capacity, such as an ability to engage in intensive cognitive processing.

An understanding of the relation between respiratory sinus arrhythmia (RSA) and sustained attention involves the relationship between RSA and vagal parasympathetic activity. RSA is the variability in heart rate that occurs at the same frequency as breathing, with heart rate acceleration shortly after the beginning of inspiration, and heart rate deceleration shortly after the beginning of expira-

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tion. RSA may be quantified by several methods (e.g., Grossman & Wientjes, 1986; Porges, 1986a, 1986b; Richards, 1986), and is a value proportional to the change in heart rate (or heart period) occurring within each breath, or at the same frequency as breathing. The co-occurrence of heart rate and respiration is controlled by rhythmic activity in the brainstem respiratory centers (Grossman, 1983; Lopes & Palmer, 1976; Spyer, 1979), and is mediated via efferent innervation from the vagus nerve to the heart (Anrep, Pascual, & Rossler, 1935; Katona & Jih, 1975; Porges, McCabe, & Yongue, 1982). Thus, RSA is a measure of vagal parasympathetic activity on the heart. And, it may be related to behavioral responses involved in attention that are under parasympathetic control (Porges, 1976, 1980).

Attention-linked heart rate responses in infants may be related to concurrent respiratory sinus arrhythmia (RSA) as well as baseline RSA. Patterns of heart rate responding might occur concurrently with changing RSA during attention. Alternatively, RSA itself may not mediate the heart rate change, but serve as an index of parasympathetic activity. Most researchers assume that heart rate responses during attention are primarily mediated by vagal parasympathetic activity rather than by sympathetic influence (e.g., Graham, 1979; Somsen, Jennings, & van der Molen, 1988). Given that RSA is positively related to vagal parasympathetic influence on the heart, attention-linked heart rate responses due to parasympathetic activity may be indexed with RSA.

There may also be developmental changes in the attention-heart rate variability relation. Heart rate variability increases over the first year of life, including respiratory sinus arrhythmia (Harper et al., 1978; Katona, Frasz, & Egbert, 1980; Richards, 1985b, 1987; Watanabe, Iwase, & Hara, 1973). Because attentional responses are coupled to RSA or the level of vagal parasympathetic activity, the co-occurring relations between attention and RSA may change with age.

The present study of heart rate variability during attention in infants had two goals. The first goal was to determine whether there was a reliable change in heart rate variability during visual attention in infants from 14 to 26 weeks of age. Two measures of heart rate variability were used: the standard deviation of heart rate (Porges et al., 1973), and an index of respiratory sinus arrhythmia (Porges, 1986a, 1986b; Porges & Bohrer, 1990). The second goal of the study was to determine the relation between changes in RSA and heart rate across different phases of visual attention. One part of the second goal was to examine the changes in RSA during stimulus orienting, sustained attention, and attention termination (see Casey & Richards, 1988; Graham, 1979; Graham, Anthony, & Zeigler, 1983; Porges, 1976, 1980; Richards, 1988a). Another part of the second goal was to determine whether the attenuated response of heart rate to a second stimulus during the phase of attention termination was related to concurrent RSA.

Methods

Subjects

Infants were recruited from birth notices published in a Columbia, South Carolina newspaper. The infants were full term, defined as having birthweight of greater than 2500 grams and gestational age of 38 weeks or greater based on the mother's report of her last menstrual cycle. The parents reported that their infant had no prenatal or perinatal medical complications. A cross-sectional design was used to sample 30 infants with 10 each at ages 14, 20, and 26 weeks. The mean testing ages of the infants were 100.31 days (SD = 3.69), 142.45 days (SD = 3.42), and 185.57 days (SD = 4.23), respectively. Fourteen additional subjects were tested but became fussy, sleepy, or inattentive during the experimental trials and were not included in the analysis. Technical or recording difficulties during the testing of 7 other subjects resulted in the exclusion of their data as well.

Apparatus

The infant was held in its parent's lap approximately 51 cm from the center of a black and white 49 cm TV monitor (19 in. TV). A single light-emitting diode (LED) was located on the bottom center of the TV screen, and blinked at a rate of 3.33 Hz when turned on. The area around the TV monitor was covered with a neutrally colored cloth in order to block extraneous visual information. A video camera lens was located above the TV, and a monitor located in an adjacent room was used by an observer to record infant fixations.

The visual stimuli were presented in a 30 cm square area on the TV monitor, subtending approximately 32° visual angle. The first stimulus was a recording of a *Sesame Street* TV program, and was presented on each trial. The "second onset" stimulus was one of three computer-generated visual stimuli. These consisted of a checkerboard pattern of 1.27 cm checks (3° visual angle per check), a series of computer-generated, concentric squares, or a small box shape moving across a diamond. The *Sesame Street* TV program and the computer-generated patterns have both been found in pilot testing and previous research (e.g., Richards, 1988a) to elicit initial visual fixation durations of at least 10 seconds for the three testing ages.

Procedure

Respiration and the electrocardiogram (ECG) were recorded for a 5-min baseline with the infant sitting on its parent's lap on a couch. The infant was awake

and alert during the baseline. Interaction between the parent and infant was not restricted during this time, and many parents talked with the infant during this period. The parent was then seated in a chair in the viewing area with the infant on the parent's lap facing the monitors. Respiration and the ECG were recorded for 5 s with no stimulus, and then the LED was turned on. A *Sesame Street* program was presented when the infant looked in the direction of the LED or TV. Recording of the ECG and respiration continued through a heart rate deceleration, and for 10 s following the return of heart rate to prestimulus level. A heart rate deceleration was defined as five successive beats with longer heart periods than the median heart period of the five heartbeats preceding the presentation of the primary stimulus. The return of heart rate to the prestimulus level was defined as five successive beats with shorter heart periods than the median prestimulus heart period, and following a heart rate deceleration. Trials were restarted if no heart rate deceleration occurred within 10 s of stimulus onset (17% of all trials), or if the infant looked away from the TV before the return of heart rate to the prestimulus level or was not looking when the second onset stimulus was presented (23% of all trials).

There were three types of experimental trials: control, immediate second onset, and delayed second onset. A "control" trial consisted of the presentation of the *Sesame Street* program through 10 s following the return of heart rate to prestimulus level. The screen then went blank for 5 s. An "immediate second onset" trial consisted of the presentation of the *Sesame Street* program until the return of heart rate to the prestimulus level. A computer-generated visual pattern replaced the *Sesame Street* program on the TV screen for 5 s, and then the screen went blank for 10 s. The "delayed second onset" trial consisted of the presentation of the *Sesame Street* program through 5 s following the return of heart rate to prestimulus level, followed by the onset of the second stimulus for 5 s and then a blank screen for 5 s. The three types of trials were randomly presented in 3-trial blocks. Each infant received a minimum of two 3-trial blocks, up to a maximum of four 3-trial blocks. The computer-generated stimuli were randomly presented in 3-trial blocks.

Quantification of Physiological Variables

For the recording of ECG, Ag-AgCl electrodes were placed on the infant's chest using disposable electrode collars. The ECG was digitized at 1000 Hz (each ms). The R-wave was identified in the ECG, and heart period was defined as the duration between successive R-waves in the ECG. Heart rate was computed on an "interval-by-interval" basis. This was done by assigning values of heart rate to equal intervals weighted by the proportion of time the beat occupied the interval. Three variables were quantified from both the baseline and experimental trials: mean heart rate of the 0.5-s intervals, standard deviation of 0.5-s intervals, and standard deviation of filtered 0.5-s intervals. This was calculated using a 500-ms interval (0.5-s by 0.5-s intervals). Means and standard deviations were calculated with the original data. Standard deviations of filtered 0.5-s intervals were calculated by first quantifying heart rate on a 0.1-s by 0.1-s basis for the respiratory sinus arrhythmia (RSA) measure. The 0.1-s values were transformed by a bandpass filter (see *Appendix A*) which passed variability from 0.49 to 1.92 Hz (29.4–115.2 bpm). The resultant variable was then "sampled" at 0.5-s intervals (every fifth 0.1-s value used), and the standard deviation of this was taken.

Respiration was recorded in both the baseline and experimental trials in order to determine whether it was closely related to respiratory sinus arrhythmia. Respiration was measured using a pneumatic chest cuff, and a pneumatic respiration transducer (Grass Instruments) quantified thoracic circumference changes due to respiration. The respiration signal was digitized on-line at 50 Hz by an IBM PC XT computer. Respiration period was quantified by locating the end-expiration points in the digitized records (20-ms accuracy), and defined as the interval between end-expirations. Each subject's mean respiration period in both baseline and experimental trials fell within the range defined by the bandpass filter for the estimate (0.49–1.92 Hz). Eighty-five percent of the individual breaths in the baseline minutes had periods within this range. More than 96% of all breaths fell within this frequency interval in the different attention phases. Respiratory period and heart rate were significantly negatively correlated in both the baseline and experimental trials, but respiratory period was not correlated with the standard deviation of the raw or filtered data for either baseline or experimental trials (see *Appendix B*).

Experimental Design for Statistical Analysis

The data for the response to the first stimulus were summed in 5-s intervals: *prestimulus:* the average from three periods was taken, from 5 s immediately preceding the onset of the first stimulus, and from 6 s following stimulus termination on the control and immediate second onset trials; *stimulus orienting:* 5 s immediately following first stimulus onset; *sustained attention:* 5 s immediately following stimulus orienting, but only if heart rate remained below the median of the five heartbeats that were below the median prestimulus beats; *pre-attention termination:* 5 s immediately preceding return of heart rate to prestimulus levels; *attention termination:* 5 s immediately following the return of heart rate to prestimulus levels. The response to the onset of the second stimulus was taken from the 10 s immediately following the return to prestimulus levels, which included the responses occurring on the control trial, the immediate second onset trials, and the delayed second onset trials.

Four types of variables were tested: mean heart rate in the 5-s phases, standard deviation of the 0.5-s values in the 5-s phases, standard deviation of the filtered 0.5-s values in each 5-s phase, and mean heart rate in the 0.5-s by 0.5-s intervals. Intervals effects were corrected

with the Huynh-Feldt correction for heterogeneity of covariances (Huynh & Feldt, 1970), and the linear, quadratic, and cubic orthogonal polynomial trends of the intervals effects were tested. For the 0.5-s by 0.5-s data, when data were directly compared with the prestimulus intervals, the raw data were used. When the prestimulus was not involved in the comparison, the mean of the 5-s prestimulus interval was subtracted from each of the 0.5-s values, indicating change from prestimulus level. Post hoc tests were corrected for error rate with a Scheffé adjustment. It was found in previous research that infants with large magnitude baseline respiratory sinus arrhythmia (RSA) show larger heart rate responses during visual attention than those with lower RSA magnitude (e.g., Richards, 1987). For the current study, low and high RSA groups were defined with median splits within each age on the filtered variable from the baseline.

Results

Heart Rate Defined Phases

The 0.5-s heart rate variables were analyzed with an Age(3) × Phase(5) × Intervals(10) ANOVA, and the variability variables were analyzed with an Age(3) × Phase(5) ANOVA. All variables showed a main effect of phase (p's<.001). This was expected because of the operational definition of these phases based on the heart rate pattern. For the 0.5-s by 0.5-s intervals, there were significant intervals effects (p's<.001) and Intervals × Phase interactions (p's<.001). The only significant effect involving age was on the filtered data variability ($F(2/27)=3.73$, $p<.05$). The mean of the variability of the filtered data increased with age (for example, prestimulus $\bar{X}$'s = 1.28, 1.43, and 2.03, for 14, 20, and 26 week olds, respectively).

Figure 1 shows the mean heart rate for all subjects changing over the attention phases. Heart rate decelerates rapidly in the first 5 s, remains low during the next 5 s, at some later point returns to baseline, overshoots the baseline level, and then slows down during the attention termination phase. This pattern was in accord with the operational definition of the attention phases. Post hoc tests compared the prestimulus intervals with each of the attention phases. The intervals effect was different in the prestimulus and the stimulus orienting, pre-attention termination, and attention termination phases (p's<.05). Mean heart rate over the 5 s was different in the prestimulus and sustained attention phases ($p<.05$), but the intervals effects were the same for these two phases.

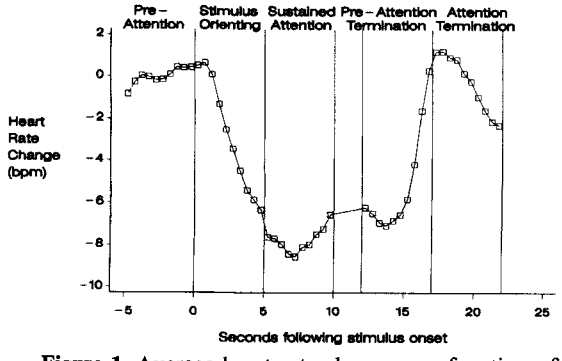

Figure 1. Average heart rate change as a function of seconds following the first stimulus onset during the heart rate defined attention phases.

Table 1

Standard deviations of raw and filtered heart rate during the heart rate defined attention phases

Phases	SD of Raw Heart Rate	SD of Filtered Heart Rate
Prestimulus	3.73	1.70
Stimulus orienting	4.40*	1.35*
Sustained attention	3.60	1.37*
Pre-attention termination	3.97	1.29*
Attention termination	3.46	1.37*
6 to 10 seconds poststimulus	3.67	1.53

*Significantly different from the prestimulus value.

The variability measures that correspond to these mean changes are presented in Table 1. Post hoc tests were performed for the phases' effects by comparing each prestimulus mean with corresponding phases' means. Table 1 indicates which of the phases' standard deviations were different from the prestimulus standard deviation. A significant increase occurred in overall standard deviation from the prestimulus to the stimulus orienting phase, which was based on the large mean shift during stimulus orienting. The variation of the filtered data showed significant decreases in the stimulus orienting, sustained attention, pre-attention termination, and attention termination phases. Heart rate variability returned to prestimulus levels by 6–10 s following stimulus termination.

Baseline High and Low RSA Infants

The effect of baseline respiratory sinus arrhythmia (RSA) on the responses was tested with an Age(3) × RSA(2) × Phases(4) × Intervals(10) ANOVA. The RSA × Phase interaction only approached statistical significance ($p=.087$). Post hoc tests showed that the low RSA group had a smaller heart rate response in the sustained attention intervals than the high RSA group ($p<.05$). It is believed that this interaction only approached statistical significance because of the smaller sample size, resulting in lower power than in previous studies (cf. Casey & Richards, 1988; Richards, 1987, 1989a) for the statistical test of the interaction.

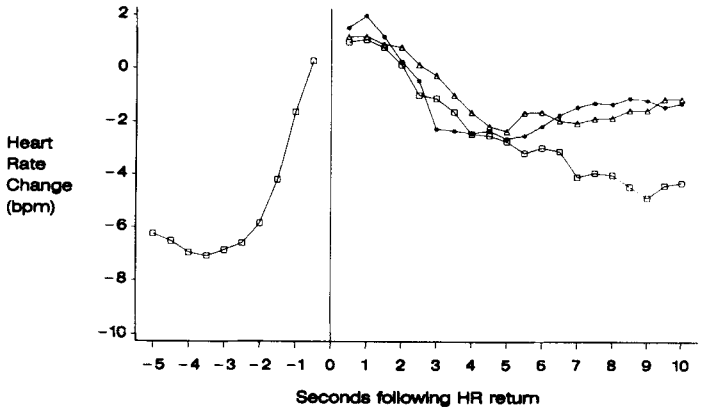

Figure 2. Average heart rate change as a function of seconds following the return of heart rate to prestimulus level for control ("△"), immediate second stimulus onset ("●"), and delayed second stimulus onset ("□") trials.

The raw and filtered standard deviation measures in the attention phases were each tested with an Age(3) × RSA(2) × Phases(5) ANOVA. There was a significant RSA effect only on the standard deviation of the filtered data ($F(1/241)=3.54$, $p<.05$). Baseline low and high RSA infants continued to have low and high RSA in the experimental trials. There were no RSA × Phase interactions, indicating that the changes in heart rate variability occurring during attention phases were equal in the low and high RSA infants. Correlations were performed between baseline and attention measures of mean heart rate, standard deviation of heart rate, and standard deviation of filtered heart rate data. Table 2 presents these correlations. Heart rate and the standard deviation of the filtered variable were significantly correlated from the baseline to the experimental trials. The correlation between the baseline RSA measure, and heart rate mean in the attention phases, ranged from −.36 to −.42, similar in magnitude to the correlation between baseline heart rate and baseline RSA.

Heart Rate Response to Second Stimulus Onset

The response to the onset of the second stimulus was analyzed with an Age(3) × Trial Type(3) × Intervals(20) ANOVA. There was a significant intervals effect for heart rate ($F(3/81)=23.97$, $p<.001$), and the Trial Type × Intervals interaction approached statistical significance ($p=.068$). Figure 2 shows the heart rate response to the second stimulus. Heart rate on the control and immediate second onset trials decreased in the first 5 s, and then began to return to prestimulus levels in the second 5 s. Heart rate on the delayed second onset trials continued decreasing in the second 5 s when the second stimulus was presented. According to post hoc comparisons, heart rate on the control trials did not differ from heart rate on the immediate second onset trials, but did differ from heart rate on the delayed second onset trials ($p<.05$).

The variability measures from the intervals following the return to the prestimulus level were analyzed for the control, immediate second onset, and delayed second onset trials with an Age(3) × Trial Type(3) ANOVA. There were no significant main effects or interactions involving the trial type effect, indicating that these variability measures were not different from each other on the control, immediate second onset, and delayed second onset trials.

Analyses were done using the baseline measure of respiratory sinus arrhythmia (RSA) to obtain low

Table 2

Correlations between variables from the baseline recording and attention phases

Baseline Variables	Correlations				
	Prestimulus	Onset	Sustained Attention	Pre-Attention Termination	Attention Termination
Mean heart rate	.75**	.71**	.78**	.73**	.74**
Standard deviation of raw data	.41*	−.17	.54**	.39*	.29*
Standard deviation of filtered data	.35*	.52**	.54**	.42**	.44**

*$p<.05$, **$p<.01$.

and high RSA groups, for the seconds following the return of heart rate to prestimulus level, for the response to the second stimulus. There were no main effects or interactions of the baseline RSA variable with the trial types or intervals.

High and Low RSA During Attention Termination

In view of the fact that the heart rate variability changed during the response to the second stimulus, we looked at high and low amounts of heart rate variability in the intervals immediately preceding the second stimulus onset. Respiratory sinus arrhythmia (RSA) was obtained on the control condition and the immediate second onset condition from the last 1.5 s of pre-attention termination, and 1 s of attention termination. RSA was obtained on the delayed second onset condition from the 2.5 s preceding stimulus onset. Based on RSA during this interval, the trials were split into high and low RSA trials, and an Age(3) × RSA(2) × Intervals (20) ANOVA was done separately for each of the three conditions. There were no significant main effects or interactions of RSA on the control and the immediate second onset trials. However, there were significant RSA × Intervals interactions for heart rate ($F(3/72)=4.55$, $p<.01$) on the delayed second onset trials.

Figure 3 shows the heart rate responses for the three conditions split by the magnitude of respiratory sinus arrhythmia (RSA) preceding the second stimulus onset. The responses on the control and immediate second onset conditions were the same for high and low RSA infants. The response of the low RSA infants on the delayed second onset trials was the same as the responses of both groups on the control and immediate second onset trials.

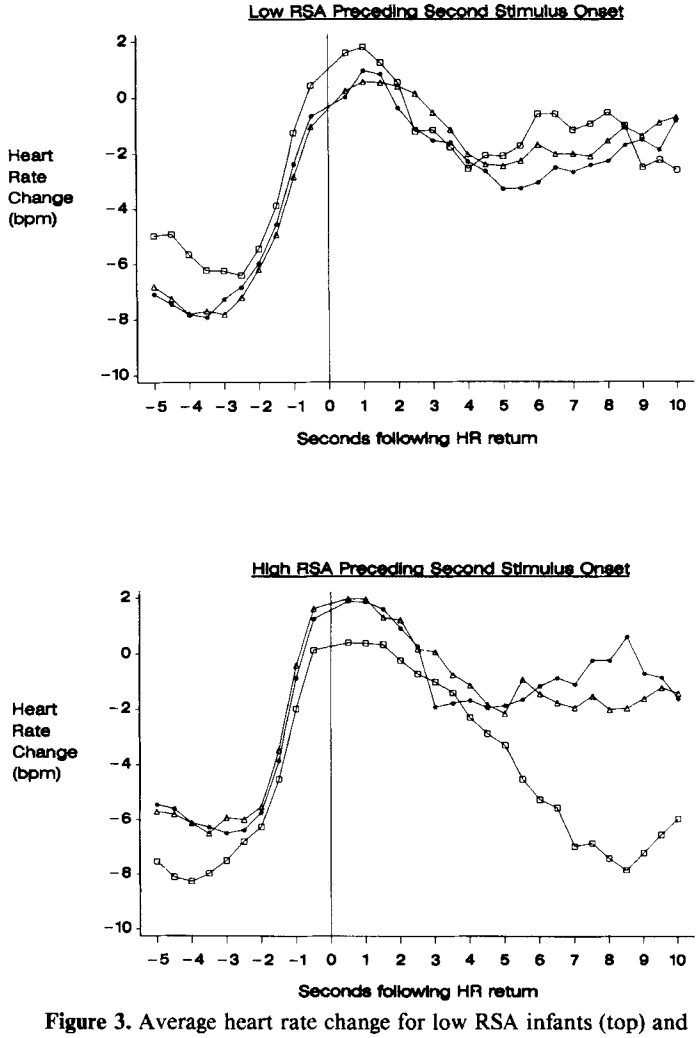

Figure 3. Average heart rate change for low RSA infants (top) and high RSA infants (bottom) as a function of seconds following the return of heart rate to prestimulus level for the control ("△"), immediate second stimulus onset ("●"), and delayed second stimulus onset ("□") trials.

However, for the delayed second onset condition for those trials when RSA was high, there was a large heart rate deceleration at the onset of the second stimulus. The trials on which extent of RSA was high produced heart rate changes equal in magnitude to the original heart rate response in the orienting phase (compare Figure 3 with Figure 1).

Discussion

Heart rate and heart rate variability showed reliable responses during the phases of attention. The pattern of heart rate response to the visual stimulus replicated previous work with infants of these ages (e.g., Casey & Richards, 1988; Richards, 1987). The heart rate response was used to define phases of attention in which to examine heart rate variability responses. The only reliable effect on heart rate variability across the attention phases was a decrease in respiratory sinus arrhythmia (RSA) in all attention phases, returning to prestimulus level in the interval of 5–10 s following stimulus termination. Baseline RSA was positively correlated with the magnitude of heart rate deceleration during sustained attention, but was not related to the heart rate response during the other phases. Baseline RSA was also not related to the heart rate variability responses during any of the attention phases. The heart rate response to the second stimulus was attenuated if the second stimulus occurred immediately upon the return of heart rate to its prestimulus level, compared to the initial heart rate response, or the heart rate response for the delayed second onset trial. The concurrent RSA magnitude was positively correlated with the heart rate response to the delayed second stimulus. There were no differences in the heart rate response to the second stimulus onset as a function of the magnitude of baseline RSA.

The results of this study are consistent with a model that postulates the existence of sequential phases of attention indexed by the heart rate response. We can speculate on the vagal activity occurring during these attention phases based on the mean heart rate and heart rate variability responses. The large deceleration of heart rate during stimulus orienting, and the sustained lowering of heart rate during sustained attention, is consistent with the hypothesis that attention-linked heart rate responses are caused by large vagal outflow increasing the period of the heartbeat. The heart rate activity during attention termination is consistent with vagal efferent flow diminished below prestimulus levels, allowing heart rate to increase above prestimulus levels. Vagal activity may take several seconds to return to prestimulus levels, and has a "refractory period" during which the response to new stimulation is attenuated.

The decrease in respiratory sinus arrhythmia (RSA) during stimulus orienting and sustained attention seems paradoxical with the proposed increase in vagal activity on the heart during these phases. This paradox may be resolved by distinguishing between the brainstem control of RSA, and higher cortical control of attention-linked, vagally mediated heart rate responses. RSA is caused by brainstem mechanisms involved in the control of rhythmic breathing activity (Grossman, 1983; Spyer, 1979). The brainstem respiratory centers affect vagal efferent control of heart function by alternately inhibiting and releasing from inhibition the vagal activity on the heart (Lopes & Palmer, 1976). Heart rate responses during attention originate in cardio-inhibitory centers in the neocortex, including the frontal cortex and the limbic system. These higher centers directly affect the vagus nerve, which results in heart rate deceleration during orienting and attention. Apparently, during stimulus orienting and sustained attention, cortical efferent traffic controlling vagal activity is stronger than, or inhibits, the gating activity of the brainstem respiratory centers. This results in a decrease in the measured RSA, even though vagal activity is very high. *Relative* magnitude of the individual differences in baseline vagal tone, measured by RSA, was conserved during the attention phases. This was evidenced by the positive correlation between baseline RSA and RSA in the experimental phases. *Absolute* magnitude of vagal parasympathetic outflow during attention may have been indicated best by the change in heart rate during these initial phases.

Testing age was significantly related to baseline respiratory sinus arrhythmia (RSA) magnitude, and RSA during the experimental trials. Baseline RSA increased with age. This is consistent with several studies showing an increase in RSA over this age range (e.g., Harper et al., 1978). As in previous studies (Richards, 1987, 1989a), mean heart rate change during sustained attention was correlated with baseline RSA, with high RSA infants showing a prolonged sustaining of the lowered heart rate during sustained attention compared with the low RSA infants. Similarly, the heart rate response during sustained attention increased with age. Heart rate variability responses during attention, however, were not different for the three age groups, or different for low and high baseline RSA groups. It has been argued (Richards, 1989a) that the developmental changes in heart rate responding during sustained attention are a result of concurrent developmental changes in RSA. Given the lack of a relation between baseline RSA and heart rate variability responses, the lack of a link between age changes in RSA and different amounts of RSA involvement during attention was expected.

The heart rate variability response during attention in infants in this study was different from the responses found in other studies with adults or school-aged children. Studies with adults or with older children, using the standard deviation of the raw heart rate as a measure of variability, have reported a decrease in heart rate variability during the sustained phase of attention (Coles, 1972; Porges & Humphrey, 1977; Porges & Raskin, 1969; Walter & Porges, 1975). Studies in which the frequencies of heart rate variability were decomposed (e.g., respiratory sinus arrhythmia (RSA) at respiration frequency; Traube-Hering-Mayer rhythm near 0.1 Hz, which involves blood pressure and baroreceptor feedback) found that the lowest frequency rhythms were more closely related to attention than was RSA (Hyndman & Gregory, 1975; Mulder & Mulder, 1981). In the present study, only RSA decreased during the stimulus orienting and sustained attention phases. The standard deviation of the raw heart rate, which contains variability due to low and high frequency variations, was not different in either the sustained attention or prestimulus phases. Porges et al. (1973, 1974) also reported that heart rate variance in newborn infants did not decrease from 6 to 10 seconds following stimulus onset.

The difference between the changes in respiratory sinus arrhythmia (RSA) in the present study and the broadband variability changes in studies with adults or school-aged children may be based on different attentional systems being tapped, or methodological differences. The mean heart rate change during attention in adults is only 1–2 bpm, compared to the 8–10 bpm changes found in infants reported in this and other studies (Richards, 1987). The large increase in vagal activity that results in a large heart rate decrease may overshadow the mechanisms controlling RSA, resulting in the RSA decrease. This study did not find evidence of changes in the low frequency band such as have been found in studies with adults (Coles, 1972; Hyndman & Gregory, 1975; Mulder & Mulder, 1981). This suggests that the type of sustained attention elicited in this study with infants may not be directly comparable to the long intervals of sustained attention observed in adults.

A direct comparison between studies of adult attention and infant attention is not straightforward. Many studies with adults have used tasks that result in longer intervals of attention (e.g., 30–60 s) than can reasonably be used with infants. The low frequency rhythms that change during adult attention cannot be reliabily quantified with durations that are usually elicited in infant attention research. For example, for infant subjects, reliability estimates from 5-s or 15-s samples for mid-frequency heart rate variability (0.117–0.253 Hz) were less than .7, and for low-frequency variability (0.038–0.097 Hz) were less than .5 (Richards, 1989b). Acceptable levels of reliability of low- and mid-frequency heart rate variability can be obtained in infants only with 25-s or 60-s sampling durations. In order to study these low frequency components of heart rate variability in infants, situations would have to be designed in which sustained alertness could be elicited over much longer intervals than found in the present study. Perhaps changing the experimental stimuli (e.g., live persons, or audiovisual stimuli) or the experimental context could accomplish this.

The difference between the heart rate response to the first stimulus and the one to the second stimulus is consistent with the hypothesis of a refractory period following attention termination during which an optimal heart rate deceleration cannot occur. This finding replicates findings from two previous studies (Casey & Richards, 1988, 1989) in which heart rate deceleration to a new stimulus was attenuated during attention termination. It also parallels findings from studies of the heart rate response to stimulus offset (Berg, 1972, 1974; Casey & Richards, 1989; Richards, 1988b). A large heart rate response at stimulus offset occurs only if the heart rate response to the stimulus has been completed for some time (Casey & Richards, 1989; Richards, 1988a). It appears that the attention termination phase must be completed for several seconds before another heart rate orienting response can occur.

The physiological basis for this refractory period may involve vagal control of heart functioning. The return of heart rate to its prestimulus level during attention termination and the continuing low level of respiratory sinus arrhythmia (RSA) during this phase suggest that high vagal activity during the previous phases has ceased, but vagal activity has not returned to prestimulus levels. The data from the delayed second stimulus onset imply that the refractory period continues as long as vagal activity is low. When vagal activity returns to its baseline level, a normal heart rate response to stimulus onset occurs. The effect of the RSA on the delayed second stimulus onset trials was not due to individual differences in RSA, because baseline RSA did not distinguish responding on those trials. As in several previous studies (Casey & Richards, 1988, 1989; Richards, 1985b, 1987, 1988a, 1989a), the heart rate response during the stimulus orienting and attention termination phases was not distinguished by baseline RSA. It was the transient return of RSA to normal levels for some infants but not others

that distinguished the end of the refractory period in attention termination.

The cause for the diminished vagal activity, and attenuated heart rate onset response during the refractory period, is not known. One possibility is that there are central processes controlling attention that actively disengage attention during the attention termination phase, and actively inhibit optimal attention processes for some duration. Posner postulates such a process called "attention disengagement," which occurs at shifts of attention and consists of disengaging attention from one stimulus and focusing on another. Attention disengagement lasts only a brief time in adults, and during that time attention cannot be re-engaged in the same location or sensory channel (Posner & Cohen, 1982; Posner & Presti, 1987). This process is an active one, and is controlled by an area in the parietal lobe of the cortex (Posner, Petersen, Fox, & Raichle, 1988). Such a process might be shown in the present paradigm by demonstrating that inhibition of infant cognitive functions (e.g., recognition memory, learning, stimulus detection) occurs concomitantly with the attenuated heart rate response during this refractory period. The inhibited cognitive functioning and the diminished heart rate response should also be closely associated with attenuated respiratory sinus arrhythmia (vagal level) during the attention termination phase. Such an association would be inconsistent with a model that attributed the attenuated heart rate response to other processes (e.g., sympathetic arousal, vagal fatigue).

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Appendix A

The standard deviation of a filtered variable was used to assess respiratory sinus arrhythmia (RSA) in the baseline and experimental trials. For long intervals of time (e.g., minimum 30 s), spectral analysis methods assess RSA well (Harper, Scalabassi, & Estrin, 1976; Harper et al., 1978; Porges et al., 1982; Richards, 1986). Previous studies (e.g., Casey & Richards, 1988; Richards, 1989a), which examined the relation between baseline RSA and attention, used this measure of RSA because the baseline was 5 min in duration. For short intervals of time (5–10 s), spectral methods cannot be used. Porges (1986a, 1986b; Porges & Bohrer, 1990) recently developed an attractive alternative to spectral analysis. This method consists of a detrending of the heart rate series, followed by a filtering of the signal to remove extremely low and high frequencies. The low and high frequencies are chosen to include infant respiratory frequencies. The variability of the filtered series thus represents variability in heart rate occurring at the same frequency as respiration, i.e., RSA.

In the present study, the heart rate values were transformed by a polynomial filter which passed variability from 0.49–1.92 Hz (29.4–115.2 bpm). The fre-

quency response of this filter was tested with broadband white noise, pure and compound sine functions, and a combination of sine functions and white noise. The transfer function of this filter had a steep rise near 0.49–0.6 Hz, a 1.0 gain (flat response) from 0.6 to approximately 1.25 Hz, and a gradual decline from 1.25 through 1.92 Hz. This transfer function compares well to the filter used by Porges (Porges & Bohrer, 1990). The spectral estimates of respiratory sinus arrhythmia (RSA) used in previous research (Richards, 1987) and the standard deviation of the filtered variable are highly correlated (e.g., $r=.79$ for the current study; $r=.90$, Richards, 1986; $r=.87$, Richards, 1989b). The spectral and filtered RSA estimates were negatively correlated with baseline heart rate (r's = $-.53$ and $-.45$, respectively, p's$<.01$), reflecting the common vagal influence on heart rate and RSA. The reliability of this measure in 5-s epochs was recently shown to be good compared with 1-min estimates, or 1-min estimates of RSA via spectral analysis methods (Richards, 1989b). For example, in that study, Cronbach's *alpha* for five 5-s samples was .75, for five 1-min samples was .85, and for five 1-min samples of spectral analysis was .82.

Appendix B

Several analyses were conducted to determine whether heart rate or heart rate variability during the baseline, or changes during attention, could be attributed to respiration period changes. In summary, respiration period was correlated with mean heart rate, but not with heart rate variability measures. Changes in respiration period during the attention phases could not be shown to be statistically related to changes in any of the heart rate derived measures.

The correlations between respiration period and the heart rate variables were examined. During the baseline, respiration period was significantly negatively correlated with heart rate ($r=-.59$), but not significantly correlated with heart rate standard deviation ($r=.13$) or standard deviation of the filtered variable ($r=.16$). Respiration period and heart rate were significantly negatively correlated over all heart rate defined attention phases (r's ranged from $-.33$ to $-.61$ for 13 possible epochs, average $r=.43$, Fisher's $Z=2.37$). The correlation between respiration period and the standard deviation of the raw values for these epochs was not significant (r's ranged from $-.25$ to .39, average $r=.14$, Fisher's $Z=0.71$), nor was the correlation between respiration period and the standard deviation of the filtered variable (r's ranged from $-.11$ to .37, average $r=.11$, Fisher's $Z=0.59$). Thus, the pattern of correlations between respiration period and the heart rate variables was similar during the baseline and experimental trials.

Respiration period lengthened during the experimental trials from the prestimulus to the stimulus periods by 34 ms (first stimulus), 46 ms (immediate second stimulus onset), and 39 ms (delayed second stimulus onset). Three analyses were done to determine whether these changes were significantly related to heart rate mean and variability changes occurring during the attention periods. First, the correlations between respiration period change from the prestimulus and heart rate mean or variability changes in the same epochs were computed. The respiration period change scores were not correlated significantly with any of the heart function change scores for any of the attention phases (e.g., stimulus orienting r's ranged from $-.10$ to .07; sustained attention r's ranged from $-.12$ to .10; pre-attention termination r's ranged from $-.05$ to .09; and attention termination r's ranged from $-.20$ to .10).

Second, subjects were grouped into one of three groups depending on their respiration period changes from prestimulus through stimulus orienting, sustained attention, pre-attention termination, and attention termination. These groups were: 1) longer respiration period in all attention phases compared to prestimulus intervals; 2) shorter respiration period in all attention phases; and 3) mixed respiration changes. This was used as a factor in ANOVAs with Age(3) and Phase(4) with heart rate mean and variability, and Age(3), Phase(4), and Intervals(10) for 0.5-s heart rate values. There were no significant effects of the respiration split variable, either in main effect or interaction, on the heart rate and heart rate variability measures. We also looked at several other ways of splitting the data on the respiration changes, including median splits on the differences, extreme (greater than 2 standard deviations) increases or decreases in respiration period, and increases or decreases in respiration period in the first breath of the attention phase. None of these groupings based on respiration changes had significant effects (main effects or interactions) on the heart rate mean and variability measures.

Third, respiration period mean was used as a covariate that changed on a trial-by-trial basis with heart rate mean and the variability measures. As expected from the correlations, respiration period as a covariate was significantly related to heart rate mean, but not standard deviation of the raw heart rate or standard deviation of the filtered heart rate. The inclusion of respiration as a covariate did not affect the pattern of statistical significance for any of the heart rate variables.

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