

Peripheral Stimulus Localization by Infants: Attention, Age, and Individual Differences in Heart Rate Variability

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The effect of attention to a focal stimulus on 3- to 6-month-old infants' peripheral stimulus localization was examined. Fixation was engaged on a central visual stimulus, and a stimulus was presented in the periphery after discrete time intervals (0 to 12 s) or until changes in heart rate (HR) occurred. Peripheral stimulus localization occurred less frequently when a significant HR deceleration had occurred (sustained attention) than when HR had returned to its prestimulus level (attention termination). A signal detection analysis showed that response bias was altered by attention and that during inattention infants with high HR variability were more likely to shift fixation away from the central stimulus independently of the presence of the peripheral stimulus. These data suggest that infant attention affects decision processes for continuing focal stimulus fixation rather than peripheral stimulus discriminability.

Peripheral stimulus localization by young infants has been studied for many years. Early research used peripheral stimulus localization to determine the extent of the visual field with localization perimetry (Aslin & Salapatek, 1975; de Schonen, McKenzie, Maury, & Bresson, 1978; Harris & MacFarlane, 1974; MacFarlane, Harris, & Barnes, 1976; Tronick, 1972). Such localization perimetry defined the effective visual field as the eccentricity at which an infant will shift fixation from a central location to a peripheral location in the presence of a peripheral stimulus. For example, Harris and MacFarlane found that 1- to 2-day-old infants would direct fixation from a central location to a peripheral stimulus up to 25° to 30° in the periphery but not 35° or 40°. Seven-week-old infants would shift fixation to the peripheral stimulus at the two larger eccentricities. Recent research has used peripheral stimulus localization (and nonlocalization) as a measure of attention to a central stimulus in young infants (Atkinson, Hood, Braddick, & Wattam-Bell, 1988; Atkinson, Hood, Wattam-Bell, & Braddick, 1992; Hood & Atkinson, 1993; Richards, 1987, 1994; Richards & Casey, 1992) or to control infant fixation direction in order to study covert visual attention (e.g., Hood, 1993; Johnson, Posner, & Rothbart, 1994). In this study, we showed that in 3- to 6-month-old infants attention to a central visual stimulus inhibits localization of a stimulus in the peripheral visual field and that individual differences in heart rate variability affect central attention and peripheral stimulus localization in young infants.

The presence of a stimulus in the central visual field affects the responses to a stimulus in the peripheral visual

field. A procedure used in early peripheral stimulus localization studies (e.g., Tronick, 1972) to direct fixation to the central visual field was to present a stimulus until the infant looked at it and then to display the peripheral stimulus in addition to the focal stimulus. The eccentricity to which localization would occur was much smaller in this competitive situation than when there was no central visual stimulus or when the central stimulus disappeared before or at the same time as peripheral stimulus onset (Aslin & Salapatek, 1975; Harris & MacFarlane, 1974). One- and 2-month-old infants, for example, make directionally appropriate eye movements toward a peripheral stimulus at 40° more than 50% of the time in the absence of a central stimulus but less than 20% of the time in the presence of a central stimulus (e.g., Aslin & Salapatek). In addition to decreasing the effective visual field size, the presence of the focal stimulus increases the latency to make a directionally appropriate eye movement if the focal stimulus remains on (Aslin & Salapatek; Atkinson et al., 1988, 1992; Hood & Atkinson, 1993; Richards, 1987, 1994).

One interpretation of the effect of a focal stimulus in the central visual field on peripheral stimulus localization has emphasized an inability to disengage attention from the focal stimulus. This interpretation emphasized the model of Posner and Petersen (1990), in which visual attention must be disengaged from the central visual stimulus before saccades to the peripheral stimulus can occur (see Hood & Atkinson, 1993). In this interpretation, shifting fixation from the central to the peripheral stimulus requires an active process to control attention disengagement, presumably controlled by brain systems located in the posterior parietal lobes (Fisher & Breitmeyer, 1987; Posner & Petersen). An exciting developmental finding in this regard is that younger infants (1 to 2 months) seemed to be more affected by the presence of the central stimulus than older infants (3 to 6 months), even so far as to be unable to shift fixation to the peripheral stimulus if focal attention was occurring (Atkin-

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son et al., 1988, 1992; Hood & Atkinson; Johnson, Posner, & Rothbart, 1991).

An alternative interpretation of the effect of a stimulus in the central visual field on peripheral stimulus localization has emphasized the focusing of attention to the central stimulus as the cause of this effect. The first systematic manipulation of central stimulus presence concluded with the interpretation that young infants demonstrated internal control of fixation by not localizing the peripheral stimulus because attention was engaged by the central stimulus (Harris & MacFarlane, 1974). Similarly, Finlay and Ivinskis (1984) and Richards (1987) have interpreted the longer latencies to shift fixation toward the peripheral stimulus as an indication of the level of attention to the central stimulus. When infants show by sustained lowered heart rate that sustained attention to the central stimulus is engaged, a peripheral stimulus presented for a fixed interval is missed (Finlay & Ivinskis, 1984) or the latency to localize a continuing peripheral stimulus is very long (Richards, 1987). Unresponsiveness to the peripheral stimulus is interpreted as indicating an enhanced level of attention to the central stimulus. In contrast, when infants show by lack of a significant heart rate response that attention is not engaged, localization of a fixed-duration peripheral stimulus is likely (Finlay & Ivinskis, 1984) or localization of a continuing peripheral stimulus occurs quickly (Richards, 1987). Focal stimulus attention results in a spatial selectivity for fixation, with fixation being directed primarily toward the location in which the attended stimulus occurs.

A problem with these attentional interpretations of the competitive influence of central stimulus fixation on peripheral stimulus localization is the absence of an attention measure that is independent of visual fixation. Studies showing a lengthening of the latency to localize fixation interpreted the fixation shift from the central to the peripheral location as concordant with the attention shift from the focal stimulus to the peripheral stimulus (Hood & Atkinson, 1993; Richards, 1987). Similarly, in studies in which the peripheral stimulus was presented for a fixed interval and the percentage of localization was the dependent variable (e.g., Aslin & Salapatek, 1975), the fixation shift toward the peripheral location (or the absence of a fixation shift) was merely another measure of fixation direction. Using localization as the measure of attention does not provide an independent (or satisfactory) measure of attention engagement during central stimulus exposure.

A potential alternative attention engagement measure is heart rate changes occurring in response to the visual stimulus. Heart rate changes occurring during visual fixation have been used to distinguish the infant's initial reaction to a stimulus (stimulus orienting), focused attention to the stimulus (sustained attention), and attention disengagement from the stimulus (attention termination) (Berg & Richards, in press; Graham, 1979; Graham, Anthony, & Ziegler, 1983; Porges, 1980; Richards & Casey, 1991, 1992). These attention phases are associated with distinctive heart rate responses and behavioral activity. For example, infant visual sustained attention is characterized by a sustained lowered mean heart rate and lack of distractibility from central

stimulus fixation in the presence of a peripheral stimulus (Richards, 1987; Richards & Casey, 1991, 1992). Attention termination is the next phase of attention. Heart rate returns to prestimulus levels during this phase. The infant may continue to fixate on the stimulus during this phase but is more easily distracted or may voluntarily look away from a visual stimulus during this phase (Casey & Richards, 1988; Richards, 1987). Thus, heart rate changes occurring in the presence of a central stimulus could provide a visual attention measure that does not use the infant's shift from the central location to the peripheral location.

This study had two primary goals. The first goal was to examine how infants' attention to a central stimulus affected the peripheral stimulus localization probability. Infants were tested at 14, 20, and 26 weeks of age (3, 4.5, and 6 months, respectively). An interesting visual stimulus was presented until sustained attention occurred or until attention waned. A peripheral stimulus was presented for 2 s during sustained attention or while the infant was fixating on the central stimulus but under focal inattention. Attention level was manipulated by presenting the central stimulus and waiting for the heart rate to slow down before presenting the peripheral stimulus (sustained attention) or waiting for the heart rate to return to its prestimulus level before presenting the peripheral stimulus (attention termination). It was hypothesized that peripheral stimulus localization percentages should be significantly lower under conditions of focal stimulus attention engagement than when attention had disengaged even though fixation had continued.

The second goal was to examine the relationship between peripheral stimulus localization and individual differences in interbeat interval (IBI) variability. Several studies with infants have shown that resting levels of IBI variability are associated with the cardiac changes occurring during visual sustained attention (Richards, 1987, 1994; Richards & Casey, 1991, 1992), with recognition memory (Linnemeyer & Porges, 1986), and with general developmental level (Fox & Porges, 1985). Of special relevance to this study are experiments showing that the duration of fixation toward a central stimulus in the presence of a distracting peripheral stimulus is correlated with levels of respiration-related variance in IBIs (respiratory sinus arrhythmia; RSA) (e.g., Richards, 1987). High-RSA infants, who have extended heart rate responses during sustained attention, look longer than low-RSA infants at a central stimulus in the presence of a distracting stimulus during sustained attention. High- and low-RSA infants' distraction latencies are the same during stimulus orienting and attention termination. Mean IBI level, variance of IBIs, and the amplitude of RSA were recorded during a 5-min baseline period. Infants were divided into high- and low-heart-period-variability groups on the basis of principal-component scores derived from these variables. It was expected that the high-IBI-variability infants would have lower localization levels than the low-IBI-variability infants, particularly during sustained attention. From 3 to 6 months of age, IBI lengthens (heart rate decreases) and RSA increases (Harper, Hoppenbrouwers, Serman, McGinty, & Hodgman, 1976; Harper et al., 1978; Katona, Frasz, & Egbert, 1980; Richards, 1987; Richards &

Casey, 1992). This fact indicates an increase in parasympathetic cardiac control over this age range. The ages chosen (14, 20, and 26 weeks) were based on these changes.

This study included several procedures important for experimental control. The sequential nature of the heart-rate-defined attention phases (stimulus orienting, sustained attention, and attention termination) was controlled for by also presenting peripheral stimuli at fixed delays from the central stimulus onset. It was expected that under these fixed-delay conditions, heart rate level at the time of presentation, as an index of sustained attention or attention termination, would be more closely related to peripheral stimulus localization levels than to delays from the central stimulus onset. It has been noted that studies of peripheral stimulus localization often fail to use appropriate control conditions (e.g., correct rejections) and fail to measure incorrect fixations (false alarms) and so cannot eliminate the probability that spontaneous fixation shifts that are not attributable to peripheral stimuli occur (Maurer & Lewis, 1991). Therefore, fixation during the entire stimulus presentation was recorded to include appropriate control conditions for estimating sensitivity and response bias parameters for a modified signal detection analysis. Sensitivity in signal detection analyses has been interpreted as reflecting sensory and information acquisition processes (stimulus characteristics, discriminability, and state of the sensory system), whereas response bias reflects subject-controlled decision processes (Baird & Noma, 1978; Green & Swets, 1966; Massaro & Cowan, 1993). Thus, it might be possible to identify the locus of the attention effect on peripheral stimulus localization with signal detection parameters. Finally, these control conditions should allow evaluation of the heart rate changes occurring in peripheral stimulus trials and in control trials to determine whether heart rate changes reflect the processing of peripheral stimuli without fixation toward them (cf. Finlay & Ivinskis, 1984, 1987; Maurer & Lewis, 1991).

Method

Infants

Infants were recruited from birth notices published in a Columbia, South Carolina, newspaper. The infants were full term, defined as having a birth weight of more than 2,500 g and a gestational age of 38 weeks or more, on the basis of the mother's report of her last menstrual cycle. The infants had no acute or chronic pre- or perinatal medical complications and were in good health at the recording session.

A total of 155 infants were sampled cross-sectionally at 14 ($n = 49$, $M = 100.9$ days, $SD = 3.57$ days), 20 ($n = 52$, $M = 143.4$ days, $SD = 4.86$ days), or 26 ($n = 54$, $M = 187.0$ days, $SD = 5.65$ days) weeks of postnatal age. Of the 155 infants, 20 had the baseline recording but were used in pilot testing for the experimental protocol, 17 had the baseline recording but were not used in the peripheral localization trials (sleepy, fussy, or not interested during testing protocol), and 28 did not successfully complete the minimum number of peripheral localization trials (fussy, missing data, or experimental protocol mistakes). Ninety infants for whom baseline data were obtained and who completed the minimum

number of localization trials therefore constituted the study population, with 30 infants each at the three testing ages (14 weeks, $M = 100.8$ days, $SD = 3.67$ days; 20 weeks, $M = 142.6$ days, $SD = 4.63$ days; and 26 weeks, $M = 187.9$ days, $SD = 5.62$ days).

Apparatus

The infant was held in his or her parent's lap approximately 51 cm from the inner edges of two black-and-white 49-cm (19-in.) TV monitors. Each screen's center was 56 cm from the infant's eyes, and the far edge was 70 cm away. The plane of the TVs was parallel to the infant's eyes. The TVs subtended 88° of visual angle; each TV subtended 44° of visual angle. There was a visual angle of 48° from each monitor's center. A single light-emitting diode (LED) that blinked at 3.33 Hz when turned on was located on the bottom center of each TV screen. A neutral-color material covered the surrounding area. A video camera was centered above the TVs, and in an adjacent room an observer judged infant fixations on a TV monitor. The session was recorded on videotape with a time code in order to synchronize physiological and experimental information for analysis.

The focal stimuli were patterns shown on the TV monitors (a Sesame Street TV program or computer-generated patterns, e.g., a series of computer-generated concentric squares of various sizes, a flashing checkerboard pattern, or a small box shape moving across a diamond). The stimuli were presented in a 30-cm² area, subtending approximately 32° of visual angle. These patterns had been found to elicit first fixation durations longer than 10 s (e.g., Richards, 1997).

The peripheral stimulus consisted of a 2° white square that repeatedly traveled vertically from the top to the bottom of the TV. The square moved at approximately 15°/s and thus traversed the TV approximately once in the 2.5 s that it was on during the localization epochs. For the 20- and 26-week-old infants, the peripheral stimulus was located 15° off center in the display area and thus was 24° from the focal stimulus display area edge and 39° from the focal stimulus center. Pilot testing showed that at this eccentricity 14-week-old infants rarely localized the peripheral stimulus in the presence of the focal stimulus. The location of the peripheral stimulus for that age group was reduced to 7° off center (16° from the edge and 31° from the focal stimulus center), producing satisfactory localization frequencies.

Procedure

Respiration and an electrocardiogram (ECG) were recorded for a 5-min baseline while the infant sat on his or her parent's lap on a couch. The parent then sat on a chair in the viewing area so that the infant was facing the TV monitors. The peripheral stimuli were presented for four trials in order to acquaint the infant with their location. These trials consisted of a 5-s period followed by a LED presentation on one TV. The purpose of the LED presentation was to attract the infant's fixation toward the TV. When the infant looked at the LED or toward the TV, the LED was turned off and the peripheral stimulus was presented on the other TV. The peripheral stimulus remained on until it was judged that the infant looked in its direction and then remained on for an additional 5 s.

The experimental trials consisted of the focal stimulus presentation and then the peripheral stimulus presentation at several delays. Between each trial there was a 5-s period with a blank screen. Then, the LED on a TV was presented until the infant looked toward that TV. A stimulus was displayed when the infant looked in the direction of the LED or the TV. After the focal

stimulus onset, the peripheral stimulus was presented for 2 s at predetermined delays, and the focal stimulus remained on. If the infant looked toward the peripheral stimulus while it was on (or within 1 s of its being turned off), the focal stimulus was turned off and the peripheral stimulus remained on for 5 s. If the infant continued to look at the focal stimulus, the trial was continued and additional peripheral stimuli were presented. The focal stimulus remained on through the first heart rate deceleration and for 5 s after the heart rate had returned to the prestimulus level, and then the TV was turned off.

The delays at which the peripheral stimulus was presented after focal stimulus onset were defined by heart rate changes or by time delays. The time-defined delays were 0 s (simultaneous presentations) and 2, 4, 6, 8, 10, and 12 s. The heart-rate-defined delays were heart rate deceleration, heart rate deceleration plus 2 s, heart rate acceleration, and heart rate acceleration plus 2 s. The delay for the heart rate deceleration epochs occurred after a significant heart rate deceleration, evaluated on-line as five successive beats with IBIs each longer than the median for the five prestimulus beats (i.e., sustained attention). The delay for the heart rate deceleration plus 2 s epochs consisted of the heart rate deceleration followed by an additional 2 s. The delay for the heart rate acceleration epochs occurred when the heart rate returned to its prestimulus level, evaluated on-line as five beats with IBIs shorter than the median for the five prestimulus beats (i.e., attention termination). The heart rate acceleration must have followed a significant heart rate deceleration. The delay for the heart rate acceleration plus 2 s epochs consisted of the heart rate acceleration followed by an additional 2 s. Additional prestimulus trials that consisted of the peripheral stimulus presented alone were carried out.

Each focal stimulus presentation could have multiple peripheral stimulus presentations. The 13 delay types (7 time defined, 4 heart rate defined, and 2 prestimulus) were randomly ordered in blocks. The first delay type for each focal stimulus presentation was the next available one unused in the current block. If the infant did not localize the peripheral stimulus for this first delay type, delay types that could occur at later delays were chosen from the current or successive block and were presented. These additional delay types were chosen with three conditions: (a) time-defined and heart-rate-defined types were alternated if available in the current block; (b) peripheral stimulus presentations had to be separated by at least 2 s; and (c) the focal stimulus presentation was ended if the criterion of heart rate acceleration plus 5 s was met. Each infant received a minimum of two peripheral stimulus presentations at each delay type.

Testing was done only if the infants maintained an alert, awake state during the entire procedure (eyes open, no fussing or crying, and responding to the protocol). Trials were restarted if no heart rate deceleration occurred within 10 s after fixation onset. They were also restarted if the infant looked away from the TV before the heart rate had returned to the prestimulus level or was not looking when the second stimulus was presented. Additional focal stimulus trials without the peripheral stimulus were carried out through 5 s after the heart rate had returned to the prestimulus level. These presentations occurred after every four focal stimulus presentations and were included to prevent the association of temporal sequencing between the focal and peripheral stimuli but were not included in any of the analyses.

Measurement and Quantification of Physiological Variables

The ECG was recorded by placing Ag-AgCl electrodes on the infant's chest and was digitized at 1,000 Hz (each millisecond)

with a microcomputer. A computer algorithm identified on-line the QRS complex in the ECG, and IBI was defined as the duration between successive R waves in the ECG. This evaluation was made on-line within 30 to 60 ms after the R-wave occurrence for the heart-rate-defined epochs. For quantitative analyses, artifact correction was done with the Cheung (1981) and Berntson, Quigley, Jang, & Boyesen (1990) algorithms, along with visual inspection of suspect beats.

Respiration was measured with a pneumatic chest cuff (Grass Instruments) and was digitized on-line at 50 Hz (each 20 ms) during the baseline period. The peak and trough of the digitized recording, representing inspiration and expiration, were identified by computer algorithms. Artifacts were eliminated by viewing the respiration recording on computer displays for each identified breath. Respiration frequency was quantified to determine the modal frequency for RSA quantification.

Individual differences in heart rate variability in past research were dealt with by separation of high- and low-variability groups with a single variable, for example, a band-pass filter measure (vagal tone; Porges, 1992) or a spectral analysis measure (Richards, 1994). However, parasympathetic cardiac control might be estimated best with mean heart rate and RSA in some linear combination¹ (e.g., multiple regression; Grossman & Kollai, 1993; Kollai & Mizsei, 1990). Thus, four variables were computed from the baseline recording: the IBI average, the standard deviation of the IBI values, a time domain quantification of RSA, and a frequency domain quantification of RSA. Each variable was calculated separately from the baseline 5 min, and an average of the five calculations was taken. The IBIs from the baseline recording were proportionally assigned to 100-ms intervals in order to achieve adequate resolution of the frequency domain RSA measure. The IBI average and the standard deviation of the IBI values were computed. The time domain measure of RSA was the standard deviation of the IBI series transformed with a band-pass filter (Porges, 1985; see Richards, 1995, or Richards & Casey, 1991). The raw IBI series was filtered with a symmetric trigonometric moving average low-pass filter (0.49 Hz), a subtraction resulting in a high-pass series, and then with a low-pass filter with weights to eliminate higher frequencies (1.92 Hz). The resulting series had variability in a fixed frequency interval (0.49 to 1.92 Hz) that includes infant respiration frequency. The standard deviation of this series was the time domain RSA measure. The frequency domain measure of RSA was computed with spectral analysis (Richards, 1995). The periodogram was computed with the Fast Fourier Transform from values assigned to the first 512 0.1-s intervals of each of the minutes (cosine tapered), yielding a frequency resolution of 0.01953 Hz. A Daniell smoothing algorithm was applied to the periodogram to obtain the power spectrum. The

¹ RSA is a measure of increases and decreases in heart rate that occur in phase with respiration expiration and inspiration. The cooccurrence of heart rate and respiration is controlled by rhythmic activity in the brain stem respiratory centers (Grossman, 1983; Lopes & Palmer, 1976; Spyer, 1979) and is mediated by 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. Parasympathetic cardiac control is also reflected in mean IBI length, IBI length being positively related to the extent of parasympathetic activity on the heart. Thus, the mean and variability of the IBI length are related to the level of parasympathetic cardiac control, and parasympathetic cardiac control might be estimated best with mean heart rate and RSA in some linear combination (Grossman & Kollai, 1993; Kollai & Mizsei, 1990).

variable was defined as the power estimates summed over 0.1953 Hz (11.71 breaths per minute) and centered at the modal respiration frequency for that baseline minute. The metric for the spectral analysis variable is the root-mean-square variation of the IBI values at the respiration frequency ranges. The time and frequency domain measures represent variability in the IBI values occurring at the same frequency of respiration, that is, the operational definition of RSA.

Peripheral Stimulus Localization Judgments

Using a TV monitor in an adjacent room, a single observer judged the infant's fixation direction during the experiment in order to control the experimental protocol. Each session was judged off-line. A time code recorded on the videotape allowed the judgment to have millisecond accuracy, although resolution was limited to a single video frame scan (half the total frame length = ~16 ms). The latencies for the first four presentations were determined by judging when the infant looked toward the peripheral stimulus and pressing a key that caused the computer to read the time code on the videotape. The observer judged the looks during the other trials as follows: looking at the right TV, looking at the left TV, or looking away. The time code on the videotape was used by the computer to determine whether the peripheral stimulus was present (localization or nonlocalization) or not (control, correct rejection, or false alarm) and, in the case of localizations, to determine the latency from the peripheral stimulus onset to the beginning of the look. Interrater agreement between two raters was computed for 5 infants at each age. The raters agreed about the occurrence of a fixation away from the focal stimulus in 98% of the trials, and correlations for latency judgments were greater than .95. Because of this high agreement level, a single observer's ratings were used for the data analysis.

Experimental Design for Statistical Analysis

Epochs in the peripheral stimulus localization trials were classified into five categories: localization, nonlocalization, control, correct rejections, and false alarms. *Localizations* were defined as the infant correctly moving fixation toward the TV with the peripheral stimulus within 3 s of its onset (referred to as *hits* in the *Signal Detection Analysis* section of Results). *Nonlocalizations* were defined as the infant continuing fixation on the TV with the focal stimulus for 3 s after peripheral stimulus onset (referred to as *misses* in the *Signal Detection Analysis* section of Results). *Correct rejections* were defined as any epoch in any trial in which a delay type criterion was met (either heart rate or time), a peripheral stimulus was not present, and the infant continued to fixate on the focal stimulus. *False alarms* were defined as looks away from the TV monitor with the focal stimulus toward the other TV monitor when no peripheral stimulus was being presented. For the IBI change analyses, *control* epochs were selected from focal stimulus fixation periods during which a peripheral stimulus was not present but that matched the time- or heart-rate-defined delay type for an existing peripheral stimulus presentation. One control epoch was chosen for each peripheral stimulus presentation (localization and nonlocalization epochs) by selection of an epoch that had a similar delay type in the nearest trial before or after the presentation, with the condition that a control epoch could be used only once. These categories were obtained from any trial with a peripheral stimulus presentation and any trial that was restarted but not from the trials with a focal stimulus presentation alone.

The results were analyzed in factorial designs. Testing age (14,

20, and 26 weeks) was a between-subjects factor. Heart period variability (low and high) was a between-subjects factor that was constructed by use of a median split within ages for the principal-component scores derived from an analysis of the four variables quantified from the baseline IBIs (see the *Baseline Heart Rate and RSA* section of Results). Delay type (seven time defined, four heart rate defined, and two prestimulus) was a within-subjects factor.

The frequencies of localization and nonlocalization were analyzed as a categorical dependent variable by use of linear categorical models. A signal detection analysis was done with the epochs defined as localizations (hits), nonlocalizations (misses), correct rejections, and false alarms. Because only one or two peripheral stimulus presentations for each delay were given to an infant, the parameters used in the signal detection analysis (d' and β) could not be calculated for individual infants for analysis with parametric statistics (e.g., analysis of variance [ANOVA]). Alternatively, the four categories' frequency distributions were analyzed with linear categorical models, and signal detection parameters were calculated with the frequencies summed over effect categories.

The linear categorical models used to analyze the localization frequencies and the signal detection analysis use maximum-likelihood estimates for response function parameters that consist of generalized logits (log ratios) of the marginal probabilities for the independent variable effects (PROC CATMOD; SAS Institute, Cary, NC). The maximum-likelihood optimization procedure results in an information matrix that provides the numerical basis for a χ^2 value for the independent variable effects based on the Wald test, with df equal to the number of parameters used from the information matrix for the effects. The Wald test represents the significance of the change in the χ^2 value for the overall model that would result if the response function for a particular effect were dropped from the model. Post hoc tests were Wald tests that were obtained by examination of the information matrix parameters from the overall model. The maximum-likelihood estimates for these models assume asymptotic normal distributions for the response functions. Post hoc tests, therefore, were chosen to obtain categories with a sufficiently large sample size to meet this assumption.

Results

Baseline Heart Rate and RSA

The mean of the baseline 0.1-s \times 0.1-s IBI values, the natural logarithm of the standard deviation of those values, the time domain (band-pass filter), and the frequency domain (spectral analysis) for the RSA measures² were analyzed with a one-way multivariate ANOVA with Age ($N = 3$) as the factor for all of the infants ($N = 155$). There was a statistically reliable age effect, Wilks's $\Lambda = .7843$, $F(8, 298) = 4.81$, $p < .0001$. The Age factor significantly affected all variables except for the standard deviation of the

² The natural logarithm for these three variance measures better approximated normally distributed variables than did untransformed values. Therefore, the natural logarithm for these three variables was used for quantitative analyses (e.g., multivariate ANOVA and principal-component analysis). The untransformed value of the standard deviation of the IBIs is reported in Table 1, whereas the RSA measures (band-pass filter and spectral analysis) are reported as natural logarithms in Table 1 for consistency with previous studies (Porges, 1992; Richards, 1994).

IBI values (Table 1). The average IBI became larger with age (decline in heart rate), and the RSA increased with age.

To choose a measure representing heart rate variability, I examined the correlations between the baseline variables. All of the measures were significantly correlated with each other (Table 2). The correlation matrix for these measures was tested for homogeneity across ages. The null hypothesis that the correlation matrix was identical across the three ages could not be rejected, $\chi^2(20, N = 152) = 26.13, p = .1614$. Thus, although the mean levels changed across ages, the relationships between the measures did not. A principal-component analysis including all infants ($N = 155$) resulted in a first principal component that accounted for 66% of the variance in the correlation matrix (Table 3). The computed score for the first principal component changed significantly across the three ages, $F(2, 152) = 9.77, p < .0001$ ($M_s = -.409, -.042, \text{ and } .412$ for 14-, 20-, and 26-week-old infants, respectively), but the other component scores did not. For other analyses, the infants completing the testing protocol ($N = 90$) were separated into high- and low-heart-period-variability groups by use of a median split within ages for the first principal-component score.³ The M_d 's were $-.532, -.149, \text{ and } .369$ for 14-, 20-, and 26-week-old infants, respectively.

Localization and Nonlocalization of the Peripheral Stimulus

Localization latency. The peripheral stimulus in the first four trials was presented until the infant localized it. The latency to localize the peripheral stimulus in these trials was analyzed to determine whether the 3-s window for localizing the peripheral stimulus in the detection trials was appropriate and whether there were any preexisting latency differences between the low- and high-heart-period-variability groups. The average latency over all infants was 986.0 ms ($SD = 574.2$). More than 98% of the trials were shorter than 3,050 ms, suggesting that the 3-s window for infants' localizing the stimulus in the subsequent detection trials was appropriate. The latency to localize the peripheral stimulus in these trials was analyzed with an Age ($N = 3$; 14, 20, and 26 weeks) \times Heart Period Variability (low and

Table 2

Correlations Between Measures (All Significantly Different From Zero at $p < .001$) for Baseline Measures of Interbeat Interval (IBI) for Infants From 14 to 26 Weeks Old

Parameter	Correlation for the indicated parameter:		
	SD of IBI	Band-pass filter RSA estimate	Spectral analysis RSA estimate
Average IBI length	.483	.433	.458
SD of IBI		.529	.556
Band-pass filter RSA estimate			.851

Note. RSA = respiratory sinus arrhythmia.

high) \times Trials ($N = 4$) ANOVA. The age main effect approached statistical significance, $F(2, 84) = 2.94, p = .0593$. The localization latency decreased over the three ages ($M_s = 1,111.7, 1,014.7, \text{ and } 855.3$ ms for 14-, 20-, and 26-week-old infants, respectively). The heart period variability main effect and interactions were not significant, suggesting that no preexisting latency differences existed between the low- and high-variability infants.

Localization percentage. The localizations (hits) and nonlocalizations (misses) in the peripheral stimulus localization trials were analyzed. Figure 1 shows the localization percentage as a function of the delay conditions, for low- and high-heart-period-variability infants. A competition effect occurred in that peripheral stimulus localization in the prestimulus trial (no focal stimulus) occurred frequently (82.6%, $N = 196$), compared with that for the 0-s (immediate peripheral stimulus) delay epoch (30.4%, $N = 115$) or with that in the epochs in which a focal stimulus was present and the peripheral stimulus was delayed from 2 to 12 s (44.0%, $N = 729$) (Figure 1). There was a large difference between localization percentages in the heart rate deceleration (39.8%, $N = 236$) and acceleration (65.4%, $N = 226$) epochs (Figure 1).

The localization or nonlocalization categorical dependent variable was analyzed with epochs from time-defined and heart-rate-defined types. The delay epochs were divided into categories depending on whether they met the following heart rate change criteria: deceleration, deceleration plus 2 s, deceleration plus 4 s, acceleration, and acceleration plus 2 s. The a priori heart-rate-defined epochs were assigned to these categories by definition, whereas the time-defined

Table 1
Changes With Age (14 to 26 Weeks) and Probability of a Significant One-Way Analysis of Variance Effect for Baseline Measures of Interbeat Interval (IBI)

Parameter	Value (ms) at testing age (weeks)			<i>p</i>
	14	20	26	
Average IBI length	390.1	407.3	419.0	<.0001
SD of IBI	28.22	28.24	29.58	<.42
Band-pass filter RSA estimate	3.19	3.88	4.79	<.0001
Spectral analysis RSA estimate	2.55	3.33	4.43	<.0015

Note. RSA = respiratory sinus arrhythmia.

³ Several analyses were done with other variables derived from the IBIs of the baseline. These included a median split based on the average IBI length and one based on the spectral analysis measure (cf. Richards, 1994). The split based on IBI length was not as sensitive to the heart rate changes or the localization results as was the spectral analysis measure or the principal-component measure. The spectral analysis measure and the principal-component measure were equally sensitive to the heart rate changes. The median split for the principal-component measure was more sensitive than either of the other variables in affecting the pattern of results for the localization analyses.

Table 3
Principal-Component Analysis of Baseline Measures of Interbeat Interval (IBI) for Infants From 14 to 26 Weeks Old

Parameter	Value in the following principal-component analysis:			
	1	2	3	4
Average IBI length	.428	.737	-.386	.022
SD of IBI	.466	.313	.826	.018
Band-pass filter RSA estimate	.543	-.445	-.153	.695
Spectral analysis RSA estimate	.551	.399	-.143	-.718
Eigenvalue	2.644	0.686	0.520	0.148
Proportion variance	.661	.171	.130	.037

Note. RSA = respiratory sinus arrhythmia.

epochs were assigned to these categories depending on whether heart rate met the deceleration or acceleration criteria at the beginning of the time epoch. In addition to testing age and heart period variability effects, this analysis tested whether the simple passage of time within the deceleration or the acceleration phase affected localization behavior. These epochs, along with the prestimulus epoch and the 0-s (immediate) delay epoch, were analyzed with an Age ($N = 3$) \times Heart Period Variability ($N = 2$) \times Delay ($N = 7$) design with linear categorical modeling. The null hypoth-

esis of homogeneity among the marginal effects was rejected for the main effects of age, $\chi^2(2, N = 1,281) = 10.90, p = .0043$; delay, $\chi^2(6, N = 1,281) = 108.67, p < .0001$; and heart period variability, $\chi^2(1, N = 1,281) = 3.95, p = .0468$.

Table 4 shows the localization percentages for these conditions, separated by low- and high-variability groups. The delay effect was attributable to the difference between localization percentages in the prestimulus trial, the 0-s delay epoch, the epochs in which the heart rate deceleration criteria were met, and the epochs in which the acceleration criteria were met. The heart rate deceleration categories were not significantly different among themselves, and the heart rate acceleration categories were not significantly different among themselves (Table 4). Localizations remained infrequent as long as the heart rate deceleration criteria continued to be met and increased in frequency as soon as the acceleration criteria were met. The age effect reflected an increase in localization percentages from 14 (46.7%, $N = 493$) to 20 (48.0%, $N = 421$) to 26 (58.8%, $N = 367$) weeks of age. This effect occurred primarily in the heart rate deceleration categories (35.8, 39.5, and 51.9%, respectively) rather than in the other categories (55.6, 56.0, and 58.0%, respectively, in the heart rate acceleration categories).

Fewer localizations were made by the high-variability group than by the low-variability group during the heart rate deceleration epochs (Table 4). The two groups showed equivalent localization percentages in the prestimulus and

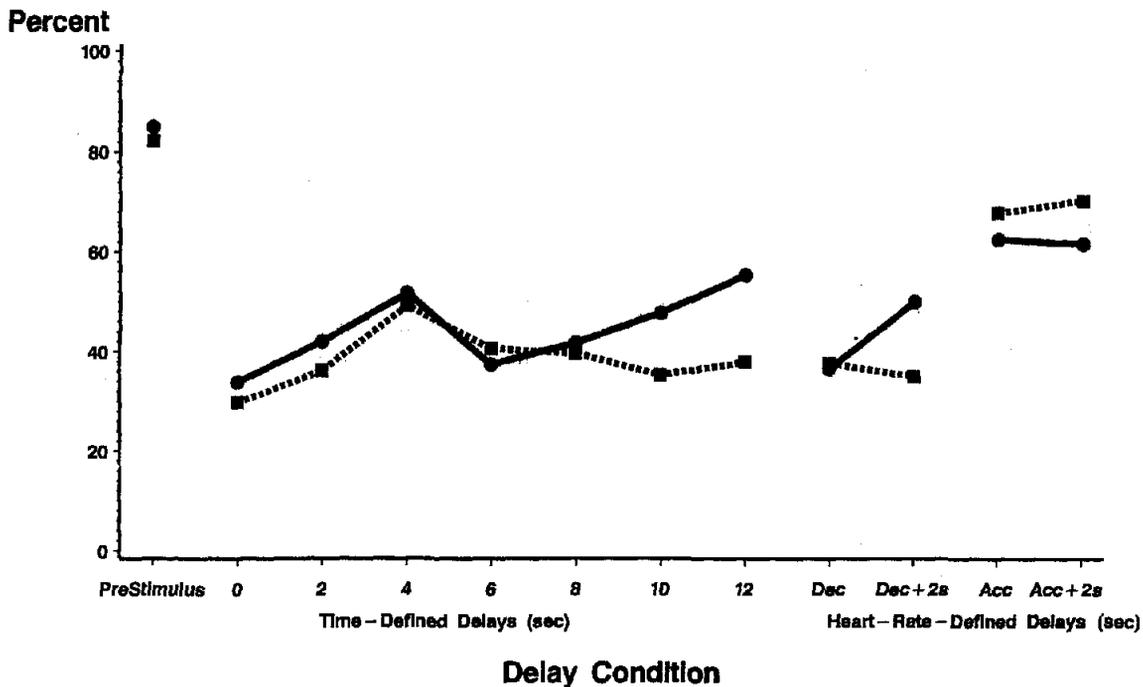


Figure 1. Frequency of peripheral stimulus localization as a function of delay type ($N = 196$ for prestimulus trials; $N = 106-132$ for delay trials; median $N = 118$). Circles represent low interbeat interval variability; squares represent high interbeat interval variability. Dec = deceleration; Acc = acceleration.

Table 4

Localization Percentages for Time-Defined and Heart-Rate-Defined Epochs Categorized by Time Within Heart Rate Deceleration and Time Within Heart Rate Acceleration Phases and Separated by Low- and High-Heart-Period-Variability Groups

Heart period variability	% localization (no. of epochs)						
	Before heart rate change		For post hoc categories defined by heart rate changes				
	No focal stimulus	0 s	Deceleration	Deceleration plus 2 s	Deceleration plus 4 s	Acceleration	Acceleration plus 2 s
Low	85.0 (100)	33.9 (59)	42.1 (128)	48.3 (91)	38.7 (80)	58.2 (91)	57.2 (103)
High	80.2 (96)	32.7 (56)	37.6 (125)	38.2 (84)	32.3 (71)	58.5 (94)	52.4 (103)

0-s delay epochs (Figure 1 and Table 4), and there was an increasingly larger differentiation between the two groups as the delay durations lengthened (Figure 1). The analyses shown in Table 4 indicate that the localization percentage differences between the two groups occurred mainly when the heart rate deceleration criteria were met.

Signal Detection Analysis

The epochs of the peripheral stimulus localization trials were classified into four categories for signal detection analysis: localization (hit), nonlocalization (miss), correct rejection, and false alarm. The signal detection analysis was done to determine whether the effect of attention to the focal stimulus on peripheral stimulus localization was attributable to a change in stimulus discriminability (d') or response bias (β) processes. All epochs were classified by detection type (hit, miss, correct rejection, or false alarm), the delay interval in which the epoch fell (early: 2.0 to 5.9 s; middle: 6.0 to 9.9 s; or late: 10.0 to 13.9 s), and the heart rate criterion status at the epoch onset (heart rate deceleration or heart rate acceleration). Signal detection parameters (percentages, d' , and β) were calculated by summing over infants within categories for which there was a statistically significant difference from the categorical modeling.

The four detection type categories were analyzed with an Age ($N = 3$) \times Heart Period Variability ($N = 2$) \times Delay ($N = 3$) \times Heart Rate Criterion ($N = 2$) design with linear categorical modeling. The null hypothesis of homogeneity among the marginal effects was rejected for age, $\chi^2(6, N = 3,246) = 21.88, p = .0013$; heart period variability, $\chi^2(3, N = 3,246) = 8.10, p = .0440$; delay, $\chi^2(6, N = 3,246) = 34.18, p < .0001$; heart rate criterion, $\chi^2(3, N = 3,246) = 42.63, p < .0001$; and an interaction between delay and heart rate criterion, $\chi^2(6, N = 3,246) = 27.68, p < .0001$.

The signal detection parameters are shown in Table 5. The heart rate criterion main effect was attributable to a difference in localization and false alarm percentages between epochs in which heart rate deceleration and acceleration were occurring. The localization percentage was smaller during heart rate deceleration than when the heart rate had returned to its prestimulus level. The false alarm percentage was larger when heart rate acceleration was occurring. The sensitivity parameter was equivalent in the

two epoch types, but there was a larger bias against responding when heart rate deceleration was occurring. The delay main effect and the interaction between delay and heart rate criterion were attributable to differences in the signal detection parameters across delay intervals during heart rate acceleration. For epochs in which heart rate had returned to its prestimulus level, there was an increase from the early to the late delay intervals in the false alarm percentage. Coupled with little change in the localization percentage, this finding led to a significant decline in the response bias parameter, indicating an increasing tendency to make a localization response in the late delay intervals when heart rate acceleration criteria had been met. Localizations and false alarms remained infrequent as long as the heart rate deceleration criteria continued to be met, resulting in no significant change in the signal detection parameters across delay intervals.

The age and heart period variability effects on the signal detection parameters were examined. The age effect resulted primarily from an increase across ages in the tendency to look away from the focal stimulus toward the other TV, regardless of the peripheral stimulus presence. This

Table 5

Localization Percentages and Signal Detection Parameters for All Trials, Categorized by Early, Middle, and Late Delay Categories and by the Status of the Heart Rate Change Criteria at the Time of the Selected Epoch

Heart rate criterion and delay interval (s)	% localization	% false alarm	d'	β
Deceleration				
2.0-5.9 ($N = 1,222$)	44.0	5.0	1.50	3.84
6.0-9.9 ($N = 730$)	37.9	4.0	1.44	4.40
10.0-13.9 ($N = 324$)	37.3	3.3	1.51	5.12
All ($N = 2,276$)	41.0	4.4	1.47	4.14
Acceleration				
2.0-5.9 ($N = 145$)	60.7	8.5	1.64	2.46
6.0-9.9 ($N = 502$)	60.0	10.5	1.51	2.13
10.0-13.9 ($N = 323$)	51.0	15.5	1.06	1.70
All ($N = 970$)	55.7	11.3	1.35	2.05
Combined				
2.0-5.9 ($N = 1,367$)	45.6	5.4	1.50	3.63
6.0-9.9 ($N = 1,232$)	47.1	6.6	1.43	3.09
10.0-13.9 ($N = 647$)	46.2	8.2	1.29	2.61

Note. N = number of epochs.

effect was shown by increases across ages (14, 20, and 26 weeks) in localization percentages (41, 43, and 56%, respectively) and false alarm percentages (3.6, 5.9, and 9.9%, respectively) and a resultant decrease in β values with age (4.84, 3.34, and 2.26, respectively). This effect was independent of the delay length or the heart rate criteria. The heart period variability effect on the signal detection parameters was of mixed direction. Peripheral stimulus localization in the heart rate deceleration epochs was much lower for the high-heart-period-variability infants than for the low ones but was equivalent in the heart rate acceleration epochs. However, the false alarm percentage for the high-variability infants was larger than that for the low-variability infants, particularly in the heart rate acceleration epochs (14.6 and 8.5%), and led to an overall smaller β value in the high-heart-period-variability group (2.77 and 3.80). Thus, relative to the low-variability infants, the high-variability infants continued to fixate on the focal stimulus when heart rate had decelerated but were more biased toward responding when heart rate had returned to prestimulus levels.

IBI Changes⁴

The IBI changes during the peripheral stimulus localization epochs are shown in Figures 2 and 3, plotted as 0.5-s \times 0.5-s values. The increasing IBI length in the heart-rate-defined epochs (Figure 2) was as expected from the experimental criteria for those trials, as was the return of IBI length toward the prestimulus level. In the time-defined epochs, there was an increase in IBI length for 3 to 4 s, then a stabilization of IBI length in the control and nonlocalization trials, and finally a return of IBI length to the prestimulus level in the localization trials (Figure 3).

IBI changes preceding localization epochs. The IBI values from the period preceding the categorized epochs in the peripheral stimulus localization trials were analyzed to determine whether IBI changes preceded localizations. The difference between the mean IBI level for the 2.5-s period immediately preceding the control, localization, and nonlocalization epochs and the mean IBI level for the 2.5-s period preceding the focal stimulus onset was analyzed. This change score for the heart-rate-defined epochs was analyzed with an Age ($N = 3$) \times Heart Period Variability ($N = 2$) \times Delay ($N = 4$) \times Localization ($N = 3$; control, localization, and nonlocalization) \times Intervals ANOVA. A similar analysis was done for the change score for the time-defined epochs. Only the interactions with the localization factor are reported, because the difference in IBI changes for the control, localization, and nonlocalization epochs was of interest.

For the heart-rate-defined epochs, there was a significant Delay \times Localization interaction, $F(6, 349) = 7.72, p < .0001$. This interaction was attributable to differences in the IBI levels during the localization epochs in the heart rate deceleration plus 2 s epochs. The control and nonlocalization epochs had equivalent IBI levels in the heart rate deceleration plus 2 s epochs (Figure 2). The IBI values

immediately preceding the epochs in which the peripheral stimulus was localized were different from those in the control and nonlocalization epochs and were returning to prestimulus levels in the heart rate deceleration plus 2 s epochs. For the time-defined epochs, there was a significant Delay \times Localization interaction, $F(12, 813) = 1.88, p = .0334$. Control, localization, and nonlocalization epochs had the same IBI levels for the 0-, 2-, and 4-s delay epochs (Figure 3). The IBI values were declining toward the prestimulus levels immediately before the epochs in which peripheral stimulus localization occurred for the later (6-, 8-, 10-, and 12-s) delay epochs (Figure 3).

IBI changes during the peripheral stimulus presentation. The IBI values during the peripheral stimulus presentation epochs were analyzed to determine whether IBI changes were different in the control and nonlocalization epochs; that is, did the peripheral stimulus affect IBI changes when the infants continued to fixate the focal stimulus? The values for the 3.0-s period during the control and nonlocalization epochs were analyzed as the difference between the mean IBI level for this period and the mean IBI level for the 2.5-s period preceding the focal stimulus onset. This change score for the heart-rate-defined epochs was analyzed with an Age ($N = 3$) \times Heart Period Variability ($N = 2$) \times Delay ($N = 4$) \times Localization ($N = 2$; control and nonlocalization) ANOVA. A similar analysis was done for the time-defined epochs. Only delays of 0 through 10 s were included in the time-defined epoch analysis because control trials for 12-s delays came only from trials in which the heart rate deceleration criterion had been met and the heart rate acceleration criterion had not yet been met.

There were no interactions with the localization factor in the heart-rate-defined epochs. This finding shows that IBI changes were the same for epochs in the presence of a peripheral stimulus when localization did not occur and control epochs with no peripheral stimulus (Figure 2). For the time-defined epochs, there was a Delay \times Localization interaction, $F(5, 1250) = 2.88, p = .0136$. For the earlier delay epochs (0, 2, 4, 6, and 8 s), there were no differences in IBI changes between epochs in the presence of a peripheral stimulus when localization did not occur and control epochs with no peripheral stimulus (Figure 3). For the 10-s epochs, the nonlocalization epochs had sustained IBI levels during the peripheral stimulus presentation, whereas the IBI levels in the corresponding control epochs were returning toward prestimulus levels (Figure 3). There was an Age \times Heart Period Variability \times Delay \times Localization interaction for the time-defined epochs, $F(10, 1250) = 1.92, p =$

⁴ Several analyses had missing data for infants for one or more of the factors in the design. For example, combinations of the localization factor and the delay type factor had cells that were not totally filled for individual infants. Because of the missing cells, the ANOVAs were computed with a general-linear-models approach that used nonorthogonal designs. The sums of squares (hypothesis and error) for the nested effects in the design were estimated by use of subjects as a class and by nesting of repeated measures (intervals and delay types) within this class variable. PROC GLM (SAS Institute) was used for the computations.

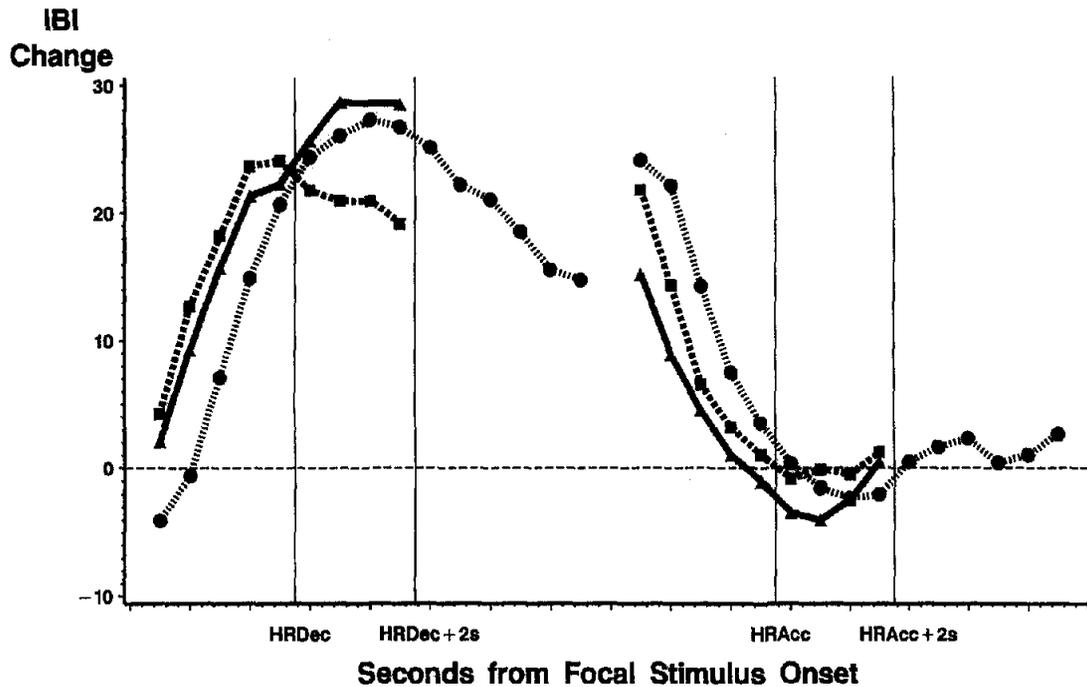


Figure 2. Interbeat interval (IBI) changes in heart-rate-defined epochs immediately before peripheral stimulus presentations as a function of peripheral stimulus detection. Squares represent localization; triangles represent nonlocalization; circles represent control. The data only come from the 2.5 s before the peripheral stimulus presentation. HRDec = heart rate deceleration (in seconds); HRAcc = heart rate acceleration (in seconds).

.0388. This interaction reflected the different IBI levels for the nonlocalization and control epochs for the older and high-variability groups only in the 6-s delay epochs. In large part, however, the control and nonlocalization epochs had the same IBI levels.

Discussion

Infants' localization percentages corresponded closely to the heart rate changes occurring at the time of peripheral stimulus presentations. This finding was shown by the lower localization percentage for the peripheral stimulus during epochs in which a significant IBI lengthening had occurred (sustained attention) than during epochs in which the IBI had returned to the prestimulus length (attention termination). This result occurred when the peripheral stimulus presentation was defined a priori (Figure 1) by IBI lengthening or shortening or when the presentations were defined by time intervals and the IBI levels were examined post hoc (Tables 4 and 5). If the peripheral stimulus onset delay was defined by a specific duration rather than by the IBI change, the return of the IBI to the prestimulus length preceded the epochs in which localization occurred. IBI length during control epochs with no peripheral stimulus was similar to IBI length during nonlocalization epochs (misses). Peripheral stimulus localization percentages during sustained attention to the focal stimulus were much

lower for infants with high IBI variability than for low-IBI-variability infants. A signal detection analysis suggested that changes in response bias accounted for the differences between attention and inattention conditions.

Several findings are consistent with the interpretation that heart rate changes occur before localizations. In the heart rate deceleration plus 2 s epochs, the IBI levels in localization epochs (hits) were returning toward the prestimulus level before the localization occurred (Figure 2). In the time-defined trials, the peripheral stimulus onset delay was not specifically defined by the IBI change. However, in those trials, the return of the heart rate to the prestimulus level preceded the epochs in which localization occurred (Figure 3). Control epochs (no peripheral stimulus) and nonlocalization epochs (misses) had equivalent IBI levels in heart-rate-defined (Figure 2) and time-defined (Figure 3) epochs. Similarly, the effects of the heart rate status on peripheral stimulus localization were not attributable simply to the sequential nature of the heart-rate-defined phases (i.e., heart rate deceleration before return of the heart rate to the prestimulus level). In the time-defined epochs, the heart rate level at the time of presentation was more closely related to peripheral stimulus localization levels than to delay from the central stimulus onset (Tables 4 and 5). These findings support the interpretations that the heart rate changes index attention status at the time of the peripheral stimulus presentation and that heart rate changes are independent of the

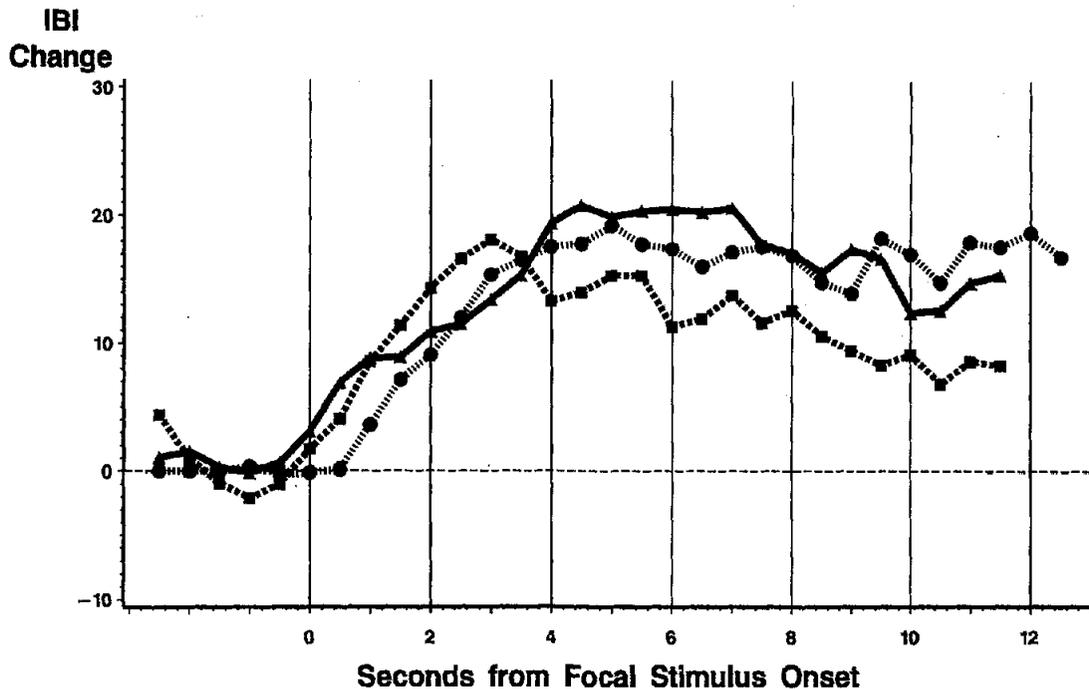


Figure 3. Interbeat interval (IBI) changes in time-defined epochs immediately before peripheral stimulus presentations as a function of peripheral stimulus detection. Squares represent localization; triangles represent nonlocalization; circles represent control. The data only come from the 2.5 s before the peripheral stimulus presentation.

peripheral stimulus themselves. These results contradict interpretations that the sustained heart rate deceleration in nonlocalization trials may be attributable to covert peripheral stimulus detection and response to the dual-stimulus configuration with a larger heart rate change (Finlay & Ivinskis, 1984, 1987; Maurer & Lewis, 1991).

The IBI changes during visual fixation can reasonably be interpreted as reflecting infants' sustained attention (IBI lengthening) and attention termination or inattention (IBI return to prestimulus length) (Berg & Richards, in press; Richards & Casey, 1992). This interpretation is based on studies of recognition memory, perception, and distraction by peripheral stimulus (e.g., Richards, 1987, 1997). These cardiac changes provide a visual attention engagement measure that does not rely on the fixation shifts that serve as the dependent variables. The experimental manipulations based on the IBI thus allow a causal interpretation of the effects of the heart rate change on the infants' fixation shifting. Focal stimulus attention results in sustained fixation on the central stimulus, and focal stimulus inattention allows fixation shifts to occur from the central to the peripheral location. This interpretation affirms early speculation (e.g., Harris & MacFarlane, 1974) that in young infants the central stimulus affects peripheral stimulus localization because of attention to the central stimulus. The attention interpretation is strengthened by affirmation of the converse proposition, that focal stimulus fixation has much less effect on peripheral stimulus localization when attention is not engaged.

The results of the present study are consistent with the conclusions of Finlay and Ivinskis (1982, 1984, 1987) and Richards (1987; see also Richards & Casey, 1992) that heart rate changes in response to the central stimulus indicate attention and have the spatial selectivity property of attention.

The peripheral stimulus localization percentage during sustained attention to the focal stimulus was lower for the infants with high IBI variability than for the low-IBI-variability infants. The effect primarily occurred when the IBI levels were significantly longer than the prestimulus IBI levels, indicating that sustained attention was occurring, as opposed to when the IBI levels had returned to their prestimulus length (Table 5). This finding implies that the high- and low-IBI variability groups differed in their localization behavior primarily during sustained attention rather than during attention termination or before the onset of sustained attention. Studies have shown that high-variability infants sustain their IBI change throughout the entire period of fixation (e.g., Richards, 1987). Thus, the high-IBI-variability infants sustain their heart rate response throughout fixation, and the IBI change is closely related to their continued fixation on the central stimulus in the presence of the peripheral stimulus. These findings are consistent with studies showing enhanced cognitive performance in high-RSA infants, including recognition memory (Linnemeyer & Porges, 1986), developmental level (Fox & Porges, 1985), and the latency to turn toward a peripheral stimulus during

sustained attention (Richards, 1987). The differences in IBI variability are relatively stable individual differences over several months in infancy (Richards, 1989, 1994; Fracasso, Porges, Lamb, & Rosenberg, 1994). The results of this study and these other studies suggest that baseline IBI variability indexes individual differences in the ability to engage in intensive cognitive processing during sustained attention (Porges, 1992; Richards & Casey, 1992).

Signal detection analyses of localization patterns suggest at least one mechanism that may be responsible for the effects of a central stimulus on peripheral stimulus localization. This mechanism is the infants' decision processes rather than detection or acquisition. Signal detection analyses provide a measure of perceptual sensitivity, stimulus discriminability, and information acquisition in the sensitivity parameter (d') and a measure of response and infant-controlled decision processes in the bias parameter (β) (Baird & Noma, 1978; Green & Swets, 1966; Massaro & Cowan, 1993). The variation in these two parameters caused by the independent variables was primarily in response bias rather than sensitivity. For example, peripheral stimulus localization was more probable during attention termination than during sustained attention, but the false alarm percentage during attention termination was more than double that during sustained attention. The response bias measure was thus much lower in attention termination than in sustained attention. This finding indicates a greater tendency to respond during attention termination independently of the sensory detection of the presence or absence of the peripheral stimulus. The increase over the three ages in peripheral stimulus localization was accompanied by a larger concomitant increase in the false alarm percentage and a decrease in the response bias. This finding indicates that the older infants had a tendency to shift fixation from the central to the peripheral location independently of the presence of the peripheral stimulus, that is, an increasing tendency to make a response. The infants with high IBI variability likewise were more likely to shift fixation away (smaller response bias parameter) from the focal stimulus, particularly during inattention.

These data suggest that attention to the central stimulus raised the threshold for the infants' response to a peripheral stimulus. The peripheral stimulus was thus less likely to elicit a shift of fixation to the peripheral location during focal stimulus attention engagement. The effect of attention was not to decrease perceptual sensitivity (sensory discrimination, stimulus discriminability, or information acquisition) to the peripheral stimulus. Rather, attention to the central stimulus biased the infant against shifting fixation to peripheral locations and biased the infant toward continuing fixation on the focal stimulus. Attention increased the overall likelihood of responding to stimuli only in the location to which attention was currently directed, that is, spatial selectivity. This interpretation is similar to that of Harris and MacFarlane (1974) that "internal control" of fixation (i.e., attention) plays a strong role in the effect of the central stimulus on peripheral stimulus localization.

The results of this study have implications for the understanding of the relationship between infant attention sys-

tems. A distinction has been made between attention-getting and attention-holding stimulus properties and the presumed cognitive systems governing them in infants (Cohen, 1972, 1973; Richards & Casey, 1992). Attention getting involves stimulus properties that guide fixation shifts and results in the orienting of receptors and attention to new stimuli. Peripheral stimulus localization is thought to be an excellent paradigm for studying this property (e.g., Cohen, 1972) because the infant must shift fixation from one location to another to bring objects to foveal vision for further exploration (Cohen, 1973; Finlay & Ivinskis, 1982; Maurer & Lewis, 1991). Attention holding is responsive to complex stimulus characteristics, including complexity, familiarity, stimulus preference, and memory. Attention holding results in sustained fixation and involves the acquisition of information from the stimulus (Richards, 1997; Richards & Casey, 1992). The lower peripheral stimulus localization probabilities during sustained attention imply that attention holding is a characteristic of heart-rate-defined sustained attention. Fixation is sustained toward the object of interest as long as this attention phase occurs and the attention-holding system attenuates the attention-getting properties of the peripheral stimulus. These results imply that the systems involved in saccadic shifts toward peripheral objects are subject to inhibition by the neural systems governing foveal stimulus processing. Such inhibition in infants seems to occur at the level of decision processes (response bias) rather than at the level of the detection and discriminability of the peripheral stimuli.

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