INFANCY, *2*(2), 135–157

THEMATIC COLLECTION: ARTICLES

Cortical Indexes of Saccade Planning Following Covert Orienting in 20-Week-Old Infants

John E. Richards

Department of Psychology
University of South Carolina

This study examined scalp-recorded, event-related potential (ERP) indexes of saccade planning in 20-week-old infants. A spatial cuing procedure was used in which the infants were presented with a central fixation stimulus and a peripheral cue. A peripheral target followed the cue on the ipsilateral or contralateral side of the cue. The procedure resulted in covert orienting of attention in these participants, reflected in behavioral (e.g., response facilitation or inhibition of return depending on cue–target stimulus–onset asynchrony) and ERP (P1 facilitation to ipsilateral target) indexes of covert orienting of attention. A presaccadic ERP that occurred over the frontal cortex about 50 msec before the saccade onset was largest when the saccade was to a target in a cued location. A presaccadic ERP potential that occurred about 300 msec before the saccade onset was largest for the saccades toward the cued location whether the target was present or not. These results suggest that saccade planning occurs in infants at this age and that infant saccade planning is controlled by cortical systems.

Visual attention may be shifted to different regions of space by overtly moving the eyes to that location or covertly moving attention without moving the eyes. Covert orienting of attention was first described by Posner (1980; Posner & Cohen, 1984). Using a spatial cuing procedure, Posner showed that a target following a cue in the

Supplementary materials to this article are available on the World Wide Web at http://www.infancyarchives.com.

Requests for reprints should be sent to John E. Richards, Department of Psychology, University of South Carolina, Columbia, SC 29208. E-mail: richards-john@sc.edu

same location (valid trial) at a very short interval will be responded to more quickly than a target appearing in a location that was not previously cued (invalid trial), even without overt eye movements toward the cued location. Alternatively, a target following a cue in the same location at intermediate intervals (e.g., 300–700 msec) will slow down the reaction time to the target relative to targets appearing in a location that was previously uncued. The speeding of reaction time at the short delays has been termed *facilitation*, and the slowing of reaction time at the intermediate intervals has been termed *inhibition of return.* This procedure has been used to measure covert orienting of attention in young infants (Hood, 1993, 1995; Hood & Atkinson, 1991; Johnson, Posner, & Rothbart, 1994; Johnson & Tucker, 1996; Richards, 2000). A recent study recorded scalp event-related potentials (ERPs) in the infant occurring before the onset of the saccade to the cued or uncued locations (Richards, 2000). It was suggested that the ERP responses occurring before the saccade to the cued location were consistent with the existence of cortical saccade planning in young infants. This study examined the ERPs associated with saccadic eye movements in 20-week-old infants to determine if saccades occurring under differing conditions involved cortical saccade planning.

There are several studies that show that covert orienting may develop in young infants. At least three studies (Hood & Atkinson, 1991, reported in Hood, 1995; Johnson & Tucker, 1996; Richards, 2000) have used the spatial cuing paradigm and have reported that 2- to 3-month-old infants do not show the inhibition of return to a previously cued location at intermediate delays, but 4-month-old infants (Johnson & Tucker, 1996), 4.5-month-old infants (Richards, 2000), and 6-month-old infants (Hood & Atkinson, 1991, reported in Hood, 1995; Johnson & Tucker, 1996; Richards, 2000) show inhibition of return. Similarly, there is agreement among studies that 6-month-old infants show facilitation of reaction times to a target following a cue at brief delays, whereas only one study has shown facilitation in infants younger than 4 months old (Richards, 2000). These studies imply that over the age range from 3 to 6 months there is an increasing ability of infants to shift attention to locations in space without overtly moving their eyes.

A recent study measured ERP responses in the spatial cuing paradigm with infants at 14, 20, and 26 weeks old (Richards, 2000). One finding in that study was that presaccadic ERP changes occurred on the trials on which a cue and target were in the same location. These presaccadic ERP changes did not occur for saccades toward a target that appeared in a different location than the cue or for saccades toward a target that had not been preceded by a cue. This presaccadic activity was a positive component in the ERP approximately 50 msec before the saccade onset, was located in the scalp regions contralateral to the saccade, and occurred primarily in the frontal and central scalp leads. It was argued that the cue and accompanying covert shift of attention resulted in an expectation about the location of the upcoming target in that location. The ERP represents specific events occurring in the cortex (Hillyard, Mangun, Woldroff, & Luck, 1995; Swick, Kutas, & Neville,

1994). The presaccadic ERP component was hypothesized to reflect cortically based saccade planning (Csibra, Johnson, & Tucker, 1997; Csibra, Tucker, & Johnson, 1998, this issue; Johnson, Gilmore, & Csibra, 1998; Richards, 2000). Of interest in this regard was the absence of the presaccadic ERP in the youngest infants (3 months) and an increasing level of the amplitude of the ERP and the spread of the ERP across multiple electrode locations for the infants at the older testing ages (4.5 and 6 months). These findings are consistent with neurodevelopmental models that posit an increasing role of the cerebral cortex in the control of attention-related eye movements (e.g., Hood, 1995; Hood, Atkinson, & Braddick, 1998; Johnson, 1990, 1995; Johnson et al., 1998; Richards, this issue; Richards & Casey, 1992; Richards & Hunter, 1998).

This study extended the study of Richards (2000). Three types of saccades may occur in the spatial cuing procedure. First, if covert orienting occurs as a result of the cue, the infant's attention would have been directed to that location, and the infant may expect a target to occur there. When a target appears in the same location, the saccade to the location could be planned by the infant. These saccades will be called *cued-exogenous* saccades in this article. The planning involved in this type of saccade is therefore not due to a familiarization procedure or a learning of a procedure and a building up of an expectation that a stimulus should occur in this location, as would be the case in the visual expectation procedure (Csibra et al., this issue; Wentworth, Haith, & Karrer, this issue). Rather, the expectation and the planning are based on the infant covertly (no eye movement) shifting attention to the cue followed by a fixation shift when the target appears. Second, there were a number of saccades in Richards (2000) to the cued location that occurred in advance of the target, which were counted as errors in that report. In addition, in a control condition with a cue occurring in a location that was not followed by a target, the infants often made a saccade to the cued location in the absence of the target. These saccades also reflect planning on the part of the infant, but the saccade is made without a specific target. These saccades are called *endogenous* saccades in this article. Third, saccades could occur to targets that occurred in a different location than the cue, or targets that appeared without a cue in either location. Given the lack of cue in the same location, the infant would be responding to these sudden-onset stimuli in a reflexive rather than planned fashion. These saccades are called *uncued-exogenous* saccades in this article. The cued-exogenous and endogenous saccades would be the result of saccade planning on the part of the infant. The cerebral cortex is closely involved in planned saccades, whether endogenous or target directed (Richards, this issue). Thus, because these two types of saccades are cortically driven, they may have a consistent relation to presaccadic ERP activity. Alternatively, the uncued-exogenous saccades would be reflexive and should be controlled by subcortical systems (Richards, this issue). Thus, it is expected that these saccades will not be consistently related to presaccadic ERP activity.

This study used a spatial cuing procedure (Hood, 1995; Hood & Atkinson, 1991; Richards, 2000) to produce covert orienting of attention in 20-week-old infants and focused on the ERP changes occurring before the saccades to the targets. The procedure duplicated that of Richards (2000) and is briefly described. A focal visual stimulus was presented for 2 sec, and a competing peripheral stimulus (cue) was presented in addition to the focal stimulus. After both stimuli were turned off, a peripheral stimulus was presented (target) on the same side (ipsilateral trial, valid trial), on the opposite side (contralateral trial, invalid trial), following no cue (no-cue control), or no target was presented (no-target control). The saccades toward the target on the ipsilateral trials were called cued-exogenous saccades because they were toward a target in an expected location. The saccades toward the target in the contralateral trials and the no-cue control trials were called uncued-exogenous saccades because they were toward a target in a previously uncued location and were unexpected. Saccades on any trials occurring toward the cued location before the target was presented, or saccades toward the cued location on the no-target control trials, were called endogenous saccades because they were toward an expected location but occurred in the absence of a specific target. The electroencephalogram (EEG) was recorded during the presentations. ERPs were computed by averaging EEG time locked to the onset of the saccade toward the target (presaccadic ERP). The EEG recording included the 19 electrodes of the international 10–20 system (Jasper, 1958) and an additional 21 electrodes for a total of 40 electrode leads. It was expected that the higher density recording would localize the scalp activity better than the traditional 19-electrode recording configuration.

A brief mention should be made of some analyses that were not done in this study. First, the ERP changes accompanying target onset were not analyzed. Several studies with adults (see Hillyard et al., 1995) have shown an enhanced P1 and N1 in the ERP to the cued targets (valid trials) relative to the uncued targets (invalid trials). This P1 enhancement was reported in infants at this age range in Richards (2000). These responses were averaged locked to the onset of the target rather than a saccade to the target. These responses were not evaluated in this study because the procedures and methods were similar to those of Richards (2000). Second, the spatial cuing procedure used in this study used three delay durations between the onset of the cue and the onset of the target, stimulus–onset–asynchrony (SOA). It was expected that the valid trials should have shorter reaction time latencies at the short SOA (facilitation), and the valid trials should have longer reaction time latencies at the longest SOA (inhibition of return). In addition, there is reason to expect the spatial cuing effect on the target-locked ERP averages is larger for the short SOAs than the long SOAs (Hopfinger & Mangun, 1998), although this SOA effect was not found with infants (Richards, 2000). However, there was no theoretical reason to expect that saccade planning before making a movement to a stimulus in the peripheral location should differ as a function of the SOA conditions. The effect of SOA on reaction times was examined to

ensure that facilitation and inhibition of return occurred to indicate that covert orienting occurred. However, the effect of SOA on ERP was not examined because of a lack of theoretical interest in a SOA–ERP relation and because of the prior study in which SOA effects were not found on similar ERP components. The Richards (2000) study demonstrated that covert orienting occurs in infants at this age range, whereas the goal of this study was to examine presaccadic ERP changes following covert orienting.

METHOD

Participants

The participants were infants recruited from birth notices published in a Columbia, South Carolina, newspaper. There were 40 infants that were tested at 20 weeks of age (M = 140.8 days, SD = 3.42; 21 boys, 19 girls). There were four groups of 10 infants each who had different EEG recording montages (see EEG Measurement and ERP Quantification). The infants were full term, defined as having birth weight greater than 2,500 g and gestational age of 38 weeks or greater based on the mother's report of her last menstrual cycle. There were 12 additional infants who became fussy or sleepy during the testing session or who did not complete enough trials to be included in the analysis because of equipment problems or poor EEG recording.

Apparatus and Procedure

The details of the apparatus and procedure may be found in Richards (2000). The infant was held in front of a TV monitor on which the stimuli were presented. After the infant fixated on a blinking dot, a dynamic black-and-white, computer-generated pattern was presented in the center of the TV monitor. After 2 sec of focal presentation, a competing stimulus (cue) was presented at 18° in the periphery for 300 msec and then both stimuli were turned off. At delays of 150, 575, or 1,000 msec (450, 875, or 1,300 msec SOA), a peripheral stimulus was presented (target) and remained on until the infant looked toward it. The three SOAs were chosen based on prior studies showing facilitation and inhibition of return for these delays (Hood & Atkinson, 1991, reported in Hood, 1995; Johnson & Tucker, 1996; Richards, 2000). There were five trial types: *ipsilateral*, where cue and target occurred in the same location; *contralateral*, where cue and target were on opposite sides; *no-target control*, where cue was presented but no target occurred; *no-cue control*, where there was no cue presentation but a target occurred; and *no-stimulus control*, where there was presentation of focal stimulus without cue or target. Each SOA and trial type combination was presented in a block of trials, and trial types were randomly

presented without replacement in these blocks. Each infant received at least two blocks (Delay × Trial Types) of trials, up to a maximum of 40 trials, and was included in the analysis only if we had EEG data from at least one of each trial type. Testing was done only if the participants maintained an alert, awake state during the procedure (eyes open, no fussing or crying, and responding to the protocol). If the infant became fussy, a short break was taken and the presentations were paused and then restarted.

EEG Measurement and ERP Quantification

The EEG was recorded from 20 locations with nonpolarizable electrodes mounted in an elastic cap (ElectroCap International). For all infants, recordings were made at the standard center, left, and right hemisphere positions spanning the scalp according to the international 10–20 recording system (Jasper, 1958; Pivik et al., 1993; 10–20 electrode location names are: F_Z, P_Z, C_Z, Fp_1, Fp_2, F_3, F_4, F_7, F_8, C_3, C_4, T_3, T_4, P_3, P_4, T_5, T_6, O_1, O_2; and non-10–20 electrode, O_Z). Also, sites were measured that were 50% of the distance between the 10–20 locations (i.e., between Fp and frontal [anterior frontal], AF_Z, AF_1, AF_2, AF_5, AF_6; between frontal and central [frontal central], FC_Z, FC_1, FC_2, FC_5, FC_6; between central and parietal [central parietal], CP_Z, CP_1, CP_2, CP_5, CP_6; and between parietal and occipital [parietal occipital], PO_Z, PO_1, PO_2, PO_5, PO_6). Figure 1 shows the location of these electrodes. Because of the time needed to apply all 40 electrodes, and the difficulty of doing so with infant participants, the 40 infants were separated into four groups that had different EEG montages: (a) 10–20 locations, anterior frontal, and parietal occipital; (b) 10–20 locations, anterior frontal, and frontal central; (c) 10–20 locations, frontal central, and central parietal; and (d) 10–20 locations, central parietal, and parietal occipital. Therefore, all groups had the 10–20 electrodes in common, and at least one set of 5 electrodes in common with other groups for the non-10–20 locations. These sites and the right mastoid were measured relative to a left mastoid reference electrode, and the EEG waveforms were algebraically rereferenced to the average of the left and right mastoids after the recording. The EEG was recorded with typical acquisition techniques (i.e., 20K amplification, 250 Hz digitization, light intensity rub and electrode gel, impedances < 5K ohms, and vertical electrooculogram [EOG] for artifacts; see Richards, 2000). Following recommendations for infant participants and human participants concerns (Pivik et al., 1993; Putnam, Johnson, & Roth, 1992), the scalp was not abraded, making this a noncritical recording situation.

The ERPs were obtained from the EEG recordings. The ERPs were made from the 4-msec interval (250-Hz) EEG recording after artifacts were removed or adjusted. The EEG was first averaged for individual infants across all SOA delays for the five cue–target conditions. The presaccadic ERP averages were made from data averaged backward in time from the onset of the EOG activity indicating that

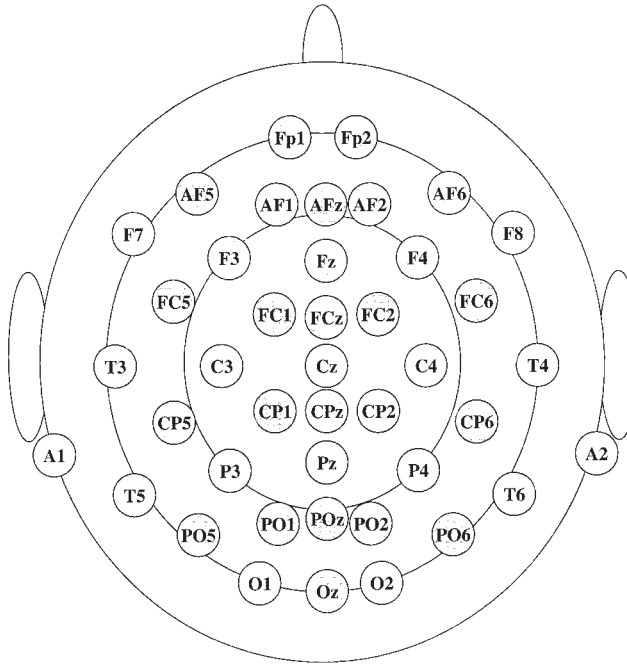

FIGURE 1 The 40-channel recording electrode locations used in the study. The 19 international 10–20 system electrodes are shown with solid backgrounds and the additional electrodes are shown with the hatched line background. The 21 additional electrodes include Oz, locations halfway between the Fp and frontal (anterior frontal), AF_Z, AF_1, AF_2, AF_5, AF_6; between frontal and central (frontal central), FC_Z, FC_1, FC_2, FC_5, FC_6; between central and parietal (central parietal), CP_Z, CP_1, CP_2, CP_5, CP_6; and between parietal and occipital (parietal occipital), PO_Z, PO_1, PO_2, PO_5, PO_6.

a saccade occurred up to 750 msec before the saccade, and from data averaged forward in time from the saccade onset for 100 msec. Measures of component amplitude were estimated on a single-trial basis using the filtered and averaged ERP response to identify appropriate locations for analysis. Peaks or troughs were identified in the EEG recording, and the maximum or minimum EEG was identified and recorded for these peaks and troughs. The peak amplitude (μV) was defined as the peak of the most extreme EEG voltage in the relevant time window over the baseline voltage, and the peak latency was the time at which this amplitude occurred (e.g., de Haan & Nelson, 1997; Richards, 2000). The location for the time points used from the single-trial analyses were determined only after constructing grand average ERP responses for that experimental condition or scalp electrode location. Topographical ERP scalp potential maps were calculated for some of the

effects. For the topographical maps, the scalp potentials were rereferenced to an average reference and interpolations were done using a third-order spherical spline technique (Ganis, Kutas, & Sereno, 1995; Nunez, 1990; Perrin, Bertrand, & Pernier, 1987; Perrin, Pernier, Bertrand, & Echallier, 1989). The scalp potential maps show the distribution of the scalp potentials at a specific point in time and are useful in visualizing the ERP data shown in figures. Details of this procedure may be found in Richards (2000).[1]

Peripheral Stimulus Localization Judgments

The localizations of the peripheral stimulus were based on an observer's judgments of fixation direction and the presence of saccades in an EOG recording (for details of EOG–saccade identification, see Richards, 2000). The horizontal EOG was recorded and used to identify saccades toward the peripheral locations where the target may have occurred. A single observer judged a videotape recording of the infant's fixation direction. Localizations were based on the observer's fixation judgments in conjunction with the existence of saccades in the EOG. A look was considered a localization when the observer judged that the infant was fixating in the direction of the TV or looked toward the peripheral stimulus and a saccade occurred in the appropriate direction. The EOG was used to ensure that no other saccade occurred before that saccade, and trials were eliminated if the infant looked away from the central stimulus when the cue presentation occurred. The latency of the localization was defined as the onset of the first localizing saccade occurring after the onset of the peripheral stimulus. The onset of the first localizing saccade was used to average EEG to compute presaccadic ERP occurring up to 750 msec before the saccade onset.

RESULTS

Localization Probability and Latency

The latency to localize the peripheral stimulus was calculated as the difference between the onset of the target and the beginning of the saccade toward the target. The latency measure was analyzed with a 3 (condition: ipsilateral, contralateral, or

[1]In addition to the presaccadic ERP averages, poststimulus averages also were done. First, the EEG changes occurring at the onset of the focal stimulus were averaged. Second, the EEG changes occurring at the onset of the target were averaged. These averages were analyzed, and results from these analyses were similar to those reported in Richards (2000). Figures showing these averages and details of the analyses are available on request from the author.

no-cue control) × 3 (SOA: 450, 875, and 1,300 msec) analysis of variance (ANOVA). The latency measure had significant skew and kurtosis, so the variable was log transformed. There was a Condition × SOA interaction that approached statistical significance, $F(4, 156) = 2.77$, $p = .0751$. Figure 2 shows the localization latencies for the three conditions and SOAs. Post hoc comparisons were done using the Scheffé error control strategy. If covert orienting to the cue occurred, reaction time should be faster on the short SOA trials for the ipsilateral condition compared to the contralateral or no-cue control trials (facilitation). Post hoc tests showed that localization latency on the ipsilateral trials was faster than on the contralateral trials at the 450 msec SOA, but not at the other SOAs ($p < .05$). However, it was also the case that the control trials (no-cue control) had faster localization latencies than the contralateral condition ($p < .05$), implying there was a processing cost to shifting attention to the cued location and then having to shift the eyes to the contralateral location. Similarly, if covert orienting occurred, there should be a lengthening of reaction time on the long SOA trials for the ipsilateral condition compared to the other conditions (inhibition of return). The ipsilateral condition was not significantly different from the other two conditions at the intermediate SOA but was significantly longer than the other conditions at the longest SOA ($p < .05$).

A goal of this study was to examine presaccadic ERP changes during cued-exogenous, uncued-exogenous, and endogenous saccades. Table 1 has the probability of looking toward the target location for exogenous and endogenous saccades

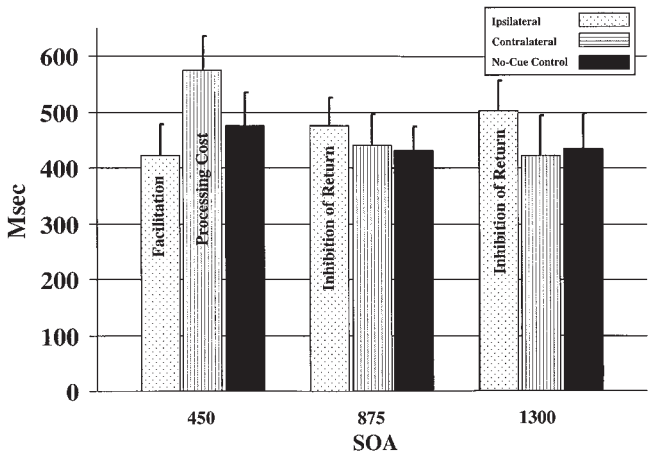

FIGURE 2 Latency to localize the peripheral stimulus when it was presented as a target. There is a facilitation and processing cost for shifting attention on the 450-msec stimulus–onset–asynchrony (SOA) trials and an inhibition of return for the ipsilateral trials on the 1,300-msec SOA trials.

TABLE 1
Exogenous (Target Present) and Endogenous (No Target) Saccades as a Function of the Presence of the Preceding Cue

Saccades	*Cue Preceding*	*No Cue Preceding*[a]
Exogenous: Look toward target	128 of 132, 97% (cued exogenous)	335 of 346, 97% (uncued exogenous)
Endogenous: Look toward cued location, no target present	204 of 276, 75% (endogenous)	77 of 149, 52% (uncued-endogenous)

[a]For the no-cue control and no-stimulus control the putative side of the cue was randomly chosen on each trial. The uncued-endogenous saccades were not included in any event-related potential analysis.

as a function of a prior cue. The exogenous saccades were defined as those saccades that occurred toward the target when it was present, either following a cue in that location (ipsilateral trials and cued-exogenous saccades) or following no cue in that location (contralateral trials, no-cue control trials, and uncued-exogenous saccades). On almost all trials, the infants looked toward the target when it was presented (~ 97%). The endogenous saccades were defined as those saccades that occurred when no target was present. This may have occurred on trials for which the infant was cued toward the peripheral location and made an eye movement to that location before the target was presented (ipsilateral, contralateral) or when the infant looked toward the cued location when no target was presented (no-target control). The presence of the cue clearly affected the endogenous saccades. The endogenous saccades that occurred following a cue were predominantly to the side of the cued location (75%), whereas the endogenous saccades that occurred when no cue preceded the saccade occurred approximately equally often on either side (52% to arbitrarily defined side).

Presaccadic ERP

The ERPs preceding the onset of a saccade toward the peripheral stimulus when it was presented as a target were analyzed. These were analyzed to determine if the ERP changes were different for endogenous, cued-exogenous, and uncued-exogenous saccades. Figure 3 shows the ERP plotted backward from the onset of the saccades. The trials were separated into endogenous saccades that occurred toward the cued location when no target was present, cued-exogenous saccades that occurred to the target following a cue in that location, and uncued-exogenous saccades that occurred to the target when a cue had not occurred in that location. These data in this figure were plotted as if the infant were making a saccade toward the left side (even and odd electrodes transposed for saccades toward the right side). There were two obvious ERP changes in these plots. First, there was a positive ERP component

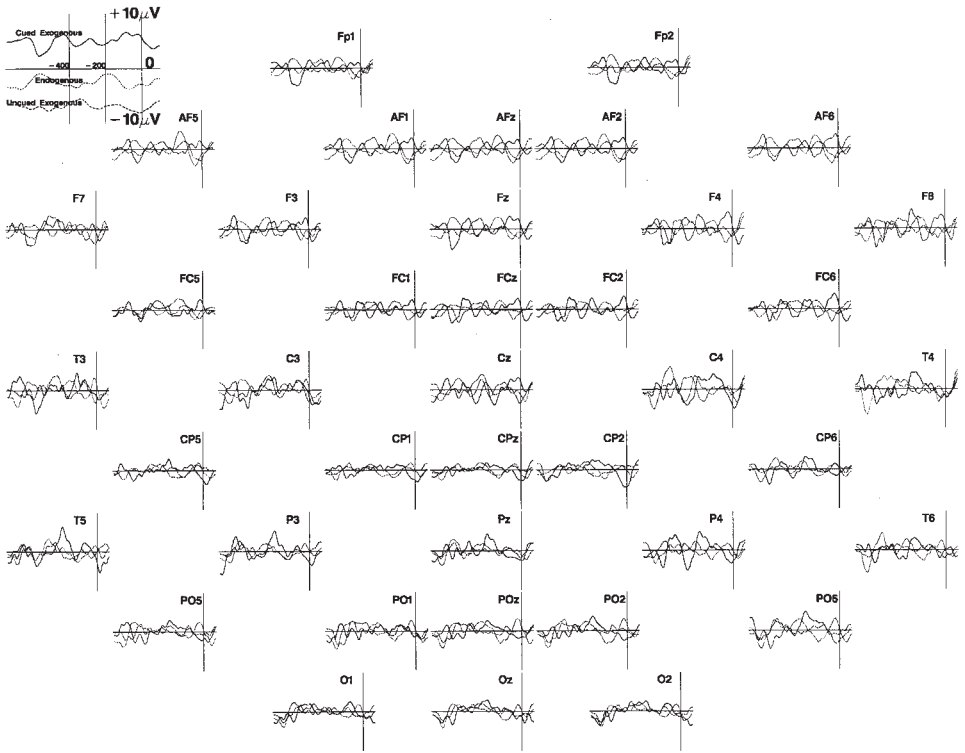

FIGURE 3 The ERP responses occurring immediately prior to the onset of a saccade to the cued location. The responses are presented separately for the 40 recording electrodes and separately for the cued-exogenous, uncued-exogenous, and endogenous saccades. The data in this figure were plotted as if the infant were making a saccade toward the left side.

that showed a peak about 50 msec before the saccade in the cued-exogenous saccades that did not occur before the endogenous or the uncued-exogenous saccades. This presaccadic positivity (PSP 50) was largest in the frontal leads contralateral to the saccade direction. Second, there was a large positive ERP occurring about 300 msec before the saccade for the cued-exogenous and endogenous saccades that did not occur in the uncued-exogenous saccades. This positive component (PSP 300) was largest in the parietal leads over the scalp contralateral to the saccade direction. Figure 4 shows the presaccadic potentials figures enlarged for F_4, FC_6, P_4, and PO_6. These four electrodes had the largest PSP 50 (F_4, FC_6) and PSP 300 (P_4, PO_6), and the locations for those components are identified in the figure.

The ERP responses preceding the onset of a saccade were analyzed using the data identified from the single-trial analysis. The peak amplitude of the positive ERP component approximately 50 msec before the saccade (PSP 50) was analyzed. Specific groups of EEG electrode groups were examined together, because it was expected based on the ERP changes (Figures 3 and 4) and prior results (Richards, 2000) that the frontal leads contralateral to the saccade would show significant effects for the PSP 50, and the parietal leads contralateral to the saccade would show significant effects for the PSP 300. These groups of EEG electrodes were (a) *frontal* leads: Fp, anterior frontal, frontal; (b) *central*: frontal central, central, central parietal, and T3 and T4; (c) *posterior*: parietal, parietal occipital, occipital; and (d) *midline*: all eight midline electrodes (see Figure 1 for electrode locations). The peak of the positive ERP component of the frontal, central, and posterior leads from each participant was analyzed with a 3 (saccade type: endoge-

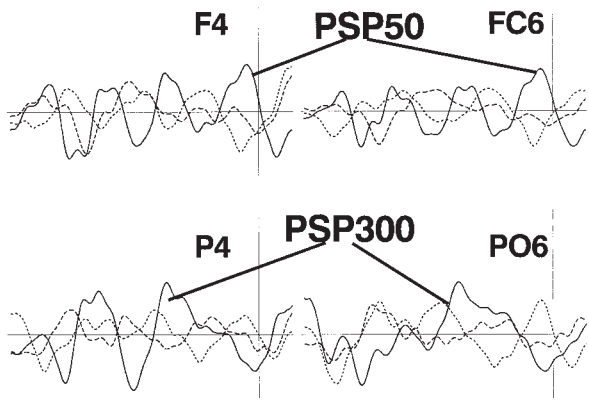

FIGURE 4 The ERP responses for four of the electrode locations shown in Figure 3. The presaccadic ERP for F_4 and FC_6 show a large presaccadic positive ERP component that occurred about 50 msec before saccade onset for cued-exogenous saccades (PSP 50). The presaccadic ERPs for P_4 and PO_6 show a large presaccadic positive ERP component that occurred about 300 msec before saccade onset for the cued-exogenous and endogenous saccades (PSP 300).

nous, cued-exogenous, uncued-exogenous) × 3 (lateral location: leads ipsilateral to saccade, midline leads, leads contralateral to saccade) ANOVA.[2] The midline leads were analyzed with a saccade type ANOVA (no lateral location factor). There were no significant effects of the saccade type or lateral location for the central, posterior, or midline leads. For the frontal leads, there was a significant interaction of saccade type and the lateral location, $F(4, 1{,}525) = 3.38$, $p = .0090$. For the cued-exogenous saccades, there was a significantly increasing size of the PSP 50 from the frontal ipsilateral leads ($M = 4.87$ μV, $SE = 0.867$) to the frontal midline leads ($M = 6.44$ μV, $SE = 1.296$) to the frontal leads contralateral to the saccade ($M = 6.97$ μV, $SE = 0.929$). For the endogenous and uncued-exogenous saccades, the difference in the PSP 50 amplitude across lateral locations was not statistically significant ($M = 3.27$ μV, $SE = 0.363$; $M = 2.28$ μV, $SE = 0.513$; and $M = 3.18$ μV, $SE = 0.344$, for the ipsilateral, midline, and contralateral lead locations, respectively). The impression in Figures 3 and 4 that the presaccadic positivity was largest in the frontal leads contralateral to the saccade direction was confirmed by these statistical tests.

The presaccadic ERP responses were analyzed for peak amplitude of the positive ERP component approximately 300 msec before the saccade (PSP 300). This was analyzed, as was the PSP 50 component, with a 3 (saccade type) × 3 (lateral location) ANOVA separately for frontal, central, posterior, and midline leads. There were two interesting effects. First, for the central leads there was a significant interaction be-

---

[2]The ANOVAs with the groups of leads were tested with an error term from an analysis using individual leads (41) and saccade type (3) in the analysis. This provided an error term to obtain appropriately conservative tests protected by the error rate of the omnibus tests (e.g., Lead × Saccade Type interaction error term) rather than by the error term of these tests with groups of leads as a factor.

Three additional analyses were done. First, the data from the four recording montage groups were analyzed. The ERPs were analyzed with group as a factor, and common electrodes were compared across groups (e.g., all 10–20, 10–20 and frontal central, and 10–20 and central parietal). There were no significant main effects or interactions involving the group factor. For the topographic plots, the data were combined irrespective of the recording montage groups. Second, the latency of the PSP 50 and PSP 300 was analyzed. There were some significant effects involving the latency factor for the PSP 300 component suggesting that this component peaked earlier for the endogenous saccades than for the cued-exogenous saccades (see Figure 4). Third, the data also were analyzed without changing the data to be ipsilateral or contralateral to the saccade (i.e., the original lead locations). In this case, there were main effects of saccade type (e.g., cued-exogeneous PSP 50 > other saccade types) but no saccade by lateral lead effect. This indicates that the responses were not lateralized in the original recording locations, but instead were ipsilateral or contralateral to saccade direction.

The data from the participants, leads, and saccade types could not provide a full factorial orthogonal design. There were different numbers of participants contributing data for each lead in the experimental conditions or for the saccade types. Because of this, the general linear models approach using nonorthogonal designs (Hocking, 1985; Searle, 1971, 1987) was used for all ANOVA analyses. The sums of squares (hypothesis and error) for the nested effects in the design were estimated using "subjects" as a class and nesting repeated measures (saccade type, lateral lead location, electrode) within this class variable. The PROC GLM of SAS was used for the computations.

tween saccade type and the lateral location, $F(4, 1459) = 2.37$, $p = .0499$. Similar to the PSP 50, the ERP that occurred at 300 msec before the saccade onset was larger on the contralateral side for the cued-exogenous saccades ($M = 9.12\ \mu V$, $SE = 0.802$) than for either the endogenous ($M = 5.49\ \mu V$, $SE = 0.465$) or the uncued-exogenous ($M = 6.23\ \mu V$, $SE = 0.463$) saccades. This may be seen in Figure 3 in the C_4, T_4, CP_2, and CP_6 leads. Second, for the posterior leads there was a significant effect of the saccade type in the leads contralateral to the side of the saccade, $F(2, 1459) = 3.04$, $p = .0480$. In this case, the PSP 300 component for the cued-exogenous and endogenous saccades was about the same size (Ms = 8.13 μV, $SE = 0.945$; and $M = 7.33\ \mu V$, $SE = 0.580$, respectively), whereas the PSP 300 ERP component for the uncued-exogenous saccades was smaller ($M = 5.26\ \mu V$, $SE = 0.481$). This may be seen in Figure 3 for the P_4, T_6, PO_2, and PO_6 leads (also see P_4 and PO_6 in Figure 4).

The presaccadic ERP responses were examined further with topographic ERP plots. Figure 5 illustrates the ERP response for the PSP 50 and PSP 300 effects, plotting the ERP amplitude differences between the cued-exogenous and the combined endogenous and uncued-exogenous saccades for the PSP 50, and between the combined cued-exogenous and endogenous saccades and the uncued-exogenous saccades for the PSP 300. The PSP 50 occurred as a large, positive activity centered above the frontal area contralateral to the saccade. This map suggests this component was centered between F_4 , F_8, AF_6, and FC_6 but was widespread between these four electrodes. The PSP 300 occurred as a positive activity centered over the parietal area contralateral to the direction of the saccade, particularly P_4 and PO_6. In distinction to PSP 50, this component was limited to a relatively small area over the parietal cortex and was smaller in magnitude.

The presaccadic ERP responses are shown as a sequence of maps in Figures 6 and 7. Figure 6 shows a sequence from 94 msec preceding the saccade through about 14 msec preceding the saccade. The top series shows the presaccadic ERP changes for the cued-exogenous saccades. The PSP 50 was a large, positive activity centered near the frontal scalp area contralateral to the saccade direction that emerged clearly at about 46 msec preceding the saccades to the target when it was in the cued location. There was no such presaccadic ERP activity for the endogenous and uncued-exogenous saccades (Figure 6, bottom series). The PSP 300 is seen in Figure 7 as a positive activity occurring over the parietal area contralateral to the saccade in the cued direction whether a target was present or not (Figure 7, top series, cued-exogenous and endogenous saccades). There was no comparable activity occurring before saccades toward the uncued location (Figure 7, bottom series, uncued-exogenous saccades).

DISCUSSION

The main goal of this study was to examine the ERP responses of 20-week-old infants preceding saccadic eye movements made to targets in a spatial cuing paradigm.

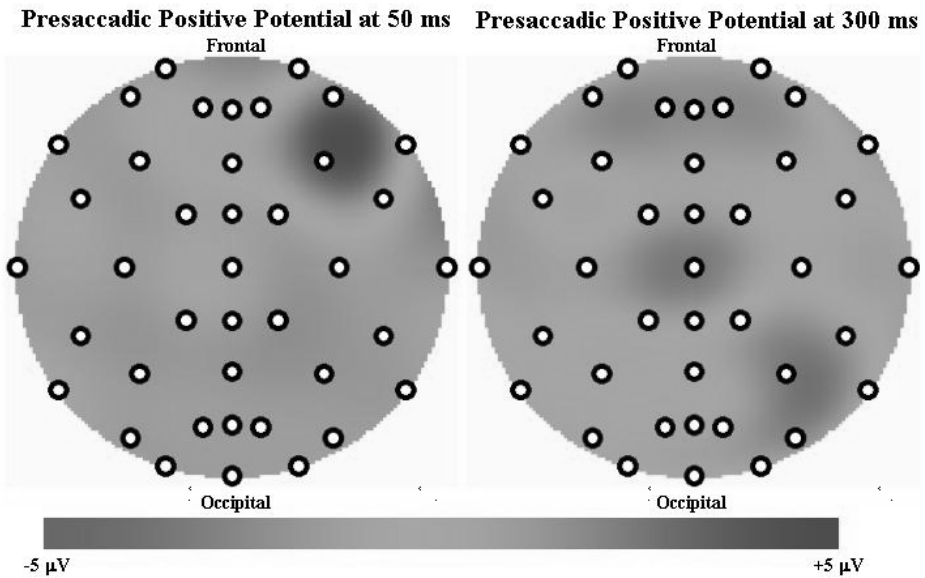

FIGURE 5 Topographical scalp potential maps for the PSP 50 and PSP 300 components for the difference between the cued-exogenous and the combined endogenous and cued-exogenous saccades (left panel), and for the difference between the combined cued-exogenous and endogenous and the cued-exogenous saccades (right panel). The maps were plotted as if the infant were making a saccade toward the left side. (A full-color version of this figure is available on the World Wide Web at http://www.infancyarchives.com)

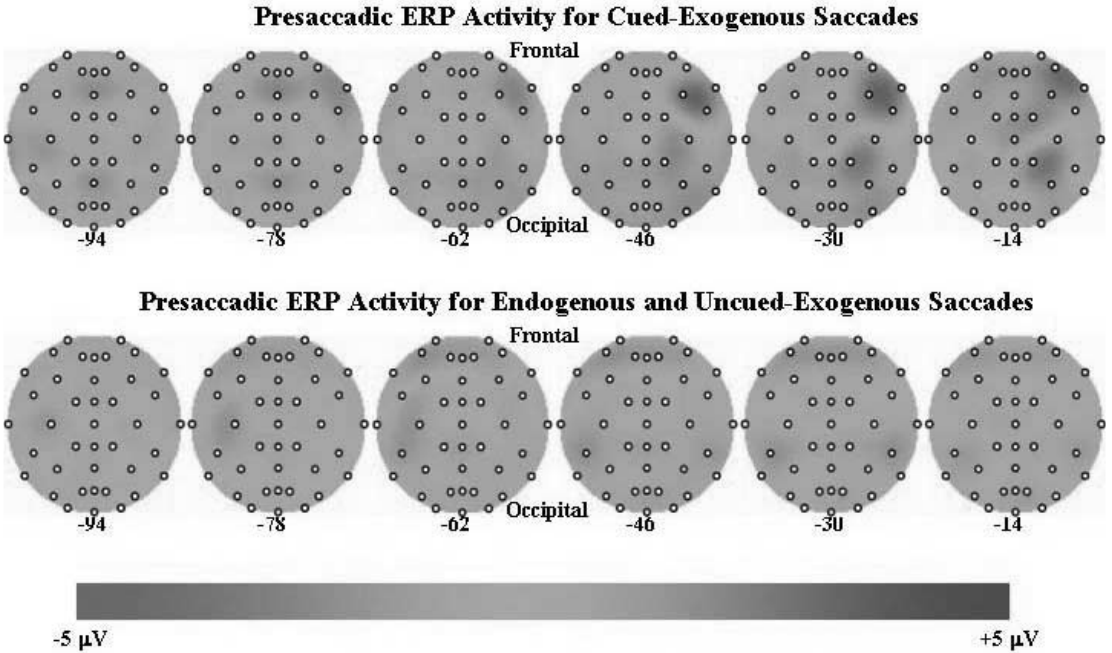

FIGURE 6 Topographical scalp potential maps for the presaccadic ERP responses for the cued-exogenous saccades and the combined endogenous and uncued-exogenous saccades. The maps are shown as a series and represent 16-msec averages of ERP from 94 msec preceding the saccade onset through 14 msec preceding saccade onset. The PSP 50 is apparent in the frontal locations contralateral to the cued-exogenous saccades (top series) beginning at –46 msec and –30 msec and declining by –14 msec. (A full-color version of this figure is available on the World Wide Web at http://www.infancyarchives.com)

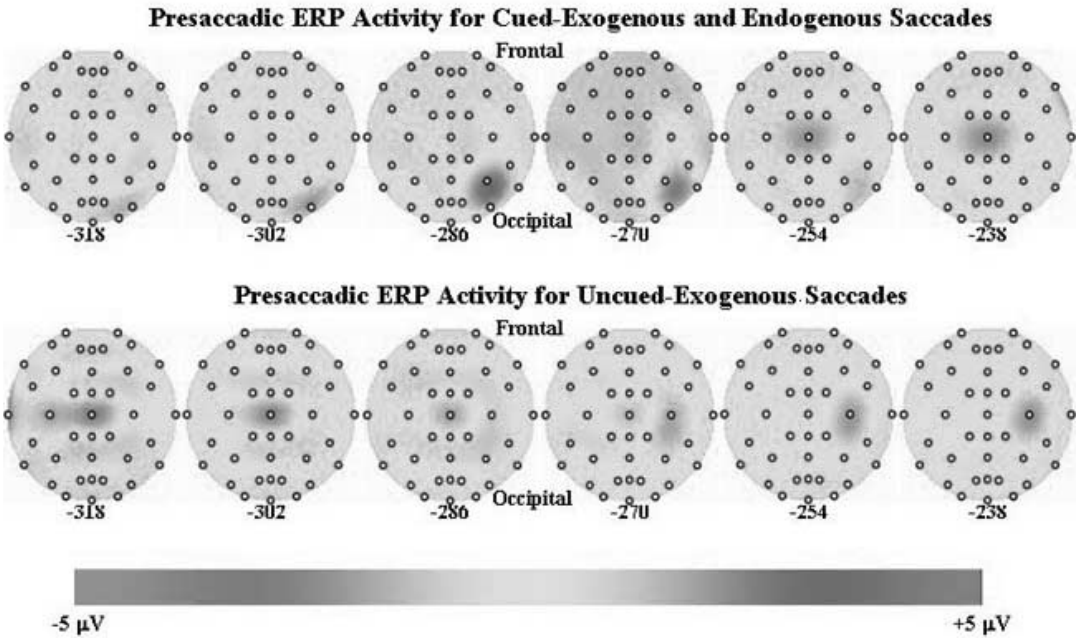

FIGURE 7 Topographical scalp potential maps comparing the combined cued-exogenous and endogenous saccades with the uncued-exogenous saccades. This figure shows the PSP 300 peaking at about –286 msec near the parietal area contralateral to the saccades in the cued-exogenous and endogenous saccades (top series). (A full-color version of this figure is available on the World Wide Web at http://www.infancyarchives.com)

It was expected that cortical saccade planning might affect saccades toward a target in a previously cued location (cued-exogenous) and saccades toward a location that had been previously cued even without a specific target present (endogenous). These two saccade types had a positive ERP component in common that occurred about 300 msec before the saccade (PSP 300). The PSP component was located in the parietal scalp region contralateral to the saccade. As in a previous study (Richards, 2000), saccades that occurred to a target in a previously cued location had a presaccadic ERP component occurring about 50 msec before saccade onset (PSP 50). The PSP 50 component was located in the scalp region contralateral to the saccade and located over the frontal scalp region. This presaccadic ERP component was absent in the endogenous saccades and in the uncued-exogenous saccades.

An assumption of this article is that the saccades toward the cued location in the spatial cuing procedure represent planned rather than reflexive saccades. Infants at 4.5 months old show both facilitation of response times and inhibition of return in the spatial cuing paradigm (Johnson & Tucker, 1996; Richards, 2000) and did so in their responses in this study (Figure 2). The existence of facilitation and inhibition of return in this spatial cuing procedure implies that the infants made a covert shift of attention when the focal stimulus and cue stimulus were presented together. The saccades to the cued location might, therefore, be considered as planned by the infants. This saccade planning to the cued location may be best appreciated in relation to the no-cue control trials. In the no-cue control trials, a cue was not given, and the target appeared in an unpredictable location. The saccade to this sudden onset target might be considered reflexive (rather than planned) and based on subcortical eye movement systems (rather than cortical systems; Richards, this issue). There were no differences in the presaccadic ERP responses between the no-cue control trials and the contralateral trials in Richards (2000). This suggests that the appearance of a target in the position contralateral to the cue also was an unexpected event. Therefore, saccades toward that location were controlled by subcortical, reflexive eye movement systems in the brain.

Given this distinction between planned and reflexive saccades in this study, the presaccadic ERP effects were consistent with the hypothesis that cortical saccadic planning occurred in infants in this study. For both the endogenous and the cued-exogenous saccades, there was a significant positive ERP response occurring about 300 msec before saccade onset. This PSP 300 occurred over the contralateral parietal area. These two saccades have in common an expectation about the location of the upcoming target. In the case of the cued-exogenous saccades, the target has already appeared, whereas in the case of the endogenous saccades, no target yet exists. Therefore, this may represent the general planning necessary to make a saccade to this location. The localization of the PSP 300 over the parietal scalp area suggests that this planning occurred in the parietal cortex. This area of the brain is integrally involved in a widespread attention system involving the parietal cortex and frontal eye fields (FEFs), and it is involved in spatial attention (i.e., pos-

terior attention system; Posner, 1995; Posner & Petersen, 1990). The parietal cortex is thought to disengage attention from one location in preparation for shifting attention to another location. In this study, this may represent the infant shifting his or her attention from the central location to the cued location independent of target presence.

The PSP 300 found in this study parallels presaccadic ERP activity reported in studies with adult participants. Studies with adult participants have reported a positive ERP component in adults that occurs about 30 msec to 300 msec prior to saccade onset (Becker, Hoehne, Iwase, & Kornhuber, 1973; Csibra et al., 1997; Kurtzberg & Vaughan, 1980, 1982; Moster & Goldberg, 1990; Thickbroom & Mastaglia, 1985a). This positive slow wave activity was centered on the contralateral parietal areas. This positive activity has been found to be larger for visually triggered saccades than endogenous saccades, but larger for predictable stimulus events than unpredictable ones (Thickbroom & Mastaglia, 1985a). The latency and location of the PSP 300 in this study was similar to this component found in adults. There also was a functional similarity between the PSP 300 component found in this study and that found in adults. Positive presaccadic ERP potentials in adults have been found to be larger, or more widespread, for saccades to expected peripheral stimuli or predicted locations and for voluntary saccades (Balaban & Weinstein, 1985; Evdokimidis, Liakopoulos, & Papageorgiou, 1991; Evdokimidis, Mergner, & Lucking, 1992; Kurtzberg & Vaughan, 1982; Thickbroom & Mastaglia, 1985a, 1985b). These presaccadic ERP components have been taken as evidence of cortical saccade planning for voluntary saccades (Balaban & Weinstein, 1985; Csibra et al., 1997; Csibra et al., 1998, this issue; Johnson et al., 1998; Kurtzberg & Vaughan, 1980, 1982; Richards, 2000).

The second ERP component found in this study was a large, positive activity occurring about 50 msec before saccade onset for the cued-exogenous saccades. This activity occurred contralateral to the saccade and in the frontal areas of the scalp. This finding was a close replication of a PSP 50 ERP component reported in Richards (2000). The 20-week-old infants in Richards (2000) had a similar component that was located closer to C_4 than to the frontal area but did have aspects over the frontal areas. The 26-week-old infants in that study had a PSP 50 of nearly the same topography as was found in this study. This PSP 50 did not occur in endogenous saccades. Thus, this component does not represent cortical saccade planning per se. Rather, it likely represents cortical targeted saccades. That is, the targets were expected to occur in the cued location, and cortical areas guided the saccade to the target. This occurred in contradistinction to the endogenous saccades for which no target was present, and in contradistinction to the uncued-exogenous saccades for which a target was present but saccade planning did not occur.

The PSP 50 ERP component was similar in some respects to that occurring in the visual expectation procedure (Wentworth et al., this issue). In the visual expectation procedure, an alternating or fixed sequence is repeatedly presented to the in-

fant. Infants in this procedure begin to anticipate the onset of the stimulus in a predictable location by making saccades to that location in advance of the stimulus onset. Wentworth et al. reported a positive ERP potential about 30 to 90 msec before saccade onset in the midline frontal lead (Fz; only midline leads recorded). This ERP component had a similar amplitude and time course as the PSP 50 component in this study. However, Wentworth et al. found this ERP component in the frontal lead primarily on the "anticipation" trials in which the infant makes an eye movement toward the upcoming stimulus location before it is presented. This type of eye movement would be similar to the cued-endogenous saccades in this study in which the PSP 50 was not found. Alternatively, the reactive saccades in Wentworth et al. did not show this component. The reactive saccades in the visual expectation procedure would be similar to the cued-exogenous saccades in this study in which the PSP 50 component was reliably found. It is possible that the anticipatory saccades in the visual expectation procedure were a result of an expectation about upcoming stimulus onset that was strongly predictable by the infant. This could therefore lead to the saccade planning for a targeted saccade. Alternatively, in this study the cue shifted attention toward the target location without a strong prediction about a target occurring in that location. Following a cue, the target could equally likely occur in the same location, a different location, or not at all. Thus, the actual presence of the target was necessary to elicit the presaccadic ERP component associated with saccade planning.

There is no readily identifiable presaccadic ERP component reported in the literature on adult ERP that corresponds to the PSP 50 reported in this study and in Richards (2000). The positive slow wave found in adult presaccadic ERP occurs primarily over the contralateral parietal cortex and at longer latencies than the PSP 50 in this study. There is a presaccadic spike potential found in adults immediately prior to saccade onset. The presaccadic spike potential in adults occurs primarily over parietal scalp leads, is extremely short in duration, occurs in the 10 to 20 msec preceding the saccade, and partially overlaps with the saccade onset (Balaban & Weinstein, 1985; Becker et al., 1973; Csibra et al., 1997; Kurtzberg & Vaughan, 1980, 1982; Weinstein, Balaban, & Ver Hoeve, 1991). The PSP 50 in this study and in Richards (2000) was centered primarily over the contralateral frontal regions, was longer in duration, differed in peak latency, and was substantially completed at the time of saccade onset. Thus, the PSP 50 component is not analogous to any presaccadic ERP components found in studies with adult participants.

The areas over which the PSP 50 occurred lie close to the FEFs of the prefrontal cortex. Richards (2000) concluded that the PSP 50 may represent activity occurring in the FEFs (or supplementary eye fields) that guide saccades to specific targets in expected locations. Studies using nonhuman primates have recorded activity in the FEFs that fires in advance of the saccade onset to attention-directed targets (Hanes, Thompson, & Schall, 1995; Schall, 1991, 1995; Schall & Hanes, 1993; Schall, Hanes, Thompson, & King, 1995). The FEFs are believed to initiate

and direct voluntary saccadic eye movement (Richards, this issue). These saccades are under volitional control (planned) rather than reflexive. The presaccadic ERP changes for these cued-exogenous saccades are evidence of cortically driven saccade planning and evidence of specific attention-directed targeted saccades.

In summary, the main goal of this study was to examine the ERP responses of young infants preceding saccadic eye movements to targets in a spatial cuing paradigm. There was evidence of nonspecific saccade planning that was potentially identified as disengaging attention from the central location to shift attention to the peripheral location (PSP 300). There also was a specific targeted saccade planning that occurred only for attention-directed targeted saccades (PSP 50). Both of these activities were absent in saccades that occurred to targets appearing in an unexpected location. These reflexive saccades are presumed to be controlled by subcortical eye movement systems and, therefore, do not show consistent relation with ERP. By 4.5 months of age (although perhaps not at 3 months; Richards, 2000), infants are able to make cortically driven planned saccades, and these saccades are distinguished from reflexive saccades by changes in the ERP immediately preceding saccade onset.

ACKNOWLEDGMENTS

This research was supported by Grant R01–HD18942 from the National Institute of Child Health and Human Development.

REFERENCES

Balaban, C. D., & Weinstein, J. M. (1985). The human pre-saccadic spike potential: Influences of a visual target, saccade direction, electrode laterality and instructions to perform saccades. *Brain Research, 347,* 49–57.

Becker, W., Hoehne, O., Iwase, K., & Kornhuber, H. H. (1973). Cerebral and ocular muscle potentials preceding voluntary eye movements in man. *Electroencephalography and Clinical Neurophysiology, 33*(Suppl.), 99–104.

Csibra, G., Johnson, M. H., & Tucker, L. A. (1997). Attention and oculomotor control: A high-density ERP study of the gap effect. *Neuropsychologica, 35,* 855–865.

Csibra, G., Tucker, L. A., & Johnson, M. H. (1998). Neural correlates of saccade planning in infants: A high-density ERP study. *International Journal of Psychophysiology, 29,* 201–215.

de Haan, M., & Nelson, C. A. (1997). Recognition of the mother's face by six-month-old infants: A neurobehavioral study. *Child Development, 68,* 187–210.

Evdokimidis, I., Liakopoulos, D., & Papageorgiou, C. (1991). Cortical potentials preceding centrifugal and centripetal self-paced horizontal saccades. *Electroencephalography and Clinical Neurophysiology, 79,* 503–505.

Evdokimidis, I., Mergner, T., & Lucking, C. H. (1992). Dependence of presaccadic cortical potentials on the type of saccadic eye movement. *Electroencephalography and Clinical Neurophysiology, 83,* 179–191.

Ganis, G., Kutas, M., & Sereno, M. (1995). Freeing the ERPs: Freeware for high quality spatial map construction and presentation. *Psychophysiology, 32,* S33.

Hanes, D. P., Thompson, K., & Schall, J. D. (1995). Relationship of presaccadic activity in frontal eye field and supplementary eye field to saccade initiation in macaque: Poisson spike train analysis. *Experimental Brain Research, 103,* 85–96.

Hillyard, S. A., Mangun, G. R., Woldroff, M. G., & Luck, S. J. (1995). Neural systems mediating selective attention. In M. S. Gazzaniga (Ed.), *Cognitive neurosciences* (pp. 665–682). Cambridge, MA: MIT Press.

Hocking, R. R. (1985). *The analysis of linear models.* Monterey, CA: Brooks/Cole.

Hood, B. M. (1993). Inhibition of return produced by covert shifts of visual attention in 6-month-old infants. *Infant Behavior and Development, 16,* 245–254.

Hood, B. M. (1995). Shifts of visual attention in the human infant: A neuroscientific approach. *Advances in Infancy Research, 10,* 163–216.

Hood, B. M., & Atkinson, J. (1991, April). *Shifting covert attention in infants.* Paper presented at the meeting of the Society for Research in Child Development, Seattle, WA.

Hood, B. M., Atkinson, J., & Braddick, O. J. (1998). Selection-for-action and the development of orienting and visual attention. In J. E. Richards (Ed.), *Cognitive neuroscience of attention: A developmental perspective* (pp. 219–250). Mahwah, NJ: Lawrence Erlbaum Associates, Inc.

Hopfinger, J. B., & Mangun, G. R. (1998). Reflexive attention modulates processing of visual stimuli in human extrastriate cortex. *Psychological Science, 9,* 441–446.

Jasper, H. H. (1958). The ten twenty electrode system of the International Federation. *Electroencephalography and Clinical Neurophysiology, 10,* 371–375.

Johnson, M. H. (1990). Cortical maturation and the development of visual attention in early infancy. *Journal of Cognitive Neuroscience, 2,* 81–95.

Johnson, M. H. (1995). The development of visual attention: A cognitive neuroscience perspective. In M. S. Gazzaniga (Ed.), *The cognitive neurosciences* (pp. 735–747). Cambridge, MA: MIT Press.

Johnson, M. H., Gilmore, R. O., & Csibra, G. (1998). Toward a computational model of the development of saccade planning. In J. E. Richards (Ed.), *Cognitive neuroscience of attention: A developmental perspective* (pp. 103–130). Mahwah, NJ: Lawrence Erlbaum Associates, Inc.

Johnson, M. H., Posner, M. I., & Rothbart, M. K. (1994). Facilitation of saccades toward a covertly attended location in early infancy. *Psychological Science, 5,* 90–93.

Johnson, M. H., & Tucker, L. A. (1996). The development and temporal dynamics of spatial orienting in infants. *Journal of Experimental Child Psychology, 63,* 171–188.

Kurtzberg, D., & Vaughan, H. G. (1980). Differential topography of human eye movement potentials preceding visually triggered and self-initiated saccades. In H. H. Kornhuber & L. Deecke (Eds.), *Motivation, motor and sensory processes of the brain* (pp. 203–208). Amsterdam: Elsevier.

Kurtzberg, D., & Vaughan, H. G. (1982). Topographic analysis of human cortical potentials preceding self-initiated and visually triggered saccades. *Brain Research, 243,* 1–9.

Moster, M. L., & Goldberg, G. (1990). Topography of scalp potentials preceding self-initiated saccades. *Neurology, 40,* 644–648.

Nunez, P. L. (1990). Localization of brain activity with electroencephalography. *Advances in Neurology, 54,* 39–65.

Perrin, F., Bertrand, O., & Pernier, J. (1987). Scalp current density mapping: Value and estimation from brain data. *IEEE Transactions on Biomedical Engineering, 34,* 283–288.

Perrin, F., Pernier, J., Bertrand, O., & Echallier, J. F. (1989). Spherical splines for scalp potential and current density mapping. *Electroencephalography and Clinical Neurophysiology, 72,* 184–187.

Pivik, R. T., Broughton, R. J., Coppola, R., Davidson, R. J., Fox, N., & Nuwer, M. R. (1993). Guidelines for the recording and quantitative analysis of electroencephalographic activity in research contexts. *Psychophysiology, 30,* 547–588.

Posner, M. I. (1980). Orienting of attention. *Quarterly Journal of Experimental Psychology, 32,* 3–25.

Posner, M. I. (1995). Attention in cognitive neuroscience: An overview. In M. S. Gazzaniga (Ed.), *Cognitive neurosciences* (pp. 615–624). Cambridge, MA: MIT Press.

Posner, M. I., & Cohen, Y. (1984). Components of visual orienting. In H. Bouma & D. G. Bouwhis (Eds.), *Attention and performance X* (pp. 531–556). Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.

Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience, 13,* 25–42.

Putnam, L. E., Johnson, R., & Roth, W. T. (1992). Guidelines for reducing the risk of disease transmission in the psychophysiology laboratory. *Psychophysiology, 29,* 127–141.

Richards, J. E. (2000). Localizing the development of covert attention in infants with scalp event-related potentials. *Developmental Psychology, 36,* 91–108.

Richards, J. E., & Casey, B. J. (1992). Development of sustained visual attention in the human infant. In B. A. Campbell, H. Hayne, & R. Richardson (Eds.), *Attention and information processing in infants and adults* (pp. 30–60). Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.

Richards, J. E., & Hunter, S. K. (1998). Attention and eye movement in young infants: Neural control and development. In J. E. Richards (Ed.), *Cognitive neuroscience of attention: A developmental perspective* (pp. 131–162). Mahwah, NJ: Lawrence Erlbaum Associates, Inc.

Schall, J. D. (1991). Neuronal activity related to visually guided saccades in the frontal eye fields of rhesus monkeys: Comparison with supplementary eye fields. *Journal of Neurophysiology, 66,* 559–579.

Schall, J. D. (1995). Neural basis of saccade target selection. *Reviews in the Neurosciences, 6,* 63–85.

Schall, J. D., & Hanes, D. P. (1993). Neural basis of saccade target selection in frontal eye field during visual search. *Nature, 366,* 467–469.

Schall, J. D., Hanes, D. P., Thompson, K. G., & King, D. J. (1995). Saccade target selection in frontal eye field of macaque: I. Visual and premovement activations. *Journal of Neuroscience, 15,* 6905–6918.

Searle, S. R. (1971). *Linear models.* New York: Wiley.

Searle, S. R. (1987). *Linear models for unbalanced data.* New York: Wiley.

Swick, D., Kutas, M., & Neville, H. J. (1994). Localizing the neural generators of event-related brain potentials. In A. Kertesz (Ed.), *Localization and neuroimaging in neuropsychology: Foundations of neuropsychology* (pp. 73–121). San Diego, CA: Academic.

Thickbroom, G. W., Mastaglia, F. L. (1985a). Cerebral events preceding self-paced and visually triggered saccades: A study of presaccadic potentials. *Electroencephalography and Clinical Neurophysiology, 62,* 277–289.

Thickbroom, G. W., & Mastaglia, F. L. (1985b). Presaccadic "spike" potential: Investigation of topography and source. *Brain Research, 339,* 271–280.

Weinstein, J. M., Balaban, C. D., & Ver Hoeve, J. N. (1991). Directional tuning of the human presaccadic spike potential. *Brain Research, 543,* 243–250.