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Localizing the Development of Covert Attention in Infants With Scalp Event-Related Potentials

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This study examined covert shifts of attention in infants aged 14, 20, and 26 weeks of age with scalp-recorded event-related potentials (ERPs). The infants were tested in a spatial cuing procedure. The reaction time to localize the target showed covert attention shifts (e.g., response facilitation or inhibition of return depending on cue–target stimulus onset asynchrony). There was a larger P1 ERP component on the valid trials than on the invalid trials or on the no-cue control trials. Presaccadic ERP potentials in response to the target were larger when it was in the cued location than when it was in uncued locations. There were increases from 14 to 26 weeks of age in the amount of inhibition of return, in the post-target-onset P1 effect, and in the presaccadic ERP potentials. These results suggest that cortical development parallels the development of covert orienting of attention and saccade planning in infants in this age range.

Visual attention may be shifted to different regions of space overtly by moving the eyes toward specific locations. Shifts of visual attention may also occur without moving the eyes, which is known as *covert orienting of attention.* Covert orienting of attention implies that information-processing resources may be shifted to a specific peripheral location. Information in that location or about that location is processed even though the eyes remain fixed on a focal visual location. Covert orienting has been demonstrated in adults in a spatial cuing procedure developed by Posner (1980; Posner & Cohen, 1984). In this procedure, the participant's fixation remained at a central location while a peripheral cue and target were presented. When the target followed the cue in the same location (valid trials) at a very short interval, the reaction time (Posner, 1980; Posner & Cohen, 1984), or the saccadic eye movement to that location (Posner, Rafal, Choate, & Vaughan, 1985; Rafal, Calabresi, Brennan, & Sciolto, 1989), was faster (*facilitation*) than it was when the cue and target appeared in different locations (invalid trials). Alternatively, when the cue and target were separated by longer intervals (e.g., 300 to 700 ms), the reaction time to the original location was slower on the valid trials than on the invalid trials. This attenuation of the reaction time has been called *inhibition of return.* The changes in reaction time as a function of the spatial relation between the cue and the target have been interpreted as indicating that attention was oriented toward the cued location in the absence of specific eye movements toward that location. This spatial cuing procedure has also been used to measure covert orienting of attention in young infants (Hood, 1993, 1995; Hood & Atkinson, 1991; Johnson, Posner, & Rothbart, 1994; Johnson & Tucker, 1996). The present study used scalp-recorded event-related potentials (ERPs) to examine infants' shifts of covert attention. One goal of the study was to determine if infants' covert shifts of attention to a peripheral cue could be indexed by ERP responses to a subsequent target. A second goal was to determine if the ERP changes occurring before the saccade in response to a peripheral stimulus were affected by covert shifts of attention to the cued location.

There are some studies that suggest that covert orienting may not exist in infants until about 6 months of age. Hood and Atkinson (1991, reported in Hood, 1995) presented 3- and 6-month-old infants with an interesting visual pattern. When the infants were fixating on this pattern, a cue was presented for 100 ms in the periphery. The infant's fixation usually remained on the focal stimulus, and there was no eye movement toward the cue when it was being presented. After a delay of 100 or 600 ms, a target appeared in the same location as the cue (valid trials) or on the opposite side from where the cue appeared (invalid trials). Trials in which no cue was presented and the target was presented (neutral trials) were also included. The 3-month-old infants showed no evidence of facilitation of the saccade toward the target at the 100-ms delay, nor did they show evidence of an inhibition of return at the 600-ms delay. The 6-month-old infants showed facilitation of the reaction time at the 100-ms delay and inhibition of return at the 600-ms delay for the valid trials. There were no differences at either delay in their responses on the invalid trials and the neutral trials. A study by Johnson and Tucker (1996) that used bilateral targets found that 4- and 7-month-old infants showed an increased probability of localizing the target that was ipsilateral to the cue at short delays (133–200 ms) and facilitated reaction times to the ipsilateral target. Alternatively, the infants showed a decreased probability of localizing the ipsilateral target at a longer delay (700 ms) and lengthened reaction times to the ipsilateral target at that delay. Johnson and Tucker did not find such an effect for 2-month-old infants. These studies imply that covert orienting of attention may emerge in the young infant between 2 and 6 months of age.

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The explanations given for the facilitation and inhibition of return found in spatial cuing involve the covert shifting of attention to the objects in the periphery. These explanations imply what might be developing in the spatial cuing tasks for young infants. The facilitation of reaction time when the cue and target are presented in the same location and when the cue–target temporal delay is brief is explained by a shift of attention to the cued location and an increased efficiency of processing at the attended location (Hillyard, Mangun, Woldroff, & Luck, 1995; Posner, 1980). Because attention is already on the target location, the sensory and perceptual processing of the target stimulus occurs more rapidly and efficiently than if attention must be shifted to this location after the target appears. Alternatively, inhibition of return is hypothesized to result from two shifts of attention—an attention shift from the central to the peripheral location followed by an attention shift back to the central location. Inhibition of return refers to a cognitive mechanism for inhibiting the return of attention to a location that has recently been processed and an apparent preference for stimuli occurring in novel locations. The lack of facilitation effects in young infants in this paradigm suggests that they do not shift attention to that location or do not benefit from such an attention shift if they do so. Another possibility is that shifts of attention in young infants take longer and may not be evident given the temporal constraints of the spatial cuing procedure (cf. Johnson & Tucker, 1996). The lack of inhibition of return in 2- and 3-month-old infants is likely a result of the infants' not shifting attention to and from the cued location when a competing focal stimulus exists. It has been reported that newborn infants show inhibition of return following overt fixation shifts from a central location to a peripheral location and back again (Simion, Valenza, Umilta, & Barba, 1995; Valenza, Simion, & Umilta, 1994; however, cf. Clohessy, Posner, Rothbart, & Vecera, 1991). This finding implies that the mechanism for inhibiting the return of attention to the recently attended location exists in 2- and 3-month-old infants but that they must not be showing the covert orienting of attention required in the spatial cuing procedure that would result in facilitation or inhibition of return. If the initial shift of attention required by facilitation does not occur, then the two shifts of attention required for showing inhibition of return should not occur, which would imply that facilitation effects might occur at younger ages than might inhibition of return.

Researchers studying the spatial cuing paradigm have included a strong neurophysiological component in their explanations of covert orienting of attention. The inhibition-of-return effect is thought to be mediated by the superior colliculus (see review by Rafal, 1998). For example, early studies showed that patients with damage to the superior colliculus showed difficulties with shifts of attention, and these difficulties interfered with the inhibition-of-return effect (Posner et al., 1985). Overt fixation shifts to and from a peripheral cue, or simply planning to make a saccade to and from a peripheral cue, result in inhibition of return in adult participants (Rafal et al., 1989). Alternatively, covert orienting initiated by endogenous cues that does not require specific planning for an eye movement to the peripheral location may not result in inhibition of return (Rafal et al., 1989). Thus, it is not covert orienting per se that results in inhibition of return. The activation of pathways in the superior colliculus responsible for saccadic fixation shifts results in inhibition of return, rather than the actual movement of the eyes or the inhibition of eye movement. Covert orienting in this view requires additional cortical structures to enhance the saccadic planning that occurs in attention shifts. Covert orienting also may require active inhibition of the final motor pathway for saccades to interrupt the reflexive saccadic movement typically generated by the superior colliculus. Such enhancement and/or inhibition and shifts of attention are hypothesized to be controlled by a "posterior attention network" (Posner, 1995; Posner & Petersen, 1990). This network includes the parietal cortex, regions of the thalamus projecting to the parietal cortex (e.g., pulvinar), frontal eye fields, and the superior colliculus.

Researchers studying infants with the spatial cuing procedure have uniformly adopted this neurophysiological perspective (e.g., Hood, 1993, 1995; Hood, Atkinson, & Braddick, 1998; Johnson, Gilmore, & Csibra, 1998; Johnson et al., 1994; Johnson & Tucker, 1996). The general conclusion of the "neurodevelopmental" approach is that the superior colliculus, which is relatively mature at birth, can support inhibition of return in early infancy (Hood, 1993, 1995; Simion et al., 1995; Valenza et al., 1994) but only for overt fixation shifts. The changes in covert attention shifts found between 3 and 6 months of age must therefore be due to cortical changes in areas such as the parietal cortex and frontal eye fields that involve saccadic planning and attention shifting (Hood, 1993, 1995; Johnson et al., 1994, 1998; Johnson & Tucker, 1996). This interpretation is consistent with the general view that in the first 6 months of life there is increasing cortical control over eye movements that occur during attention and increasing cortical control over general processes involved in attention shifting (e.g., Hood, 1995; Hood et al., 1998; Johnson, 1990, 1995; Johnson et al., 1998; Richards & Casey, 1992; Richards & Hunter, 1998). However, the work on spatial cuing and covert orienting with infants has used only measures of reaction time and no direct measures of brain function. One outcome of the current study may be the identification with scalp-recorded electrical activity of specific areas of the cortex that show developmental changes consistent with the behavioral changes thought to represent the covert orienting of attention found in the spatial cuing procedure.

The present study used scalp-recorded ERPs to aid in the study of covert orienting in young infants. The electroencephalogram (EEG) is a recording of spontaneous electrical activity in the brain that is measured on the scalp. Scalp EEG is caused by action potentials summed over large numbers of neurons, synapses, or neural pathways primarily in the cerebral cortex and in thalamocortical connections. The EEG may be time-locked to specific psychological or experimental events and averaged over multiple trials, resulting in averaged ERPs. An ERP has varying positive and negative electrical waves labeled components. These components are hypothesized to be related to specific events occurring in the cortex. The events in the cortex are hypothesized to be closely related to psychological processes. These components include those such as the P1 (or P100), N1, P2, N2, and P3 (or P300) as well as various slow waves (see de Haan & Nelson, 1997, for a discussion of these components in infants; see Hillyard et al., 1995, or Swick, Kutas, & Neville, 1994, for a discussion of these components in adults).

The ERP may be useful in two respects for studying the spatial cuing procedure. First, the effects of selective spatial attention on the early components of the ERP have been demonstrated in several studies of the spatial cuing procedure in adults (Eimer, 1996, 1997; Harter, Miller, Price, LaLonde, & Keyes, 1989; Hill-

yard, Luck, & Mangun, 1994; Mangun & Hillyard, 1991). In these studies, participants were presented with a cue that indicated the hemifield to which attention should be directed. The location to which attention was to be directed was indicated by an endogenous cue (i.e., an arrow) at the central location (Eimer, 1996; Harter et al., 1989; Mangun & Hillyard, 1991) or at an expected location (Eimer, 1997) or was indicated by an exogenous cue (a peripheral cue; Hillyard et al., 1994; Hopfinger & Mangun, 1998). The participants were presented with valid or invalid trials. These studies found a validity effect in the ERP for the positive component occurring near 100 ms (P1), and for the negative component occurring at approximately 175 ms (N1), after the onset of the target. The amplitude of the P1 and N1 components was larger on the valid trials than on the invalid (or neutral) trials. This validity effect occurred primarily in the occipital scalp areas contralateral to the hemifield in which the target appeared. These early ERP components are thought to reflect sensory and perceptual processes. These findings suggest that the covert orienting of attention affects the early stages (sensory and perceptual) of visual information processing in the brain rather than the decision or response stages of processing (Eimer, 1996, 1997; Hillyard et al., 1994, 1995). The effect of spatial cuing on these ERP components has not been studied previously in infants. I examined these components in the current study to determine if such early perceptual gating occurs in infants during covert orienting of attention. I also used these components as a tool for the "covert" assessment of covert orienting in infants. The youngest infants in this study may not show an effect of the spatial cuing parameters on localization latencies (e.g., Hood, 1995; Hood & Atkinson, 1991; Johnson & Tucker, 1996). However, they may show some sensitivity to the presence of the peripheral stimulus or to the spatial or temporal relations between the cue and the target in their ERP responses.

Second, the ERP responses preceding the onset of the saccade toward the target may be useful in this spatial cuing procedure. Several studies have examined EEG changes time-locked to the onset of a saccade, particularly those changes occurring immediately before the saccade onset, the "presaccadic ERP." There are three presaccadic ERP components that have been identified that may be useful for this research. First, an early negativity in the ERP has been reported that begins up to 1 s prior to saccade onset and has its maximum values over the vertex (Becker, Hoehne, Iwase, & Kornhuber, 1973; Kurtzberg & Vaughan, 1980, 1982; Moster & Goldberg, 1990). Second, a positive component (or slowly increasing positive wave) has been found about 30–300 ms prior to saccade onset and occurs primarily over parietal areas contralateral to the saccade direction (Becker et al., 1973; Csibra, Johnson, & Tucker, 1997; Kurtzberg & Vaughan, 1980, 1982; Moster & Goldberg, 1990). Third, several studies have reported a sharp positive spike potential in the presaccadic ERP over parietal scalp leads just prior to saccade onset (10–20 ms; Balaban & Weinstein, 1985; Becker et al., 1973; Csibra et al., 1997; Kurtzberg & Vaughan, 1980, 1982; Weinstein, Balaban, & Ver Hoeve, 1991). These presaccadic ERP changes have been hypothesized to reflect cortical areas involved in saccade planning (Balaban & Weinstein, 1985; Csibra et al., 1997; Csibra, Tucker, & Johnson, 1998; Johnson et al., 1998; Kurtzberg & Vaughan, 1980, 1982). Indeed, these potentials were larger, or more widespread, for saccades occurring in response to expected peripheral stimuli or predicted locations (Evdokimidis, Liakopoulos, & Papageorgiou, 1991; Evdokimidis, Mergner, & Lucking, 1992; Kurtzberg & Vaughan, 1982; Thickbroom & Mastaglia, 1985) and were larger for voluntary than for reflexive saccades (Balaban & Weinstein, 1985; Evdokimidis et al., 1991, 1992). The presaccadic ERP changes may be useful in the spatial cuing paradigm because the facilitatory effects of cuing on saccadic reaction time (Posner et al., 1985; Rafal et al., 1989) may occur if covert orienting toward the cued location results in an expectation or prediction about the location of the upcoming target. If saccade planning occurs toward this expected location, then the presaccadic ERP components should be larger for the valid trials than for the invalid trials or the neutral trials. These presaccadic ERP changes were studied recently in infants by Csibra et al. (1998), who found little evidence of the presaccadic ERP components they found in a comparable study of adults (Csibra et al., 1997). However, these studies (Csibra et al., 1997, 1998) used a procedure involving exogenous orienting to a cue in an unexpected location and might not have been optimal for presaccadic potentials related to target predictability. These presaccadic ERP responses have not been studied in the spatial cuing procedure, and specific hypotheses about their occurrence cannot be advanced.

In summary, I had two goals for the present study. The first was to determine if covert orienting of attention in young infants could be indexed with ERP responses. Infants were tested in a spatial cuing procedure at 14, 20, or 26 weeks (3, 4.5, or 6 months) of age with scalp-recorded EEG. These three ages were chosen because previous studies have shown the development of ERP components of covert orienting of attention to occur in this age range (Clohessy et al., 1991; Hood, 1993, 1995; Hood & Atkinson, 1991; Johnson et al., 1994; Johnson & Tucker, 1996). I examined covert orienting with ERP components to determine if experimental manipulations that should have resulted in behavioral indexes of covert orienting (facilitation or inhibition of return) also affected infants' ERP responses. In this regard, the ERP may be used as a psychophysiological measure that may show covert orienting of attention in the absence of visual localization latencies. Young infants (14 weeks old), who are not expected to show facilitation or inhibition of return in saccadic response latencies, may show ERP changes indicating that covert orienting occurs. The second goal of this study was to examine the ERP changes in relation to the initiation of eye movements toward the peripheral stimulus when it appeared as the target. Presaccadic ERP changes may show that covert orienting occurred and resulted in an expectation or prediction about target location. The presaccadic ERP components should be larger for valid trials if the infant expects a peripheral stimulus in that location. This study may aid in the identification of specific areas of the cortex that show development over this age that parallels the development of covert orienting of attention found in the spatial cuing procedure.

Method

Participants

The participants were infants recruited from birth notices published in a Columbia, South Carolina newspaper. The infants were full term, which was defined as having a birth weight greater than 2,500 grams and a gestational age of 38 weeks or greater based on the mother's report of her last menstrual cycle. A cross-sectional design was used to sample 35 infants ranging in age from 14 to 26 weeks. The ages of the infants at

testing were 14 weeks (N = 12, M = 103.5 days, SD = 4.83, 7 boys and 5 girls), 20 weeks (N = 11, M = 140.6 days, SD = 2.87, 8 boys and 3 girls), and 26 weeks (N = 12, M = 183.5 days, SD = 4.25, 5 boys and 7 girls). Five additional infants were tested who became fussy or sleepy during the testing session (2 at 14 weeks, 2 at 20 weeks, and 1 at 26 weeks). Eight infants were tested who did not complete enough trials to be included in the analysis because of equipment problems or poor EEG recording. The infants had no acute or chronic pre- or perinatal medical complications and were in good health at the time of recording.

Apparatus

The child was held on the parent's lap approximately 55 cm from a 49-cm (19-in) TV monitor. The TV subtended 44° of visual angle. A neutral-colored piece of material covered the surrounding area. A video camera was above the TV, and in an adjacent room, an observer judged the participant's fixations on a TV monitor. The session was recorded on videotape with a time code in order to synchronize fixation changes, stimulus information, and physiological measures.

A blinking dot was used to attract fixation to the central portion of the TV. This was a 2° square that changed from white to dark at 3 Hz. Stimuli were presented in the focal visual field that were known to elicit attention in infants in this age range. These were dynamic, black-and-white computer-generated patterns (e.g., a series of concentric squares of varying size, a flashing checkerboard pattern, a small box shape moving across a diamond). The movement in the focal stimulus was stopped 100 ms before the competing peripheral stimulus was presented. These stimuli were presented in a 10° square area. The peripheral stimulus (cue and target) was a rectangle shape (2° horizontal and 6° vertical) that changed dynamically in a sine wave grating. The inside edge of the peripheral stimulus was 18° from the center of the TV (approximately 8° from the edge of the focal stimulus). A *Sesame Street* movie was presented every 4 trials to keep the infant occupied while adjustments were made.

Procedure

The parent sat in a chair in the viewing area, and the infant sat on the parent's lap facing the TV monitor. Testing was conducted only if the infant maintained an alert, awake state during the procedure (eyes open, no fussing or crying, responding to the protocol). If the infant became fussy, a short break was taken and the presentations were paused and then restarted.

A spatial cuing procedure (see Hood, 1995, for a diagram of different conditions; Hood & Atkinson, 1991) was used to examine covert orienting. The experimental trials consisted of the focal stimulus presentation and cue and target presentations. At the start of each trial, a small blinking square was presented in order to orient the infant's fixation to the center of the TV. When the infant looked at the TV, the focal stimulus was presented. A new focal stimulus was presented on each trial. After 2 s of focal stimulus presentation, a competing stimulus (the cue) was presented in the periphery for 300 ms in addition to the focal stimulus, and then both stimuli were turned off. At delays of 150, 575, or 1,000 ms (450-, 875-, or 1,300-ms stimulus onset asynchronies, or SOAs), a peripheral stimulus was presented (the target). The peripheral stimulus remained on until the infant looked away from the focal stimulus location (toward or away from the target location). If the infant looked toward the peripheral stimulus, it remained on for an additional 2.5 s, followed by a 2.5-s intertrial interval.

There were five conditions defined by the cue and target presentations: *valid* trials—the cue and the target were presented in the same location; *invalid* trials—the cue and the target were on opposite sides; *no-target control* trials—the cue was presented without a target presentation; *no-cue control* (often termed *neutral*) trials—no cue presentation, but a target was presented; and *no-stimulus control* trials—presentation of the focal stimulus alone, without cue or target. If there was a shift of attention to the competing peripheral stimulus, then one would expect that localization latency and ERP responses to the target might differ on the valid and invalid trials. The no-cue control trials should show the appropriate behavioral and ERP averages occurring in response to a peripheral stimulus in an unexpected location, and the processing cost of shifting attention to an inappropriate location should be shown by a comparison of responses on invalid and no-cue control trials (Eimer, 1996, 1997; Hillyard et al., 1994, 1995). The no-target control and no-stimulus control trials may account for saccadic eye movements that might occur toward a location where a cue previously appeared but no target was present, for saccades toward a remembered location rather than to an overt target, and for control values of localization with no peripheral stimulus.

The trials on which the peripheral stimulus was presented as a target (valid, invalid, and no-cue control) were factorially combined with the SOA delays (450, 875, and 1,300 ms), whereas the trials with no peripheral stimulus presented as a target (no-target control and no-stimulus control) used only 1,300-ms SOA delays. This resulted in a total of 11 trial types that were presented randomly within an 11-trial block. Each infant received at least two trial blocks and was included in the analysis only if he or she had EEG data from each trial type. To obtain as many trials as possible, I continued the trials as long as the infants were not fussy. The number of trials for each infant ranged from 22 to 41 (M = 27.9 trials, Mdn = 27 trials). The number of trials for the entire sample of infants for each of the 11 trial types ranged from 54 to 60 (M = 57.6 trials, Mdn = 58 trials).

Measurement and Quantification of Physiological Variables

The horizontal electrooculogram (EOG) was recorded with 6-mm Ag–AgCl electrodes that were placed posterior to the outer canthus of each eye with the use of disposable electrode collars. The EOG was digitized at 1000 Hz (each millisecond) with a microcomputer. The EOG was amplified at 2K, and a direct current (DC) recording was made. The saccades were separated from the composite EOG record with an algorithm used by Matsuoka and Ueda (1986; Matsuoka & Harato, 1983; Richards & Holley, 1999; Richards & Hunter, 1997). A third-order differential filter was used to identify saccades, and a computer-based editing program was used to verify the onset–offset of each saccade. The onset–offset of the saccade and the EOG amplitudes (in microvolts) at the beginning and end of the saccades were recorded. The vertical EOG was measured with an alternating current (AC) recording to detect blinks or other eye-movement artifacts.

The EEG was recorded from 20 locations with nonpolarizable electrodes that were mounted in an elastic cap (ElectroCap International, Eaton, OH) and located at standard center, left-hemisphere, and right-hemisphere positions spanning the scalp according to the International Federation 10/20 recording system (Jasper, 1958; Pivik et al., 1993), which uses the following electrode location names: F_Z, P_Z, C_Z, F_{p1}, F_{p2}, 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. The 20th location was a non-10/20 electrode location called O_Z. 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 a Grass Neurodata Acquisition system (Astro-Med, Inc., West Warwick, RI) with bandpass filters set at 0.1 and 100 Hz, was amplified by 20K with a 60-Hz notch filter, and was digitized at 250 Hz (every 4 ms). Three caps were used, with the cap size determined by the circumference of the infant's head (38–42 mm, 42–46 mm, and 46–50 mm). Each electrode location was filled with Omni-Prep, a light-intensity rub was done, and then the electrode was filled with a separate recording gel. The electrodes were adjusted until impedance for all electrodes was < 5K ohms. During this preparation, which took 10–12 min, a second experimenter entertained the infants with toys, a child "busy box," clown faces, and so forth. In conformance with recommendations for infants and human participants (Pivik et al., 1993; Putnam, Johnson, &

Roth, 1992), the infants' scalps were not abraded, which made this a noncritical recording situation.

Quantification of Event-Related Potentials

The ERPs were obtained from the EEG recordings. The EEG recordings were first inspected for artifacts (e.g., a change in EEG > 100 μV) or poor quality, and individual channels or locations within trials were eliminated from the analyses if these occurred. Trials or portions of trials containing eye-movement artifacts, defined as EOG changes > 150 μV in either the vertical or the horizontal direction or blinks in the vertical EOG, were eliminated from the analyses. Epochs that contained eye movements with EOG changes < 150 μV were adjusted with regression techniques to remove the EOG-related artifacts in the EEG (de Haan & Nelson, 1997; Gratton, Coles, & Donchin, 1983; Kenemans, Molenaar, Verbaten, & Slangen, 1991; Nelson, 1994), but only as long as the eye movements were unrelated to the experimental events (e.g., movements within a stimulus). Epochs that contained eye movements related to the experiment (e.g., eye movement in response to a competing stimulus or the saccade in response to the peripheral stimulus) were not used in any analyses.

The ERP averages were made from the 4-ms-interval (250-Hz) EEG recording after artifacts were removed or adjusted. The EEG was digitally filtered with a 0.1–30-Hz bandpass filter, because the speed of the signals for the ERP averages are in this range. Averaging the EEG across trials increases the detectibility of the components in the ERP by increasing the signal-to-noise ratio. The literature on adult covert orienting and ERP[1] suggested no specific hypotheses concerning the ERP averages that related age or the experimental manipulations to the SOA delays. Therefore, SOA was not considered in calculating the ERP averages, which increased the number of trials going into each individual's averages (SOA effects were tested on the single-trial ERP data; see next paragraph). The EEG was first averaged for individual infants across all SOA delays for the five cue–target conditions. The number of trials making up each average is given in the caption to the figure showing that ERP average (see Results section.) The data for the ERP averages were taken from these infants' individual averages, and the grand averages shown in the figures in the Results section were based on the individual infants' averages for the appropriate stimulus type. The poststimulus ERP averages were calculated from 100 ms before the event through 1 s after the event of interest (focal stimulus onset, cue onset, and target onset). The presaccadic ERP averages consisted of data averaged (a) backward in time from the onset of the EOG activity indicating that a saccade had occurred to 750 ms before the saccade and (b) forward in time from the saccade onset for 100 ms.

In addition to calculating the ERP averages, I estimated measures of component peak latency and amplitude on a single-trial basis using the averaged ERP responses to identify the locations in which to take the single-trial measures. The use of ERP component analysis on a single-trial basis allowed testing of all the experimental factors in the analysis (EEG lead location, experimental condition, and SOA delay). Peak latency and amplitude were analyzed in a multistep procedure. First, the event-locked EEG on single trials was filtered, in order to emphasize the components that were hypothesized to be of interest (Farwell, Martinerie, Bashore, Rapp, & Goddard, 1993; Ruchkin, 1988; Smulders, Kenemans, & Kok, 1994). Such filtering eliminates unwanted frequencies in the EEG signal on single-trial EEG recordings in a manner analogous to the manner in which averaging techniques do. The EEG signal was filtered with a digital bandpass filter from 3 to 8 Hz for the single-trial data.

Second, the mean voltage in the prestimulus period (or in the postsaccadic period for presaccadic ERPs) was subtracted from the voltage in the poststimulus period. Peaks or troughs were identified in the EEG recording, and the maximum or minimum EEG was identified and recorded for these peaks and troughs (Luck & Hillyard, 1994a, 1994b). Initially, the amplitude and latencies were examined for relevant peaks in windows identified for adult durations (Luck & Hillyard, 1994a; also see McIsaac & Polich, 1992, and Neville & Lawson, 1987) and for infant durations (Karrer & Monti, 1995; McIsaac & Polich, 1992; Shucard, Shucard, & Thomas, 1987). The peak latencies that were examined for the postonset ERP components were as follows: P1, 50–150 ms; N1, 150–200 ms; P2, 150–300 ms; and N2, 175–300 ms. The time periods for presaccadic activity were based on data from adult studies (e.g., Balaban & Weinstein, 1985; Becker et al., 1973; Csibra et al., 1997; Kurtzberg & Vaughan, 1980, 1982; Moster & Goldberg, 1990) and pilot data, because there have been no studies of infant presaccadic activity in this paradigm. The peak latencies that were examined for the presaccadic ERP components were as follows: premotor negativity (PMN), up to 1 s before saccade; presaccadic positive potential (PSP), from 300 to 30 ms before saccade; and spike potential (SP), 10–20 ms before saccade.

Third, the peak amplitude (in microvolts) 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 (de Haan & Nelson, 1997; Luck & Hillyard, 1994a, 1994b). The area under the curve for the component was defined from the start and end of the identified component and was an additional measure of amplitude.

Fourth, the locations for the time points used from the single-trial analyses were determined only after constructing grand average ERP responses. Thus, the data for these analyses came from the single-trial peak-picking procedure, and the relevant time epochs came from the periods defined by the grand average ERPs.

Topographical ERP scalp potential maps were created to show some of the effects. For the topographical maps, the scalp potentials were rereferenced to an average reference, and interpolations were made with the use of 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 (see color figures in the Results section) show the distribution of the scalp potentials at a specific point in time and are useful in visualizing the ERP data shown in the other figures.

Judgments of Peripheral Stimulus Localization

Each session was judged offline by a single observer. A time code recorded on the videotapes allowed the judgments to be made with millisecond accuracy, though resolution was limited to a single video scan (0.5 × total frame length ≈ 16 ms). The observer judged the infant as looking toward the center stimulus, looking toward the right or left peripheral stimulus, or looking away from the TV. The time code on the videotape was synchronized with the computer clock in order to synchronize the physiological measures with fixation.

Localizations were based on the observer's fixation judgments in conjunction with the existence of saccades in the EOG. First, the infant had to be looking in the direction of the focal stimulus at the onset of the cue and target for the data to be used in the analysis. Second, localizations were defined according to the presence of saccades in the EOG and the localization judgment of the observer. A look was considered a localization when (a) the observer judged that the infant's eyes moved from the focal stimulus to any location near the peripheral stimulus, (b) a saccade occurred in the EOG recording in the appropriate direction, and (c) no other saccade occurred before that saccade. I also counted as localizations those instances in which a saccade in the appropriate direction occurred but the observer did not judge that a localization took place. On these trials, the

[1] After this study was completed and submitted for publication, Hopfinger and Mangun (1998) reported finding P1 validity effects for short SOA delays but not for long ones. Because the grand averages from each participant in the present study were summed over the SOA levels, I could not differentiate the average ERP responses by SOA level. The single-trial analysis, however, permitted a test of the SOA as a factor in the analysis.

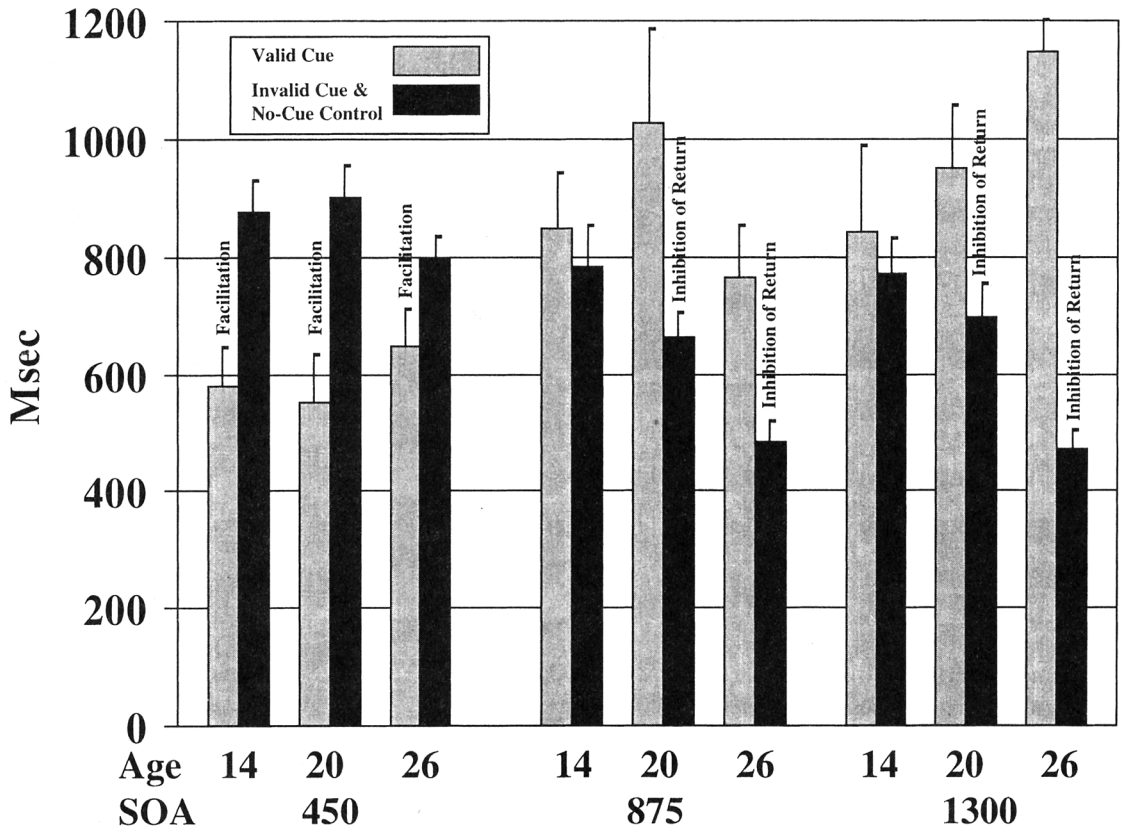

Figure 1. Latency (in milliseconds) to localize the peripheral stimulus when it was presented as a target. Latencies are presented separately for the three testing ages (in weeks), the three stimulus onset asynchrony (SOA; in milliseconds) conditions, and the valid cue trials and combined invalid-cue/no-cue-control trials. The error bars are the standard errors of the mean.

observer had to have judged that the infant was looking in the direction of the TV, and the saccade amplitude had to be similar to those in effect when the observer judged that a localization *had* occurred. This procedure (combining observer judgments and the existence of saccades) has been used in previous studies and has resulted in a high degree of interobserver agreement (Hicks & Richards, 1998; Richards & Hunter, 1997). The latency of the localization was defined as the onset of the first localizing saccade occurring after the onset of the peripheral stimulus.

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. If covert orienting to the cue occurred, at short SOAs reaction time should be faster on the valid trials than on the invalid or no-cue control trials (facilitation). Conversely, at long SOAs there should be a lengthening of reaction time on the valid trials compared with the other conditions (inhibition of return). Figure 1 shows the localization latencies for the valid trials and the other peripheral stimulus trials, separately for each age group. The localization latencies for the invalid and no-cue control trials were combined for this figure because the latencies on these two trial types did not differ overall or interact with the other factors. Significant facilitation may be seen for all three ages at the shortest SOA. Only the 20- and 26-week-old infants showed a significant inhibition of return at the 875- and 1,300-ms SOAs.

The latency measure was analyzed with an Age (14, 20, or 26 weeks) × Condition (valid, invalid, or no-cue control) × SOA (450, 875, or 1,300 ms) analysis of variance (ANOVA).[2] The latency measure had significant skew and kurtosis, so the variable was log-transformed, and the median latency of each participant was chosen for the valid, invalid, and no-cue control trials. There was a significant interaction of condition and delay, $F(4,$

[2] The ANOVAs for many of the analyses were performed with a general linear models approach that used a nonorthogonal design because of the unequal distribution of looks across factors and because of the different numbers of looks in the experimental conditions (see Hocking, 1985; Searle, 1971, 1987). 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 (e.g., experimental condition, SOA) within this class variable. The "PROC .GLM" of SAS (1996) was used for the computations. The duration dependent variables (e.g., look duration per stimulus) were log-transformed before analysis to obtain a variable consistent with a normal distribution.

Table 1
Number and Percentage of Looks Ipsilateral and Contralateral to the Cue Without the Target Present

Cue–target condition	Ipsilateral to cue		Contralateral to cue		Other trials (look at target or look away)	
	N	%	*N*	%	*N*	%
Cue present, look before target onset (valid and invalid)	71	22	19	6	227	72
Cue present, no target (no-target control)	118	71	45	27	4	2
No cue present[a], look before target onset (no-cue control)	6	4	7	4	134	92
No cue present[a], no target (no-stimulus control)	73	45	78	48	12	7

[a] For the no-cue control and no-stimulus control conditions, the putative side of the cue was randomly chosen on each trial.

124) = 2.92, $p = .0238$. This effect was examined separately for the 450- and 1,300-ms SOAs. The latencies for the invalid and no-cue control trials did not differ overall, nor did they interact with age or SOA. The latencies were significantly shorter (facilitation) for the valid trials than for the other trials at the 450-ms SOA ($p < .001$), and this effect did not differ for the three testing ages. The latencies were significantly longer (inhibition of return) for the valid trials than for the other trials at the 1300-ms SOA, but this effect was significant only for the 20- and 26-week-old infants ($p < .001$) and not the 14-week-old infants.

There were trials on which the infant looked away from the central stimulus before the target was presented (valid, invalid, and no-cue control trials) and on which the infant looked away when no target was presented (no-target control and no-stimulus control trials). I analyzed the side toward which the saccade was directed on these trials to determine (a) if the infant looked more toward the side ipsilateral to the cue, which would indicate an effect of the cue presence or a memory-driven saccade, and (b) what the infant did when no cue was present. Table 1 shows the probability of the infant's looking toward the side of the cue (a) before the peripheral stimulus occurred (valid and invalid trials), (b) when the cue was present but there was no target (no-target control trials), (c) when there was no cue but there was a target (no-cue control trials), and (d) when there was no cue and no target (no-stimulus control trials). It is evident from this table that the presence of the cue heavily influenced these memory-driven saccades. The saccades occurring before the target appeared or when no target appeared were heavily biased toward the side ipsilateral to the cue ($N = 189$) rather than to the side contralateral to the cue ($N = 64$), $\chi^2(1, N = 253) = 56.06$, $p < .0001$. In addition, infants made more saccades toward the ipsilateral side of the cue before the target appeared ($N = 71$) than toward either side when no cue was present ($N = 13$), $\chi^2(1, N = 84) = 31.67$, $p < .0001$. Finally, on trials without a cue, any saccades toward the periphery occurred approximately equally often on either side (i.e., $N = 79$ ipsilateral to and $N = 85$ contralateral to the arbitrarily "cued" side), $\chi^2(1, N = 164) = 0.22$, $p = .6396$. These saccades to the side of the previously presented cue did not show a systematic bias for the three testing ages.

Peripheral-Stimulus-Onset ERP

The ERPs in response to the onset of the peripheral stimulus when it was presented as a target were analyzed[3] to determine if the presence of a competing stimulus (cue) during presentation of the focal stimulus—that is, of a valid or invalid cue—affected the infant's subsequent response to the peripheral stimulus when it was a target. Figure 2 shows the ERP responses to the peripheral stimulus for the valid, invalid, and no-cue control trials, presented as difference scores from the trials on which no peripheral stimulus was presented as a target. The data in Figure 2 were plotted as if the peripheral stimulus was always presented on the left side, so the even-numbered electrodes (e.g., O_2) represent the contralateral leads and the odd-numbered electrodes (e.g., O_1) represent the ipsilateral leads. There was a significant positive component corresponding to the P1 component of the ERP. This component was largest for the valid trials, particularly at the most posterior leads on the contralateral sides (e.g., O_2, T_4, and T_6), but this difference between the valid trials and the other conditions also appeared at other recording locations. The latency of this component was about 135 ms for the three occipital leads. There also was a significant negative component, occurring at about 260 ms for the occipital

[3] The ERPs in response to the onset of the focal stimulus also were examined and analyzed. There was a significant positive component corresponding to the P1 ERP component that occurred primarily at the occipital (and other posterior) electrodes. This component peaked at between 100 and 150 ms for all three ages and did not appear to differ in amplitude across the three ages. There also was a large negative ERP component that occurred primarily at the frontal leads, with a maximum deviation at the F_Z electrode. The intervals effects (4-ms samples) were analyzed with repeated measures ANOVAs and adjusted with the Huynh-Feldt ϵ-adjustment to the degrees of freedom to control for inflated error rates with psychophysiological measures (Huynh & Feldt, 1970; Jennings & Wood, 1976; Keselman & Keselman, 1988; Pivik et al., 1993). There were expected effects of epochs (4-ms samples), differences between the EEG lead locations for the epochs, and significant age effects on the negative ERP component. As expected, there were no effects of experimental condition (valid, invalid, no-cue control, no-target control, or no-stimulus control) on the ERP response to the onset of the focal stimulus.

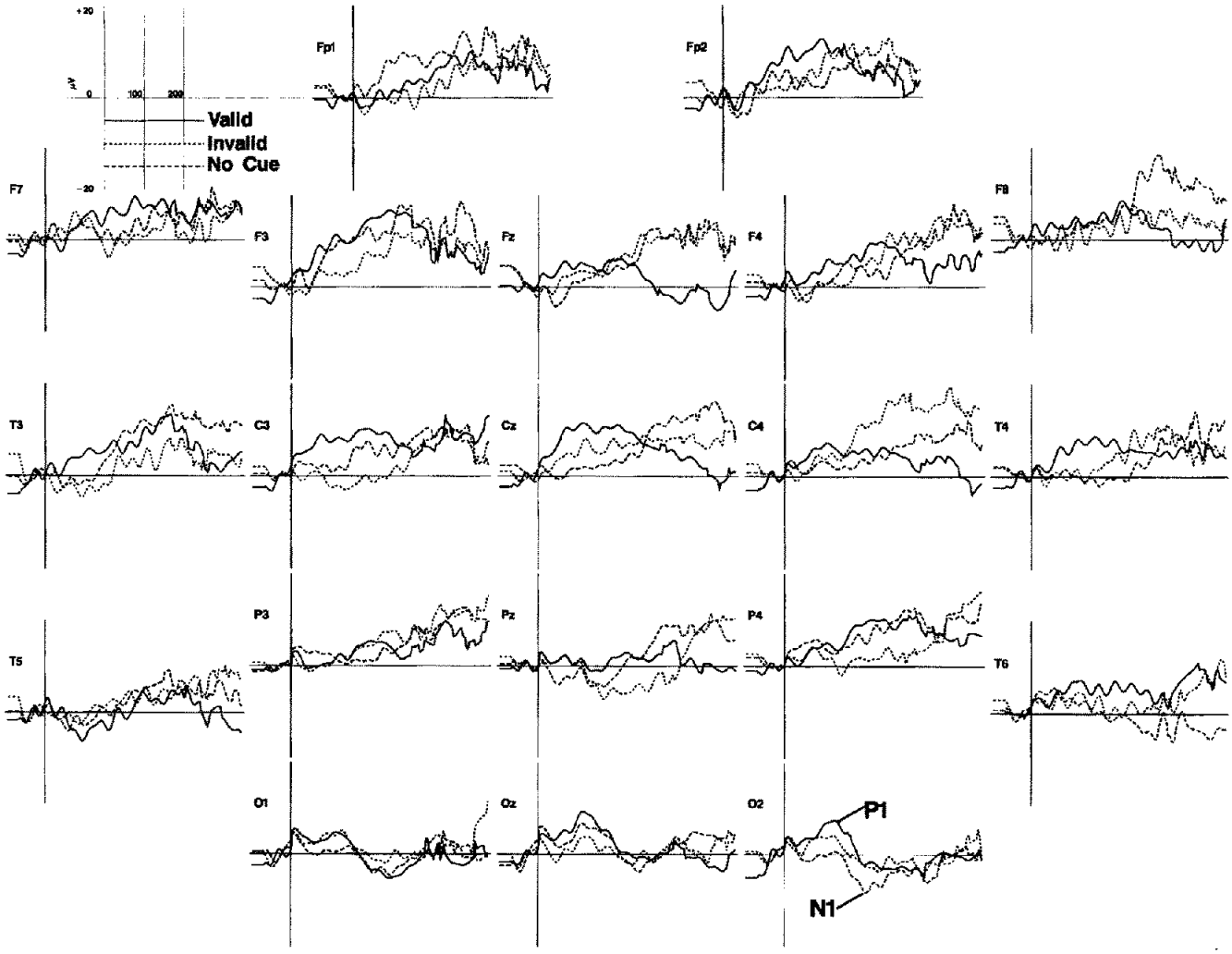

Figure 2. Event-related potential (ERP) responses to the onset of the peripheral stimulus when it was presented as a target. The responses are presented separately for the 20 recording electrodes and for the trials on which the cue and target were on the same side (Valid), the cue and target were on different sides (Invalid), and no cue appeared but the target was presented (No Cue). The data are presented as differences from the ERP responses on trials in which a peripheral stimulus was not presented as a target. The data for the electrode locations were reversed to the opposite hemisphere for the trials on which the peripheral stimulus was presented on the right side, so the even-numbered electrodes represent recording sites contralateral to the stimulus and the odd-numbered electrodes represent recording sites ipsilateral to the stimulus. The approximate locations of the P1 and N1 components are identified for the O_2 electrode. The median numbers of trials for each electrode going into the grand average were 126, 135, and 129 for the valid, invalid, and no-cue control conditions, respectively.

leads, perhaps corresponding to the N1 component. The N1 component did not appear to differ among the valid, invalid, and no-cue control trials.

The ERPs in response to the onset of the peripheral stimulus were also analyzed using the data from the single-trial identification of components. Only the data from the three occipital electrodes were analyzed because of the interest in the attention effects found with these electrodes.[4] The peak amplitude, component area, and peak latency were analyzed with an Age (3) × EEG Lead (3: ipsilateral occipital, contralateral occipital, and O_Z) × Experimental Condition (3) ANOVA. There was a significant effect of testing age for the P1 peak amplitude, $F(2, 31) = 3.14$, $p = .0573$, for the P1 component area, $F(2, 31) = 3.87$, $p = .0319$, and for the N1 component area, $F(2, 30) = 4.47$, $p = .0199$. In each case, the amplitude of the 14-week-old infants' response was larger than that of either the 20- or the 26-week-old infants' responses. There was a main effect of experimental condition on the P1 peak

[4] The peripheral-stimulus-onset data also were analyzed for epochs effects, as were the focal-stimulus-onset data. There were expected effects of epochs, differences between the electrode sites for the epochs, and age by epochs effects on the occipital sites, all of which confirm the effects found in the single-trial data analysis.

amplitude, $F(2, 46) = 3.24$, $p = .0500$. Consistent with the ERP plots shown in Figure 2, the response to the target on the valid trials was the largest, followed by the response to the invalid trials, and then by the response to the no-cue control trials (Ms = 29.79, 27.91, and 22.08 μV for the valid, invalid, and no-cue control trials, respectively). However, post hoc tests[5] showed a different pattern for the three electrodes. There was no difference in the ERP response for the experimental conditions at the ipsilateral occipital electrode ($F < 1.0$). The ERP responses for the two cue trials were approximately equal at the O_Z electrode and significantly larger than that for the no-cue control trials ($p < .05$). At the contralateral occipital electrode, the ERP responses for the valid trials were the largest, followed by the responses for the invalid trials, and then the responses for the no-cue control trials ($p < .05$; Ms = 30.25, 26.19, and 19.77 μV for the valid, invalid, and no-cue control trials, respectively). Post hoc tests also showed that this effect was different for the three testing ages. The 14-week-old infants did not differ significantly between the three testing conditions at any of the occipital electrodes, whereas the 20- and 26-week-old infants showed the difference between experimental conditions at the O_Z and contralateral occipital electrodes. There was a main effect of experimental condition on the N1 component area, $F(2, 55) = 3.33$, $p = .0433$. The N1 component area was larger on the two cue trials (Ms = -25.75 and -26.65 μV for the valid and invalid trials, respectively) than on the no-cue control trial ($M = -20.72$ μV). Thus, whereas the peak N1 amplitude did not differ among the three experimental conditions, the total area of the N1 component was larger on the cued trials. There were no significant effects on the latency of the peak amplitude for the P1 component or the latency of the peak amplitude for the N1 component.

The single-trial data also were analyzed with SOA as a factor in the analysis.[6] There were no effects involving this factor that were significant in the analysis. An examination of the means for this factor revealed no pattern of SOA on the ERP responses to the onset of the peripheral stimulus when it was presented as a target.

The age differences found in the single-trial component analysis were examined further with topographical ERP plots. Figure 3 shows the ERP response at the contralateral occipital electrode to the peripheral stimulus for the valid, invalid, and no-cue control trials, plotted separately for the three testing ages. There was an enhancement of the P1 response for the 20- and 26-week-old infants on the valid cue trials, a smaller response on the invalid cue trials, and the smallest response on the no-cue control trial. The 14-week-old infants had a larger overall P1 response, but it did not differ for the experimental conditions. Figure 3 also shows topographical potential maps for this P1 effect, plotting the ERP amplitude difference between the valid and no-cue control trials. These maps show that the amplitude of the P1 component was localized to the contralateral occipital area for the 26-week-old infants. The ERP for the 20-week-olds showed a wider spread of this P1 effect, which occurred at the contralateral parietal (i.e., P_4), temporal (i.e., T_4 and T_6), and occipital (i.e., O_2) sites. A smaller positive P1 effect appeared to be present in the 14-week-old infants over these same locations, although this effect was not significant in any of the analyses.

Presaccadic ERP

The ERPs preceding the onset of a saccade toward the peripheral stimulus when it was presented as a target were analyzed to determine if the presence of the cue affected the subsequent ERPs occurring before localization of the target. Figure 4 shows the ERP plotted backward from the onset of the saccade (Second 0) for the valid, invalid, and no-cue control trials. The data in this figure are plotted as if the infant was making a saccade toward a stimulus on the left side (even-numbered and odd-numbered electrodes were switched when the peripheral stimulus was on the right side). There were two obvious ERP changes in these plots. First, there was a large ERP response *during* the saccade on the contralateral frontal-temporal leads (F_{p_2}, F_8, T_4, and T_6 in Figure 4; labeled "Saccadic" on the F_8 graph). This response was likely due to the effect of the EOG change, or the contralateral muscle activity, resulting from the saccade. Second, there was a positive component that showed a sharp peak about 50 ms before the saccade. This was largest in the frontal leads and in the valid cue condition. This peak is referred to as the PSP and appears to be a specific ERP component rather than overall positive slow wave activity. This peak is identified in Figure 4 on some of the frontal leads. There also was a presaccadic negativity in several of the leads that occurred about 400–500 ms prior to saccade onset and a positive component that occurred about 300 ms prior to saccade onset.

The ERP responses preceding the onset of a saccade were analyzed using the data identified from the single-trial analysis. The peak amplitude and latency of the positive ERP component (PSP) approximately 50 ms before the saccade were analyzed with an Age (3) × EEG Lead (20) × Experimental Condition (3) ANOVA. There was only one significant effect on the PSP, the interaction among age, EEG lead, and experimental condition, $F(76, 976) = 1.48$, $p = .0060$. I examined this effect by looking at post hoc Age × Experimental Condition effects in groups of EEG leads. The Age × Experimental Condition effect was significant for the frontal and central leads, but not for the posterior (e.g., parietal and occipital) leads, and it occurred bilaterally. The impression given in Figure 4 that this effect occurred primarily on the

[5] In this and other post hoc tests, the Scheffé method was used to control for inflation of testwise error rate. The error mean squares for each post hoc comparison were obtained from the error term for the omnibus interaction for that post hoc evaluation. The significance level of the post hoc tests was $p < .05$ for all tests, and these individual probabilities are not reported in the text.

[6] The single-trial ERP data were analyzed with SOA as a factor, as was done by Hopfinger and Mangun (1998) in a study that reported P1 validity effects for short SOA delays but not for long ones. The peak amplitude of the P1 and N1 components was analyzed with an Age (3) × EEG Lead (3) × Experimental Condition (3) × SOA (3) ANOVA. The main effect of SOA and the interactions of the SOA factor with the other experimental factors were not statistically significant. The peak amplitude of the PSP preceding the saccade also was analyzed with an Age (3) × EEG Lead (20) × Experimental Condition (3) × SOA (3) ANOVA. Again, the main effect of SOA and the interactions involving SOA were not statistically significant. If the SOA had an important influence on the P1 or N1 effects, it should have manifested in this single-trial ERP analysis. It is doubtful that an effect of SOA would have appeared in the grand average plots (see Figures 2, 3, and 4) if the grand averages had not been summed over the SOA levels.

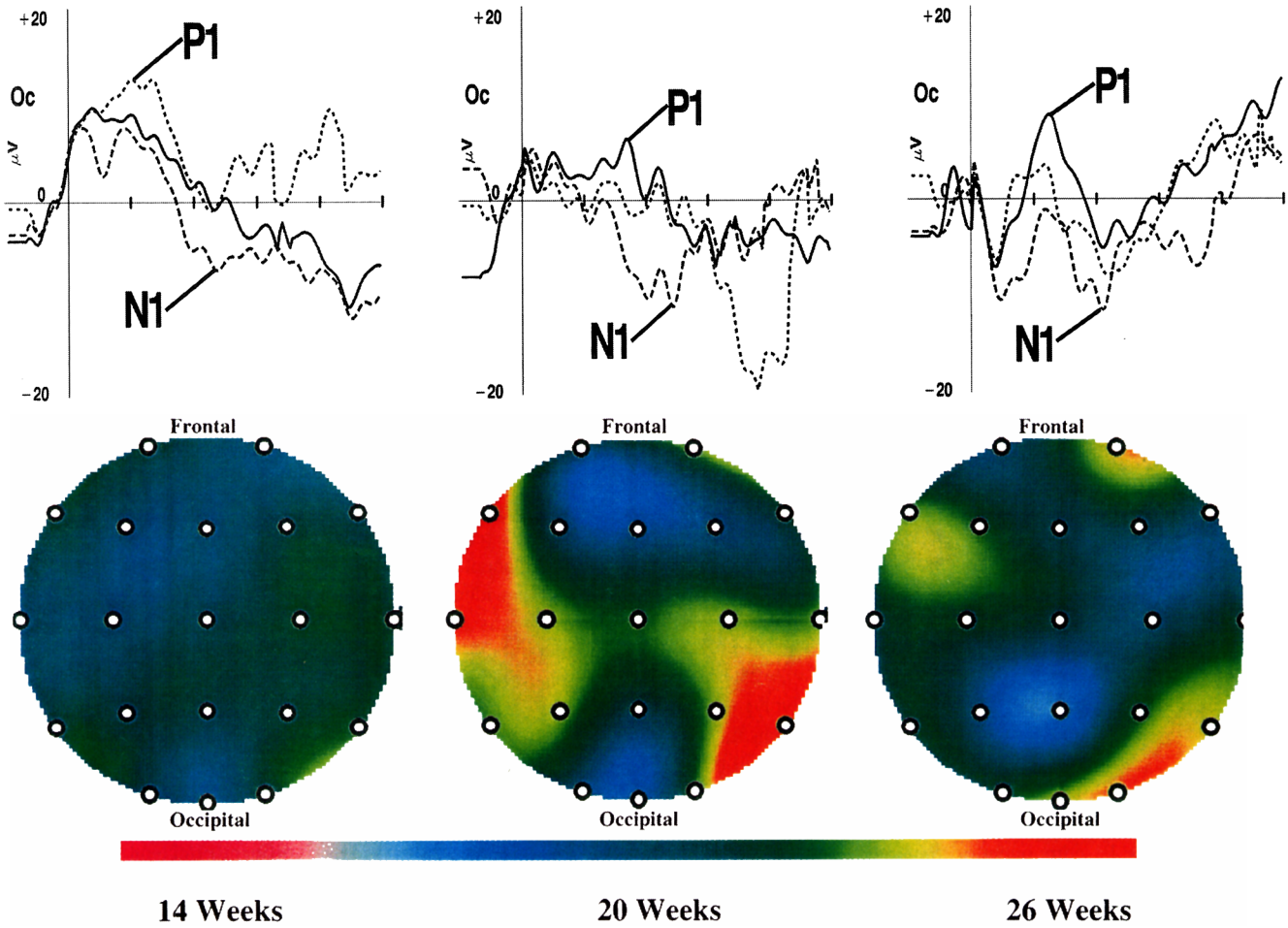

Figure 3. Top row: The event-related potential (ERP) responses at the contralateral occipital (Oc) electrode to the onset of the peripheral stimulus when it was presented as a target. The responses are presented separately for the three testing ages and separately for the valid (solid line), invalid (small dashes), and no-cue control (long dashes) trials. The data are presented as differences from the ERP responses on the no-stimulus control trial. The approximate locations of the P1 and N1 components are identified on each figure. Bottom row: Topographical scalp potential maps for the P1 effect for the three testing ages. These maps plot the difference between the valid and no-cue control trials for the peak potential, which occurred between 50 and 200 ms (M = 135 ms) following peripheral stimulus onset.

valid cue trials was confirmed by the post hoc analyses. That is, the peak responses were not significantly different for the invalid and the no-cue control trials, but the peak responses for both of those conditions were significantly different from that for the valid trials. For example, across all frontal leads, the mean level of this response was 5.88 μV (SE = 0.628) for the valid trials, 3.40 μV (SE = 0.388) for the invalid trials, and 4.01 μV (SE = 0.424) for the no-cue control trials. The difference across the three testing ages in this effect was such that the PSP did not change across age for the invalid and no-cue control trials combined (4.52 μV [SE = 0.371], 3.71 μV [SE = 0.405], and 4.37 μV [SE = 0.402] for the 14-, 20-, and 26-week-old infants, respectively) but significantly increased across the three testing ages for the valid trials (2.91 μV [SE = 0.546], 5.68 μV [SE = 0.868], and 9.70 μV [SE = 1.033] for the 14-, 20-, and 26-week-old infants, respectively). In summary, on the valid trials, in which the cue predicted where the target would occur, the saccades were accompanied by the PSP, primarily in the frontal EEG leads, and the PSP amplitude increased over the three testing ages. On the invalid and no-cue control trials, in which the cue did not signal the location of the target stimulus or no cue was present, the PSP was smaller and did not change significantly over the three ages. The latency of the PSP peak also was analyzed, but there were no significant effects involving experimental condition for this variable. The peak, area, and latency of the presaccadic negativity (peak around 400–500 ms prior to onset) and the presaccadic positive component (peak at 300 ms prior to onset) were analyzed. There were no significant experimental effects for these two components.

The presaccadic ERP responses were examined further with topographical ERP plots. Figure 5 illustrates the ERP response for the valid trials and for the invalid and no-cue control trials combined. These are shown as a sequence of maps beginning approx-

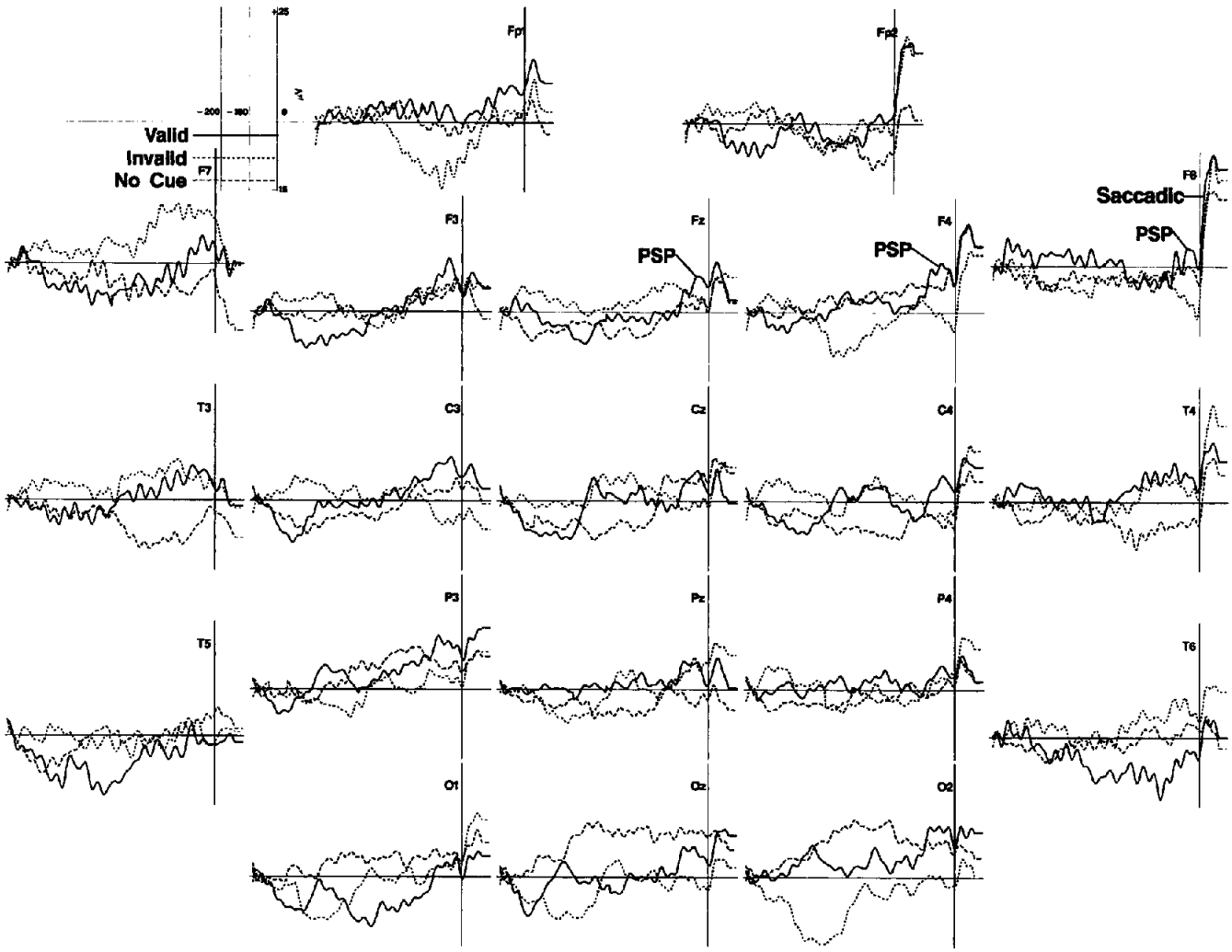

Figure 4. The event-related potential (ERP) responses occurring immediately prior to the onset of a saccade to the peripheral stimulus when it was presented as a target. The responses are presented separately for the 20 recording electrodes and separately for the valid, invalid, and no-cue control trials. The data are plotted backward from the onset of the saccade, which is represented as occurring at Second 0. The data in this figure were plotted as if the infant was making a saccade toward the left side (even-numbered and odd-numbered electrodes were switched for saccades toward a peripheral stimulus on the right side). The approximate location of the presaccadic positive potential (PSP) component is identified for the F_Z, F_4, and F_8 electrodes. The median numbers of trials for each electrode going into the grand average were 84, 112, and 119 for the valid, invalid, and no-cue control experimental trials, respectively.

imately 92 ms before the saccade and ending 28 ms after the saccade. The top series shows the ERP responses for the valid trials. There was a large amount of positive activity centered above the contralateral central area (i.e., C_4) and occurring at about −44 ms that corresponded to the PSP effect. The map suggests that this was centered at C_4, although this effect occurred over a wide range of electrodes (see Figure 4). This positive activity did not appear in the scalp potential maps for the invalid and no-cue control trials (bottom series). The large ERP change occurring during the saccade was reflected in these maps as positive activity centered on the contralateral frontal electrodes (i.e., F_8) and was fairly intense. This activity occurred in all three experimental conditions. Figure 6 shows the ERP response at −44 ms (PSP) for the valid trials separately for the three testing ages. There was almost no activity occurring in the 14-week-old infants, a localized positive activity in the 20-week-old infants centered at the contralateral central electrode (i.e., C_4), and a widespread contralateral activity in the frontal and central electrodes for the 26-week-old infants.

Discussion

The overall goal of this study was to examine the development of covert orienting of attention in infants from 14 to 26 weeks of age. A change in covert orienting over this age range should be reflected in the localization latency of a peripheral target when preceded by cues. Spatial and temporal cue–target relations also

should affect the localization latencies. The ERP was used (a) to examine the influence of covert orienting to the cue on subsequent responses to the target and (b) to examine presaccadic ERP changes occurring in response to the target. The ERP also may be useful in a "covert assessment" of covert orienting by providing a measure of the effect of the cue–target spatial-temporal relation in addition to the saccadic localization latencies.

The latency of the saccade to localize the peripheral target was affected by the spatial and temporal relations between the cue and target, and this effect changed over the three testing ages. First, all three ages showed faster localization of the target when it was presented at a brief delay in the same location as the cue than when it was presented in the opposite hemifield or when no cue appeared. This facilitation of the reaction time when the cue and target were presented in the same location is generally explained by a covert shift of attention to the cued location and more efficient processing of targets at the cued location (Hillyard et al., 1995; Posner, 1980). There was not an overt shift of fixation because the EOG indicated that the eyes remained in the central location. Second, infants at the older two ages also showed inhibition of return. When the cue and target were in the same location and the delay consisted of longer intervals (e.g., SOAs of 875 or 1,300 ms), 20- and 26-week-old infants showed slower localization of the target than when the target was presented in the opposite hemifield or when no cue appeared. At the longest delay (1,300 ms), this shift was larger for the 26-week-old infants than the 20-week-old infants. The youngest infants did not show evidence of inhibition of return at either the 875- or 1,300-ms delays. The interpretation of inhibition of return has been that attention was shifted from the focal to the peripheral location by the cue and then back to the focal location by the central stimulus and that these shifts were then followed by a difficulty in shifting attention to the previously processed location (Posner, 1980; Posner & Cohen, 1984).

The age changes in the effect of the cue–target relation on saccadic localization were partially consistent with previous findings on infants in this age range. The facilitation of saccadic reaction times to the target found at all three testing ages has not previously been reported in 3-month-old infants but has been found only in 4- and 6-month-old infants (e.g., 3- and 6-month-old infants, Hood & Atkinson, 1991; 2-, 4-, and 6-month-olds, Johnson & Tucker, 1996). Most studies have reported facilitation of the response time at short delays for the older infants, although Hood (1993) reported that 6-month-olds showed inhibition of return at a short delay (180-ms cue–target onset asynchrony, 0-ms cue–target gap; but 6-month-olds in the same experimental condition showed facilitation in a study by Hood & Atkinson, 1991). The inhibition of return found in the older two ages is consistent with findings that 4- (Johnson & Tucker, 1996) and 6-month-old infants (Hood & Atkinson, 1991; Johnson et al., 1994; Johnson & Tucker, 1996) showed longer reaction times to a target in the same location as the cue at intermediate delay intervals. It has also been shown in these two ages with bilateral targets that at these intermediate intervals a preference exists for localizing the invalid target rather than the ipsilateral target (Johnson et al., 1994; Johnson & Tucker, 1996). Shifting of attention to a target (facilitation) and shifting of attention to and from a target (inhibition of return) must operate covertly because these findings occurred in the absence of overt saccades toward the cued location, that is, fixation remained on the central location until the target was presented.

Post-Target-Onset ERP

The spatial relation between the cue and the target significantly affected the ERP to the onset of the target. There was a larger positive ERP component occurring at about 135 ms when the cue and target were in ipsilateral hemifields (valid trials) than when the cue and target were in contralateral hemifields (invalid trials) or when a cue did not precede the target (neutral trials). The validity effect on this positive ERP component did not occur (or was very small) in the 14-week-old infants, occurred at larger levels in the 20-week-old infants, and was at its largest in the 26-week-old infants. The ERP component occurred at the contralateral occipital leads for the target onset (peripheral visual field; see Figures 2 and 3). This ERP component was similar to the P1 (i.e., P100, or first positive ERP component) found in adult ERP recordings (Hillyard et al., 1995; Swick et al., 1994). A negative ERP component occurred around 260 ms after onset of the target and was larger at the contralateral posterior leads and larger on the cued trials relative to the neutral trial. This ERP component was similar in form to the N1 component found in adult participants, although in adults this component generally occurs around 175 ms after stimulus onset (e.g., 150–200 ms in Luck & Hillyard, 1994a, 1994b). The enhanced P1 on the valid trials was similar to that found in studies of spatial cuing procedures that used adult participants (e.g., Eimer, 1997; Harter et al., 1989; Hopfinger & Mangun, 1998; Mangun & Hillyard, 1991). This early ERP component reflects sensory and perceptual processes and suggests that covert orienting of attention affects the early stages of processing rather than the later stages (Eimer, 1996, 1997; Hillyard et al., 1994, 1995). The existence of this component in the older infants in the present study is a "covert" demonstration that covert orienting occurred in response to the peripheral stimulus when it was presented as a cue. The results from this study indicate that infants were shifting attention to the cued location covertly and that this early sensory-perceptual gating occurs in infant attention as it does in adult attention.

There was a developmental dissociation between the age changes in the P1 ERP component and the age changes in saccadic localization latencies. There was a gradual increase in the validity effect in the P1 component over the three testing ages. The 14-week-old infants showed no distinction in this ERP component between the experimental conditions, the 20-week-old infants showed an intermediate validity effect for this positive ERP component, and the 26-week-old infants showed the largest validity effect for this ERP component. However, infants at all three ages showed a facilitation of saccadic localization latencies on the valid trials compared with the invalid or neutral trials (see Figure 1). The developmental dissociation between this facilitation and the changes in the P1 ERP component was complemented by the findings for inhibition of return and presaccadic ERP changes. Infants at the oldest two testing ages (20 and 26 weeks) showed the validity effect in the ERP components, the facilitation of saccadic localization latencies, the inhibition of return, and validity effects in the PSP component of the presaccadic ERP. The 14-week-old infants showed a validity effect only for the facilitation of the

reaction time at short SOAs. The effects demonstrated by the 20- and 26-week-old infants showed gradual age changes. The 20-week-old infants showed intermediate levels of inhibition of return (280-ms difference between valid trials and other trials at the 1,300-ms SOA; see Figure 1), intermediate levels of the P1 validity effect (see Figure 3), and intermediate levels of the PSP component in the presaccadic ERP (see Figure 6). The 26-week-old infants showed the largest effects for inhibition of return in saccadic localization latencies (675-ms difference between valid trials and other trials at the 1,300-ms SOA; see Figure 1), for post-target-onset ERP responses, and for presaccadic ERP components.

Several findings in this study indicate that the 14-week-old infants were sensitive to the presence of the peripheral stimulus when it was presented as a cue along with the focal stimulus. The 14-week-old infants showed facilitation effects on saccadic localization latencies (see Figure 1), that is, a validity effect on saccadic reaction times. They also showed anticipation errors in the direction of the cue (see Table 1). In addition, the 14-week-olds showed a large ERP component at the occipital electrodes contralateral to the target before looking toward it (e.g., see Figures 2 and 3). This "covert" assessment of target responsivity showed that the infants' P1 response was sensitive to the target presence but not to the cuing conditions preceding it. Other studies have shown that infants are sensitive to characteristics of stimuli in the periphery such as flicker, form, contour, size, and spatial density (e.g., Hicks & Richards, 1998; Lewis, Maurer, Burhanpurkar, & Anvari, 1996). Thus, the 14-week-old infants in the current study were sensitive to the presence of the peripheral stimulus without looking toward it (cue) or before looking toward it (target). The lack of the full range of spatial covert orienting effects was not due to inadequate or immature peripheral stimulus sensitivity.

The facilitation of the saccadic localization latencies on the valid trials for the 14-week-old infants, together with the absence of validity effects on P1, inhibition of return, and presaccadic ERP components, suggests that the peripheral stimulus was automatically processed without covert shifts of attention by the 14-week-old infants. An exogenous cue in the spatial cuing procedure may elicit reflexive saccadic programming rather than attentional saccadic programming. This saccadic programming may cause short-delay facilitation effects, such as those found in this study (Hillyard et al., 1994; Hopfinger & Mangun, 1998). In addition, saccades in the short-delay condition may consist of those that were programmed before the target onset as well as target-elicited saccades. The faster localization latencies would therefore be due to a combination of preprogrammed saccades and target-elicited saccades. This explanation would be consistent with the large number of saccades made before target onset to the same side of the cue (see Table 1). The processing of the peripheral stimulus by the 14-week-old infants included the location of the stimulus, a reflexive saccadic planning toward the stimulus location that resulted in facilitation on the valid trials, and the appearance of the sensory-perceptual P1 component in response to the target for all target conditions.

The 20- and 26-week-old infants showed the full range of effects that would be expected to indicate covert shifts of attention in the spatial cuing procedure. The facilitation of the saccadic reaction time coupled with the validity effects on P1 indicate that the 20- and 26-week-old infants shifted attention to the cued side and were more efficient at processing the information in the attended location (Hillyard et al., 1995; Posner, 1980). Inhibition of return is hypothesized to result from an attention shift from the central to the peripheral location followed by an attention shift back to the central location. Infants at the oldest two ages showed this effect in the current study, indicating that they were able to keep processing resources on the central stimulus and to shift processing resources to the peripheral cue in parallel. This resulted in continued fixation on the focal stimulus (focal stimulus attention) and processing of the stimulus location of the peripheral cue, which led to inhibition of return and validity effects in the ERP. The P1 and presaccadic ERP effects (see next section) found in the older infants are consistent with the interpretation that attention shifts occurred in these infants and resulted in the full range of spatial cuing procedure effects. These results suggest that there was a gradual increase in the level of covert orienting of attention over these three ages. This interpretation of the present results implies that facilitation occurred without covert shifts of attention (i.e., no inhibition of return and no ERP validity effects in 14-week-olds) whereas inhibition of return was positively correlated among the age groups with other indexes of covert orienting (e.g., graded ERP effects).

Presaccadic ERP

The presaccadic ERP components in this study cannot be readily identified with analogous presaccadic ERP changes occurring in adult participants. First, a presaccadic positivity was found about 300 ms prior to the saccade onset. This presaccadic positivity was similar in some respects to a positive ERP component found in adults that occurs about 30–300 ms prior to saccade onset (Becker et al., 1973; Csibra et al., 1997; Kurtzberg & Vaughan, 1980, 1982; Moster & Goldberg, 1990). However, in the present study this activity was not limited to or centered at the contralateral parietal areas, as has been reported in studies with adults. Second, there was a positive ERP component occurring about 50 ms before saccade onset (labeled "PSP" in Figure 4) that occurred at several EEG leads when the saccade was toward a target that had been preceded by a cue occurring in that location (valid trials). This PSP component in the current study bears only superficial similarity to the positive spike potential found in adults. The spike potential occurs primarily over parietal scalp leads immediately prior to saccade onset in adults, is extremely short in duration, and occurs in the 10–20 ms 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 et al., 1991). The PSP component found in the current study had a different distribution (frontal–central), differed in peak latency (50 ms before saccade onset), and was longer in duration. The latency differences in the presaccadic ERP components between this study and adult studies may be due to differing latencies of infant and adult ERP components (cf. McIsaac & Polich, 1992). However, the different scalp locations argue against such an analogous identification. Thus the PSP in this study does not seem to be easily identified as analogous to any of the presaccadic ERP components found in studies with adults.

There was, however, a functional similarity between the PSP component identified in the current study and the presaccadic ERP

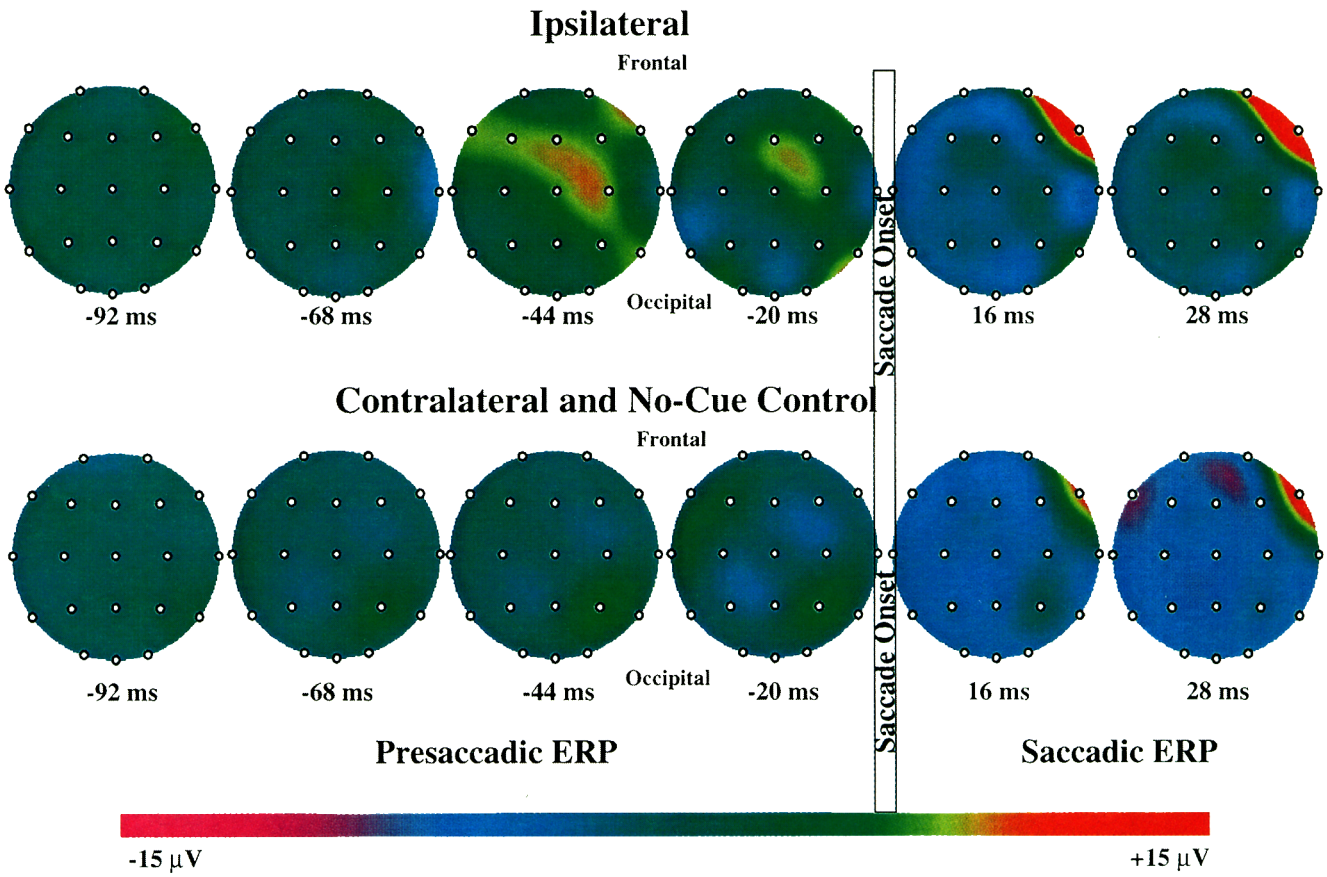

Figure 5. Topographical scalp potential maps for the presaccadic event-related potential (ERP) responses for the valid trials and the combined invalid and no-cue control trials. The maps are shown as a series and represent 12-ms averages of ERP responses taken from 92 ms preceding the saccade onset through 28 ms during the saccade. The presaccadic positive potential (PSP) is evident on the valid trials at about −44 ms preceding saccade onset, and the positive ERP activity occurring during the saccade is evident for all trial types at the frontal electrodes at 16 and 28 ms following saccade onset.

changes that have been found in adults. Positive and negative 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 et al., 1991, 1992; Kurtzberg & Vaughan, 1982; Thickbroom & Mastaglia, 1985). These presaccadic ERP changes reflect cortical areas involved in saccade planning (Balaban & Weinstein, 1985; Csibra et al., 1997, 1998; Johnson et al., 1998; Kurtzberg & Vaughan, 1980, 1982). The shift of attention to the cued location may have resulted in an expectation or prediction about the location of the upcoming target and thus in the faster processing of the target in that location. Thus, the presaccadic ERP component was larger for the valid trials than the invalid or neutral trials precisely because saccade planning occurred toward the expected location. If this interpretation is correct, it would suggest that the PSP component reflected saccade planning in cortical areas for the two older groups of infants.

The findings from this study may be profitably compared with those from recent studies that examined presaccadic ERP changes in adults and infants (Csibra et al., 1997, 1998). Those studies presented participants with a focal stimulus and with a peripheral stimulus presented simultaneously with the focal stimulus (the "overlap" condition) or after a brief delay (the "gap" condition). Adults in this paradigm showed the presaccadic slow wave activity and spike potential in both conditions (Csibra et al., 1997). However, 6-month-old infants tested in a similar manner (Csibra et al., 1998) showed no evidence of these specific presaccadic ERP components. The authors concluded that saccades toward the peripheral targets for infant participants in their studies were under subcortical control (e.g., the superior colliculus) and did not involve cortical saccade planning. The lack of a specific spike potential in the current study is probably in line with these findings. However, the procedure in the Csibra et al. (1997, 1998) studies involved exogenous orienting to a cue in an unexpected location. The infants in those studies may not have developed an expectation about the location of the target because there was no previous cue to indicate where it would occur. In the present study the cue may have explicitly indicated to the infant where attention should be shifted, resulting in enhanced post-target-onset ERP responses (P1 component), expectations about the appearance of the target in that location, and cortical saccade planning as reflected in the presaccadic PSP component of the ERP.

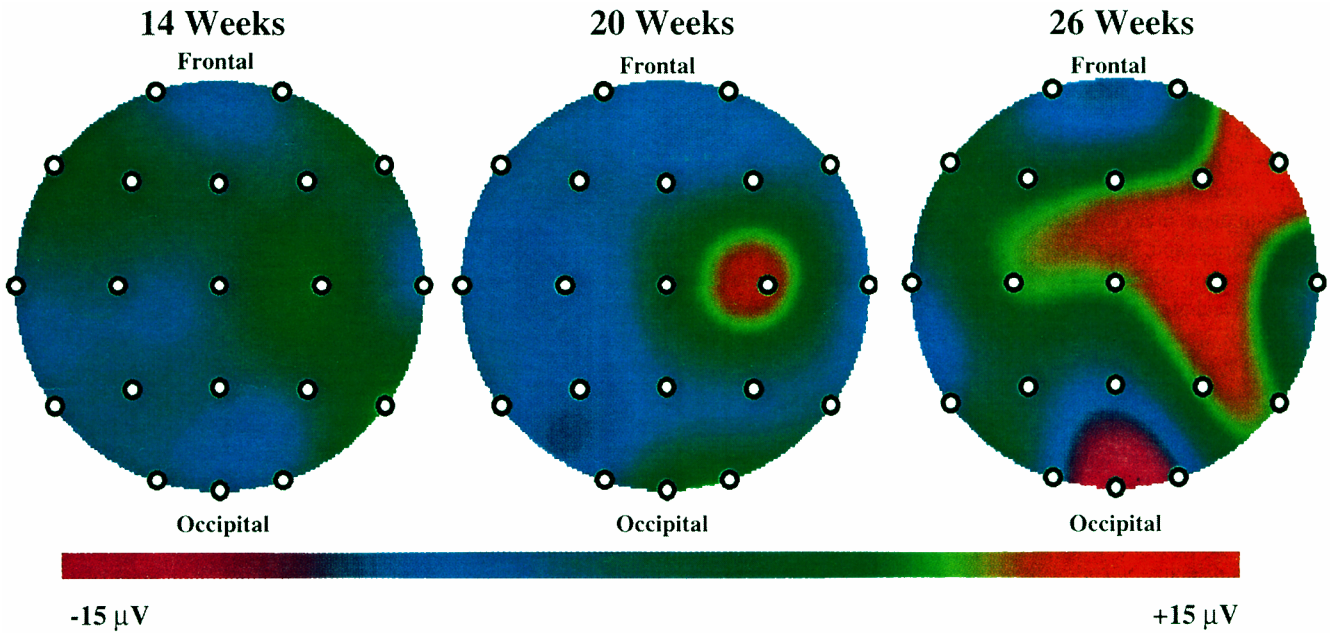

Figure 6. Topographical scalp potential maps for the presaccadic positive potential (PSP) effect occurring in the valid trials, separately for the three testing ages. These maps plot the peak potential for 12 ms centered at 44 ms before the onset of the saccade toward the peripheral stimulus.

Infant Covert Orienting of Attention and Cortical Development

One implication of this research is the identification of specific areas of the cortex that may develop over this age range and that may be responsible for ERP responses in the spatial cuing procedure. It should be acknowledged that the localization of cortical areas involved in cognitive processing simply through the use of scalp-recorded ERP is tenuous. The relatively small number of electrodes in this study, the difficulty of inferring cortical sources from scalp ERP, and the lack of converging measures of cortical activity limit the generalizations that may be made from EEG and ERP recording. An additional problem with infants in this age range is that there are large changes in skull size over this age range and dramatic changes in cortical area. Thus, inferring cortical activity sources from scalp ERP may be difficult because of changes in the skull, changes in the underlying brain structures, or changes in the relation between scalp location (or underlying skull location) and cortical areas. For example, it appears that the P1 effect for the 20-week-old infants occurred over the posterior aspect of the temporal lobe (e.g., T_6 in Figure 3) whereas for the 26-week-old infants it occurred over the occipital lobe (e.g., O_2 in Figure 3). This difference in scalp potential distribution may have been caused by age changes in the relative position of the same cortical area. Converging evidence from structural imaging (i.e., magnetic resonance imaging [MRI]) or functional imaging (e.g., positron emission tomography, functional MRI) would be necessary to confirm these cortical locations.

There is evidence from studies of adults that the generators of the P1 and N1 components and the validity effects found in studies of spatial cuing come from extrastriate occipital areas of the cortex. For example, using high-density scalp recording of ERP, current source density topographical maps, identification of brain structures with MRI, dipole source modeling, and ingenious experiments over the visual hemifields and quarterfields, Hillyard, Mangun, and others (see Hillyard et al., 1995) have shown that the cortical sources of the P1 and N1 effects are the lateral extrastriate visual areas of the occipital cortex rather than striate areas. This finding suggests that these effects occur at an intermediate level in the visual pathway rather than in thalamic (lateral geniculate nucleus) or striate visual areas. The 14-week-old infants in the current study showed substantial P1 and N1 ERP components in response to the targets that occurred in all three experimental conditions. The 20- and 26-week-old infants showed the validity effects found in adults with spatial cuing procedures. This extrastriate occipital area was sufficiently mature in the youngest infants to show the early ERP components, and developmental changes occurred in attentional modulation of this area. This change in attentional modulation may have occurred through the development of the attentional selectivity of this area itself or through the development of cortical areas other than the striate or extrastriate occipital cortex. One candidate for such developmental change would be an increasing top-down control of these visual pathways by other attention systems (i.e., Area PG of parietal cortex, or the "posterior attention system"; Posner, 1995; Posner & Petersen, 1990). These findings also indicate that the covert orienting of attention in infants at the two older ages affects relatively early stages of visual information flow through the brain. Attention gates the flow from these predominantly sensory-perceptual cortical areas to other areas involved in spatial attention and stimulus processing.

There have been no studies of adults in the spatial cuing paradigm that used ERP measurement, saccades, and presaccadic ERP changes. The studies of presaccadic ERP components have used only EEG and ERP and have not used modern localization tech-

niques. Given this caveat, however, it is tempting to suggest that the presaccadic ERP component found in the present study may have been generated from cortical areas involved in saccade planning to expected stimulus locations. The presaccadic ERP component identified in this study occurred in the central and frontal regions and was located predominantly over the scalp regions contralateral to the saccadic eye movement. These areas lie close to the supplementary eye fields (SEFs) of the premotor area of the frontal cortex and the frontal eye fields (FEFs) of the prefrontal cortex. Several studies of nonhuman primates that used single-cell recording techniques have shown that cells in the FEF and SEF fire in advance of saccade onset to attention-directed targets (Hanes & Schall, 1996; Hanes, Thompson, & Schall, 1995; Schall, 1991a, 1991b, 1995; Schall & Hanes, 1993; Schall, Hanes, Thompson, & King, 1995). The presaccadic firing of cells in these two areas has a time course similar to that of the PSP found in the present study. The FEF and SEF areas initiate and direct saccadic eye movement by inhibition or disinhibition of the superior colliculus, or they may initiate saccadic eye movements by affecting brainstem motoneurons directly (Schall, 1995; Schiller, 1985, 1998). The FEF and SEF generate volitional eye movements (planned saccades), whereas the superior colliculus initiates and directs reflexive saccadic eye movements. If the ERP activity preceding saccade onset in the infants in this study represents activity in the SEF or FEF cortical areas, this would be consistent with the position that the saccades on the valid trials represent an expectation or prediction about the location of an upcoming target and would be evidence of cortically driven saccade planning in infants at the two oldest ages.

The conclusions regarding the neurodevelopmental changes occurring in covert orienting of attention may be summarized as follows: The response of the 14-week-old infants to the peripheral stimulus when it was presented as a cue appeared to be tapping relatively automatic processes. One such process may be an activation of the cells in the superior colliculus responsible for reflexive peripheral stimulus localization. The reflex localization was inhibited by the focal stimulus attention during the cue presentation. Facilitation of the saccadic localization latencies for the valid trials occurred as a result of this automatic activation. Other indexes of covert orienting of attention to the peripheral stimulus were absent in the 14-week-old infants. There was a gradually increasing role of attention in the responses of the 20- and 26-week-olds in the spatial cuing procedure, as demonstrated by a change in the level of the inhibition of return at the longest cue–target delay, an increase in the level of the P1 effect over the three testing ages, and an increase in the cortical activity indicating saccadic planning over the three testing ages. This increasing role of attention from 14 to 26 weeks of age involved an increasing influence of the cortex in this spatial cuing procedure. This interpretation of these findings is consistent with theoretical models that have hypothesized an increasing role for the cerebral cortex over this age range in controlling the shifting of attention and attention-related eye movements (e.g., Hood, 1995; Hood et al., 1998; Johnson, 1990, 1995; Johnson et al., 1998; Richards & Casey, 1992; Richards & Hunter, 1998).

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