CHAPTER

14

Development of Covert Orienting in Young Infants

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ABSTRACT

Adults can shift attention to different regions of space without moving the eyes, that is, covert orienting of attention. Covert orienting implies that information processing may occur for stimuli in peripheral locations. The purpose of this chapter is to review evidence that in the first 6 months of life, infants are able to shift attention throughout space covertly. These studies show that there is an increasing efficiency from birth to 6 months with which infants shift spatial attention. Some cortical areas that may be involved in the development of spatial attention are suggested.

I. INFANTS CAN SHIFT ATTENTION COVERTLY TO PERIPHERAL STIMULI

Several studies have shown that covert orienting can occur in young infants. The spatial cuing procedure developed by Posner (Posner, 1980; Posner and Cohen, 1984) (see Chapters 16, 31, and 64) was adapted by Hood (1995) to study covert orienting in infants. In Posner's procedure, the participant's fixation remained at a central location while a peripheral cue and target were presented. Two reaction time effects are used to show covert shifts of attention: response facilitation and inhibition of return (see Chapter 64). Response facilitation occurs when the cue and target occur close in time in the same location. Response slowing (inhibition of return) occurs when the cue and target are separated further in time and the cue and target are in the same location. Hood presented infants with an interesting visual pattern in the center. When the infant is fixating on this pattern, a stimulus is presented in the periphery (analogous to "cue") in addition to the central stimulus. Infants will not shift fixation from the center pattern to the peripheral pattern during the brief presentation of this peripheral stimulus (Richards, 1987, 1997, 2002). The peripheral stimulus and central stimulus are then removed, and the peripheral stimulus is presented in the periphery (analogous to "target"). The eye movement from the center position to this peripheral stimulus is the dependent measure. The target can be presented on the same side as the cue ("valid trials") or on the opposite side ("invalid trials"), cannot be presented ("no-target control"), or can be presented on a trial without the cue being presented ("neutral"). Hood and Atkinson (1991, reported in Hood, 1995; Hood, 1993) tested 3- and 6-month-old infants in this procedure. The reaction time of infants at 3 months of age for valid, invalid, and neutral trials was no different between a target presented at 200-ms stimulus onset asynchrony (SOA) or 700-ms SOA, that is, no facilitation or inhibition of return. The reaction time of the 6-month-old infants was facilitated on the 200-ms-SOA validly cued trials, relative to neutral or invalid trials. Reaction time was slowed on the 600-ms-SOA trials, that is, inhibition of return. There were no differences at either delay in the responses on the invalid or the neutral trials.

Other aspects of covert orienting have been studied in infants. Johnson and Tucker (1996) used a similar procedure for cuing the infant, and then presented bilateral stimuli in the "target" period. They found that 4- or 7-month-old infants had an increased probability of localizing the target that was ipsilateral to the cue at short delays (SOA of 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 (SOA of 700 ms) and lengthened reaction times to the ipsilateral target at that delay. They did not find such an effect for

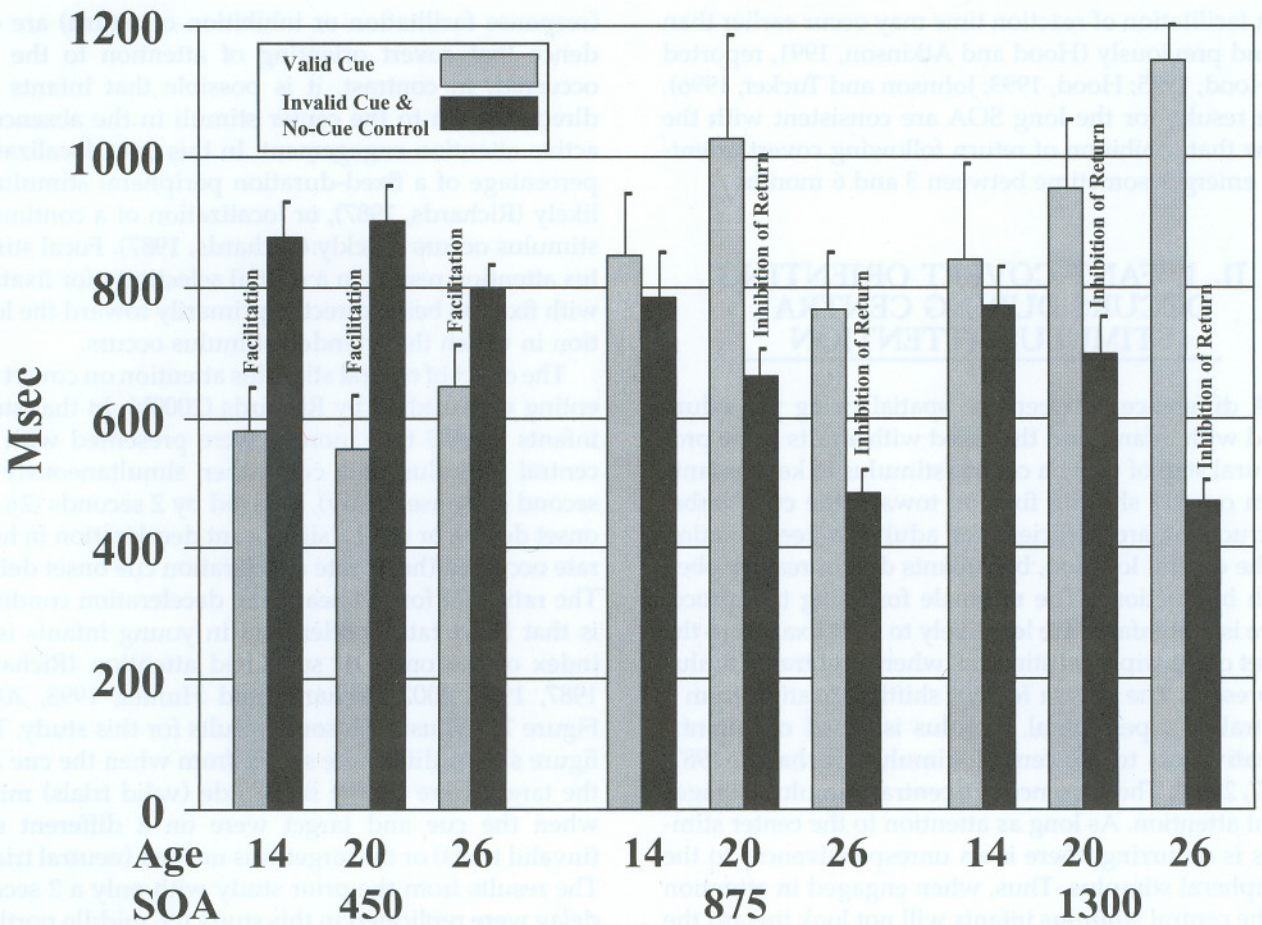

FIGURE 14.1 Latency to localize the peripheral stimulus when it was presented as a target. These figures are presented separately for the three testing ages, the three SOA conditions, and the valid cue and invalid cue/neutral trials. Response facilitation is shown by a faster response time for the valid targets than the invalid/neutral targets, and inhibition of return is shown by a slower response on the validly cued trials. Reprinted, with permission, from Richards (2000a).

2-month-old infants. Newborn infants appear to show inhibition of return following overt shifts of fixation (Valenza et al., 1994). In this procedure infants were given a peripheral cue to which they shifted fixation, and then fixation was returned to the center. A bilateral target was then presented. Newborns were more likely to look toward the target contralateral to the side to which fixation had earlier been directed. This suggests that the mechanism for inhibition of return exists at birth, but that "covert" orienting of attention does not occur at 2 to 3 months of age but does occur by 4 or 6 months.

A recent study examined covert orienting in infants aged 3, 4.5, and 6 months (14, 20, and 26 weeks) (Richards, 2000a). The spatial cuing procedure adapted for infant participants (Hood, 1995) was used. This study presented the central stimulus for 2 seconds, then the central and peripheral stimulus ("cue") together for 300 ms, turned both stimuli off, and then presented the target stimulus at SOAs of 300, 875, or 1300 ms. Figure 14.1 shows the latency to localize the target as a function of testing age and the spatial cuing conditions. No difference was found in the responses in the invalid and neutral conditions, so these are averaged together. The latency to localize the peripheral stimulus when it was presented as a valid target was faster than when it was presented as an invalid/neutral target at a SOA of 450 ms, for all three testing ages. This indicates that response facilitation occurred at all three ages. The reaction times at the two longer SOAs (875 and 1300 ms) were longer on the valid trials for the two older ages, there was no difference for the 3-month-old infants between valid and invalid/neutral trials. There was a regularly increasing amount of inhibition of return. The differences between the reaction time on the valid and invalid/neutral trials at the 1300-ms SOA were 70, 260, and 670 ms for the 14-, 20-, and 26-week-olds, respectively. This study shows

that facilitation of reaction time may occur earlier than found previously (Hood and Atkinson, 1991, reported in Hood, 1995; Hood, 1993; Johnson and Tucker, 1996). The results for the long SOA are consistent with the view that inhibition of return following covert orienting emerges sometime between 3 and 6 months.

II. INFANT COVERT ORIENTING OCCURS DURING CENTRAL STIMULUS ATTENTION

A difference between the spatial cuing procedure used with infants and that used with adults is the procedural step of using a central stimulus to keep infants from overtly shifting fixation toward the cue. Verbal instructions are sufficient for adults to keep fixation at the central location, but infants do not readily obey such instructions! The rationale for using this procedure is that infants are less likely to shift fixation to the onset of a peripheral stimulus when a central stimulus is present. The reason for not shifting fixation from a central to a peripheral stimulus is based on infants' attentiveness to the central stimulus (Richards, 1987, 1997, 2002). The presence of a central stimulus engages focal attention. As long as attention to the center stimulus is occurring, there is an unresponsiveness to the peripheral stimulus. Thus, when engaged in attention to the central stimulus infants will not look toward the "cue," and thus subsequent reaction time effects (response facilitation or inhibition of return) are evidence that covert orienting of attention to the cue occurred. In contrast, it is possible that infants can direct fixation to the center stimuli in the absence of active attention engagement. In this case, localization percentage of a fixed-duration peripheral stimulus is likely (Richards, 1987), or localization of a continuing stimulus occurs quickly (Richards, 1987). Focal stimulus attention results in a spatial selectivity for fixation, with fixation being directed primarily toward the location in which the attended stimulus occurs.

The effect of central stimulus attention on covert orienting was studied by Richards (2000b). In that study infants from 3 to 6 months were presented with the central stimulus and cue either simultaneously (0-second cue onset delay), delayed by 2 seconds (2 s cue onset delay), or until a significant deceleration in heart rate occurred (heart rate deceleration cue onset delay). The rationale for the heart rate deceleration condition is that heart rate deceleration in young infants is an index of the onset of sustained attention (Richards, 1987, 1997, 2002; Richards and Hunter, 1998, 2002). Figure 14.2 illustrates some results for this study. This figure shows difference scores from when the cue and the target were on the same side (valid trials) minus when the cue and target were on a different side (invalid trials) or the target was uncued (neutral trials). The results from the prior study with only a 2 second delay were replicated in this study (cf. middle portions of panels for Fig. 14.2 with Fig. 14.1). The immediate

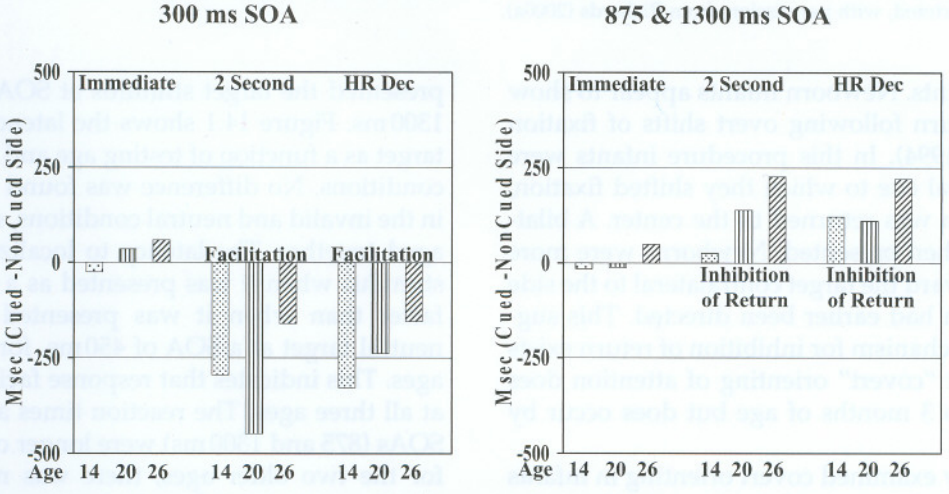

FIGURE 14.2 Difference scores for latency to localize the peripheral stimulus when it was presented as a target. The onset between the central stimulus and cue, i.e., cue onset delay, was either immediate (simultaneous), 2 seconds, or after a significant heart rate deceleration. For the 300-ms SOA, a faster reaction occurred to the validly cued target than to the invalid/neutral targets (top figures), i.e., response facilitation. For the 875 and 1300 ms SOAs, a slower reaction time occurred for the validly cued targets, i.e., inhibition of return. Reprinted, with permission, from Richards (2000b).

condition, when the central stimulus and cue were presented simultaneously, resulted in no difference between the valid and invalid/neutral trials for either SOA at any age (left-hand side of panels for Fig. 14.2). When the cue was presented contingent on the occurrence of a heart rate deceleration, indicating sustained attention was engaged, facilitation of response time and inhibition of return occurred at all three testing ages (right-hand side of panels for Fig. 14.2).

The finding that facilitation and inhibition of return occur for the heart rate deceleration condition (Fig. 14.2) indicates that attention engagement must be well underway for infants to show covert orienting of attention. This implies that this is a different task for infants than adults in that it demands focal stimulus attention be engaged in parallel with the covert orienting of attention to the cued location. The older infants apparently were able to keep processing resources on the central stimulus and 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 leading to inhibition of return and facilitation. The youngest infants, however, also may shift attention to the peripheral cue but not shift attention back to the center stimulus. Thus, there is a relative automatic processing of the peripheral stimulus information, a reflexive saccadic planning toward the peripheral stimulus that was inhibited, and response facilitation. However, apparently the young infants cannot shift attention from the peripheral location back to the center location and, therefore, do not show inhibition of return. These studies imply that one developmental change over this age range is an increasing flexibility in the spatial attention system resulting in the ability to orient to multiple locations in space under a wide variety of stimulus situations.

III. CORTICAL BASES OF SPATIAL ATTENTION DEVELOPMENT?

The inhibition of return effect is thought to be mediated by the superior colliculus (Rafal, 1998). It is thought that the activation of pathways in the superior colliculus responsible for fixation shifts and the inhibition of those pathways during the spatial cuing procedure result in inhibition of return (also see Chapters 16 and 64). The response facilitation that occurs at short SOAs is hypothesized to be due to the enhancement of sensory processing of information in the attended portion of visual space (Hillyard et al., 1995). This is shown elegantly in studies of selective spatial attention and the enhancements of the early components of the event-related potentials (ERP) in scalp-recorded electrical potentials (Hillyard et al., 1995) (see Chapters 84 and 85). Researchers studying infants in the spatial cuing procedure have adopted this neurophysiological perspective (Hood, 1993, 1995; Johnson and Tucker, 1996; Richards, 2000a, 2001, 2003; Richards and Hunter, 2002). The general conclusion of the "neurodevelopmental" approach is that the superior colliculus, which is relatively mature at birth, supports inhibition of return in early infancy (Hood, 1993, 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 involving saccadic planning and attention shifting. This interpretation is consistent with the general view that that there is an increase in the first 6 months of life of cortical control over eye movements that occur during attention and increasing cortical control over general processes involved in attention shifting (e.g., Hood, 1995; Richards, 2003; Richards and Hunter, 1998, 2002).

Some studies have used scalp-recorded ERPs to study covert orienting in infants. The ERP represents specific events occurring in the cortex and may be useful in identifying the cortical areas involved in the development of covert orienting Richards (2000a, 2001) studied infants at 14, 20, and 26 weeks of age. The spatial cuing procedure adapted for infants was used and ERPs were measured at the beginning of the target onset, or immediately before saccade onset. Figure 14.3 (bottom) shows the ERPs changes occurring at the target onset for the occipital electrode contralateral to the target for the valid, invalid, and neutral trials. A large positive deflection occurring at about 135 ms following stimulus onset may be seen for all three testing ages (P1?). This positive potential was the same size for all three cuing conditions for the youngest ages, slightly larger for the valid condition than the other two conditions for the 20-week-old infants, and the largest for the valid condition for the 26-week-old infants. This enhanced first positive component has been found for valid trials in adults and is called the "P1 validity effect" (Hillyard et al., 1995) (see Chapters 16, 36, 84, and 85).

A second ERP component was found in these studies. Presaccadic ERP is calculated as EEG changes occurring backward in time from the onset of the saccade to the target. The presaccadic ERP reflects cortical areas involved in saccade planning (Richards, 2000a, 2001, 2003; Richards & Hunter, 2002). A presaccadic ERP component was found in frontal electrode sites about 50 ms before saccade onset, located in scalp regions contralateral to the saccade. Figure 14.3 (top

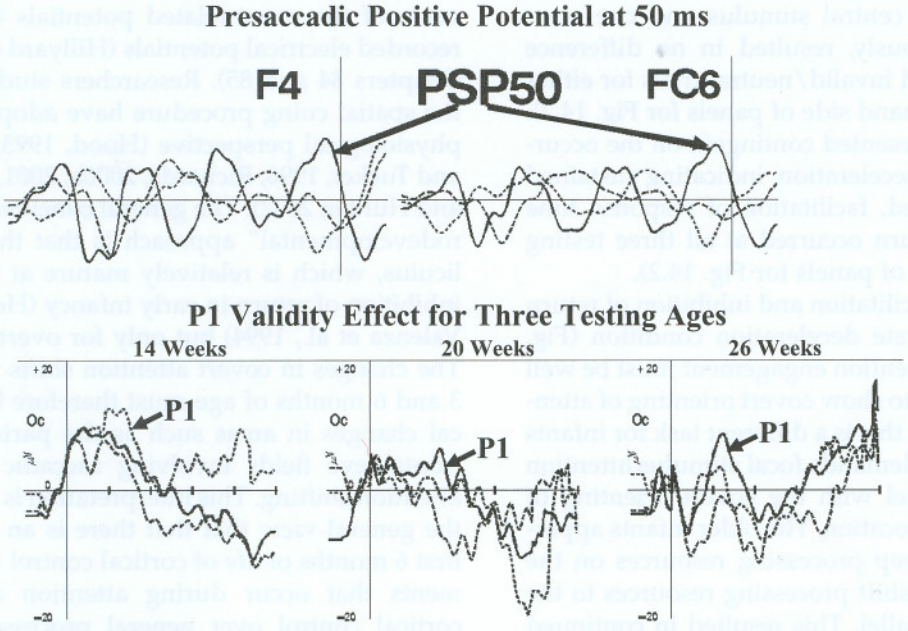

FIGURE 14.3 Top: ERP responses on the contralateral occipital electrode to the peripheral stimulus onset when it was presented as a target. The responses are presented separately for the three testing ages, and separately for the valid (solid line), invalid (small dashes), and no-cue control (long dashes) trials. The data are presented as the difference from the ERP on the no-stimulus control trial. The approximate locations of the P1 and N1 components are identified on each figure. Bottom: Presaccadic ERP component occurring about 50 ms before saccade onset. The presaccadic ERPs for F_4 and FC_6 show a large presaccadic positive ERP component that occurred about 50 ms before saccade onset for cued exogenous saccades.

figures) is an ERP recording from these electrodes (Richards, 2001). This positive component occurred primarily for saccades toward a target that occurred in the same location as the cue, that is, valid trials, and did not occur (or was smaller) for saccades toward a target that appeared in a different location than the cue (invalid trials), or for saccades toward a target that had not been preceded by a cue (neutral trials). Also, this presaccadic ERP component was absent in the youngest infants (3 months), and the amplitude of the ERP and the spread of the ERP across multiple electrode locations increased for infants at the older testing ages (4.5 and 6 months).

The cortical locations that generate these covert orienting and saccade planning effects have been examined with "equivalent current dipole analysis" ("brain electrical source analysis"; Richards, 2003; Richards and Hunter, 2002) (see Chapter 84). Figure 14.4 (top) shows a topographical scalp potential map for the presaccadic ERP component and an equivalent current dipole analysis of the presaccadic ERP. A hypothesized current dipole located in the area of the frontal eye fields generates a scalp potential map that closely corresponds to the recorded ERP. This analysis is consistent with the interpretation that the eye movements to the target in the planned location involve cortical areas that control planned eye movements (see Chapters 21 and 22). Figure 14.4 (bottom) shows some analyses of the P1 validity effect done with a 128-channel EEG system. Some of the dipoles were located in the primary visual area of the occipital cortex (Brodmann area 17; Fig. 14.4, Primary Visual Cortex). The activation of these dipoles did not show the validity effect (cf. Chapter 84). Some of the dipoles were located in the fusiform gyrus (Brodmann area 19; Fig. 14.4, Fusiform Gyrus). This area is one of the pathways from the primary visual area to the object identification areas in the temporal cortex ("ventral processing stream"). The dipoles located in the fusiform gyrus were those whose activation showed the P1 validity effect (cf. Chapters 16, 36, 84, and 85). The analysis of the cortical bases of the covert orienting effects suggests that specific brain areas may be identified that also show development and that form the basis for the changes in covert orienting seen in infants in this age range.

There are two implications of the work showing the ERP components accompanying covert orienting in young infants and their cortical bases. A first and very general implication is that brain areas involved in the control of sensory processing (e.g., P1 validity effect) and in the control of saccade planning (presaccadic

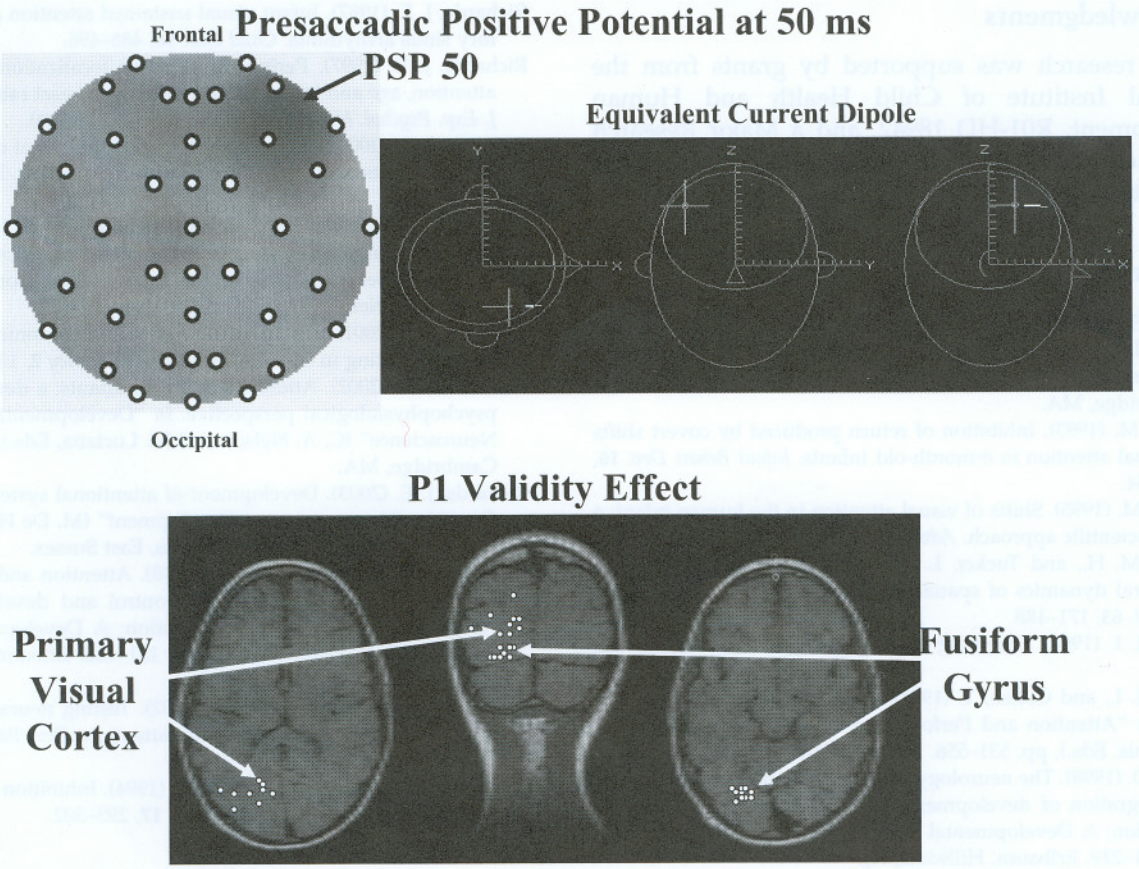

FIGURE 14.4 Top: The topographical scalp potential map is for the presaccadic ERP component (Fig. 14.3), plotted as the difference between the valid and the invalid/neutral trial saccades, plotted as if the infant were making a saccade toward the left side. The equivalent current dipole analysis resulted in a dipole located in the frontal eye fields. Bottom: Equivalent current dipole locations for individual infant participants for a component reflecting the "P1 validity effect" in a spatial cueing task. The dipole locations are plotted on a MRimage from a young child. Reprinted, with permission, from Richards (2003) and Richards and Hunter (2002). (See color plate)

ERP component) may form the basis for changes in attention to peripheral stimuli in young infants. These techniques may prove useful in identifying the cortical areas that show developmental changes that parallel the behavioral responses. Second, there are some specific implications for the development of covert orienting with these findings. Recall that infants at the earliest ages tested in these studies (14 weeks and 3 months) show response facilitation at levels comparable to those of the oldest infants. This implies that the age changes in the cortical areas involved with sensory processing (P1 validity effect, fusiform gyrus) or age changes in the cortical areas involved with saccade planning (presaccadic ERP, frontal eye fields) do not form the basis for the response facilitation. This suggests that a subcortical mechanism is responsible for this effect. A likely candidate is the activation of the pathways in the superior colliculus that are involved in peripheral saccadic eye movements (see Chapters 16 and 64). The covert orienting to the exogenous stimulus occurs without cortical involvement and is present at very early ages. Alternatively, other changes in spatial attention may be based on these cortical areas. The interpretations of the increase in inhibition of return (Figs. 14.1, 14.2) were that there was increased flexibility from 3 to 6 months in moving attention throughout space. This is accompanied in the brain by changes in the enhancement of cortical areas responsible for sensory processing (fusiform gyrus) (see Chapter 84) and the increasing sophistication of attention-based saccade planning (frontal eye fields) (see Chapters 21 and 22). 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; Richards, 2003; Richards and Hunter, 1998, 2002).

Acknowledgments

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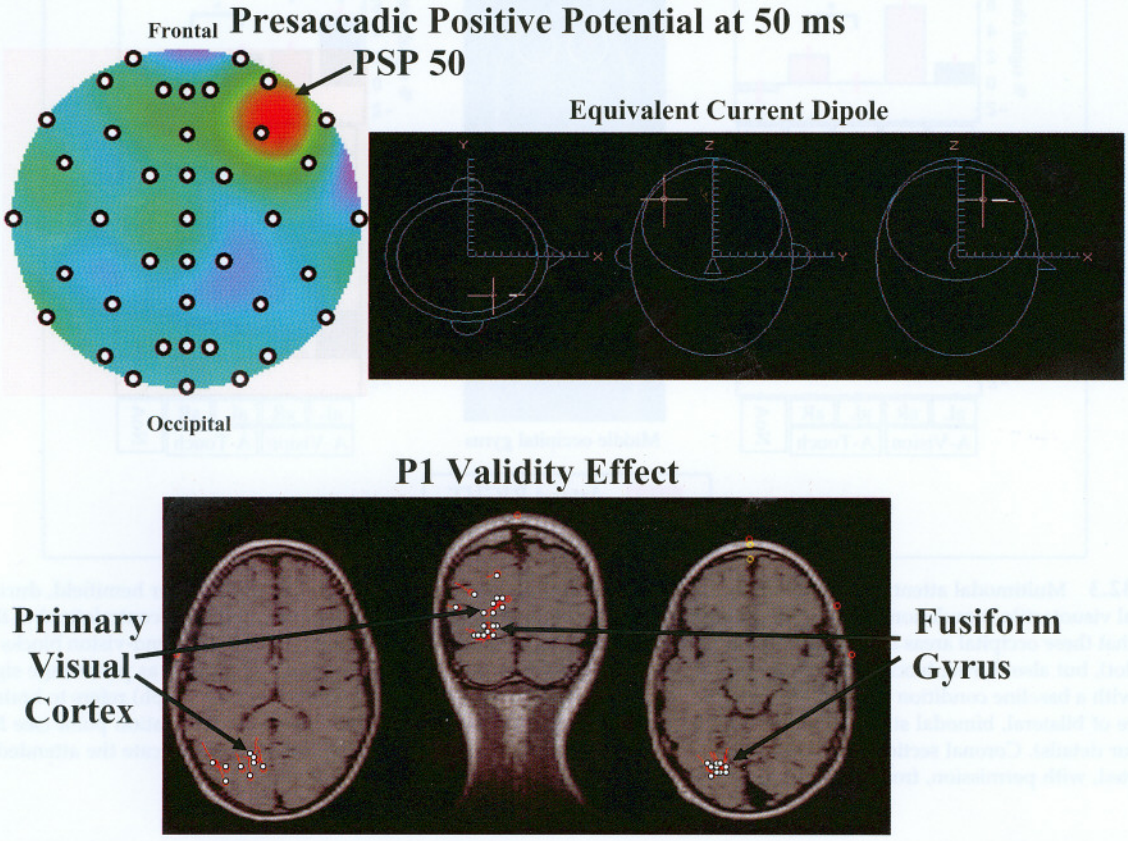

FIGURE 14.4 Top: The topographical scalp potential map is for the presaccadic ERP component (Fig. 14.3), plotted as the difference between the valid and the invalid/neutral trial saccades, plotted as if the infant were making a saccade toward the left side. The equivalent current dipole analysis resulted in a dipole located in the frontal eye fields. Bottom: Equivalent current dipole locations for individual infant participants for a component reflecting the "P1 validity effect" in a spatial cueing task. The dipole locations are plotted on a MRimage from a young child. Reprinted, with permission, from Richards (2003) and Richards and Hunter (2002).

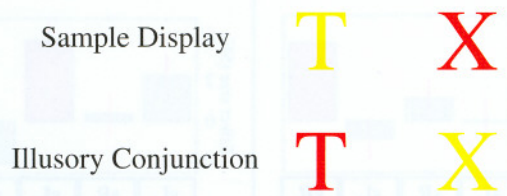

FIGURE 24.1 Example of illusory conjunctions. The sample display contains two shape features (T and X) and two color features (yellow and red). Illusory conjunctions occur when the colors and shapes are incorrectly bound in perception as represented in the lower part of the figure.

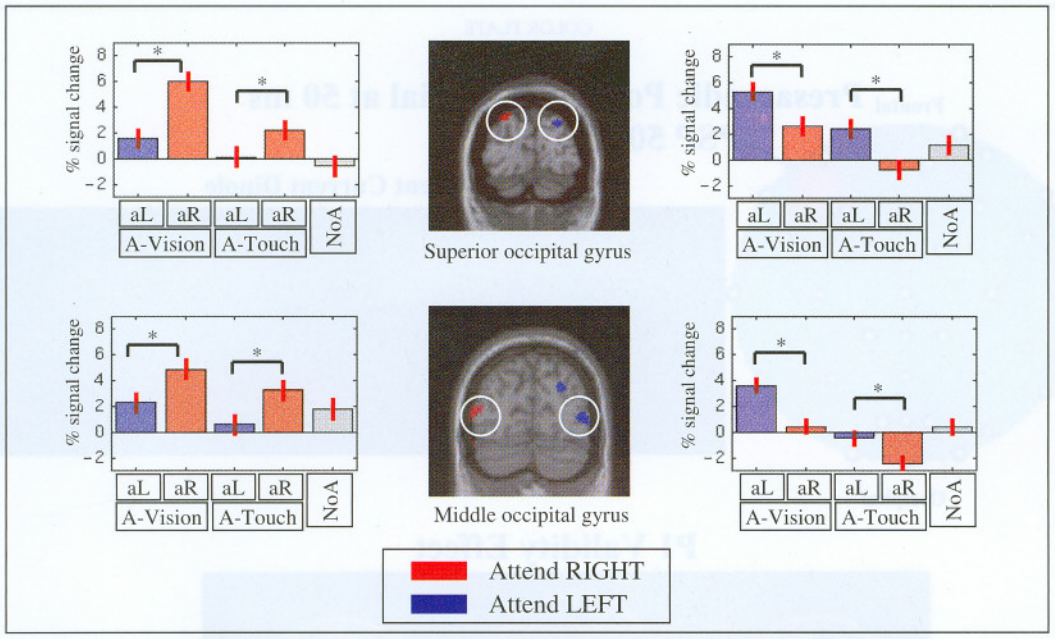

FIGURE 32.3 Multimodal attentional modulation in visual cortex when attending covertly to one or the other hemifield, during bimodal and bilateral visuotactile stimulation. Plots refer to the voxel at the maxima of the activated cluster, always found contralateral to the attended side. Note that these occipital areas displayed differential activity depending on the attended side not only for attend-vision blocks (bars 1 and 2 in each plot), but also during blocks of tactile attention (see bars 3 and 4 in each plot). Activities are expressed as percentage signal change compared with a baseline condition without any peripheral stimulation. The NoA condition (fifth bar in each graph) refers to brain activity in the presence of bilateral, bimodal stimulation, but with attention directed to a central task performed on the fixation point (see Macaluso et al., 2002a, for details). Coronal sections are taken at $y = -90$ (top slice) and $y = -80$ (bottom slice). aL and aR indicate the attended side. (*$P <$ 0.05). Adapted, with permission, from Macaluso et al. (2002a).

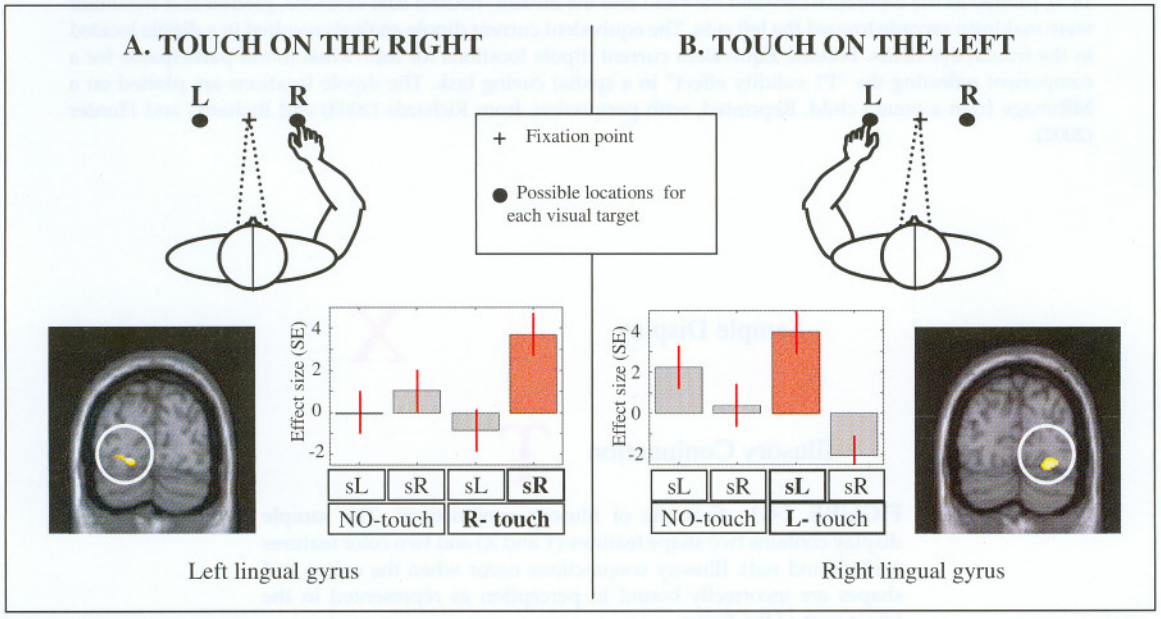

FIGURE 32.4 Effect of multisensory spatial correspondence on visual responses in occipital cortex (see Macaluso et al., 2000b). For each group (A: group receiving tactile stimulations to the right hand, B: group with touch to the left hand), we cartoon the spatial relation between possible visual and tactile input (top), and plot brain activity from the critical maxima for four experimental conditions: sl, stimulate left visual field; sR, stimulate right visual field; in the presence of touch (on left or right hand) or the absence of touch. The sections ($y = -82$ and $y = -80$, for left and right hemisphere activation, respectively) show the anatomical location of the cross-modal interaction (highest activity for contralateral visual stimulation with concurrent touch at same location) observed in the hemisphere contralateral to the location where spatially congruent multimodal stimulation could be presented. The signal plots show the amplification of visual responses when vision and touch were stimulated at the same contralateral location (see red bars in both graphs). Effect sizes are expressed in standard error (SE) units. For display purposes, the anatomical sections shown were thresholded at P-uncorrected = 0.05.