Differential Frontal Cortex Activation
Before Anticipatory and Reactive Saccades in Infants

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Neural correlates of anticipatory and reactive saccades were studied in 4-month-old infants by recording high-density event-related potentials. Infants were presented with a fixed sequence of stimulus presentation to which they rapidly showed anticipatory saccades, as well as continuing with some reactive (stimulus-driven) saccades. As in a previous study, no clear evidence was found for adultlike, saccade-related potentials, although some presaccadic differences between reactive and anticipatory saccades were observed. Infants also showed different stimulus offset-related effects preceding the 2 types of trials with a right-frontal positivity when an anticipatory look follows, but only left-frontal positivity when a reactive saccade follows.

Although even newborns are capable of making reactive saccades to conspicuous visual targets, there is evidence for substantial developments in the ability to plan and execute saccades over the first year of life (for reviews, see Johnson 1990, 1995; Richards, this issue). Although several theoretical accounts have attempted to relate these changes to underlying neural growth, until recently it has been difficult to directly test these accounts. In a previous study (Csibra, Tucker, & Johnson, 1998), we examined saccade-related potentials in 6-month-old infants in a gap paradigm. Infants viewed trials in which a central fixation stimulus disappeared some time (200 msec) prior to the presentation of a peripheral target in an unpredictable location (gap trials), and trials in which the peripheral target appeared before the offset of the central stimulus (overlap trials). Like adults, infants were slower to
make a saccade in the overlap trials than in the gap trials. Strikingly, infants did not show clear evidence of the presaccadic electrical brain activity consistently observed in adults and associated with cortical saccade planning processes. They did, however, show a left-frontal positive event-related potential (ERP) component that we suggested reflected cortical disinhibition of the colliculus initiated by fixation stimulus offset.

In the gap paradigm we employed, both trial types involved reactive saccades, because the participant could not predict the location where the visual target would be presented. Even though presaccadic potentials are observed in most saccade situations in adults (Balaban & Weinstein, 1985; Csibra, Johnson, & Tucker, 1997; Kurtzberg & Vaughan, 1982), one possible reason we did not observe any presaccadic potentials in our group of 6-month-old infants is that no volitional control of eye movements was required, making it more likely that collicular circuits alone could initiate the saccades we observed. To address this question, in this study we adapted the anticipatory looking paradigm developed by Haith and colleagues (Haith, Hazan, & Goodman, 1988; Haith & McCarthy, 1990) while recording high-density ERPs from 4-month-old infants.

In one model of the neurodevelopment of saccadic control (Johnson, 1990), anticipatory and volitional saccades were claimed to be supported by a pathway extending to frontal structures, such as the frontal eye fields, that matured at approximately 3 months of age. In the same model, reactive saccades were attributed to other pathways involving the superior colliculus and parietal cortex. The expectation from this model is that frontal areas should be activated differentially preceding anticipatory saccades in infants of 6 months. A different possibility is that in the adult paradigms in which presaccadic potentials have been recorded over parietal sites, adults were endogenously generating their saccades. If this is the case, then we expect these parietal components to be present in anticipatory, but not in reactive, saccades in infants. On the other hand, if infants of 4 months are simply not capable of activating the same cortical eye movement planning pathways as adults, we would expect not to observe these components in either type of saccade.

METHOD

Participants

The participants were 10 infants (5 boys and 5 girls) aged 17.4 weeks to 19.3 weeks ($M = 18.3$ weeks, $SD = .67$ weeks) at the time of the test. All the participants were full-term infants with no known birth or other complications. The data from an additional 20 infants were excluded from the analysis because of fussiness or not paying attention to the stimuli ($n = 8$), failing to make anticipatory eye movements in one or the other direction ($n = 6$), or excessive electroencephalogram (EEG) arti-
facts \((n = 6)\). The latter 6 participants’ data were included in the behavioral analyses but were excluded from the electrophysiological analyses. The attrition rate \((2/3)\) in this study was high but not unprecedented in infant ERP research (see, e.g., Csibra et al., 1998; de Haan & Nelson, 1997), and it was due to the fact that we applied rather stringent criteria for participant exclusion (discussed later). Because these criteria included behavioral performance requirements (certain number of anticipations), our sample represents only those 4-month-olds who made frequent anticipatory eye movements. The selection of this sample, however, was necessary for the main question of this study (i.e., the comparison of the neural substrates underlying anticipatory vs. reactive saccades).

**Apparatus**

The infants sat on their parent’s lap at 60 cm distance from a 40 cm \(\times\) 29 cm \((36.9° \times 27.2°)\) computer monitor within an acoustically and electrically shielded and dimly lit sound booth. A video camera was mounted on the wall behind the monitor centered on the infant’s face that allowed the experimenter to monitor the participant’s gaze and to record his or her behavior. A loudspeaker was placed below the monitor for presentation of the acoustic stimuli. The brain electric activity of the participant was measured by a Geodesic Sensor Net (Tucker, 1993) comprising 62 electrodes evenly distributed across the scalp. The electrodes were connected to an EGI NetAmps amplifier through a head box located on an arm above the participant’s head.

**Stimuli**

The stimulus presentation monitor had a dark gray background on which the four stimulus locations were marked as light gray squares (see Figure 1). The size of the squares was 10 cm \(\times\) 10 cm, and their center was 10 cm from the monitor center, leaving a 4.1-cm gap between them. The stimuli were color cartoon figures randomly selected from a set of 282 pictures in each trial.

The participant’s attention was drawn to the monitor by flashing the first stimulus at the first stimulus location (upper left square) in 200 msec on–200 msec off cycles. If necessary, the experimenter could also trigger various computer-generated sounds (e.g., an animal voice, a bell, etc.) to get the infant’s attention. When the infant looked at the flashing stimulus, the experimenter initiated the experimental stimulus sequence. The picture stopped flashing and remained on screen for 800 msec, which was followed by an 800-msec interstimulus interval (ISI). After this, as long as the infant was watching the stimulus monitor, the visual stimuli appeared at the next location in the prespecified sequence: upper left, upper right, lower left, and lower right (see Figure 1). Each visual stimulus in the sequence started as a smaller,
6.25 cm × 6.25 cm image in the center of the square and expanded into the full size in 120 msec. This “looming” appearance was accompanied by a short beep. The image then stayed on for a further 680 msec, resulting in 800 msec total stimulus duration. The ISI was always 800 msec. These display parameters were selected because they yielded the highest rate of anticipations in our pilot studies.

The visual angle of the full-size stimulus was 9.5° on the fovea and 9.2° viewed at the periphery from the previous stimulus location. A saccade from the center of the previous stimulus location to the center of the next stimulus location required a magnitude of 13.4° (left to right) or 18.9° (right to left).

Procedure

After the infant became familiar with the laboratory environment, the electrode net was mounted on the participant’s head and fixed by elastic chinstraps. Then, the infant was seated in front of the stimulus monitor and his or her attention was drawn to the screen and the stimulus sequence started. If the infant started to cry, became inattentive, or touched the electrode net, the experiment was interrupted and the child was soothed. The experiment was terminated when it had to be interrupted a second time for any of the aforementioned reasons. Participants typically completed between 70 and 150 trials before the session was terminated.

EEG Recording

The brain electric activity was measured simultaneously at 62 scalp locations that included electrodes at the outer canthi of the two eyes (see Figure 2). The reference
electrode was at the vertex (Cz in the conventional 10–20 system). The electrical potential was amplified with 0.1–100 Hz bandpass, digitized at 500 Hz sampling rate, and the continuous EEG was stored on computer disk for offline analysis.

Behavioral Analysis

The participant’s looking behavior was coded on the basis of the video recordings before the ERP analysis. A trial was considered valid if (a) the infant was looking at the previous stimulus when it went off and (b) made his or her first eye movement toward the next stimulus location either during the ISI or during the first 600 msec of the next stimulus duration without blinking. Our criteria for including saccades

![Figure 2](image-url) FIGURE 2 Scalp locations of the 62 electrodes of the infant version of the Geodesic Sensor Net as compared to electrode locations of the standard international 10–20 system. The reference point is at the vertex. Dashed lines mark the frontal and occipital electrode groups used in the statistical analyses.
was inclusive, with any saccade initiated in the appropriate horizontal direction for the next location being used. Saccades for which the electrooculogram (EOG) marker could not be found (discussed later) were excluded from further analyses.

Thus, the EOG analysis served also to ensure reliability of the behavioral analysis.

**EOG Analysis**

The horizontal EOG was reconstructed from the EEG signal by subtracting the electrical signal on the electrode at the outer canthus of the right eye from the corresponding signal of the left side. Saccades were identified manually as a monotonic slope in either direction lasting at least 20 msec and with a slope of more that 1 μV/msec. The first sampling points of these slopes were taken as the onset of the saccade. We adopted a conservative definition of anticipatory saccades, with an eye movement being classified as anticipatory if its onset occurred either before the presentation of the next stimulus or during its first 120 msec (while it was expanding). The saccadic reaction time (SRT) is expressed relative to the target onset; negative SRT (and positive SRTs below 120 msec) indicates anticipatory onset.

**ERP Analysis**

The continuous EEG was segmented into trials that consisted of 1,600 msec EEG starting 100 msec before a stimulus offset, and the trials that were coded invalid on the basis of the behavioral analysis were discarded. The valid trials were segmented further into offset-locked and saccade-locked segments. The offset-locked segments started 100 msec before stimulus offsets and ended 600 msec after them; the saccade-locked segments started 550 msec before saccade onsets and ended 150 msec after them. Both kinds of segments were baseline corrected by subtracting the average voltage amplitude in the pre-offset 100 msec from the whole segment. Any segment in which the EEG exceeded the 200 μV range was rejected as artifact. Further artifacts and recording failures were identified by trial-by-trial inspection of the EEG. Participants were required to contribute at least 10 artifact-free trials with anticipatory and 10 with reactive eye movements and at least 2 artifact-free trials in both directions (right and left) within both trial types (anticipatory and reactive). The 6 infants who did not meet these criteria were excluded from further analyses.

The raw EEG trials were averaged separately for the offset-related and saccade-related ERPs according to saccade type (anticipatory vs. reactive) and saccade direction (left vs. right). The averaged ERPs were rereferenced to the average voltage (Lehmann, 1987) and the scalp surface maps were created by spherical scalp interpolation (Perrin, Pernier, Bertrand, & Echallier, 1989).
RESULTS

Behavioral Measures

Sixteen infants met the behavioral criteria of completing at least 40 valid trials including at least four anticipatory responses. The mean proportion of anticipatory saccades was 30.1%. This relatively high anticipation rate was partly due to the fact that infants with no or very few anticipatory responses were excluded from the sample (but, see Wentworth, Haith, & Karrer, this issue, for an even higher rate of anticipation). Although the average number of valid trials did not differ between the two sides (32.6 and 30.7 toward the right and left side, respectively), the proportion of anticipatory saccades was different: 41.2% and 18.9% to the right and left, respectively, $t(15) = 3.374, p < .005$. An analysis of the average SRTs of the anticipatory responses revealed that these occurred earlier toward the right ($-100.4$ msec) than toward the left ($-23.6$ msec) side, $t(15) = 2.261, p < .05$. There was no side difference between the latencies of the nonanticipatory responses: $335.1$ msec to the right and $347.6$ msec to the left, $t(15) = 1.098$.

The same pattern of results remained when we removed the data from the 6 participants who had too few trials without artifact-contaminated EEG and were therefore excluded from the ERP analysis (see Figure 3). For the 10 participants

FIGURE 3  Distribution of saccadic reaction times pooled across the participants whose data were included in the ERP analyses ($n = 10$).
who were included in the subsequent ERP analyses, the proportion of the anticipatory saccades was 51.4% to the right and 22.7% to the left, \( t(9) = 4.426, p < .002 \). The average latency of anticipatory saccades was \(-128.6\) msec and \(-32.2\) msec to the right and to the left, respectively, \( t(9) = 2.624, p < .05 \). The latency of the nonanticipatory saccades was not significantly different to the right (323.9 msec) and left (336.6 msec).

**Offset-Related ERPs**

Figure 4 shows the ERPs time locked to the offset of the visual stimuli on either side of the screen. (Note that although this figure shows the ERPs generated by stimuli from both sides, we treated the ERPs from the two sides separately in the statistical analyses and contrasted them in the side factor.) The offset of the foveal visual stimuli evoked a characteristic triphasic wave over the occipital scalp: a negative peak around 80 msec latency, a positive peak around 120 msec latency, and a greater negative deflection with 170 msec peak latency. These waves appear to be not dependent on the type of the subsequent saccade (anticipatory vs. reactive). We performed three-way (2 Sides [right, left] \( \times \) 2 Saccade Types \( \times \) 6 Electrode Sites) analyses of variance (ANOVAs) on the mean amplitude of the 60- to 100-msec, 100- to 140-msec, and 140- to 200-msec postoffset intervals at the six occipital electrode sites (35, 36, 37, 38, 39, 43; see Figure 2). None of these analyses yielded a significant effect or interaction of these factors.\(^1\) With regard to the later parts of the ERPs, the offset-related occipital responses before anticipatory and reactive saccades appear to deviate from each other (see Figure 4). This difference is probably due to some early anticipatory eye movements (a small proportion of them occurred as early as 600 msec before the next stimulus onset, i.e., 200 msec after the previous stimulus). However, ANOVAs on the mean amplitudes of the 200- to 500-msec postoffset interval at occipital sites did not reveal any significant effects.

Further brain responses to the stimulation offset occurred at frontal locations as an elongated positive wave starting at about 100 msec after stimulus offset and reaching its maximum between the 200- to 300-msec postoffset interval. We performed a four-way ANOVA on the average amplitude in this interval with the factors of saccade type (2: anticipatory, reactive), side (2: right, left), hemisphere (2: right, left), and electrode location within hemisphere (7: see Figure 2). This ANOVA yielded a significant side effect, \( F(1, 9) = 6.73, p < .05 \), showing that the ERPs were more positive after the offset of the stimulus on the right side of the screen, and a highly significant Saccade Type \( \times \) Hemisphere interaction, \( F(1, 9) = 16.01, p < .005 \), showing greater difference before the two types of saccade over

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\(^1\)Here and in the subsequent analyses, where more than two electrode sites were included in the ANOVAs, the levels of significance were adjusted according to the Greenhouse–Geisser procedure.
the right-frontal cortex than over the left-frontal areas. No other main effect or interaction was significant. To explain the interaction, we performed Saccade Type × Side × Electrode Site ANOVAs on the amplitudes measured over the two hemispheres. There was no significant effect in the data measured over the left-frontal areas, and the ANOVA on the amplitudes of the right-frontal leads revealed only a significant effect of saccade type, $F(1, 9) = 5.21, p < .05$. This pattern of results in-

**FIGURE 4** Stimulus offset-related, grand-average, ERPs (collapsed across left and right directions). See Figure 2 for the scalp location of electrodes. The vertical bar marks the stimulus offset. Positive is plotted upward.
icates that the frontal positivity was greater before anticipatory saccades than before reactive saccades over the right-frontal areas only (Figure 5). We tested this result in two further analyses. A three-way (Side $\times$ Hemisphere $\times$ Electrode Location) ANOVA of the ERPs before anticipatory trials revealed two main effects: side, $F(1, 9) = 5.81, p < .05$; and hemisphere, $F(1, 9) = 7.39, p < .05$. A similar ANOVA on the frontal postivities before reactive saccades did not reveal any significant effect. One possibility is that this hemispheric difference is due to the fact that more anticipatory eye movements were performed toward the right side, and these might have contaminated the ERPs over the right-frontal cortex. However, only a minute fraction of saccades (three saccades to the left and two saccades to the right in 10 participants) fell into the interval analyzed earlier (see Figure 3), which could not have been sufficient to result in this significant difference. Furthermore, the absence of interaction between the factors side and saccade type suggests that the differential amount of the frontal activity before anticipatory and reactive saccades was not due to the differential proportion of right versus left eye movements in the two trial types.

The earlier results show hemispheric asymmetries in frontal activity before anticipatory versus reactive saccades but do not tell us which saccade types were preceded by higher cortical activity. We tested this question by comparing the average

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**FIGURE 5** Scalp surface maps of the average voltage amplitudes in the 200- to 300-msec postoffset interval before anticipatory (left) and reactive (right) saccades.
voltage amplitudes in the earlier interval to the zero (i.e., baseline) level. For this comparison we chose the electrode site (15) that displayed the strongest offset-related, left-frontal effect in our previous study (Csibra et al., 1998). Before anticipatory saccades, the average positivity was 6.61 μV, t(9) = 1.43, ns; before reactive saccades, this measure was 12.49 μV, t(9) = 3.43, p < .005. In contrast, the symmetrical counterpart of the aforementioned electrode site (57) produced an average of 17.85 μV amplitude in this interval before anticipatory eye movements, t(9) = 4.64, p < .001, and 11.18 μV before reactive saccades, t(9) = 1.95, ns.

Saccade-Related ERPs

Figure 6 shows the saccade-related potentials for the anticipatory and reactive saccades. Similar to our previous results (Csibra et al., 1998) and to other researchers’ findings (Richards, 2000; Vaughan & Kurtzberg, 1989), there is no evidence of the characteristic spike potentials that precede the saccades performed by adult participants (see, e.g., Csibra et al., 1997). In fact, the presaccadic activity in this study is better characterized as slow-wave deflections than phasic, time-locked potential changes. The only exception from this general picture is the parietal (Pz, electrode 34) positivity prior to anticipatory trials peaking about 110 msec before saccade onset. As this peak was not identifiable in all the individual saccade-related ERPs, we analyzed this potential on the basis of the mean amplitude of the 80- to 140-msec presaccadic interval. A two-way (Side × Saccade Type) ANOVA revealed a significant saccade type main effect, F(1, 9) = 8.36, p < .02, demonstrating a higher positivity before the anticipatory saccades than before the reactive saccades. Note, however, that none of the neighboring electrode sites showed the same effect, which suggests either very localized neural activity or a spurious statistical effect. Note, also, that the visual inspection of the individual averages of the 10 participants revealed that only 5 of them showed a clearly more positive potential before anticipatory saccades in the aforementioned interval, and 2 of them showed the reverse pattern.

The brain activities prior to anticipatory and reactive saccades also differ at the occipito-temporal and frontal sites. These long-lasting differences were statistically tested by entering the average voltage amplitude of the presaccadic 500 msec into ANOVAs. At the occipital sites a three-way ANOVA (Saccade Type × Side × Electrode Location [35, 36, 37, 38, 39, 43]) revealed only a strong main effect of saccade type, F(1, 9) = 30.7, p < .001. We found a similar effect when comparing the amplitudes at the two occipito-temporal sites (31, 47), F(1, 9) = 13.48, p < .01; no interaction with either the side or hemisphere factor was found. To analyze the frontal slow-presaccadic deflections, we performed a similar four-way ANOVA as we did with the offset-related frontal effect (see earlier). This analysis yielded a main effect of saccade type, F(1, 9) = 5.74, p < .05, and a Saccade Type × Hemi-
sphere interaction, $F(1, 9) = 7.91, p < .05$. These effects indicate that the frontal potentials prior to reactive saccades were more positive than the potentials prior to anticipatory saccades, and that this effect was greater over the left-frontal cortex. There was also a main effect of electrode location, $F(6, 54) = 3.68, p < .05$, and an Electrode Location × Hemisphere interaction, $F(6, 54) = 3.21, p < .05$, indicating that this effect was not equally present at all electrode sites (see Figure 6).

**FIGURE 6**  Saccade-related, grand-average, ERPs (collapsed across left and right saccades). See Figure 2 for the scalp location of electrodes. The vertical bar marks the onset of the saccade identified by the electrooculogram. Positive is plotted upward.
DISCUSSION

The main aim of this experiment was to study the difference between endogenously and exogenously generated saccades to investigate further the neural mechanisms of eye movement planning processes in infants. Beginning with our behavioral observations, we successfully replicated the earlier results (Haith et al., 1988; Haith & McCarthy, 1990) that 4-month-old infants readily make anticipatory saccades toward the next location in a predictable stimulus sequence. Our stimulus sequence was more complex than that used in many anticipatory looking paradigms, and this might have contributed to our participants making more anticipatory saccades to the right than to the left. However, others have reported a similar asymmetry in the proportion of anticipatory saccades even with perfectly symmetrical stimuli (e.g., Chawarska, 1999).

The saccade-related potentials and ERPs recorded from the reactive saccades in this experiment replicate our findings in a previous study (Csibra et al., 1998) in several respects, despite being conducted with different stimuli and experimental conditions. First, as in our earlier study (and in other researchers’ findings; see Vaughan & Kurtzberg, 1989), there was no clear evidence of adultlike presaccadic potentials recorded over parietal sites. Although it remains possible that the spike potential is present but very poorly time locked to the eye movement onset in infants, this observation is consistent with a surprising lack of functional development in parietal eye movement centers at 4 to 6 months of age. However, we do not wish to conclude that there is no cortical influence over reactive saccades at this age, because in this study we also replicated our previous finding of a left-frontal positivity locked to fixation offset before reactive saccades. This effect had a very similar scalp profile and time course to that observed previously (compare the right side of Figure 5 to Figure 3 in Csibra et al., 1998). We proposed that this fronto activity reflects frontal eye field (or perhaps other frontal regions) disinhibition of collicular circuits, resulting in more rapid saccades to peripheral targets than would otherwise be possible (Csibra et al., 1998).

Turning to the anticipatory saccades, although we still saw no convincing evidence of a spike potential preceding saccade onset, at one parietal lead (No. 34, Pz) a significant positivity preceded the anticipatory saccades. Due to the very focal nature of this effect, we remain cautious about its significance and await the results of attempts to replicate it. The major contrast between anticipatory and reactive saccades we observed was a difference in the stimulus offset-related effects. Preceding reactive saccades, the disappearance of the foveal stimulus elicited a left-frontal activity, just like in our previous study. In contrast, when the subsequent eye movement was anticipatory, there was a prominent right-frontal activation following the fixation stimulus offset. Because the left-frontal positivity in these anticipatory trials failed to reach a level significantly different from the base-
line, we conclude that this right-frontal effect replaced the left-frontal one before anticipatory eye movements.

When looking at the saccade-locked effects (not time locked to stimulus offset), we note that the saccades in the reactive and anticipatory trials were performed in a different visual environment: There was no conspicuous stimulus in the visual field prior to the anticipatory eye movements, whereas the reactive saccades were preceded by the onset of a large, peripheral moving (looming) image. We hypothesize that the occipital differences we observed in the presaccadic potentials can be attributed to these visual factors. The concurrent left-frontal sustained positivity prior to reactive saccades could also be a consequence of the visual stimulation, though its lateralization contradicts this interpretation. This effect did not interact with the direction of the eye movement, and therefore we believe it reflects endogenous left-frontal activity. Because of the 800-msec long ISI, its time course rules out the possibility that this frontal effect was simply another manifestation of the offset-related left-frontal effect. Rather, it may reflect a reactivation of the same frontal eye movement centers after the new target appears.

These results, and those of Csibra et al. (1998), provide support for some aspects of Johnson’s (1990) model of the neurodevelopment of saccadic control, as well as clearly requiring modification to other aspects of the original model. The fact that we are unable to observe presaccadic potentials over parietal sites is not consistent with Johnson’s (1990) claim that these circuits could influence saccades by 3 or 4 months. It remains possible that there are saccade paradigms that will elicit such potentials in 4-month-olds. However, it would still need to be explained why 4-month-old infants only show parietal activation in a subset of the tasks in which it is seen in adults and 12-month-olds (Csibra et al., 1997; Csibra et al., 2000). An aspect of these findings that is broadly consistent with the Johnson (1990) model is the differential activation of frontal structures in anticipatory trials and their putative role in disinhibiting collicular activity. However, the fact that reactive saccades are preceded by left-frontal activity and anticipatory saccades by right-frontal activity on stimulus, offset was not anticipated.

One possible explanation of the right-frontal effect seen in anticipatory trials is that it involves endogenous disengagement from the foveated stimulus while it is still on. This would explain both the absence of the offset-evoked left-frontal activity and the early eye movements in anticipatory trials. In this case, the right-frontal effect would reflect spatiomotor anticipatory processes and is consistent with positron emission tomography results in adults, which demonstrated that the right-dorsolateral prefrontal cortex (DLPC) plays a major role in the generation of self-initiated movements (Jahanshahi et al., 1995). Another result obtained with adults that is consistent with the right-frontal effect we observed in infants comes from a recent transcranial magnetic stimulation study in which stimulation of the right DLPC 200 msec after a fixation stimulus offset (in a gap paradigm) resulted in more rapid saccades to a target (Müri et al., 1999). Müri et al. concluded
that stimulation of the right DLPC at this time point reduces its inhibition of the superior colliculus, resulting in the facilitation of target-directed saccades. The same effect was not found at other time points or with stimulation of the right-parietal cortex. These findings suggest that certain parts of the frontal cortex play an important role in the preparation of both endogenously and exogenously generated eye movements. Our results are consistent with the hypothesis that these areas have already begun to operate at 4 months of age, whereas the posterior eye movement regions lag behind this development.

Our findings to date correspond to some degree to those reported by Richards (this issue). He replicated our previous finding (Csibra et al., 1998) that reactive saccades to peripheral targets do not elicit any presaccadic potentials in 6-month-old infants, suggesting a collicular basis to these saccades. Our results also support the proposal that the frontal eye fields may be involved in eye-movement-related trials, although in our paradigms this activity appears to be aligned to both fixation stimulus offset and saccade onset. In many saccade paradigms, it can be difficult to dissociate cue–target-related effects from saccade-related effects, and thus, further research on this question is required. Our results suggest that much of the behavioral difference between anticipatory (endogenous) and reactive (exogenous) saccades is attributable to frontal cortical processes (disengagement) during the preceding stimulus.

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