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ORIGINAL PAPER

The Relation between Infant Covert Orienting, Sustained Attention and Brain Activity

Wanze Xie[1] · **John E. Richards**[1]

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Abstract This study used measures of event-related potentials (ERPs) and cortical source analysis to examine the effect of covert orienting and sustained attention on 3- and 4.5-month-old infants' brain activity in a spatial cueing paradigm. Cortical source analysis was conducted with current density reconstruction using realistic head models created from age-appropriate infant MRIs. The validity effect was found in the P1 ERP component that was greater for valid than neutral trials in the electrodes contralateral to the visual targets when the stimulus onset asynchrony (SOA) was short. Cortical source analysis revealed greater current density amplitude around the P1 peak latency in the contralateral inferior occipital and ventral temporal regions for valid than neutral and invalid trials. The processing cost effect was found in the N1 ERP component that was greater for neutral than invalid trials in the short SOA condition. This processing cost effect was also shown in the current density amplitude around the N1 peak latency in the contralateral inferior and middle occipital and middle and superior temporal regions. Infant sustained attention was found to modulate infants' brain responses in covert orienting by enhancing the P1 ERP responses and current density amplitude in their cortical sources during sustained attention. These findings suggest that the neural mechanisms that underpin covert orienting already exist in 3- to 4.5-month-old, and they could be facilitated by infant sustained attention.

Keywords Infant covert orienting · Sustained attention · ERPs · Cortical source analysis

Covert orienting refers to the shift of attention to a peripheral location without an overt eye movement. Prior studies with behavioral measures and event-related potentials (ERPs) have advanced our understanding of infant covert orienting and its effects on early visual processing (e.g., Hood 1993, 1995; Johnson et al. 1994; Richards 2000a, 2001, 2005, 2009). For example, infants were observed to show distinct ERPs (e.g., P1, N1) responses to spatially cued stimulus compared to non-cued (neutral) stimulus (Richards 2000a, 2005). These ERPs findings shed light on the neural correlates of infant covert orienting at the scalp level, but provide limited information about the underlying cortical sources. Infant sustained attention is a type of endogenous attention accompanied by a decrease in infants' heart rate (HR) that reflects voluntary attention allocation and enhanced brain alertness (Colombo 2001; Richards 2009). Infant sustained attention has been found to affect a wide variety of cognitive processes, such as information processing, stimulus orienting, and recognition memory (Courage et al. 2006; Mallin and Richards 2012; Reynolds et al. 2010). The facilitation effect of sustained attention on these cognitive processes raises the possibility that sustained attention would enhance infant brain activity involved in covert orienting. The current study aimed to examine the cortical activity related to infant covert orienting and determine the effect of sustained attention on infant brain activity involved in covert orienting.

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✉ Wanze Xie
xiew@mailbox.sc.edu

[1] Department of Psychology, and Institute for Mind and Brain, University of South Carolina, Columbia, SC 29208, USA

The development of infant covert orienting has been studied with the spatial cueing procedure developed by Posner (1980). In this procedure, the infant's fixation remains on a central stimulus while a peripheral cue and target are presented. The target may be presented at the same side of the cue ("valid trials"), at the opposite side of the cue ("invalid trials"), without a preceding cue ("neutral trials"), or may not be presented after a cue ("no-target trials"; e.g., Hood 1995; Richards 2000a, 2001, 2005). The stimulus onset asynchrony (SOA) refers to the time period between the cue and target onsets. The reaction time (RT) or saccade latency to the target is normally shorter for valid than neutral and invalid trials (validity effect) and shorter for neutral than invalid trials (processing cost effect) when the SOA is short (e.g., 450 ms). Infants showed different patterns of responses to the targets when a long SOA was used. Infants' RT to the target is typically longer for valid than neutral and invalid trials when the SOA is long (e.g., 1350 ms), which is termed as the inhibition of return (IOR; Richards2000a, 2005). The validity effect has been observed in 3- and 4-month-old, but the IOR has not been observed in infants until 4.5–6 months of age (Johnson et al. 1994; Johnson and Tucker 1996; Richards 2000a, 2001, 2005; Varga et al. 2010).

Infant sustained attention plays an important role in infant cognitive processes, such as stimulus orienting, information processing, and recognition memory (see Colombo 2001; Richards 2009, 2010 for reviews). Infant sustained attention has been well defined by infant HR changes derived from electrocardiogram (ECG) recordings (Reynolds and Richards 2007). Infant HR decreases and remains at a lower level compared to the pre-stimulus baseline during sustained attention. The decrease of infant HR is accompanied by an increase of brain arousal and alertness (Richards and Casey 1991; Richards 2008; Xie and Richards 2016), which facilitates information processing and allocation of attention resources and improves task-related performance (Reynolds and Richards 2007; Reynolds et al. 2010). For example, infant sustained attention was found to benefit saccadic localization of peripheral stimuli, which is a process involving attention orienting and saccade planning (Hunter and Richards 2003; Mallin and Richards 2012). Infant sustained attention was also found to improve infant behavioral performances in a spatial cueing paradigm (Richards 2000b, reported in Richards 2004). The authors of these studies argued that there should be improvement in early visual processing and localization of peripheral stimulus when attention engagement is well underway. These findings from the prior studies by Richards and colleagues suggest that the neural mechanisms underpinning the behavioral signatures of covert orienting are also likely to be influenced by infant sustained attention.

Studies with ERP measures have shed light on the neural correlates of infant covert orienting (e.g., Csibra et al. 1998, 2001; Richards 2000a, 2001, 2005). Richards (2000a, 2001, 2005) has studied the development of infant covert orienting and relevant ERP components from 3 to 6 months of age. The P1 component in the occipital electrodes was found to be larger for valid than invalid and neutral trials in 4.5- and 6-month-old, but no difference was found in 3-month-old. The N1 component, a negative deflection following the P1, was found to have parallel effects with the P1 (Richards 2000a, 2005). Richards (2005) also applied cortical source analysis to examine the brain regions that might generate the scalp recorded ERPs involved in infant covert orienting. Cortical sources that might generate the P1 validity effect were found to be located at the Brodmann's areas (BAs) 18 and 19 (extrastriate cortex). Cortical sources for the N1 validity effect were localized to the BAs 7, 18, 19 (central occipital and parietal regions). Richards also found other sources underlying the P1 and N1 located in the temporal lobe including the inferior temporal gyrus and fusiform gyrus. The source localizations of the infant P1 and N1 components are mostly in line with the active brain regions found in adult covert orienting using cortical source localization techniques (e.g., Clark et al. 1995; Fu et al. 2008; Martinez et al. 1999, 2001) and other neuroimaging techniques (e.g., Corbetta et al. 2005; Di Russo et al. 2002; Munneke et al. 2008; Yamagishi et al. 2005). It should be noted that prior studies looking at infants' ERP components did not find the effect of SOA duration on the validity effect (Richards 2000a, 2001, 2005), which was inconsistently with the existing behavioral findings. The lack of an SOA effect on infants' ERP responses may be due to the usage of only one SOA condition (Richards 2005) or the within-subject design for SOA duration, which resulted in insufficient power to detect the effect.

An accurate MRI model that describes the materials inside the head and their relative conductivity is beneficial for source analysis of ERPs (Michel et al. 2004; Reynolds and Richards 2009). Age-specific MRIs may be especially important for pediatric populations (e.g., infants and young children) due to the neuroanatomical differences that would be a poor fit with an adult MRI template (Reynolds and Richards 2009; Richards and Xie 2015). A significant advance in cortical source analysis with infant participants is to use MRI models from similarly aged infants, such as MRIs from infants with similar head shape and size (Reynolds et al. 2010) or an age-appropriate MRI average (Hamalainen et al. 2011; Ortiz-Mantilla et al. 2012). Preliminary research has indicated that using age appropriate MRIs with similar head sizes to the participant lead to similar cortical source analysis results to using an MRI from the individual participant (McCleery and Richards

2012). One methodological improvement of the source analysis in the current study compared to Richards (2005) was the use of individual infant MRI from the Neurodevelopment MRI Database (Richards et al. 2015b; Richards and Xie 2015). A single 6-month-old infant MRI was used by Richards (2005) to create the head model for 3- and 4.5-month-old participants. Whereas the ideal might be to obtain structural MRIs from the infants tested in the psychophysiological experiment (Guy et al. in press; Hamalainen et al. 2011; Ortiz-Mantilla et al. 2012), we used individual MRIs from a "library" of MRIs chosen from our Neurodevelopment MRI Database (Richards et al. 2015b; Richards and Xie 2015). Therefore, an MRI close in shape, size, and age was chosen from the Neurodevelopmental MRI Database for each participant as the anatomical representation for the cortical source analysis.

The first goal of the present study was to investigate the effect of covert orienting on infant cortical activity. We measured infant ERPs and applied cortical source analysis with realistic head models to measure current density amplitude in the cortical sources. The current study tested separate groups of infants in the short and long SOA conditions to increase the number of trials and presumably enhance the power for finding a significant difference between the two SOA conditions in the ERPs validity effect. It was expected to find the validity effect in infant ERPs and cortical source activity in the short but not the long SOA condition, which was in line with previous behavioral findings. The processing effect on infants' behavioral and cortical responses has typically been observed by comparing the neutral and invalid trials (e.g., RT invalid > RT neutral). We hypothesized that the processing effect would be found at the current ages (3 and 4.5 months) mainly in the short SOA condition. Given the absence of literature on IOR in infants younger than 4.5–6 months, we hypothesized that the IOR effect may not be found in the current participants who were 3- to 4.5-month-old.

A second goal was to determine the effect of HR defined sustained attention on infant cortical activity underpinning the early stages of visual processing involved in covert orienting. We improved the experiment procedure by using continuous presentations in the spatial cueing paradigm and interesting pictures as backgrounds (Mallin and Richards 2012; Pempek et al. 2010). Experimental trials within a block were presented continuously without inter-trial gaps. These improvements would elicit sustained attention and improve infant engagement (Mallin and Richards 2012). We hypothesized that infant sustained attention would enhance infant P1 and N1 responses and the activity in the cortical sources that are responsible for generating the ERPs. Given the sharp development of infant sustained attention from 3 to 6 months, we also expected the effect of sustained attention would become more specialized to the cortical regions involved in infant covert orienting with age.

Methods

Participants

Twenty-one infants were tested at 3 (M = 105.1 days, SD = 4.62, 11 F/10 M) and another 21 at 4.5 (M = 146.1 days, SD = 5.01, 7 F/14 M) months of age. The infants were born full-term (gestational age of 38 weeks or greater), weighed greater than 2500 g at birth, and without pre- or perinatal medical complications. The participants consisted of 33 Caucasians, 7 African-Americans, 1 Hispanic, and 1 Asian. Infants were randomly assigned to either the short SOA (3 months: $N = 11$; 4.5 months: $N = 10$) or long SOA (3 months: $N = 12$; 4.5 months: $N = 9$) conditions. An additional 18 infants were tested but excluded from analyses because of fussiness during the testing session ($N = 8$) or insufficient and noisy data ($N = 10$).

Apparatus and Stimuli

Apparatus used in this study consisted of a color monitor, two cameras, and computers. A 29″ color video monitor (NEC Multisync XM29) was used. The center of the monitor was located approximately 55 cm from infant's eyes. A camera was located above the monitor to record infant visual fixations. A second camera was located above and behind the participant and toward the presentation display to record the stimulus presentation. These video recordings were used for both online and offline visual judgments of infant looking. Microsoft visual C++ programs were used for presentation and experimental control. Visual stimuli used in this study included 13 Sesame Street dynamic characters (e.g., "Big Bird" and "Elmo") and 8 computer-generated visual patterns (e.g., checkboards and rotating concentric squares whose size varying in time) as fixation stimuli and cues and targets, and 5 static backgrounds (Fig. 1; also see Richards 2005; Mallin and Richards 2012).

Procedure

Infants were tested with the spatial cuing procedure in a continuous presentation paradigm and were held on a parent's lap approximately 55 cm from the center of the monitor. Figure 1 illustrates the presentation paradigm and examples for the visual stimuli used in the current study.

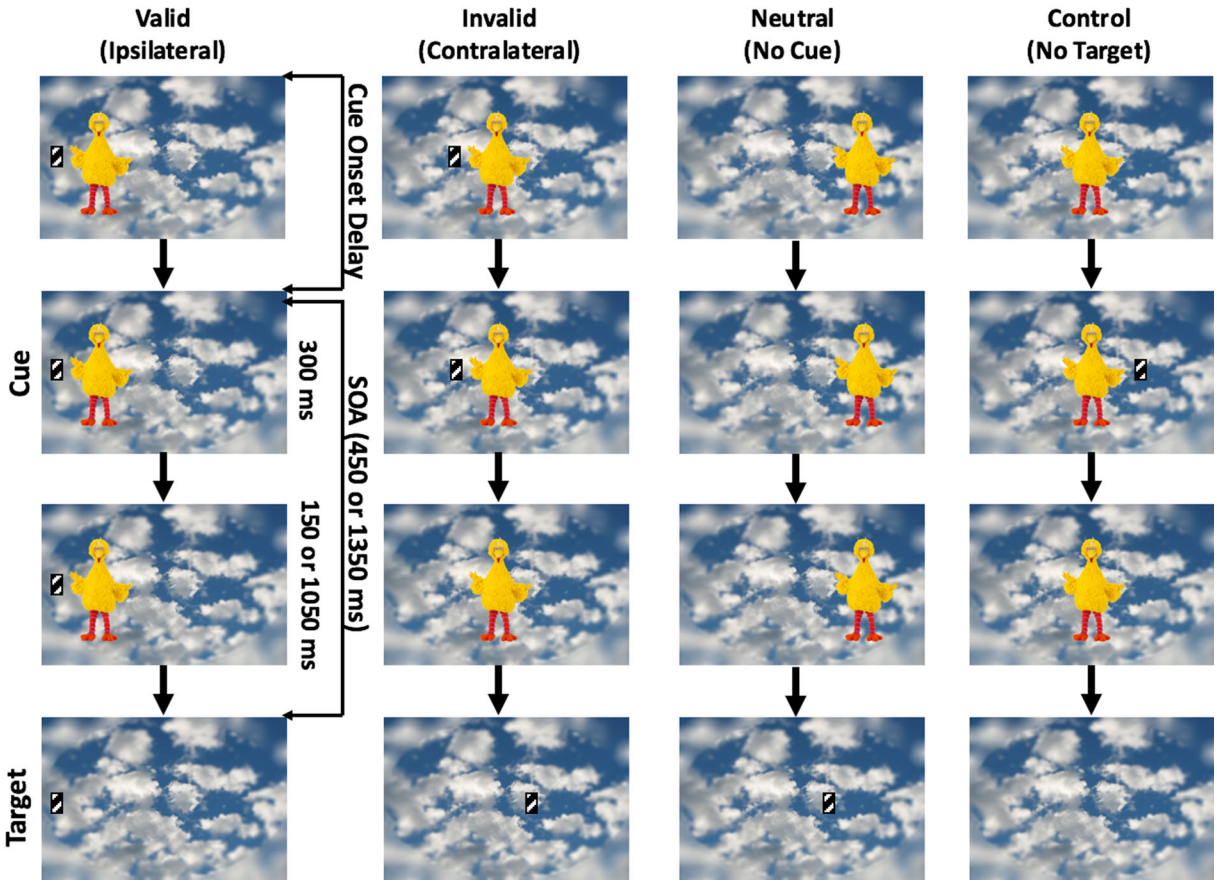


Fig. 1 Demonstration of the continuous spatial cueing paradigm used in the current study. There were four types of trials in the procedure: valid, invalid, neutral, and control. Examples of the visual stimuli are shown in this figure. Sesame Street characters (e.g., the "Big Bird" in this figure) were used as the fixation stimuli, which were randomly presented at one of three horizontal positions on the screen (left at 28 % of screen size, center at 50 % of screen size, or right at 72 % of screen size). The geometric patterns were used as cues and targets, and the cues were presented either on the left or the right side of the fixation stimulus. Either short (450 ms) or long (1350 ms) SOA was used between the cue and target onsets. These stimuli were presented on a background image

As shown in Fig. 1, a fixation stimulus was randomly presented at one of three horizontal positions on the screen (left at 28 % of screen size, center at 50 % of screen size, or right at 72 % of screen size), centered in the vertical dimension, and measured approximately 17° visual angle. The fixation stimulus was animated, but its size and location were constant in the horizontal position, which means that there was no variation in the left and right edges of the fixation stimulus. When the infant looked toward the animated fixation stimulus judged by the experimenter, a peripheral cue stimulus was presented in a 2.25° × 5° area approximately 2° visual angle from the right or left of the fixation stimulus. The cue was presented for 300 ms and then turned off. The central stimulus was frozen when the cue was presented in order to reduce the possibility of infants moving their eyes toward the cue before the target onset (e.g., Richards 2005; Markant et al. 2015a). After the cue was turned off, the fixation stimulus was left on the screen for 150 ms in the short SOA condition and 1050 ms in the long SOA condition. The fixation stimulus was removed at the end of the SOA and the target was presented. The target had the same size with the cue. It remained on the screen until the infant looked away from the location of the fixation stimulus (toward or away from the target). The presentation procedure included four cue-target presentation sequences, valid (ipsilateral), invalid (contralateral), neutral (no cue) and control (no target). The four presentation sequences were presented randomly without replacement in each set of four trials, so that each presentation sequence had an equal probability of appearing in the four sequential positions within the four trials. The valid trials had the target ipsilateral to the cue. The invalid trials had the target contralateral to the cue. The neutral trials had no cue preceding the target. The control trials had a cue, but no target. This procedure was repeated continuously for a block that lasted for 55 s, followed by a 5 s interblock interval with a blank screen, and then a new block started with a new background stimulus.

Target Stimulus Localization Latency

Infant looking was judged based on review of the video recording. A single observer determined if the infant was looking toward the fixation stimulus when the cue was presented, continued looking toward the fixation stimulus until target presentation, and then looking toward or away from the target when it was presented. The observer was blind to the experiment condition and stimulus type. Trials were used only if the infant was looking toward the fixation stimulus before the target was presented. It should be noted that we excluded trials in which the infants moved their eyes before the target onset, although infants are expected to show anticipatory saccades at times at this age (Haith et al. 1988; Johnson et al. 1994; Richards 2000a, 2005).

The latency to localize the target stimulus was determined with reaction times defined by saccades in the electrooculogram (EOG). This was calculated as the difference between the onset of the target and the beginning of the saccade in the EOG recording. More details about the EOG recording and identification of saccades can be found in prior studies (Hunter and Richards 2003; Mallin and Richards 2012; Richards 2005).

HR Defined Attention and Inattention

Attention phases were defined according to the changes in HR during viewing of the continuous stimulus presentations. Two Ag–AgCl electrodes were placed on the infant's chest with disposable electrode collars to record ECG. The phase of "stimulus orienting" was defined as the time period before the significant HR deceleration occurred when the fixation was directed to the stimulus; the phase of "sustained attention" was defined as the time when the HR decelerated and remained below the prestimulus level; the phase of "attention termination" referred to the period when HR returned back to the prestimulus level while the fixations were still on the presentations. It should be noted that the recordings of the ECG and electroencephalogram (EEG) signals and the stimulus presentations were synchronized. Thus, each experimental event/trial was categorized into sustained attention or inattentive (stimulus orienting and attention termination) state depending on the HR-defined attention phases. Details about using ECG data to define attention phases in a continuous presentation paradigm have been described elsewhere (e.g., Mallin and Richards 2012; Pempek et al. 2010; Reynolds et al. 2010).

EEG Recording, Segmenting, and Analysis

The electroencephalogram was recorded with the 128-channel EGI (Electrical Geodesics Incorporated, Eugene, OR) "Geodesic Sensor Net" (GSN) and EEG recording system (Johnson et al. 2001; Tucker 1993; Tucker et al. 1994). The EEG signal was referenced to the vertex recorded with 20 K amplification, at a sampling rate of 250 Hz (4 ms samples) with band-pass filters set at 0.1–250 Hz. The vertex-referenced EEG was algebraically recomputed to an average reference and digitally filtered to 1–45 Hz.

The EEG recordings were inspected for artifacts, poor recordings, and blinks, and if these occurred individual channels within trials were eliminated from the analyses or substituted with adjacent channels. EEG changes >150 μV in the horizontal and vertical direction were defined as eye movements and blinks respectively. Large EEG changes (>200 μV) were detected as artifacts and eliminated using computer algorithms. A linear interpolation was conducted to correct for any rejected channels using the five closest electrodes if there were less than 12 electrodes that were missing or had bad data. At least 5 good trials in an experimental cell (cue-target condition, sustained attention-inattention condition) were required for a participant's data to be included in the analysis. This minimum number of artifact-free trials per condition is lower than the usual criterion (e.g., ~10) used in infant ERP studies (DeBoer et al. 2007); but may be acceptable given good trials and our quantification procedure (cf. Stets and Reid 2011; Stets et al. 2012; also see Supplemental Information). The segmenting of the EEG was done based on target onset. The segments were from 50 ms before target onset through 300 ms following target onset. The procedures used to detect EEG artifact, segment ERPs, and average ERPs were completed using the EEGLAB and ERPLAB toolboxes (Delorme and Makeig 2004; Lopez-Calderon and Luck 2014) within MATLAB (MATLAB R2014a, the Mathworks, Inc.).

The ERP analyses focused on the P1 and N1 components with the measurement of mean amplitude at the P1 and N1 latencies. The mean ERP for the P1 was defined as the mean EEG between 90 and 110 ms, minus the preceding trough at 60–70 ms, and the N1 was defined as the mean EEG between 165 and 175 ms, minus the preceding P1 interval. These latencies were determined by first computing the overall grand average and determining the P1 and N1 latencies (e.g., Figure 3), and were similar to analyses from previous studies (e.g., Richards 2000a, 2005). This peak-to-trough, or mean minus trough corrections were calculated to mitigate the effects of slow waves and negative trends on these components. This type of correction has been utilized in ERP studies with pediatric populations (e.g., Kuefner et al. 2010; Peykarjou et al. 2013). The P1 and N1 amplitudes were calculated from these latencies separately for every acceptable ERP trial rather than averaging EEG segments across conditions before extracting the ERP amplitude (see Supplemental

Information). For the source analysis, the peak ERP latency was chosen in the time window from 80 to 150 ms for the P1 source analyses, and from 130 to 200 ms for the N1 source analyses. We used wider windows here than those in the ERPs analyses with mean amplitudes so that the peak latencies for the P1 and N1 could be picked up accurately for all the participants. EEG data was baseline corrected using the P1 and N1 peak latencies for source analysis. For both the mean ERP and the peak analyses, we excluded trials where there were pre-target eye movements, or where a saccade to the target occurred before the latency range of that component (e.g., 150 ms for P1 component; 200 ms for N1 component). ERP measurements were done using the ERPLAB toolbox (Lopez-Calderon and Luck 2014) in MATLAB.

Electrodes in the occipital area were grouped into six sets of electrodes and labeled as "virtual 10–10 clusters" depending on the closest 10–10 locations for the ERP analyses. The clusters were Occipital1, Occipital2, OccipitalZ, Inion1, Inion2, InionZ, which were chosen based on previous research (Richards 2000a, b, 2005). Please see the Supplemental Information Fig. 1 and 2, and Supplemental Table 1 for more information about the grouping of these electrode clusters and their locations on infant MRIs. We flipped the right and left channels for the trials when the target was presented on the right side, as if the peripheral target was always presented on the "left side" of the participant. As a result, the even-number virtual channels (Inion2, Occipital2) represent the contralateral leads to the target and the odd-numbered electrodes (Inion1, Occipital1) represent the ipsilateral leads.

Cortical Source Analysis

The cortical source analysis of the P1 and N1 ERP components was conducted with the Fieldtrip (FT; Oostenveld et al. 2011) computer programs and in-house custom MATLAB scripts. Our cortical source analysis consisted of four major steps that are selection of anatomical MRIs, construction of realistic head models, definition of regions of interests (ROIs), and source reconstruction (i.e., current density reconstruction; CDR). Detailed information for the procedures in each step is provided in the Supplemental Information.

We selected an MRI close in head size to each participant from the Neurodevelopment MRI Database (Richards et al. 2015b; Richards and Xie 2015) as the anatomical MRI representation of infant head and brain. We conducted external head measurements for the participants in the current study so that an MRI from the database with the closet head size was chosen for each participant.

The infant MRIs were segmented and realistic head models were created. MRIs were segmented into component materials including scalp, skull, cerebral spinal fluid (CSF), white matter (WM), gray matter (GM), nasal cavity, and eyes (Guy et al. in press; Richards 2013; See Supplemental Information Fig. 7). The inclusion of the highly conductive CSF compartment and the distinction of GM and WM have been found to show strong influence on the EEG forward model solution (Vorwerk et al. 2014). The GM volume and the eyes were used as the source volume for source analysis (Supplemental Information Fig. 4). The segmented MRIs were then transformed to wireframes to complete the finite element method (FEM) models. Finally, an electrode placement map was constructed for each participant's head model with methods that have been recently developed for adults (Richards et al. 2015a; Supplemental Information Fig. 1).

Twenty-three brain regions were chosen for ROI analysis based on past identification of dipoles responsible for generating scalp measurements (e.g., ICA clusters and the P1 and N1 components) in infant and adult spatial orienting studies (Martinez et al. 1999; Richards 2005; see Supplemental Information Fig. 3). These ROIs included the separate left and right volumes for the anterior fusiform gyrus, middle fusiform gyrus, medial inferior occipital lobe, lateral inferior occipital lobe, middle occipital lobe, superior occipital, parahippocampal gyrus, posterior inferior temporal gyrus, posterior middle-superior temporal gyri, and temporal pole (20 ROIs). A single bilateral ROI was used for the lingual gyrus, central occipital lobe, and parietal lobe (3 ROIs). The location of these brain regions (ROIs) was defined based on stereotaxic atlases created for each MRI (Fillmore et al. 2015; Sanchez et al. 2012). Supplemental Information Table 2 lists the ROIs and the atlases and corresponding structures used to generate each ROI.

Source reconstruction was conducted with realistic head models and the CDR technique. The electrode locations, source locations, and FEM model generated in the previous steps were used to create the forward model. The forward model was used to estimate the lead-field matrix that represents the linear relation between the activity in the cortical sources and the measurements in electrodes on the scalp. Source reconstruction was conducted with the ERP data and the lead-field matrix. Current density amplitude in the dipole source (GM and eyes) locations was estimated with the CDR technique and the exact-LORETA (eLORETA; Pascual-Marqui 2007; Pascual-Marqui et al. 2011) as the constraint for the CDR technique. The ERP data surrounding the P1 and N1 peaks was used to estimate the current density amplitudes (i.e., CDR values) for every location in the source volume model. The CDR values were then summed over each source location in a ROI and divided by the total volume of the ROI. This resulted in the average

μAmpere per mm^3 ($\mu A/mm^3$) for each ROI. For statistical analysis, the CDR value was averaged with the time window (±10 ms) around the P1 and N1 peaks separately. More information about using the CDR technique and realistic head models for cortical source analysis can be found elsewhere (Guy et al. in press; Richards 2013; Supplemental Information).

Statistical Analysis Strategies

Statistical analyses were performed with mixed-design ANOVAs to examine the effect of the experimental factors on the dependent variables. The design for the statistical analyses included the experimental factors of participant age (2: 3.0 months old, 4.5 months old) and SOA condition (2: short, long) as between-subjects factors, and attention phase (2: sustained attention, inattention) and cue-target validity (3: valid, invalid, neutral) as within-subject factors. The factors of attention phase and cue-target validity were analyzed separately in the ANOVAs because we did not have a sufficient number of trials in the individual cells of attention phase X cue-target validity analysis. The P1 and N1 mean amplitudes and peak latency were analyzed as dependent variables. For the ERP, the data from the six channels (Occipital1, OccipitalZ, Occipital2, Inion1, InionZ, Inion2) were tested as a repeated measure and we used the Huynh–Feldt-Lecoutre ε-adjustment of the degrees of freedom to correct for inhomogeneity of repeated measures covariance matrices (Greenhouse and Geisser 1959; Huynh and Feldt 1976; Lecoutre 1991). We used a general linear models approach to the ERP ANOVAs. This included using individual trial data in the analysis, inclusion of number or trials as an explicit factor in the ANOVA to account for differences across participants in number of good trials, and unweighted Type 1 estimation of SS for the ANOVA (cf. Vossen et al. 2011; and see Supplemental Information). It should be emphasized that the number of trial information was only used to protect the analysis from being biased by participants with small numbers of trials. If the cells were completely balanced the results from this analysis and a typical ANOVA would be identical (Vossen et al. 2011). Post hoc tests were Sheffé corrected to account for multiple comparisons, and all significant tests were reported at $P < 0.05$. For the CDR values obtained from source analysis we restricted the analyses to the factors that showed significant effects on the P1 and N1 ERP components. For these analyses we used an error protection strategy with an error term from an omnibus test with the CDR values similar to the omnibus test for the significant ERP effect, and did single df comparisons with a Sheffé corrected significance value of $P < .05$.

Results

Localization Latency and Probability

The presentations were done in trial blocks of 55 s in duration, with a minimum of 10 blocks for each participant (M = 12.72; STD = 2.18, range = 10–20). The blocks had continuous presentations of the fixation stimulus, peripheral cue and target, with the average number of presentations (trials) for participants ranged from 44 to 143 (M = 80.90, SD = 23.52). The center stimulus occurred approximately equally often in the center, right or left position (frequency = 32.82, 33.56, and 33.62 percent, respectively over all trials), and the four cueing types (valid, invalid, neutral, no-target) occurred approximately equally often (frequency = 24.50, 25.19, 25.19, and 25.19 percent, respectively over all trials).

Behavioral analysis focused on the saccade latency to the peripheral target as a function of cue-target validity, age, and SOA condition. There was a significant interaction between cue-target validity and SOA condition on the saccade latency, F (2, 82) = 14.23, $P < 0.001$. Simple effect tests showed that the main effect of cue-target validity was significant in both short, F (2, 39) = 13.61, $P < 0.001$, and long, F (2, 43) = 4.11, $P = 0.0232$, SOA conditions. Figure 2 shows the mean latency changing as a function of the cue-target validity in the two SOA conditions. The results from the multiple comparisons of different cue-target validity levels are also shown in Fig. 2. The validity (RT: valid < neutral; valid < invalid) and processing cost effects (RT: neutral < invalid) were found in the short SOA condition; however, an IOR effect (RT: valid > neutral) was not found in the long SOA condition. Table 1 summarizes these behavioral findings.

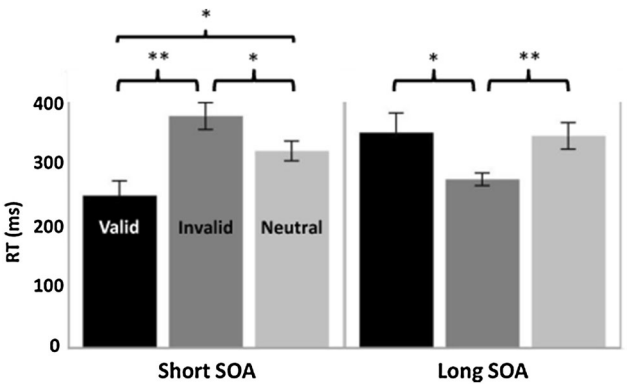


Fig. 2 Mean latency (in milliseconds) to localize the peripheral target in the three cue-target validity conditions, separately for the short and long SOA conditions. The error bars are the standard errors of the means. $**P < 0.01$; $*P < 0.05$. Bars for the short SOA condition show the validity (valid < invalid, valid < neutral) and processing cost (neutral < invalid) effects, while bars for the long SOA condition did not show the IOR effect (valid = neutral)

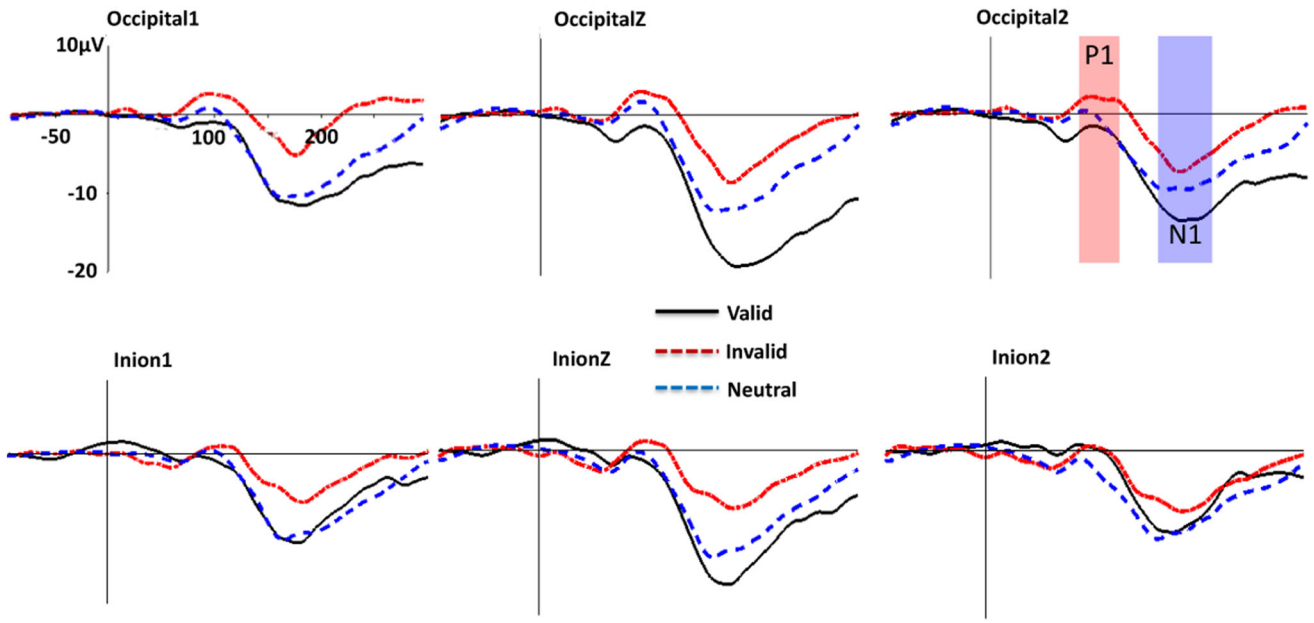


Fig. 3 Overall P1 and N1 ERP components for the valid (*black*), invalid (*red*), and neutral (*blue*) trials in the six virtual channels averaged for the short and long SOA conditions. The latencies for P1 and N1 are shown on the Occipital1 figure. The ERPs are shown from 100 ms preceding stimulus onset through 300 ms following stimulus onset averaged over 3- and 4.5-month groups

Localization probability was calculated to determine infant eye movements in the spatial cueing paradigm. We calculated the probability that saccades were made toward the target (target-driven, exogenous saccades) in the no cue condition and toward the cue side before the target was presented (memory-driven, endogenous saccades) in the valid, invalid, and no target conditions. In the no cue condition, infants looked toward the target on 89.26 % ($N = 507$) of the trials and toward the other side on only 10.74 % ($N = 61$) of the trials. In the no target condition, infants looked toward the cue side on 87.89 % ($N = 334$) of the trials and toward the other side on 12.11 % ($N = 46$) of the trials. On the trials where both a cue and a target (valid, invalid) were presented, infants' saccades might occur after the cue onset before the presence of the target or after the target onset. Among the saccades infants made after the cue onset but before the presence of the target, 88.72 % ($N = 716$) of them were toward the cue side and 11.28 % ($N = 91$) of them were toward the opposite side of the cue. Among the saccades infants made after the target onset, they looked toward the target with a probability of 93.70 % ($N = 669$) and toward the opposite side with a probability of 6.30 % ($N = 45$). There was no systematic bias for the two ages or the two SOA conditions.

Grand Average ERP Results

The number of accepted EEG trials was about 50 % of the original trials. Target presentations were done approximately equally during sustained attention and inattention (M's = 11.74 and 12.93 trials, respectively; range = 5 to 32 trials). The valid and invalid conditions had approximately equal numbers of presentations over participants (M = 8.47 and 9.19 trials, respectively; range = 5–20 trials), and slightly larger number of the neutral condition (M = 10.33 trials, range = 5–25 trials). The slightly larger average number of trials for the neutral target condition was due to saccades that occurred before the P1 and N1 intervals on the cue-present trials; the number of trials for the three cueing types was not significantly different, $P = 0.115$.

Cue-Target Validity Effect on ERPs

Figure 3 shows the overall ERP responses at the 6 virtual channels as a function of cue-target validity. The P1 is evident as a small positive ERP component peaking approximately 100 ms after stimulus onset. The N1 occurs about 170 ms following the stimulus onset. It can be seen that there is a negative trend (i.e., a negative slow wave) underlying the P1 and N1 components. Table 1 summarizes the major findings from the ERP component analysis.

Analyses on the mean amplitudes of P1 and N1 responses were performed to determine the effect of cue-target validity and its interaction with other factors on the P1 and N1 responses. The analysis of P1 amplitude revealed a main effect of channel location, F (5, 185) = 2.51, $\varepsilon = 0.7971$, $P = .044$, and an interaction between channel location, SOA condition, and cue-target validity, F (10, 359) = 2.63, $\varepsilon = 0.7971$, $P = 0.0086$. Post

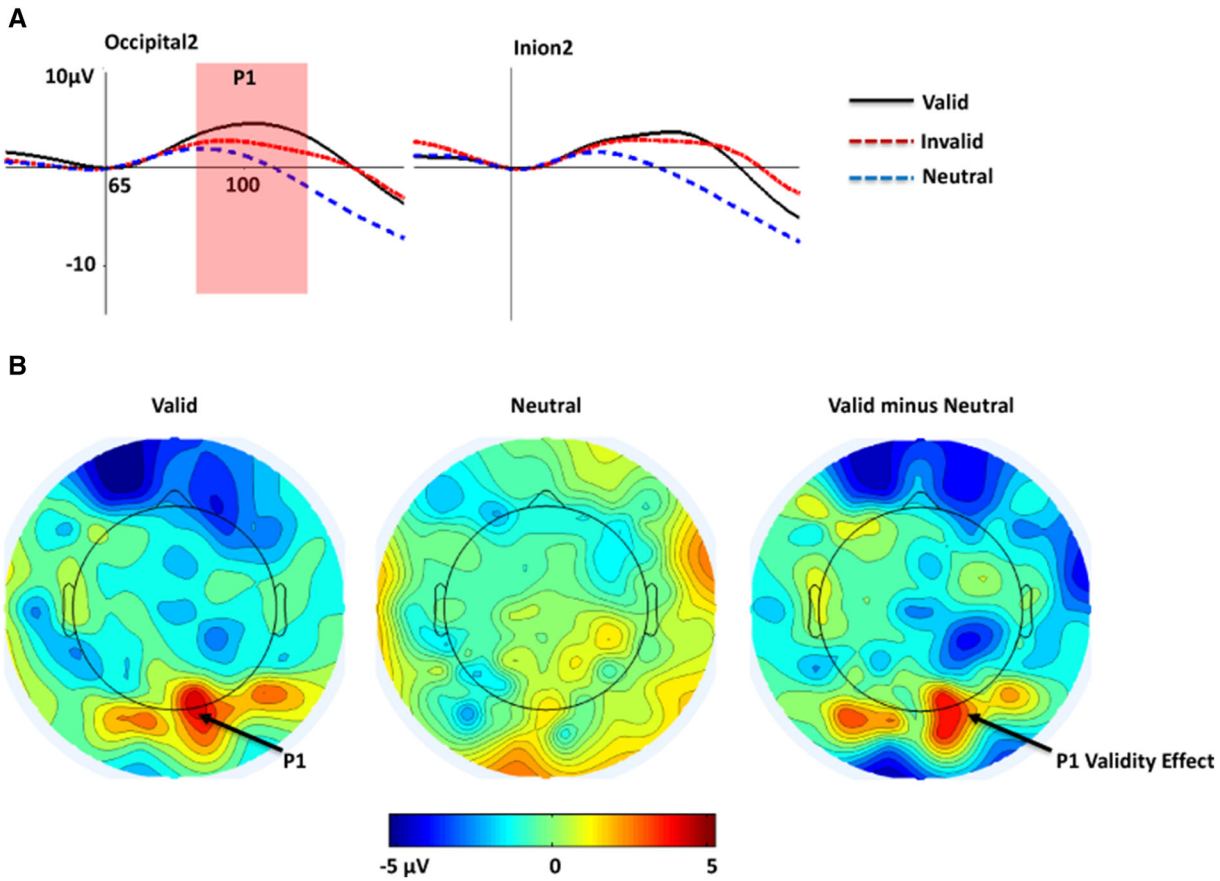


Fig. 4 A. Comparison of the P1 component for valid (*black*), invalid (*red*), and neutral (*blue*) trials in the Occipital2 and Inion2 virtual channels, only for the short SOA condition. The ERPs are plotted as the difference from the negative trough (65 ms) through 200 ms after the stimulus onset. The validity effect is represented by the P1 ERP amplitude being larger on the valid than neutral trials. B. topographical scalp potential maps illustrating the P1 validity effect. The mean amplitude at the P1 latency (90–110 ms) is shown for valid and neutral trials, and a difference map

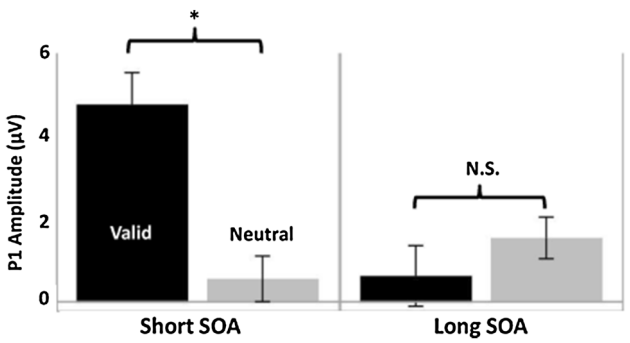
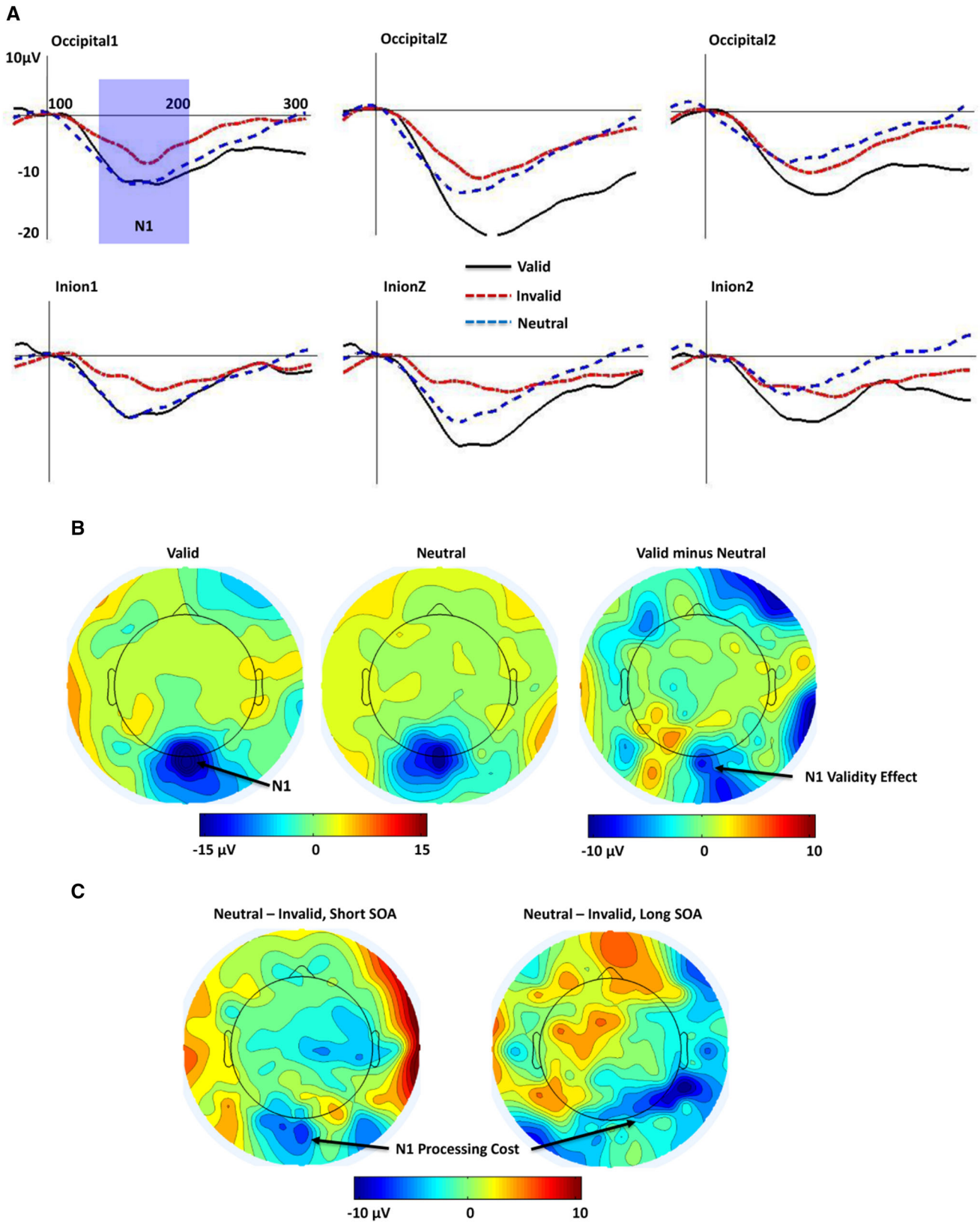


Fig. 5 Bar graphs of the P1 amplitude for the valid (*black*) and neutral (*silver*) trials separately for the short and long SOA conditions, averaged across the Occipital2 and Inion2 virtual channels. Error bars represent standard errors of means. The difference between the neutral and valid trials in the long SOA is in the direction of an IOR effect, but did not reach statistical significance. The P1 ERP component amplitude was larger on the valid trials in the short than in the long SOA. $*P < 0.05$; "N.S." refers to non-significant result

hoc tests showed a validity effect, i.e., valid P1 amplitude > neutral P1 amplitude, for both the Occipital2 and Inion2 electrodes only in the short SOA condition. Figure 4a shows the ERP for the P1 from the trough at 60–70 ms through about 125 ms after stimulus onset, separately for the three cue-target validity types, but only in the short SOA condition. The P1 ERP component was larger on the valid than on the neutral trials. Topographical scalp potential maps are shown in Fig. 4b for the ERP activity at the peak of the P1 (90–110 ms) for the valid and neutral trials, and a difference map. The validity effect occurs slightly on the right side of this figure, which represents the scalp location contralateral to the target.

An IOR effect is usually found by comparing the valid and neutral trials on the long SOA, and there is a corresponding difference between the valid trials on the short and long SOA. Figure 5 is a bar graph of the P1 amplitude for the valid and neutral trials separately for the short and long SOA conditions. The valid and neutral trials were not significantly different for the long SOA condition.


A
Occipital1
10μV
100
200
300
-10
-20
N1
OccipitalZ
Occipital2
Valid
Invalid
Neutral
Inion1
InionZ
Inion2
B
Valid
Neutral
Valid minus Neutral
N1
N1 Validity Effect
-15 μV
0
15
-10 μV
0
10
C
Neutral – Invalid, Short SOA
Neutral – Invalid, Long SOA
N1 Processing Cost
-10 μV
0
10

◀**Fig. 6** **a** The N1 responses in the six virtual channels to valid (*black*), invalid (*red*), and neutral (*blue*) trials in the short SOA condition. The ERPs are plotted as the difference from the positive peak (100 ms) through 300 ms after stimulus onset. **b** A topographical scalp potential map of the mean amplitude (165–175 ms) for the valid and neutral trials, and a difference map at the peak N1 latency. These maps show the spread of the N1 across the occipital and inion electrodes in the contralateral locations and the larger N1 response to valid than neutral trials. **c** A topographical scalp potential map for the mean (165–175 ms) difference between the neutral and invalid trials, separately for the short and long SOA conditions. The N1 mean amplitude is more negative for neutral than invalid trials in both SOA conditions, suggesting the cost of processing effect

However, the valid trials on the short SOA elicited a larger P1 response than the valid trials on the long SOA for the Occipital2 and Inion2 electrodes. This effect should not be attributed to overall lower brain activation in the long SOA condition because there is no difference in the P1 response to neutral trials between the short and long SOA conditions, and no main effect for SOA condition. Instead, this effect provides converging evidence on the validity effect on the valid trials when the SOA is short.

The analysis of N1 revealed a main effect of channel type, $F(5, 185) = 4.33, \varepsilon = 0.5722, P = .0072$, an interaction of channel type with SOA condition, $F(5, 185) = 2.68, \varepsilon = 0.5722, P = .0532$, and an interaction of channel type, SOA condition, and cue-target validity, $F(5, 354) = 2.95, \varepsilon = 0.5722, P = .0099$. There was a significant validity effect (valid N1 amplitude > neutral and invalid N1 amplitude) for the short SOA condition. This occurred for all three inion electrodes (Inion1, Inion2, InionZ), and was close to significant for OccipitalZ and Occipital2. Figure 6a shows the N1 ERP response for the six virtual electrodes, separately for the cue-validity types, and only for the short SOA. The N1 on the valid trials was larger than the N1 on the neutral and invalid trials. As with the P1 validity effect (Fig. 4a), the validity effect occurred primarily on the contralateral occipital and inion virtual electrodes, although the effect on the O2 electrode did not reach the significant level. Figure 6b is a topographical scalp potential map of the valid, neutral, and a different map for the N1 latency. The map shows the spread of the N1 across the occipital and inion electrodes in the contralateral locations.

The N1 amplitude on the neutral trials was greater than the N1 amplitude on the invalid trials, i.e., a "cost of processing" effect for Occipital1, Inion1, and InionZ in the short SOA condition and Occipital2 in the long SOA condition. Figure 6c is a topographical scalp potential map for the difference between the neutral and invalid trials, separately for the short and long SOA conditions. The N1 effect may be seen in both SOA conditions, though it appears to be in different locations. An IOR effect would occur if the N1 response for valid trials was smaller than that for neutral trials when the SOA is long. The post hoc comparison was not significant for the analysis of the IOR effect.

Sustained Attention Effect on ERPs

Analyses focused on the effect of sustained attention and its interaction with other factors on infant P1 and N1 responses. For the mean amplitude of P1, the main effect of attention phase was close to statistical significance, $F(1, 36) = 3.73, P = .0612$, and there was a significant interaction between age, SOA condition, and attention phase, $F(1, 36) = 6.87, P = .0127$. A post hoc test was done to examine the interaction of SOA and attention phases separately for the 3- and 4.5-month-old. This interaction was significant for the oldest age group but not for the youngest age group. Figure 7 shows the mean P1 amplitude for the attentive and inattentive periods, separated for the two age groups and the two SOA conditions. The P1 amplitude in sustained attention was larger than the P1 amplitude in inattention for both SOA conditions for the 3-month-old (left panel in Fig. 7). The right panel in Fig. 7 shows the

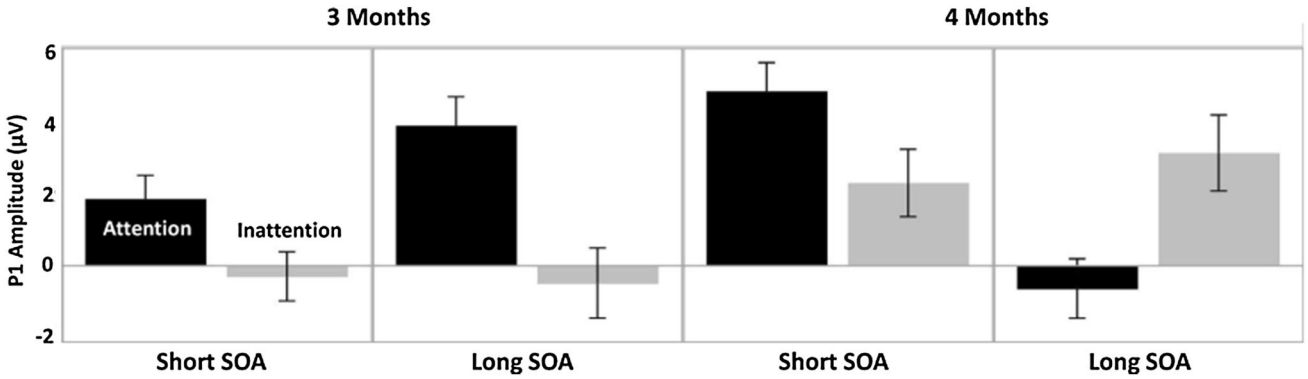


Fig. 7 Bar graphs for the P1 mean amplitude during the attentive (*black*) and inattentive (*silver*) periods, separated for the two ages and SOA conditions. Error bars represent standard errors of means. Larger P1 responses were elicited during (sustained) attentive periods than inattentive periods. This effect is consistent across the short and long SOA conditions for 3 months, but is only shown in the short SOA condition for 4 months

mean P1 amplitude for the 4.5-month-old, whose attention effect on the P1 amplitude was different across SOA conditions. The P1 amplitude during sustained attention was larger than the P1 amplitude during inattention only for the short SOA condition, and there was an opposite effect for the long SOA condition.

For the mean amplitude of N1, there were main effects of channel location, interactions between age and channel location, SOA condition and channel location, and age, SOA location, and channel location. However, none of these effects showed an interaction with attention phase and so were not further examined.

Source Analyses Results

Source analyses focused on the mean current density reconstruction amplitude (CDR value) in the ROIs that are potential cortical sources of the P1 and N1 ERP components. Table 1 summarizes the major findings from the cortical source analysis.

The first analysis was conducted to determine the effect of cue-target validity on CDR value around the P1 ERP peak latency in the ROIs. The ERP analysis showed a P1 validity effect in the short SOA condition with no effect of testing age. The CDR values were therefore tested as a "validity effect" with valid trials versus the neutral and invalid trials for the short SOA condition for each ROI segment. Four ROIs that were contralateral to the target had a significant validity effect: medial inferior occipital lobe, lateral inferior occipital lobe, middle fusiform gyrus, and posterior inferior temporal gyrus. Figure 8a shows the CDR values during the time period 40 ms preceding and following the P1 peak for the valid, invalid, and neutral conditions in the four ROIs that showed significant effects. The data from the parietal lobe was included in this figure for comparison. The valid trials had larger CDR values over this time period than the other two validity types. Figure 8b shows bar graphs for the average CDR values across this time period, separately for the three conditions. Average CDR value for the valid trials was greater than that for the invalid and neutral trials, and the later two conditions did not differ from each other. Figure 9 shows the CDR plots for the valid, neutral + invalid, and their difference on an average 3 months MRI brain template. The 2D images at the top part of the figure shows the difference between the valid and the mean of invalid and neutral trials (validity effect) in multiple axial slices. The validity effect was primarily shown in the inferior posterior occipital and temporal regions. The 3D images at the bottom part of the figure depicts the validity effect in the brain areas surrounding the four ROIs that showed significant results.

The second analysis evaluated the CDR amplitude around the N1 peak latency as a function of the cue-target validity conditions. The ERP analysis showed a N1 validity effect in the short SOA condition when comparing the

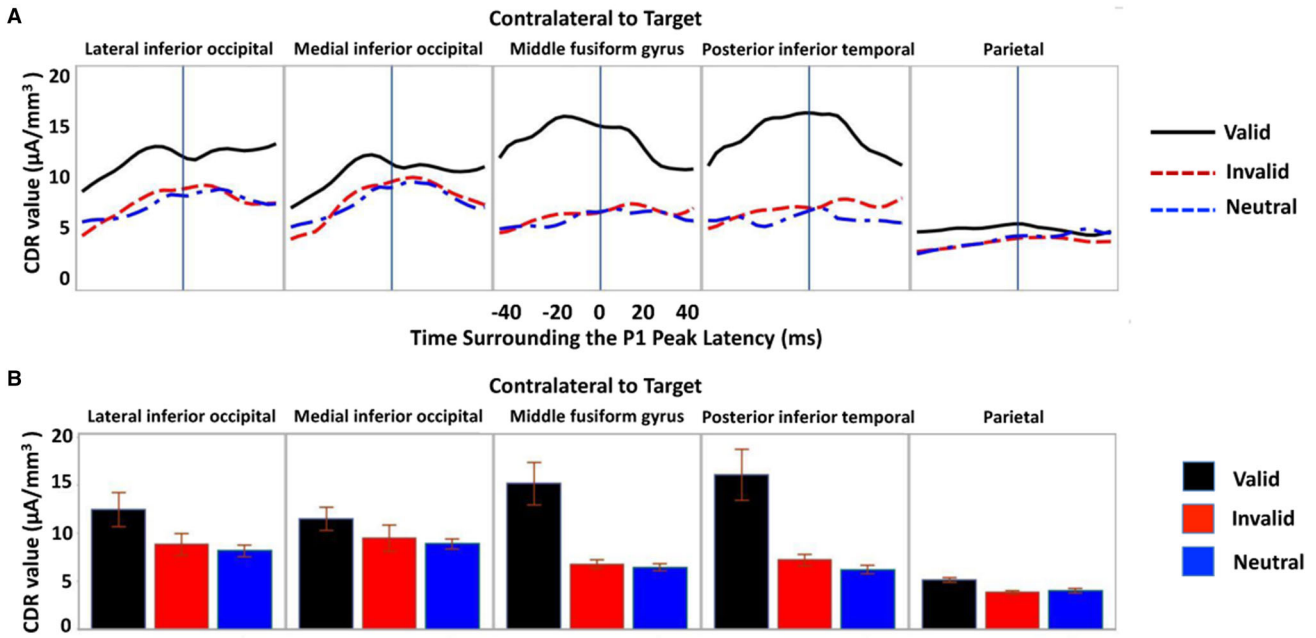


Fig. 8 **a** CDR value from −40 ms to +40 ms surrounding the P1 ERP peak latency for the valid (*black*), invalid (*red*), and neutral (*blue*) trials in the four ROIs that showed significant validity effects in the short SOA condition. These four ROIs are contralateral to the target location. The "Parietal" ROI is included in this figure for comparison. **b** Bar graphs of the Average CDR values (±10 ms) surrounding the P1 peak in the four ROIs that showed significant validity effects in the short SOA condition

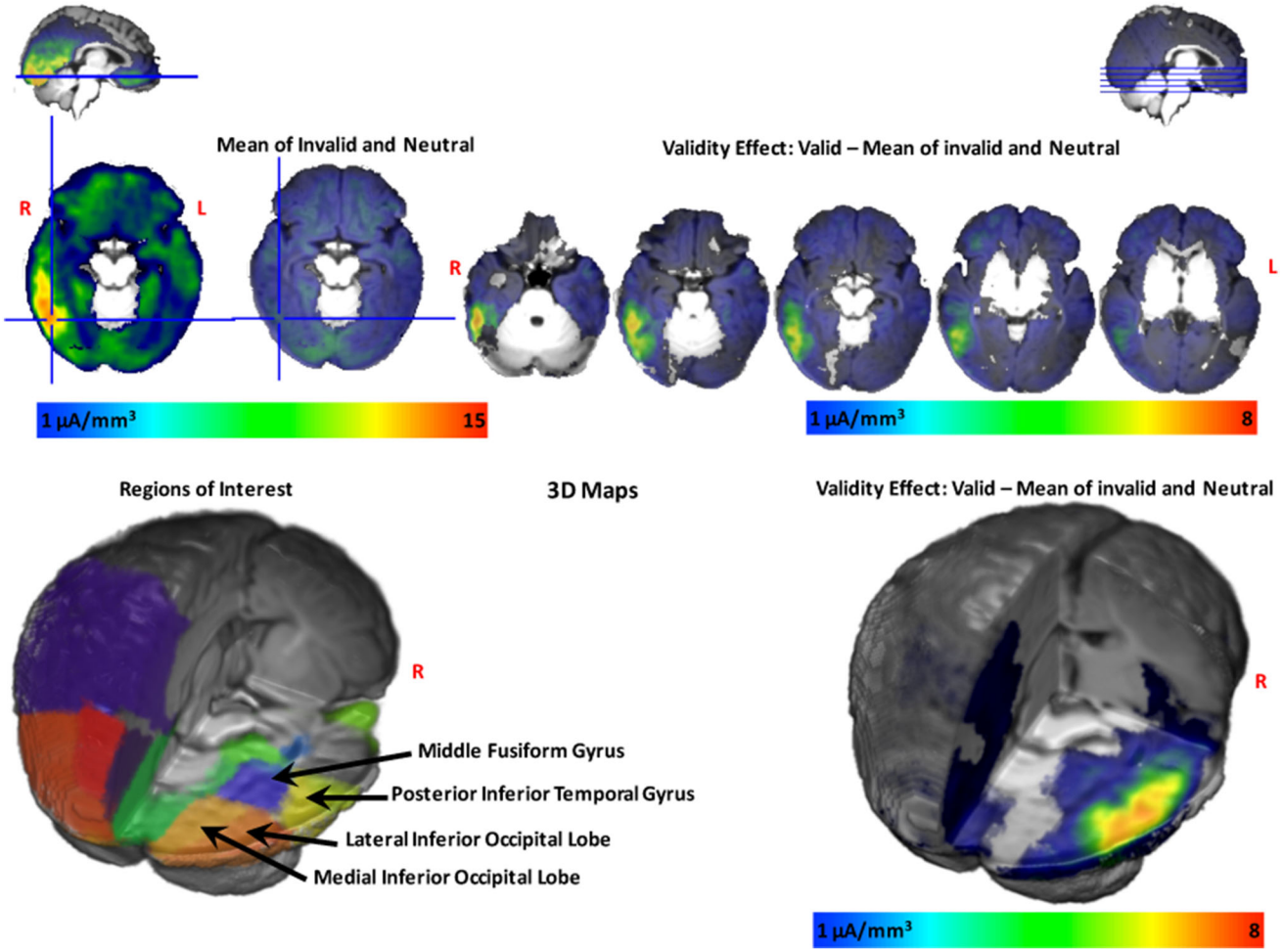


Fig. 9 **a** 2D maps of the validity effect on the CDR value (±10 ms) surrounding the P1 peak in brain source volumes for the valid trials (*first from left*), mean of the invalid and neutral trials (*second from left*), and the difference between them (*maps on the right side*). The five images (slices) taken from the bottom of the cortex to the middle occipital lobe demonstrate the validity effect (valid > invalid and neutral) primarily in the ventral temporal and posterior occipital regions contralateral to the target location. Left side of the brain in these 2D maps refers to the contralateral side to the target location. **b** 3D maps of the locations of the four ROIs that showed significant validity effects (*left*) and CDR value difference between the valid and the mean of invalid and neutral trials in brain source volumes (*right*). Right side of the brain in these 3D maps refers to the contralateral side to the target location. These data are shown in an average 3-month-old brain template

valid and neutral and invalid trials. We found no significant effects of the cue-target validity effect in the CDR values for this analysis.

The third analysis evaluated the CDR amplitude around the N1 peak latency comparing the neutral and invalid trials. The ERP analysis showed a N1 processing cost effect in both short and long SOA conditions. We compared CDR for the invalid and neutral trials from both SOA condition. A processing cost effect was found in four ROIs contralateral to the target: posterior inferior temporal gyrus, posterior middle and superior temporal gyri, medial inferior occipital lobe, and lateral inferior occipital lobe. A fifth contralateral area, middle occipital lobe, and a bilateral area, lingual gyrus, were close to statistical significance (P's −0.06 and 0.07, respectively). Figure 10a shows the CDR values during the time period 40 ms preceding and following the N1 peak for invalid and neutral trials in the four contralateral ROIs that showed significant processing cost effect. Figure 10b shows the mean CDR value over this time period for the two conditions. The neutral trials evoked greater CDR values than the invalid trials in these four ROIs. Data from the parietal lobe ROI is included in Fig. 10a, b for comparison. Figure 11 shows the difference of CDR value between neutral and invalid trials averaged over participants on the average 3 months MRI template. The processing cost effect extended from contralateral inferior occipital areas to middle occipital and middle-superior temporal areas. The 3D images at the

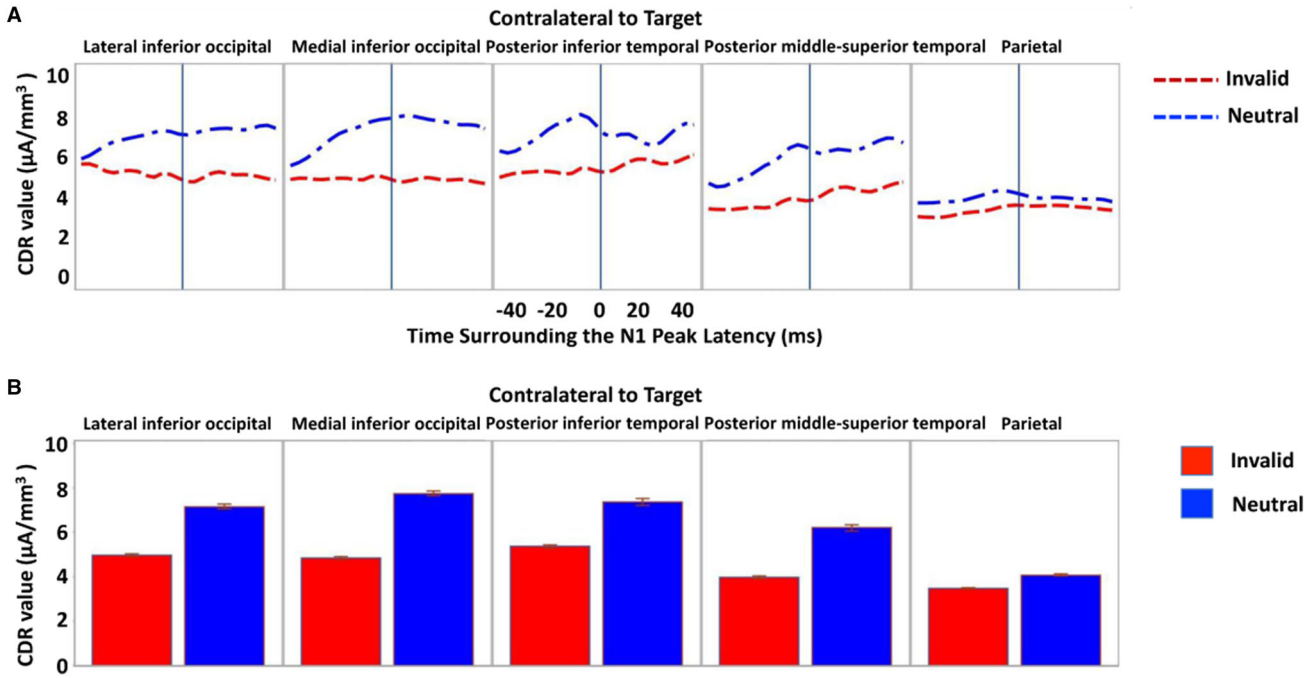


Fig. 10 **a** CDR value from −40 ms to +40 ms surrounding the N1 ERP peak latency for the invalid (*red*) and neutral (*blue*) trials in the four ROIs that showed significant processing cost effects. These four ROIs are contralateral to the target location. The "Parietal" ROI is included for comparison. **b** Bar graphs of the Average CDR values (±10 ms) surrounding the N1 peak in the four ROIs that showed significant processing cost effects

bottom part of the figure depicts the processing cost effect in the middle occipital and posterior middle-superior temporal regions.

Cortical source analyses also examined the effect of sustained attention on the current density amplitude around the P1 peak latency in the ROIs. The ERP analysis showed a larger ERP response during sustained attention for both SOA conditions for the 3-month-old participants, and a similar effect in the 4.5-month-old group for the short SOA condition. We compared the CDR value during sustained attention and inattention for the 3-month-old irrespective of SOA condition, but for the 4.5-month-old only for the short SOA condition. For the 3-month-old group, we found larger CDR values during sustained attention than inattention for four posterior contralateral ROIs and three anterior ventral temporal regions. The attention effect was found in the contralateral superior and middle occipital lobes, contralateral lateral and medial inferior occipital lobes, and in the ipsilateral parahippocampal gyrus, anterior fusiform gyrus, and temporal pole. For the 4.5-month-old, the attention effect was primarily shown in the posterior inferior occipital regions including the lateral inferior occipital gyrus on both sides, contralateral medial inferior occipital lobe, and posterior inferior temporal gyrus. Figure 12 shows the mean CDR value surrounding the peak latency of the P1 component for the ROIs that showed significant attention effect separately for the two testing ages. The attention effect was widespread for the 3-month-old group but this effect became localized to the posterior occipital ROIs in the 4.5-month-old group.

Discussion

The goal of the present study was to examine the effects of spatial cueing and sustained attention on brain activation underlying covert orienting in 3- and 4.5-month-old infants. We employed a spatial cueing paradigm with short and long SOAs to study infant covert orienting and continuously presented the experimental trials to elicit infant sustained attention. We defined attention phases based on infants' HR changes, measured their ERPs, and conducted cortical source analysis using realistic head models. Our first hypothesis was that infants would show the effects of covert orienting on brain activity, such as the validity and processing cost effects in the short SOA condition. Our second hypothesis was that sustained attention would enhance infants' brain activation in covert orienting. Table 1 summarizes the findings. We found validity and cost of processing effects on infants' behavior and brain activity. The P1 amplitude was greater

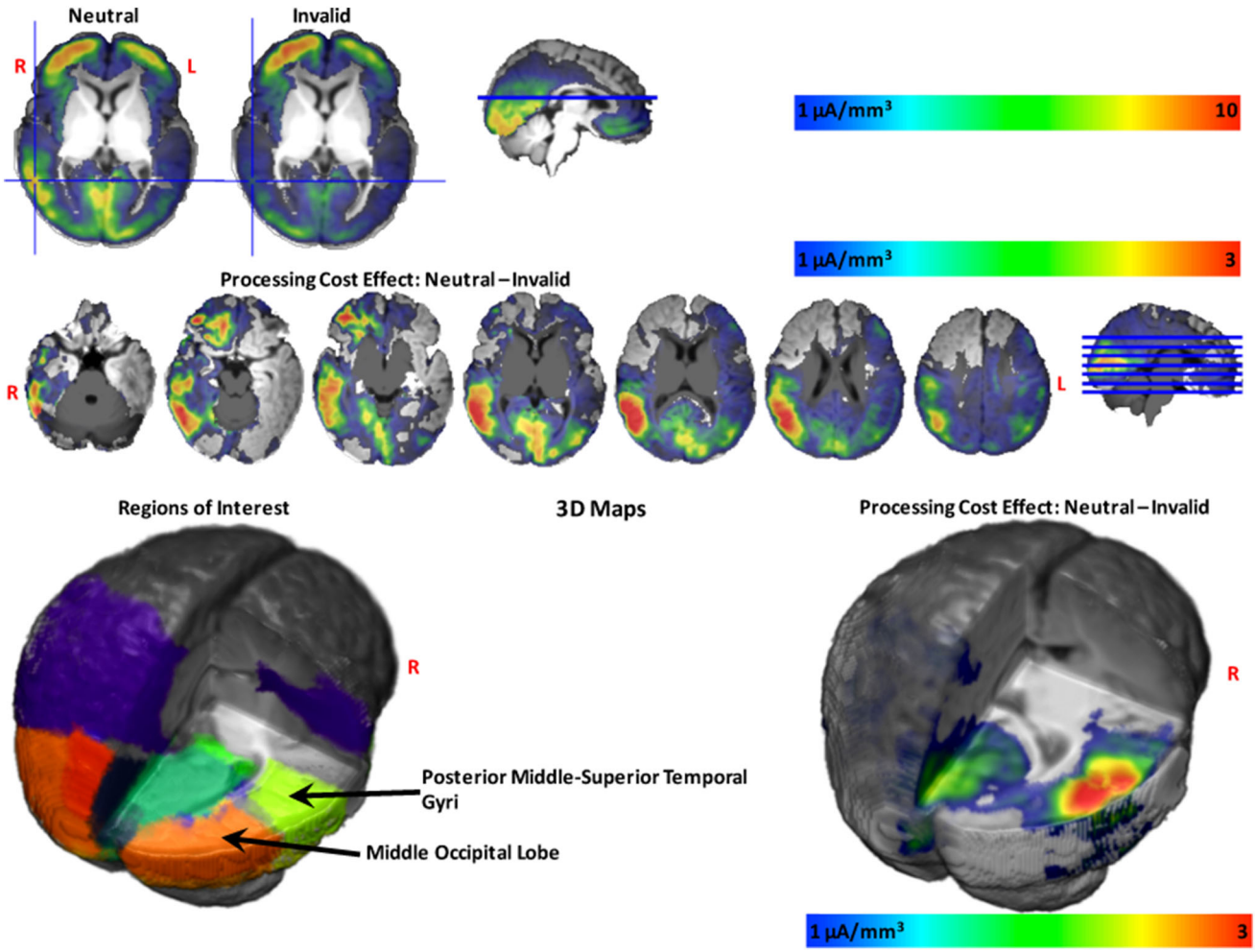


Fig. 11 **a** 2D maps of the processing cost effect on the CDR value (±10 ms) surrounding the N1 peak in brain source volumes for the neutral trials (*first from left*), invalid trials (*second from left*), and the difference between them (*maps on the right side*). The five images (slices) taken from inferior to superior occipital lobe demonstrate the processing cost effect (neutral > invalid) primarily in the posterior inferior occipital and ventral and middle temporal regions contralateral to the target location. *Note*: left side of the brain in these 2D maps refers to the contralateral side to the target location. **b** 3D maps of the locations of two of the four ROIs that showed significant processing cost effects (*left*) and CDR value difference between the neutral and invalid trials in brain source volumes (*right*). Right side of the brain in these 3D maps refers to the contralateral side to the target location. These data are shown in an average 3-month-old brain template

for valid than neutral trials and the N1 amplitude was greater for valid than neutral and invalid trials in the short SOA condition. Source analysis showed greater current density amplitude (CDR value) for valid than neutral and invalid trials in the cortical sources of the P1 component located in the posterior occipital and ventral temporal regions contralateral to the targets. The processing cost effect was indicated by greater N1 ERP response and increased current density amplitude in the lateral inferior occipital and middle and superior temporal ROIs for neutral than invalid trials. We did not find an IOR effect in the latency of eye movements nor the ERP responses, i.e., in the long SOA condition, the RT to valid trials did not take longer than neutral trials and ERPs amplitudes for valid and neutral trials were non-significantly different.

The primary goal of the behavioral analysis was to examine the effect of cue-target validity on infant saccadic localization of peripheral targets in an improved spatial cueing paradigm with the continuous presentation paradigm. The validity and processing cost effects on infant localization latency (RT) found in the current study replicated previous findings (Hood 1993, 1995; Johnson et al. 1994; Johnson and Tucker 1996; Richards 2000a, 2001, 2005). These effects of covert orienting on localization latency suggest that a spatial cue can influence

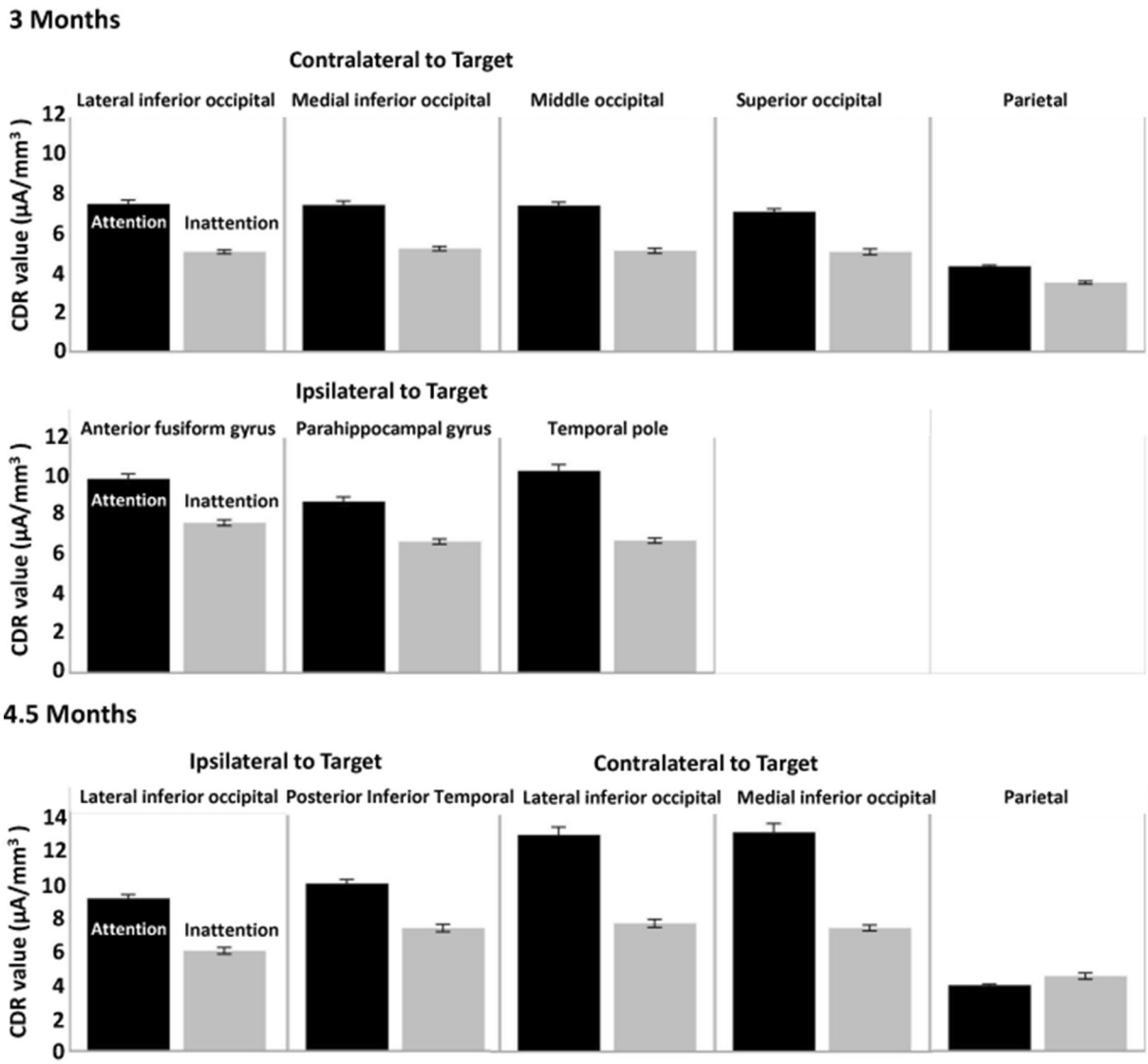


Fig. 12 Bar graphs of average CDR value (±10 ms) surrounding the P1 peak latency for sustained attention (*black*) and inattention (*silver*) periods in the ROIs that showed significant attention effect, separately for the 3- and 4-month-old. Note that the attention effect is widespread for the 3-month-old, but this effect became localized to the posterior occipital ROIs for the 4.5-month-old. The "Parietal" ROI is included for comparison

infant saccadic localization by causing a covert shift of attention to the peripheral cued location. We did not find an IOR effect on infants' localization latencies. This lack of effect was consistent with the majority of previous studies that failed to show the IOR effect in infants younger than 6 months of age (Hood 1993, 1995; Johnson and Tucker 1996; Varga et al. 2010). It should be noted that Richards (2000a) found an IOR effect in 4.5 months of age. However, the IOR effect in the 4.5-month-old group in that study was much smaller than the IOR effect in the 6-month-old group. It is plausible that the development of the mechanisms underlying IOR undergoes substantial changes in the first 6 months of life and does not become well established until the second half of the first year.

Effects of Infant Covert Orienting on Brain Activity in the Spatial Cueing Paradigm

The validity effect on infant ERP responses found in the current study suggests that covert orienting affected brain activity related to early visual processing. There was a larger response in the P1 and N1 ERP components in the short SOA condition for the valid than for the neutral (in the P1 and N1) and invalid (only in the N1) trials. This

Table 1 Summary of the major findings from the behavior, ERPs, and cortical source analyses

Analyses	Variables	Results	Figures
Behavior	RT	(i). The validity effect was found in the short SOA condition.	Figure 2
		(ii). The IOR effect was not found in the long SOA condition.	Figure 2
		(iii). No main effect or interaction involving age was found.	
ERPs	P1	(i). The validity effect: There is an an interaction between channel location, SOA condition, and cue-target validity, i.e. valid > neutral found in the contralateral posterior electrodes only for the short SOA condition.	Figure 4, 5 Figure 5 Figure 7
		(ii). No IOR effect: Valid P1 is not smaller than Neutral and invalid P1 with long SOA. However, valid P1 amplitude with long SOA < Valid P1 with short SOA.	
		(iii). The attention effect: Sustained attention elicited larger P1 in both SOA conditions for 3-month-old, but only in the short SOA condition for 4.5-month-old	
	N1	(i). The validity effect: There is an an interaction of channel type, SOA condition, and cue-target validity, i.e., valid > neutral and invalid in part of the ipsilateral and contralateral posterior virtual channels (I1, Iz, I2, O2, Oz) for the short SOA condition.	Figure 6a, b Figure 6a, c
		(ii). No IOR effect: Valid N1 is not smaller than Neutral and Invalid N1 with long SOA	
		(ii). The processing cost effect: neutral N1 amplitude > invalid N1 amplitude in both short (O1, I1, Iz) and long (O2) SOA conditions.	
Cortical source activity	CDR around the P1 peak	(i). The validity effect: Valid trials elicited greater current density amplitude (CDR value) than neutral and invalid trials in the posterior inferior occipital and ventral temporal ROIs contralateral to the target location in the short SOA condition.	Figure 8, 9 Figure 12
		(ii). The attention effect: Sustained attention elicited greater CDR value in both 3- and 4.5-month-old. This attention effect is widespread for 3-month-old but became more localized to the posterior ROIs by 4.5 months.	
	CDR around the N1 peak	(i). The processing cost effect: Neutral trials elicited greater CDR value than invalid trials in the posterior inferior occipital and inferior and middle-superior temporal ROIs contralateral to the target location.	Figure 10, 11

finding is in line with previous adult research that found larger P1 and N1 peaks in valid than invalid and neutral trials with a short SOA (e.g., Mangun and Hillyard 1991; Martinez et al. 1999). The lack of a greater P1 ERP response to the valid than the invalid trials was inconsistent with our hypothesis or the behavioral and N1 ERP findings. The small numbers of trials in part of the data might lead to insufficient power to detect this effect in the statistical analyses, although the P1 amplitude seemed to be larger for the valid than invalid trials in the contralateral channels (Fig. 4a). The validity effect on adult P1 and N1 components shows the facilitation of covert orienting on the cortical activity involved in the early visual processing of a stimulus (Hillyard et al. 1995). Our current finding suggests that the neural mechanism of this facilitation effect of covert orienting is already established in infants as young as 3–4.5 months.

One novel finding in this study is that the validity effect on infants' ERPs responses was only shown in the short SOA condition but not in the long SOA condition. This finding supports our current and previous behavioral reports that covert orienting facilitated saccadic localization of peripheral targets only with a short SOA in infants at 3–4.5 months (Richards 2000a, 2001). However, previous infant research did not find a significant effect of the SOA duration on the ERPs validity effect at these ages (Richards 2000a, 2005). We designed the current study to include separate groups of infants in the short and long SOA condition, and we employed a continuous presentation procedure with interesting stimuli. The current procedures resulted overall in more presentations per condition, and in more trials in which the EEG was successfully used. These changes likely enhanced the sensitivity of our procedures to detect the discrepancy between the effects of short and long SOAs on infants' ERP components in a spatial cueing task.

We also found a "cost of processing" effect in both of infants' behavior and the ERP responses. The eye movement latency to the targets was longer in the invalid trials than in the valid trials (Fig. 2), and there was a larger N1 ERP response to neutral than invalid trials (Fig. 6). This cost of processing implies that the shift of attention to an invalid location resulted in attenuated brain activation to the target, and corresponding longer latencies to the target. The processing cost effect on the N1 response found here is consistent with adult literature (e.g., Mangun and Hillyard 1991; Martinez et al. 1999).

The failure to find an IOR effect on infant behavior and ERP components in the long SOA condition suggests the

delayed development of IOR in infancy. To our knowledge, neural correlates of the IOR effect have not been shown during the first year of life (c.f. Richards 2000a, 2005). The absence of the IOR effect on infant brain activity might due to the protracted developmental trajectory of the mechanisms underlying the IOR effect compared to those underlying the validity effect (Colombo 2001). The current findings together with the existing literature suggest that the mechanisms underlying the IOR effect might not be well established until the second half of the first year (Amso and Johnson 2005, 2008; Hood 1995; Johnson and Tucker 1996; Richards 2001). The IOR effect is critical for distractor suppression in visual processing and attention relocation in visual search, which in turn plays a vital role in learning and memory encoding (Markant and Amso 2013; Markant et al. 2015b). Future research should investigate the development of the neural mechanisms underlying IOR in the first year of life, perhaps including infants at ages 6 months and older.

Cortical Source Analysis of ERP Responses During Spatial Cueing

Source analysis of the ERPs with the CDR technique provides converging evidence for the validity effect on infant brain activation. The validity effect on the current density amplitude surrounding the P1 ERP peak was shown in the contralateral medial and lateral inferior occipital lobe and posterior ventral temporal regions. The finding of distinct CDR value for valid trials in these ROIs in the striate and extrastriate cortex is consistent with previous adult (e.g., Fu et al. 2008; Hillyard et al. 1995; Martinez et al. 1999, 2001) and infant research (Richards 2005). Richards (2005) localized potential dipoles generating the P1 and N1 to the striate and extrastriate cortical areas. The engagement of the visual cortex in covert orienting implies specific mechanisms by which spatial attention modulates visual information processing in these brain regions (Martinez et al. 1999). The finding of the present study suggests that these mechanisms already exist in infants as young as 3 and 4.5 months of age.

The finding of the validity effect along ventral temporal areas (middle fusiform gyrus, posterior inferior temporal gyrus) was comparable to Richards (2005) that also localized the validity effect to dipoles in the temporal gyrus. These medial temporal regions along the "ventral pathway" are thought to be involved in pattern and object identification. Thus, the finding of the validity effect in the ventral temporal regions in addition to the occipital ROIs suggests that shifting attention to a valid location enhances not only visual processing of basic information (e.g., color, brightness) but also high-level visual processing (e.g., object identification). Recent studies have highlighted the interactions between infant spatial attention and social perception of faces (Hayden et al. 2012; Markant et al. 2015a, b), eye gaze (Farroni et al. 2004; Reid et al. 2004), and human biomechanical motion (Daum and Gredeback 2011, Rohlfing et al. 2012). For instance, Daum and Gredeback (2011) investigated how the observed goal-directed manual grasping actions modulated the infants' spatial attention in a modified spatial cueing paradigm. Targets presented at one side of the screen were either congruent or incongruent with the grasping direction of a hand presented at the center of the screen. Infants' gaze shift to the targets was found to be faster in the congruent than the incongruent condition, suggesting the effect of social perception of hand gesture on infant spatial attention (Daum and Gredeback 2011). Thus, the development of brain networks for spatial attention observed in the current study may contribute to the development of brain networks underlying social perception.

We failed to find the validity effect on the CDR amplitude around the N1 ERP peak latency. It is possible that distinct brain activation for the valid trials around the N1 ERP peak occurred in the boundaries of the "hypothesis-based' (i.e., a priori seed-selection) ROIs used in the current study. The validity effect in these regions might be counterbalanced by the averaging processing of the source activity for the entire ROI. Future research may try "data-driven" approaches (e.g., dipole analysis, independent component analysis) to examine the validity effect around the infant N1 ERP peak latency.

The processing cost effect on current density amplitude supports the idea that shift of attention to an invalid location may attenuate brain activity to visual target. Neutral trials in the short SOA condition caused greater current density amplitude surrounding the latency of the N1 peak in contrast to invalid trials. This effect was found in the ROIs contralateral to the targets along the extrastriate cortex, as well as in the middle and superior occipital and temporal regions. Invalid shift of attention may influence the same neural mechanisms underlying visual processing that are elevated in the validity effect. This processing cost effect has not been shown in previous infant research (e.g., Richards 2000a, b, 2005). The separation of the SOA conditions and the application of the continuous presentation paradigm in the current study should have contributed to the finding of the processing cost effect in infants as young as 3–4.5 months.

Effect of Sustained Attention on Early Visual Processing in Covert Orienting

The current study provides evidence for the effect of sustained attention on the early stages of visual processing in infant covert orienting. We found greater P1 ERP

amplitude and increased current density amplitude around the P1 peak latency in brain regions involved in early visual processing (Martinez et al. 1999; Richards 2005) during sustained attention than inattention. Sustained attention is hypothesized to increase general arousal (Reynolds and Richards 2007; Richards 2009, 2010) and information processing efficiency (Reynolds et al. 2010; Richards 2010; Richards et al. 2010). The current finding of enhanced brain activity to visual targets during sustained attention is consistent with the hypothesis that sustained attention enhances brain alertness, which in turn facilitates infant information processing. Prior studies have shown the effect of sustained attention on localization of peripheral stimulus, a process involving attention orienting and saccadic planning (Hunter and Richards 2003; Mallin and Richards 2012; Richards 2004). Combined with the current findings, it is plausible that the effect of sustained attention on the early visual processing in spatial orienting facilitates the response to a peripheral stimulus.

The interaction of sustained attention, SOA duration, and age on the P1 response suggests the intertwined development of sustained attention and covert orienting in early infancy. The P1 ERP amplitude was larger during sustained attention than inattention in both short and long SOA conditions for 3 months; however, this effect was only found in the short SOA condition for 4 months (Fig. 7). It is possible that the effects of sustained attention on the younger ages was due to extended exogenous attention orienting during this spatial cueing task, but a briefer time course of the attention effect on the older infants. There are substantial changes in infant sustained attention over this age range (see review by Colombo 2001; Richards 2009). Perhaps one change is the efficiency with sustained attention enhances the brain areas that affect covert orienting. We do not offer a specific mechanism for this effect.

Another age-related change is that the effect of sustained attention on brain activation becomes more focalized with age. We found increased current density amplitude around the P1 peak latency in both ventral temporal and occipital ROIs during sustained attention at 3 months. It is intriguing that the sustained attention effect becomes more restricted to occipital ROIs at 4.5 months (c.f. Fig. 10). It is plausible that the neural mechanisms involved in early visual processing and the spatial cueing task become more specialized with age. Further investigation on the interaction between the factors of cue-target validity, sustained attention, SOA duration, and age is needed to advance our understanding of the relation between development of sustained attention and covert orienting.

One limitation of the current study is that our experimental design did not provide enough trials to test the effects of the attention phases on the cue-target validity responses for the ERP analyses. The attention and cue-target validity factors must be within-subject manipulations. This design resulted in insufficient number of trials in individual cells (e.g., valid attention, valid inattention) for the analysis of the interaction of attention phase and cue-target validity. There is potential for an impact of the small number of trials in some participants on the ERPs and cortical source analyses. For example, the lack of the effect of infant sustained attention on the N1 ERP component was inconsistent with our hypothesis. This might due to some interaction of infant sustained attention and spatial cueing effects, which was not assessed in the current study. Our minimum number of artifact-free trials per condition was lower than the usual criterion (e.g., $\sim$10) used in infant ERP studies (DeBoer et al. 2007); but may be acceptable given good trials and our quantification procedure (cf. Stets and Reid 2011; Stets et al. 2012). In addition, we used a general linear models approach to the analyses in which individual trials rather than participant-based averages were used for the ERP analyses (Vossen et al. 2011). This allowed us to use an unweighted means approach to the ANOVA effects and would ameliorate the bias that may occur by participants with small numbers of trials, or by large standard errors for these participants. Given our number of subjects in the experiment and the unweighted general linear models approach, we believe that this issue is unlikely to affect the interpretation of our current significant results. A future design consideration that might increase the numbers of trials for the ERP analyses would be to employ only valid and neutral or valid and invalid trials in the presentation paradigm. Please refer to the Supplemental Information for further discussion of this issue.

Conclusion

The present study examined the effect of covert orienting and sustained attention on infant brain responses in a spatial cueing task. We found the validity and processing cost effects on cortical activation at 3–4.5 months, which are the ages when infants start to show distinct behavioral responses to different spatial cueing trials. The consistency between the behavioral changes and the cortical data indicates that the development of the neural mechanisms of covert orienting underpins the changes in infant behavioral responses in a spatial cueing task. The lack of an IOR effect at these ages suggests a delayed development of the neural mechanisms underlying IOR. A novel finding in this study was the effect of infant sustained attention on brain activation involved in covert orienting. The increase of brain arousal during infant sustained attention benefits the information processing at the earliest stages.

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Compliance with Ethical Standards

Conflict of Interests The authors have approved the manuscript and agree with its submission. These authors declare no conflict of interest.

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