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Visual Omitted Stimulus Potentials Are Not Retinotopic

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Significance Statement

Unexpectedly omitted stimuli do not elicit a retinotopic potential generated in the primary visual cortex, suggesting that they are processed in a different pathway from stimulus perception.

Declarations of interest: none

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Tomomi Ishida: Conceptualization, Methodology, Investigation, Data curation, Formal analysis, Visualization, Project administration, Writing–Original Draft

Hiroshi Nittono: Conceptualization, Methodology, Writing–Review & Editing

Abstract

Omitted stimulus potentials (OSPs) are elicited in response to the omission of expected stimuli and are thought to reflect prediction errors. If prediction errors are signaled in the sensory cortex, OSPs are expected to be generated in the sensory cortex. The present study investigated the involvement of the early visual cortex in the generation of OSPs by testing whether omitted visual stimuli elicit brain responses in a spatially specific manner. Checkerboard pattern stimuli were presented alternately in the upper and lower visual fields, and the stimuli were omitted in 10% of the trials. Event-related potentials were recorded from 33 participants. While a retinotopic C1 component was evoked by real visual stimuli, omitted stimuli did not produce any response reflecting retinotopy but did elicit a visual mismatch negativity, which was larger for omitted stimuli expected in the lower visual field than for those in the upper visual field. These results suggest that omitted visual stimuli are processed in a different pathway than actual stimuli.

Keywords: Event-related potential, Omission, C1, Visual mismatch negativity, Early visual cortex, Prediction

1. Introduction

The study of unexpected stimulus omission is effective for investigating the mechanism of prediction errors, which are discrepancies between predicted and actual sensory signals. This approach allows the examination of pure prediction error signals based on top-down predictions because it can isolate exogenous responses evoked by sensory stimuli from endogenous responses to the omission itself. Prediction errors are assumed to arise in the early sensory cortex and propagate to higher-order regions [1–4]. However, evidence of this theoretical process is still lacking. Omitted stimulus potentials (OSPs) are event-related potentials (ERPs) that reflect the processing of prediction errors when stimuli are unexpectedly omitted. If prediction errors are first signaled in the early sensory cortex, initial OSPs should be generated there. This study focuses on the visual modality to examine the involvement of the early sensory cortex in the generation of OSPs.

Previous studies of scalp-recorded ERPs have reported omission N1 (oN1; auditory: [5–7], visual: [8]), omission mismatch negativity (oMMN; [9–11]), and omission visual mismatch negativity (omission vMMN; [12]) when predicted stimuli are omitted. These OSPs have shorter peak latencies in the auditory modality than in the visual modality [8,13–15]. This latency difference is consistent with latency differences in sensory evoked potentials, which partly reflect differences in the speed of converting sensory input into electrical signals [16]. Therefore, at least some part of the OSPs are specific to each sensory modality.

However, the modality specificity of omission-related brain responses has not been clearly tested. In the visual modality, fMRI studies have shown that omission of expected visual stimuli elicits stimulus-property-specific activity in the primary visual

cortex, V1 (orientation: [17,18]; contrast: [18]; position: [19]). In contrast, ERP studies have not suggested that V1 contributes to OSPs. Czigler et al. [12] recorded ERPs for the omission of stimuli predicted in the lower visual field. The lower visual field was chosen because the vMMN was known to be larger for deviant stimuli presented in the lower visual field than in the upper visual field [20,21]; for a review, see [22]. The results showed that an occipital-predominant vMMN occurred at around 200 ms when a stimulus was unexpectedly omitted, but no significant electrical activity appeared at an earlier latency. Similarly, Stange et al. [23] found no retinotopic potentials when a circular grating pattern was unexpectedly omitted in either the upper or lower visual field. Thus, it is possible that OSPs are not primarily generated in V1.

In the visual modality, C1 is the earliest scalp-recorded evoked potential, appearing 50 ms after stimulus onset and peaking at 80–100 ms [24–26]. The most striking feature of the C1 is that it shows negative polarity when stimuli are presented in the upper visual field and positive polarity when stimuli are presented in the lower visual field. This polarity reversal can be explained by the cruciform model [25]. The orientations of the current dipoles are reversed between the upper and lower visual fields by mapping stimuli to the upper and lower banks of the calcarine fissure of the striate cortex ([27]; the negative pole is oriented superiorly for the upper visual field and inferiorly for the lower visual field). Given this retinotopic polarity reversal on the scalp, the main source of C1 is thought to be V1. Therefore, if a C1-like retinotopic potential occurs after omitted stimuli, it provides evidence of the involvement of the early visual cortex in the generation of OSPs.

In the present study, we recorded OSPs using a task that is optimal for C1 recording. Although Stange et al. [23] made a similar attempt to examine a C1-like

potential after unexpected stimulus omissions, their C1 amplitude was rather small (approximately −1 μV). To address this issue, we used a black-and-white checkerboard pattern to obtain a clear retinotopic C1. Specifically, the pattern was presented horizontally in the upper or lower visual field following the procedure of Kessler and Heinrich [28]. Considering that the polarity reversal point of C1 appears to be located 10°–20° below the horizontal meridian [26,27], we presented stimuli across the entire monitor in the area 6° above or below the horizontal meridian [29]. To control for the influence of evoked potentials from preceding stimuli on the OSP period, we used a predictable stimulus sequence in which stimuli were presented twice alternately in the same visual field (e.g., upper, upper, lower, lower). The stimuli were omitted with equal probability at each position. We had two hypotheses. If omitted stimuli elicit a C1-like potential with retinotopic features (i.e., negative for the upper visual field and positive for the lower visual field), this provides evidence that OSPs are generated in the early visual cortex. If not, there is no evidence of the involvement of the early visual cortex in the generation of OSPs. In addition, if the vMMN is elicited by stimulus omission, it should be larger when a stimulus is expected in the upper visual field than when a stimulus is expected in the lower visual field.

2. Methods

2.1. Participants

We used a sample size comparable to those used in recent OSP and C1 studies, for example, 29 participants in Ishida and Nittono [8] and 24 participants in Qin et al. [29]. Taking into account the possibility of data exclusion, 40 undergraduate and graduate students were recruited. All participants had self-reported normal vision. The study

protocol was approved by the Behavioral Research Ethics Committee of the Osaka University School of Human Sciences, Japan (HB023-043), and written informed consent was obtained from all participants. Participants received 2,500 Japanese yen as monetary compensation. After excluding data based on the exclusion criteria described below, data from 33 participants (23 males and 10 females, 18–34 years old, $M = 23.1$ years old) were used for the analysis. All were right-handed according to the Japanese version of the FLANDERS handedness questionnaire [30].

2.2. Stimuli

Fig. 1 shows a sample of the stimuli and a schematic representation of the experiment. A black-and-white checkerboard pattern was presented on a ViewPixx monitor (VPixx Technologies, Quebec, Canada; 120 Hz, 53.04 cm x 29.84 cm) at a viewing distance of 57 cm. The check size was 0.13° of the visual angle in both dimensions [28]. The checkerboard was presented binocularly to either the upper or lower visual field, 6° above or below the horizontal meridian, and flashed for 150 ms [29] with a 350-ms SOA. The contrast of the checkerboard was 95% [28], and a gray background (51.6 cd/m^2) was isoluminant with the mean luminance of the checkerboard. The luminance of the room was 220 lux. A small fixation dot (0.2°) [27] was presented at the center of the monitor. The timing of stimulus onset was measured by StimTrak with the Photo Sensor (Brain Products, Gilching, Germany) and compared to the timing of stimulus triggers recorded with an electroencephalogram (EEG). Since stimulus onset was delayed by an average of 10 ms from trigger onset, which is a standard specification of ViewPixx, all stimulus triggers were shifted backward by 10 ms during preprocessing.

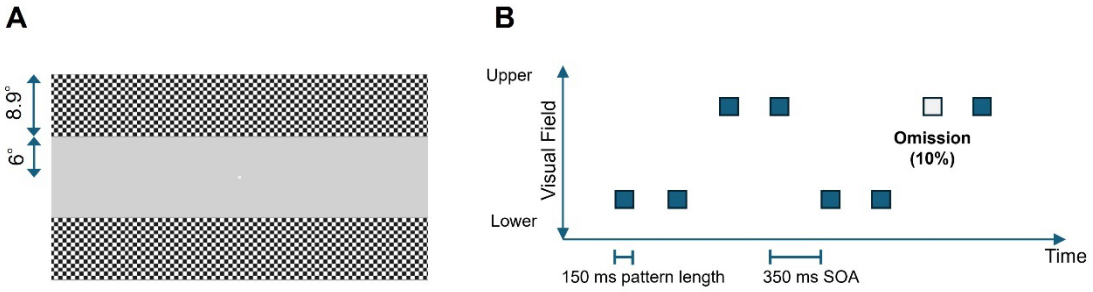

Fig. 1. Schematic representation of the experiment. Panel A: Size and location of the upper and lower checkerboard patterns with a central fixation point. Panel B: The checkerboard was flashed twice in the upper or lower visual fields. Stimuli were randomly omitted in 10% of the trials. Black squares represent stimulus presentation, and the white square represents stimulus omission.

2.3. Procedure

A chin rest was used to minimize head movement. The checkerboard stimuli were presented twice, alternating between the upper and lower visual fields. The experiment consisted of five blocks. Participants were instructed to rest their eyes for 15 seconds after every 200 trials (approximately 1.5 minutes), with a longer break after each block (1,000 trials, approximately 8 minutes). Omissions occurred with equal probability at each position ($p = 0.1$ overall), so the position of the preceding stimulus was counterbalanced. Stimuli were always presented in the first two trials of the blocks, immediately after the short breaks, and in the four trials following each omission trial. For each visual field, 5,000 trials were performed, with 2,250 stimulus trials and 250 omission trials. Participants were asked to maintain fixation on the central dot throughout the task. To ensure fixation, participants were also asked to respond to changes in the brightness of the fixation dot (catch trial) by pressing a button (approximately 0.7%, 1–2 trials per 200 trials). Trials with a late button press (more

than 1,000 ms after the onset of the catch trial) or no button press were considered error trials. Including electrode preparation and breaks between blocks, the entire experiment took approximately two hours.

2.4. Exclusion criteria

Seven participants were excluded from the analysis based on the following criteria: those who had 87 trials (35% of 250 trials) or more rejected omission trials due to ocular artifacts (n = 7) and those who had 70% or fewer hits and/or 15% or more false alarms on the catch trial (n = 3, all excluded by the first criterion). For the remaining participants (n = 33), the mean hit rate was 95.7%, and the false alarm rate was 0.04%.

2.5. EEG recording

EEG data were recorded from 64 sites using Ag-AgCl active electrodes (ActiveTwo system, BioSemi, Netherlands) at a sampling rate of 2048 Hz and a 0–400 Hz bandpass filter. A reference electrode (Common Mode Sense [CMS], active electrode) and a ground electrode (Driven Right Leg [DRL], passive electrode) were also placed on the scalp. Additional electrodes were placed on the left and right mastoids for offline re-reference. Vertical and horizontal electrooculograms were recorded from four additional electrodes placed lateral to the outer canthi of the eyes and above and below the right eye (UltraFlat active electrodes).

2.6. EEG data reduction

EEG data were analyzed using EEGLAB ([31], 2004; Version 2023.1) on MATLAB R2023b (The MathWorks Inc., Natick, MA). The data were resampled to 512 Hz, and a digital bandpass filter of 0.1–40 Hz was applied [29]. Ocular artifacts were removed using independent component analysis. Trials preceded by a catch trial or button press within 1,000 ms and trials followed by a catch trial or button press were excluded from the

analysis [27]. The epochs 100 ms before and 300 ms after stimulus onset were averaged separately for each condition (upper or lower visual field × stimulus presentation or omission). Epochs with a voltage difference greater than 100 μV were removed. The rejection rates were 13.3% for the upper visual field omission trials, 14.4% for the lower visual field omission trials, 16.6% for the upper visual field stimulus trials, and 16.2% for the lower visual field stimulus trials. Baseline correction was performed by subtracting the mean amplitude of the 100-ms prestimulus period from the amplitude of each point of the entire averaged waveform.

The peak of the C1 was identified at Pz [28] in an interval of 50–110 ms on the difference waveform between the ERPs for the upper and lower visual field stimuli. The mean amplitude of the 20-ms interval centered on this peak was used to test the C1 hypothesis. This interval was also used for the analysis of C1-like potentials in the omission trials. The peak of the omission vMMN was identified at Pz in an interval of 100–200 ms [12] on the waveform for the lower visual field omissions. The mean amplitude of the 40-ms interval centered on this peak was used to test the vMMN hypothesis. The same interval was used for the analysis of the upper visual field omission trials. The current dipoles of the C1 for the upper and lower visual field stimuli were estimated from the grand average waveforms using the EEGLAB DIPFIT plugin ([31]; Version 2023.1) on MATLAB R2023b (The MathWorks Inc., Natick, MA) at the C1 peak latency.

2.7. Statistical analysis

To examine whether the stimulus-evoked and omission-elicited potential showed retinotopic features (i.e., more negative for the upper visual field than for the lower visual field), a one-tailed paired *t*-test was performed on the mean amplitudes of the C1 interval

between the upper and lower visual fields separately for the omission and stimulus trials. To examine the presence of the vMMN, a one-tailed one-sample *t*-test against zero was performed. Additionally, a cluster-based permutation test implemented in the Fieldtrip toolbox [32] was performed to assess the presence of vMMN without a priori knowledge. A two-tailed one-sample *t*-test against zero was performed on a point-by-point basis over the 300 ms poststimulus period for all electrodes. The number of iterations was 5,000. The significance level was set at .05.

3. Results

Fig. 2 shows the waveforms and topographies for ERPs for the stimulus and omission trials. The peak latency of C1 was 110 ms, and the mean amplitude of the 100–120 ms interval was calculated. The C1 was significantly more negative for the upper visual field stimulus trials (M = −1.75 μV, SD = 1.32 μV) than for the lower visual field stimulus trials (M = 1.60 μV, SD = 1.63 μV), $t(32) = -8.11$, $p < .001$, $dz = -1.41$. The C1 was predominant in the parieto-occipital area for both the upper and lower visual field stimulus trials. Single current dipoles fitted to the C1 peak latency were located in the occipital cortex. The negative poles of the dipoles were oriented superiorly in the upper visual field and inferiorly in the lower visual field. In contrast, no clear electrical response was observed in the omission trials. The mean amplitudes of the C1 interval were not significantly more negative for the upper visual field (M = 0.21 μV, SD = 1.00 μV) than for the lower visual field (M = −0.11 μV, SD = 0.76 μV), $t(32) = 1.75$, $p = .955$, $dz = 0.30$.

For the vMMN, the peak latency was 160 ms, and the mean amplitude of 140–180 ms was calculated. The mean amplitudes were significantly negative from baseline for the lower visual field omission trials (M = −0.45 μV, SD = 0.68 μV), $t(32)$

= −3.80, $p < .001$, $dz = -0.66$, but not for the upper visual field omission trials ($M = 0.02$ μV, $SD = 0.87$ μV), $t(32) = 0.15$, $p = .561$, $dz = 0.03$. The omission vMMN was predominant in the occipital area. The cluster-based permutation test showed a significant negative potential at the occipital electrodes in an interval of 106–156 ms only for the lower visual field omission trials (see Supplementary Material).

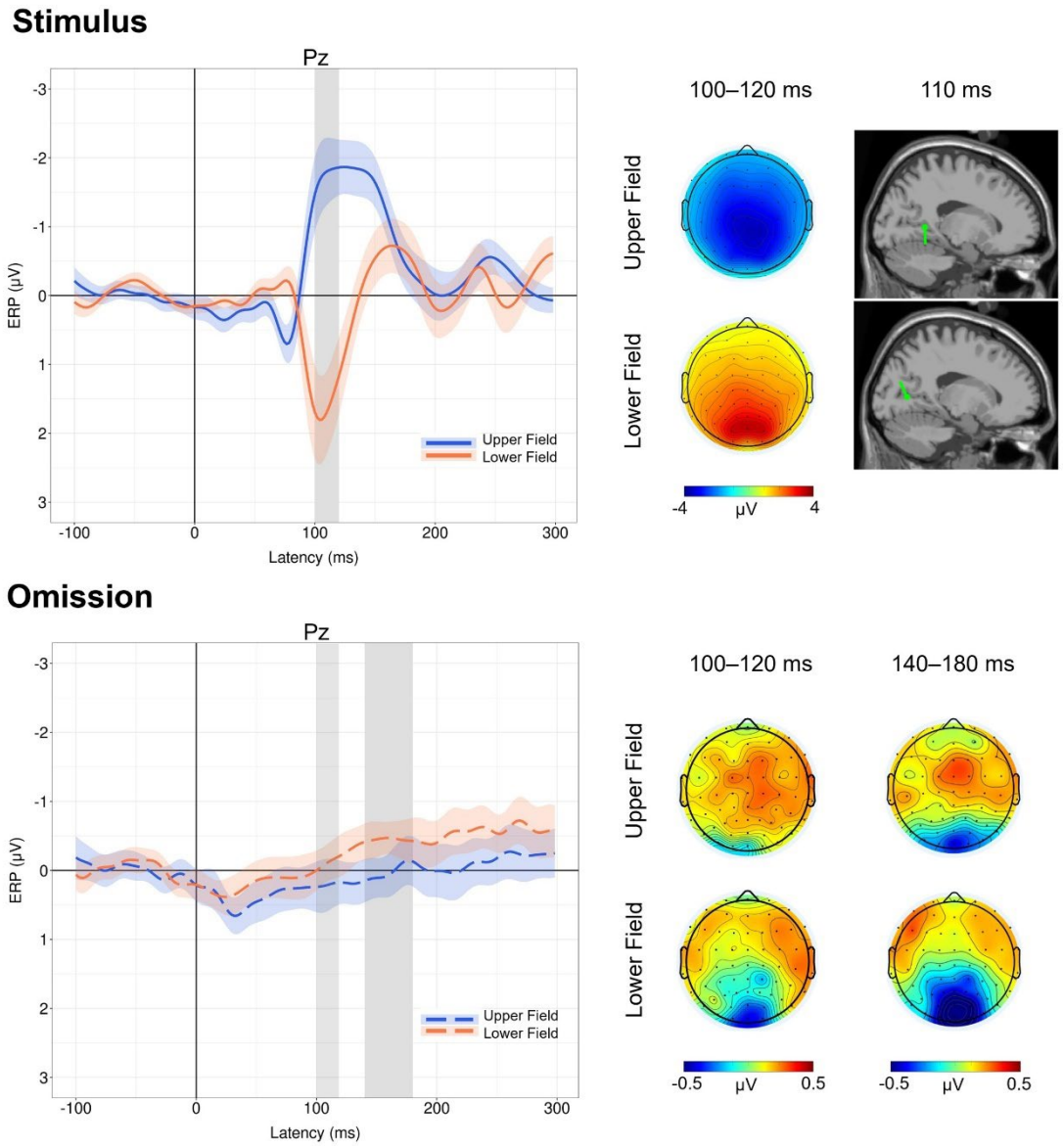

Fig. 2. Grand mean ERP waveforms, scalp topographies, and current dipole models for

each visual field. The upper panel shows the waveforms for stimulus trials: upper visual field (blue) and lower visual field (orange) at Pz with 95% confidence interval. The scalp topographies of the C1 interval (100–120 ms) and the single current dipole models at 110 ms (sagittal section) are also shown. The lower panel shows the waveforms for omission trials: upper visual field (blue) and lower visual field (orange) at Pz with 95% confidence interval. The scalp topographies of the C1 interval (100–120 ms) and the omission vMMN interval (140–180 ms) are also shown.

4. Discussion

In the present study, we compared ERP responses to unexpectedly omitted stimuli expected at different spatial locations to examine whether OSPs involve retinotopic responses. A clear C1, evoked activity in the early visual cortex primarily in V1, was observed for actual visual stimuli. This is evident from the waveforms, scalp topographies, and current dipoles. Checkerboard patterns in the upper visual field evoked a negative potential, and those in the lower visual field evoked a positive potential, peaking at 110 ms and predominant at the parieto-occipital sites. Vertically inverted dipoles were estimated for these potentials. In contrast, no C1-like response was observed in the omission trials. No polarity inversion was found in the C1 interval. Therefore, the present study failed to demonstrate the involvement of V1 in the generation of OSPs. Nevertheless, a negative occipital-predominant ERP component peaking at 160 ms appeared after stimulus omission, which was significantly negative from the baseline when a stimulus was expected in the lower visual field. This component is considered the omission vMMN in terms of scalp distribution and latency [12] and the lower visual field advantage [20,21].

The results of the present study suggest that prediction errors may be processed at early stages in a different region from stimulus perception. While fMRI studies have

shown that the omission of expected visual stimuli elicits V1 activity [17,18,19], activation is only observed in the deep layers, but not in the middle and superficial layers [18,33]. In the auditory modality, Dercksen et al. [7] reported discrepancies between oN1 and N1 topographies. They interpreted this result in light of findings in other species [34,35]; sound omissions activate only the nonlemniscal pathway, which is thought to feed prediction errors, whereas actual stimuli activate not only the nonlemniscal pathway but also the lemniscal pathway, which is thought to feed sound information. If omitted visual stimuli are also processed in a different pathway than actual stimuli, it is plausible that part of early visual cortex is not involved in the generation of OSPs.

However, it is also possible that although the present experimental paradigm was optimal for recording C1, it was not optimal for measuring omission responses. The relatively long SOA (350 ms) in this study may not be a sufficient temporal predictor. Previous OSP studies have shown that omission responses do not occur under nonattentive conditions when the SOA is longer than 150–200 ms, which is in the temporal window of integration (auditory: [9]; visual: [12]). Although the omission vMMN was found in this study, the amplitude was smaller than that found at shorter SOAs [12]. A replication of the present study with a shorter SOA could provide useful information. Alternatively, temporal prediction could be improved by self-stimulation. It is known that oN1 is discernible only when the timing of the omission is accurately predicted by the participant's button press (auditory: [5,7]; visual: [8]). If no retinotopic electrical response is elicited by the omission of self-generated stimuli, this would further support the present finding that different pathways are involved in actual and omitted visual stimuli.

In the present study, the C1 had a relatively long peak latency (110 ms), although it

showed typical retinotopic features. This is probably due to the combined effects of stimulus characteristics, such as luminance, contrast, and check size, which are known to significantly affect C1 properties [24,25,36–38].

The vMMN was significant only for omissions in the lower visual field. This result is consistent with previous studies showing that the vMMN is more pronounced when deviant stimuli are presented in the lower visual field than in the upper visual field [20,21]. The superiority of the lower visual field in vMMN generation reflects the superiority of automatic stimulus processing in the lower visual field, which has been shown in behavioral studies [39,40]. The present result suggests that stimulus omission processing may also be more effective in the lower visual field. Since the amplitude of the omission vMMN differed between the upper and lower visual fields, the spatial information of an omitted stimulus should be encoded before the vMMN is elicited. However, the sign of the earlier effect could not be determined in the present study.

In conclusion, the present study replicates previous findings that unexpected stimulus omissions do not elicit retinotopic responses. No evidence was obtained for the involvement of V1 in the generation of OSPs. Although there are some methodological limitations, the results suggest that unexpected omissions are processed in a different pathway from stimulus perception.

Ethics approval and consent to participate: This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Behavioral Research Ethics Committee of the Osaka University School of Human Sciences (HB023-043). Written informed consent for participation was obtained from all participants included in the study.

Declaration of generative AI in the writing process: The authors used DeepL Write (DeepL, Cologne, Germany) for English editing during the preparation of this work. After using this tool/service, the authors reviewed and edited the content as necessary and take full responsibility for the content of the publication.

Availability of data and materials: The datasets analyzed during the current study are publicly available at https://osf.io/8a23c/.

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Supplementary Material

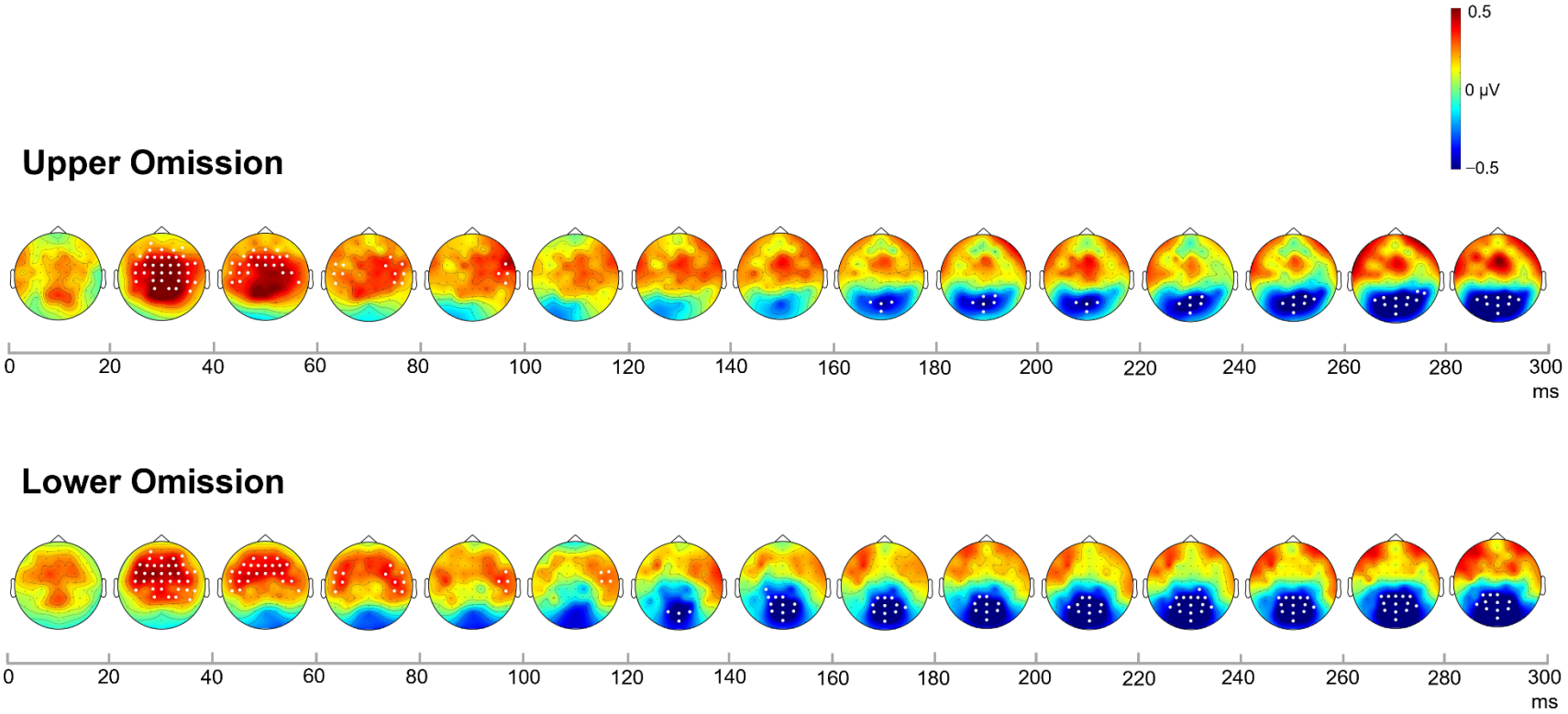

Fig. S1. The results of the cluster-based permutation test. The upper panel shows the scalp topographies for the upper visual field omission and the lower panel for the lower visual field omission. White stars in the topographies indicate electrodes where potentials were significantly different from zero according to the cluster-based permutation test ($p < .05$).