The parietal cortex and attentional modulations of activities of the visual cortex

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We recorded high density event-related brain potentials (ERPs) from a patient with focal left parietal damage in a covert visual orienting task requiring detection of targets in the attended or unattended hemifield. A positivity peaking at 120 ms (PI) to the left visual field stimuli was enlarged when attended than unattended and was localized to the right extrastirate cortex. However, spatial attention did not influence the ERPs to the right visual field stimuli.

The leftward cue elicited an enlarged PI relative to the rightward cue. The results suggest that human parietal cortex is critical for the attentional modulation of the neural activities in the extrastriate cortex associated with stimuli in the contralateral hemifield. *NeuroReport* 15:2275–2280 © 2004 Lippincott Williams & Wilkins.

Key words: Attention; Event-related potential; Extrastriate cortex; Parietal cortex; PI

INTRODUCTION

Brain lesion studies have shown that human parietal cortex plays a key role in guiding spatial attention. Parietal lesions in the right hemisphere usually induce neglect of stimuli in the contralateral hemispace [1–3] and may also lead to deficits of disengaging visual attention from precued locations [4,5]. Transcranial magnetic stimulation (TMS) of the parietal cortex that generates temporal inhibition of the parietal activity also produces contralateral neglect in healthy subjects [6,7]. Functional neuroimaging studies have shown strong evidence for the involvement of the parietal cortex in shift of spatial attention. The research recording regional blood flow using PET [8] or hemodynamic responses using fMRI [9–11] has found enhanced activations in bilateral parietal cortex in a variety of tasks manipulating spatial attention, suggesting that the parietal cortex is engaged in shift of attention in space.

However, to what degree the parietal cortex contributes to the attentional modulation of the neural activities of the visual cortex remains poorly understood. Prior event related potential (ERP) studies have shown that visual stimuli presented at one hemifield elicit larger amplitudes of a positive component wave (P1) when the stimuli are attended relative to unattended [12,13]. Because the P1 peaks at about 100 ms after sensory stimulation and has generators in the extrastriate cortex [12,14,15], it has been proposed that the neural activity of the extrastriate cortex underlying early sensory-perceptual processing is modulated by spatial attention. This proposal is strengthened by the fMRI studies that found evidence for the extrastriate activity being enhanced by spatial attention [16–18]. However, there has been little evidence for the involvement of the parietal cortex in the execution of the attentional modulation of the extrastriate activities. Han *et al.* recently recorded hemodynamic responses using fMRI from a patient with focal left parietal lesion while the patient was asked to detect targets briefly presented in the left visual field (LVF) or the right visual field (RVF) in separate blocks of trials [19]. Relative to a passive viewing condition, sustained attention to the LVF induced stronger activation in the patient's right extrastriate cortex. However, no activation in the left extrastriate cortex was observed in association with attention to the RVF. Several pieces of evidence suggest that the patient's left extrastriate was intact. First, the patient detected all the RVF targets; second, the anatomical image showed that the lesion was limited to the left superior parietal lobe; third, the fMRI results showed that the left extrastriate cortex was activated by the RVF stimuli, similar to the activation in the right extrastriate cortex induced by the LVF stimuli. The results indicate that the neural activity of the left extrastriate cortex was not modulated by spatial attention even though this brain region was intact.

We examined further how the patient's left parietal damage influenced the attentional modulation of the neural activities of the extrastriate cortex by recording high density ERPs. Unlike the paradigm used in our previous fMRI study that examined the effect of sustained spatial attention guided by instructions, the present experiment employed a precueing paradigm that shifted subjects' attention trial by trial. We examined if the early ERP components arising from the extrastriate cortex are influenced by spatial attention that shifts between the LVF and RVF dynamically under the circumstance that the left parietal cortex was damaged.
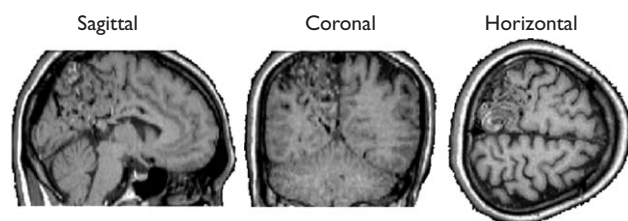
Fig. I. MR images showing the angiomas in the patient's brain. The left superior posterior parietal lobe and the posterior part of the cingulate cortex were occupied by angiomas.

MATERIALS AND METHODS

Subjects: Patient QC was a 17-year-old right handed male, who was a student in a Chinese high school in September, 2002, when he suffered epileptic seizures twice. Anatomical MR scan revealed angiomas in his left superior posterior parietal lobe, extending into part of the posterior cingulate cortex of the left hemisphere (Fig. 1). The left striate and extrastriate cortices were intact. Neurological examination disclosed no movement problem. His ability to read Chinese characters and sentences appeared unaffected. He had a normal visual field and there was no indication of neglect or extinction on confrontation testing. The visual acuity of the right eye (20/20) was better than that of the left eye (5/20). All the tests reported here were conducted in October 2002. Six healthy subjects (2 male, ages between 18–19 years) participated in the study as controls. All the controls were right handed, had normal or corrected-to-normal vision. Informed consent was obtained from both the patient and controls according to the guidelines of Department of Psychology, Peking University.

Stimuli and procedure: Stimuli were square-wave modulated black and white checkerboard patterns, circular in overall form and displayed on a grey background. The checks were aligned with the horizontal and vertical axes of the screen. An oddball paradigm was used in which circular checkerboards appeared randomly to the left or right of the fixation that was located at the center of a computer monitor. Subjects responded to a small percentage of small checkerboard patterns (targets) by a button press while they ignored large checkerboard patterns (nontargets). The center of target and nontarget stimuli was equally distant from the fixation (4.7°). The stimuli appeared at either the location indicated by precues (valid condition) or the location opposite to the cue direction (invalid condition). Target and nontarget stimuli subtended visual angles of $1.7 \times 1.7°$ and $3.0 \times 3.0°$, respectively, at a viewing distance of 120 cm. Each of the black or white checks subtended a visual angle of $0.38 \times 0.38°$ in both target and nontarget stimuli. Each trial began with the presentation of small black dot ($0.17 \times 0.17°$) in the center of the screen serving as fixation. The duration of the fixation varied randomly between 1000 and 1500 ms. The fixation was then overlapped with an arrow ($0.56 \times 0.56°$) pointing to the LVF or RVF, serving as a cue to direct the subjects' attention. 1000 ms later the checkerboard stimuli appeared in the LVF or the RVF. The checkerboard stimuli lasted for 100 ms and then disappeared with the cues. While maintaining fixation on the central dot or arrow, the patient and the controls were required to detect the occurrence of target stimuli that appeared on 20% of the

trials by pressing a button with the right index finger. 70% of target and nontarget stimuli appeared in the hemifield to which the arrow pointed, whereas 30% of target and nontarget stimuli appeared in the hemifield opposite to the direction of the arrow. There were 50 practice trials followed by 1000 trials in 10 blocks.

ERP data recording and analysis: The EEG was recorded from 120 scalp electrodes, which were labeled with numbers from 1 to 120. Electrodes 59–71 were arranged along the midline of the skull. Other electrodes were located approximately symmetrically over the two hemispheres. The skin resistance of each electrode was <5 kΩ. The recording from an electrode at the right mastoid was used as reference. Eye blinks and vertical eye movement were monitored with electrodes located below the right eyes. The horizontal electro-oculogram was recorded from electrodes placed 1.5 cm lateral to the left and right external canthi. The EEG was amplified (band pass 0.15–70 Hz) and digitized at a sampling rate of 250 Hz. The ERPs in each stimulus condition were averaged separately off-line with averaging epochs beginning 200 ms before stimulus onset and continuing for 1000 ms. Trials contaminated by eye blinks, eye movements, or muscle potentials exceeding $\pm75\,\mu V$ at any electrode were excluded from the average. ERPs and difference waves were measured with respect to the mean voltage during the 200 ms pre-stimulus interval. Mean voltages of ERPs were obtained from posterior electrodes at successive 20 ms intervals starting 60 ms after stimulus onset and continuing until 600 ms post-stimulus. Similar to the previous study [20], to obtain repeated measures in the patient, mean amplitudes in different conditions were calculated for each of the 10 blocks of trials at electrodes over the occipital, temporal, and parietal electrodes. To obtain repeated measures in the controls, mean amplitudes in different conditions were calculated from ERPs of each subject that were obtained by averaging the results of the 10 blocks of trials. The mean amplitudes from the patient and controls were then respectively subjected to ANOVAs with cue validity (valid *vs* invalid), visual field (left *vs* right), and hemisphere (electrodes over the left or right hemispheres) as independent variables.

Current source density was calculated using low resolution electromagnetic tomography (LORETA) [21] to localize the generators of ERPs in specific time window. The position of each electrode was measured with a probe for sensing the 3D position of the probe tip with respect to a magnetic field source in the head support. The digitized fiducial landmarks corresponding to the electrode coordinates were coregistered with fiducial landmarks identified on whole-head MR. The current source density was then calculated and the maximum of the results was mapped onto the 3D boundary element head model constructed from the patient's MR images to estimate locations of sources with respect to brain anatomy. The 3-dimensional coordinates of the maximum of current source density in the patient's head model were transformed to the coordinates of Talairach and Tournoux [22] by marking the anterior and posterior commissures on the patient's MR scan. For the controls the mean scalp location of each electrode was estimated by averaging each electrode location across all subjects and was used for current-source-density analysis. The 3D realistic-head boundary-element model

was constructed based on the MR images from a randomly selected subject. The CURRY program (Neurosoft. Inc.) was used for these analyses.

MR image acquisition: Brain imaging was performed using a 1.5 T GE Signa MR scanner with a custom head coil. The patient's anatomical images were obtained with a standard 3D T1-weighted sequence (resulting in a 256 × 66 × 256 matrix with 0.938 × 2.0 × 0.938-mm spatial resolution, TR=585 ms, TE=minimum).

RESULTS

Patient: The patient responded correctly to 72% and 76% of the LVF and RVF targets. Reaction times in the valid and invalid conditions were 521 *vs* 547 ms for LVF stimuli and 534 *vs* 561 ms for the RVF stimuli, respectively. Figure 2a shows ERPs to nontarget stimuli recorded at occipito-temporal electrodes from the patient. The ERPs were characterized with a positive wave peaking between 100 and 140 ms (P1), which was followed by a negativity peaking between 140 and 220 ms (N1). There were also a long-latency negativity between 260 and 320 ms (N2) and a positivity between 350 and 550 ms (P3). The main effects of cue validity ($F_{(1,9)}=5.39$, $p<0.04$) and hemisphere ($F_{(1,9)}=9.95$, $p<0.01$) were significant in a time window of 100–140 ms, corresponding to a occipital positive wave (P1) at the occipito-temporal electrodes. As there was also a reliable interaction of cue validity × visual field × hemisphere ($F_{(1,9)}=5.66$, $p<0.04$), separate analyses were conducted for the LVF and RVF stimuli, respectively.

For the LVF stimuli the main effect of hemisphere was significant ($F_{(1,9)}=15.4$, $p<0.004$), indicating that the P1 amplitude was larger at electrodes over the right than left hemispheres. The main effect of cue validity was also significant ($F_{(1,9)}=7.88$, $p<0.02$) due to the fact that stimuli elicited larger P1 amplitudes in the valid relative to invalid conditions. The difference between valid and invalid conditions was larger at electrodes over the right than left hemispheres, producing a significant interaction of cue validity × hemisphere ($F_{(1,9)}=10.6$, $p<0.01$). Current source density analyses showed that electrical activities between 100 and 120 ms had generators in the right extrastriate cortex (BA 18; Fig. 2b). The Talairach coordinates of the extrastriate activities were 9.6, −80.1, −1.2 in the valid and 8.1, −79.6, −1.3 in the invalid condition. The ANOVAs also revealed that the amplitudes of the P3 had larger amplitudes over the right than left occipital areas between 400 and 460 ms ($F_{(1,9)}=0.82$, $p<0.04$). For the RVF stimuli, however, no significant cue validity effect was observed in any time window. Current source density analyses did not come to a good solution because of a low signal-to-noise ratio. There was only a reliable effect of hemisphere between 360 and 440 ms over the parietal regions ($F_{(1,9)}=12.5$, $p<0.01$), suggesting that the amplitudes of the P3 were larger over the right relative to left hemispheres.

To study the brain activities elicited by attentional cues, ERP waveforms for the left and right pointing cues were calculated (Fig. 3). The cue-related potentials were characterized with a positivity peaking between 100 and 160 ms (P1) followed by a negativity peaking between 170 and 240 ms (N1) at occipito-temporal electrodes. The P1 was of larger amplitudes for the left than right pointing cues ($F_{(1,9)}=9.13$, $p<0.02$). In contrast, the N1 was of larger amplitude for the right than left pointing cues ($F_{(1,9)}=9.64$, $p<0.02$). In addition, the N1 showed larger amplitudes over the right than left hemispheres ($F_{(1,9)}=14.57$, $p<0.004$).

Controls: The controls correctly responded to 99% of the LVF and RVF targets. RTs in the valid condition (LVF:
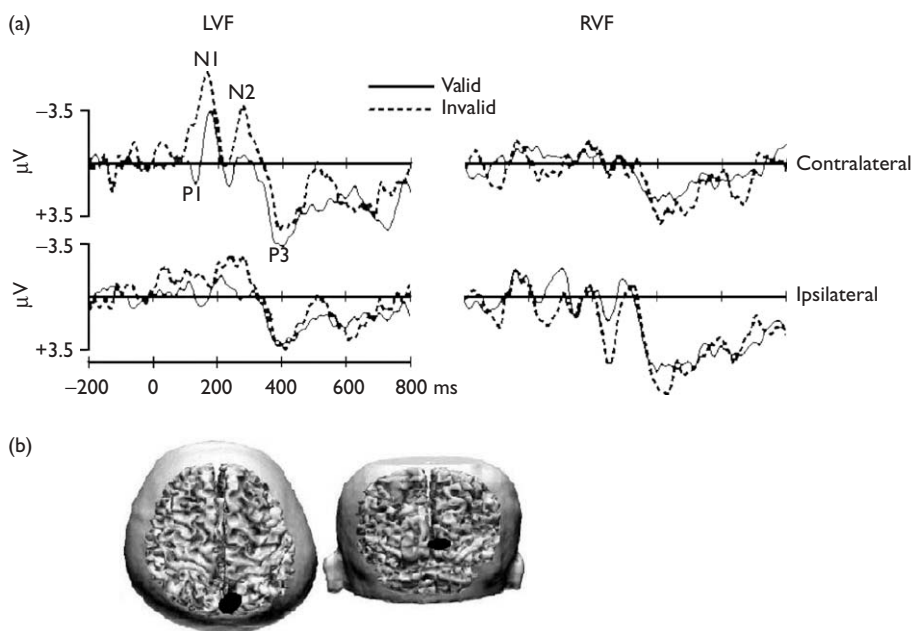

Fig. 2. (a) The ERPs recorded from the patient in association with the LVF and RVF nontarget stimuli in the valid and invalid conditions at occipito-temporal electrodes. (b) The current source density of the P1 wave between 100 and 120 ms elicited by the LVF stimuli in the valid attention condition. The maximum current source density is shown in the patient realistic-head model built based on his MR images.
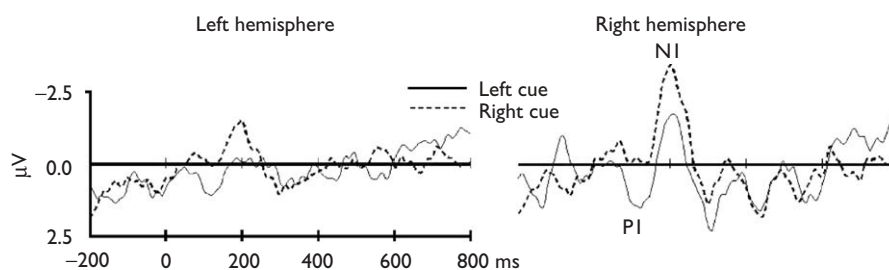
Fig. 3. The ERPs recorded from the patient in association with the attention cues at occipito-temporal electrodes.
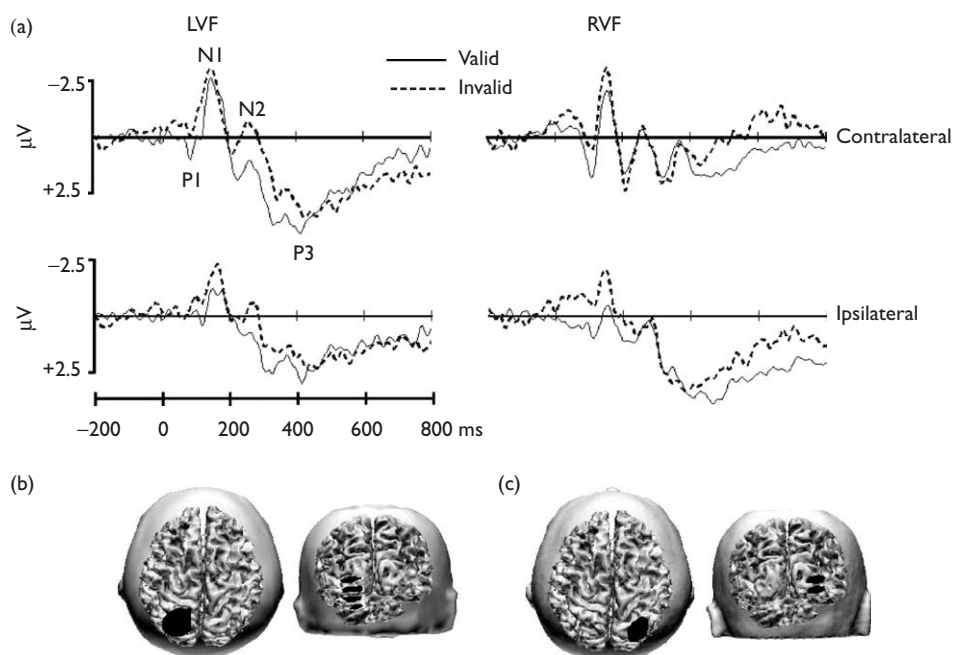

Fig. 4. **(a)** ERPs recorded from the controls in association with the LVF and RVF nontarget stimuli in the valid and invalid conditions at occipito-temporal electrodes. **(b)** The current source density of the P1 wave between 80 and 100 ms elicited by the RVF stimuli in the valid condition. The maximum current source density is shown in a realistic-head model built based on the MR images of a representative subject. **(c)** The current source density of the P1 wave between 80 and 100 ms elicited by the LVF stimuli in the valid condition.

463 ms, RVF: 448 ms) and were faster than those in the invalid condition (LVF: 496 ms, RVF: 475 ms; $F(1,5)=16.3$, $p<0.01$). Figure 4a shows ERPs recorded at lateral occipito-temporal electrodes from the controls. ANOVAs showed a significant effect of cue validity at occipital-temporal electrodes at 100–140 ms ($F(1,5)=14.2$, $p<0.02$), indicating that the P1 was of larger amplitudes in the valid than invalid conditions. However, there was no reliable interaction of cue validity × visual field ($F<1$), suggesting that the P1 effect did not differ between the LVF and RVF stimuli. The mean amplitude of the N1 wave between 140 and 160 ms was larger in the invalid compared with valid conditions ($F(1,5)=9.10$, $p<0.03$). Current source analysis of the P1 component showed maximum activities in the extrastriate cortex contralateral to stimulated hemifields for both LVF and RVF stimuli (Fig. 4b,c). The Talairach coordinates of the extrastriate activities were −27.2, −70.7, −1.2 (RVF, valid); −26.9, −29.7, −9.9 (RVF, invalid); 23.0, −72.0, −2.3 (LVF, valid); 15.4, −72.2, −0.3 (LVF, invalid).

Figure 5 shows cue-related potentials recorded from the controls, which were characterized by a positivity at 80–130 ms (P1) and a following negativity at 140–200 ms (N1). ANOVAs did not show significant difference in mean ERP amplitudes between the left and right pointing cues in any time window from 80 to 200 ms after stimulus onset ($F<1$).

DISCUSSION

This study examined the role of human parietal cortex in attentional modulation of the neural activities of the visual cortex by comparing the ERPs recorded from a patient with focal left parietal lesion and the healthy controls. The controls responded faster to the attended than to the unattended targets, indicating a cue validity effect on behavioral responses, consistent with previous observations [23]. The ERP data from the controls showed that an early ERP component (i.e., the P1) was enlarged to the stimuli at
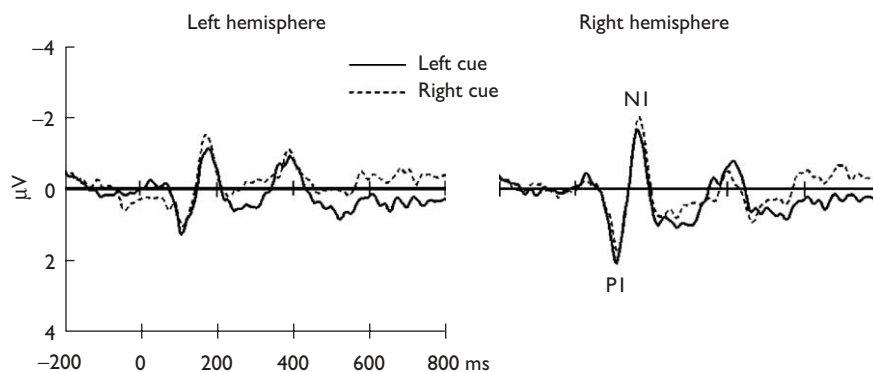
Fig. 5. The ERPs recorded from the controls in association with the attention cues at occipito-temporal electrodes.

cued relative to uncued locations. Moreover, the P1 effect did not differ between LVF and RVF stimuli. The current source density analysis revealed the generators of the P1 component in the extrastriate cortex contralateral to the stimulated hemifield. The ERP results are in line with previous work that has shown enhanced neural activities in the extrastriate cortex associated with attended relative to unattended stimuli (e.g., indexed by the P1 effect) [12–14].

The patient also responded faster in the valid than invalid conditions and this cue validity effect did not differ between the LVF and RVF stimuli. This is different from the results of a prior study with this patient that used a high percentage of targets and found the cue validity effect on RTs only to the LVF stimuli [19]. Thus low target probability might weaken the effect of parietal lesions on the behavioral responses. However, the patient's ERP results showed asymmetric P1 attention effects associated with the LVF and RVF stimuli. Similar to the results of the controls, the P1 to the LVF stimuli was enhanced when the LVF was attended than when unattended. The current source density analysis suggests that the neural activity between 100 and 120 ms after stimulus onset had a generator in the right extrastriate cortex. The results provide evidence that the patient's right extrastriate activity was modulated by covert spatial attention induced by precues. Nevertheless, neither the early nor late ERP components to the RVF stimuli were influenced by cue validity, contrasting with those to the LVF stimuli.

One possibility is that the left parietal cortex is necessary for the top-down attentional modulation of the extrastriate activity associated with the RVF stimuli. The left parietal damage deteriorated the attentional enhancement of the visual activities in the valid condition and thus resulted in equal strength of the extrastriate activity in the valid and invalid conditions. Alternatively, the absence of the attentional modulation of the P1 was due to the fact that the left extrastriate activity in association with the sensory processing of the RVF stimuli was decreased as a consequence of lacking the feedback from the left parietal cortex. Indeed, as one may notice, the P1 and N1 waves to the RVF stimuli were much less clear than those to the LVF stimuli, which unlikely resulted from damage of the left visual cortex since our prior anatomical examination and the fMRI results showed that the patient's left visual cortex was intact [19]. Nevertheless, the second account is a stronger version of the first one, suggesting that the sensory responses of the visual

cortex are eliminated after the parietal damage. Whatever the case, the lack of attentional modulation of ERPs to the RVF stimuli implies that the left parietal cortex is engaged, at least to a certain degree, in the attentional modulation of the activities of the left extrastriate cortex. However, the left parietal cortex might contribute little to the attentional modulation of the activities of the right extrastriate cortex since the attentional modulations of the ERPs to the LVF stimuli were similar to the results of healthy subjects [12–14]. In addition, because the lack of attentional modulation of ERPs to the RVF stimuli was observed when the right parietal cortex was intact, it further implies that the right parietal cortex may not play a critical role in the attentional modulation of the activities of the left visual cortex. Thus it may be conjectured that generally the parietal cortex in each hemisphere executes spatial attention gain control of activities of the ipsilateral visual cortex. In addition, the current ERP results are consistent with those of our previous fMRI study [19], which, however, employed a sustained attention paradigm and showed lack of attentional modulation of the left extrastriate activity after the left parietal damage. Therefore it appears that the parietal cortex is involved in attentional modulation of the early sensory-perceptual processing in the visual cortex regardless of whether spatial attention is focused on one hemifield or shifts between two hemifields dynamically.

One may notice the discrepancy between the current results and previous findings in that previous studies suggest that the right parietal cortex is engaged in directing attention to the both the LVF and RVF in normal subjects [8] whereas the patient reported here showed deficits of orienting of attention to the RVF although his right parietal cortex was intact. The right parietal cortex is possibly engaged in controlling rightward shifts of attention through the left parietal cortex. When the left parietal cortex is damaged, the role of the right parietal cortex in attentional modulation of left occipital activities related to the RVF stimuli is then weakened.

Similar to the previous study [24], we found that directions of central cues used for guiding spatial attention did not influence the amplitudes of the cue related potentials (such as P1 and N1) in normal subjects. However, the cue related potentials recorded from the patient's occipito-temporal regions were influenced by the cue directions. The left cue elicited an enlarged P1 over the occipital cortex relative to the right cue whereas the

following N1 was enlarged by the right cue. Since the P1 may reflect a facilitation of early sensory-perceptual processing of visual stimuli whereas the N1 may represent the orienting of attention to a task-relevant stimuli [25], the patient's results suggest that the left parietal damage led to both impairment of the representation of rightward information at an early stage of visual processing and difficulty of directing attention to the contralateral hemifield. It is possible that the deficits of cue-related processing resulting from the left parietal damage also contributed to the lack of attentional modulations of the extrastriate activities related to the RVF stimuli.

CONCLUSION

The current study provides ERP evidence that focal left parietal damage degraded the attentional modulations of the left extrastriate activities. The effect was confirmed under the condition that the left visual cortex was intact. The findings support the proposition that the parietal cortex plays an important role in human attention networks to modulate the neural activities of the visual cortex.

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