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The role of human parietal cortex in attention networks

Shihui Han,[1,2] Yi Jiang,[1] Hua Gu,[3] Hengyi Rao,[1] Lihua Mao,[1] Yong Cui[3] and Renyou Zhai[3]

[1]*Department of Psychology, Centre for Brain and Cognitive Sciences, Peking University,* [2]*Key Laboratory of Mental Health, Institute of Psychology, Chinese Academy of Sciences, and* [3]*Department of Radiology, Beijing Chaoyang Hospital, Beijing, People's Republic of China*

Correspondence to: Shihui Han PhD, Department of Psychology, Peking University, 5 Yiheyuan Road, Beijing 100871, People's Republic of China
E-mail: shan@pku.edu.cn

Summary

The parietal cortex has been proposed as part of the neural network for guiding spatial attention. However, it is unclear to what degree the parietal cortex contributes to the attentional modulations of activities of the visual cortex and the engagement of the frontal cortex in the attention network. We recorded behavioural performance and haemodynamic responses using functional MRI from a patient with focal left parietal damage in covert visual orienting tasks requiring detection of targets at the attended or unattended locations. While the patient's reaction times to left visual field stimuli were speeded by valid relative to invalid cues, attention to LVF stimuli was associated with enhanced activities in the right extrastriate cortex, right parietal and cingulate cortices, and bilateral frontal cortices. However, the patient's behavioural and neural responses to right visual field stimuli were not influenced by cue validity. The results are discussed in terms of the role of human parietal cortex in the neural network underlying voluntary attentional control.

Keywords: cue validity; ERP; fMRI; parietal cortex; spatial attention

Abbreviations: BA = Brodmann area; ERP = event related potential; fMRI = functional MRI; LVF = left visual field; RT = reaction time; RVF = right visual field

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Introduction

Human observers can shift visual attention to specific locations in the visual field to facilitate processing of the stimuli at attended locations. There has been converging evidence that several brain areas are engaged in guiding spatial attention. Studies of neurological patients showed that damage of the right parietal or frontal cortex may result in neglect of stimuli in the left hemispace (Heilman, 1979; Damasio *et al*., 1980; Mesulam, 1981; Bisiach *et al*., 1984). In a covert visual orienting task in which subjects respond to targets at cued or uncued locations without overt saccadic eye movements (Posner, 1980), lesions of the parietal lobe lead to deficits of disengaging visual attention from cued locations (Posner *et al*., 1984, 1987). Recent transcranial magnetic stimulation studies have also shown that temporary lesions of the parietal cortex generated by repeated transcranial magnetic stimulation impaired the detection of visual stimuli contralateral to the stimulated hemisphere (Hilgetag *et al*., 2001; Müri *et al*., 2002) or induced contralateral neglect in healthy subjects (Fierro *et al*., 2000; Bjoertomt *et al*., 2002). These findings suggest that the parietal cortex plays an important role in shifts of attention in space.

Functional neuroimaging studies of normal subjects have frequently observed enhanced activations in the parietal, frontal, and cingulate areas in association with spatial attention. For example, increased regional blood flow measured with PET was visualized in superior parietal and frontal cortices in a task of shifting attention relative to a central detection task (Corbetta *et al*., 1993). Activations were observed in the right parietal cortex when attending to the left visual field (LVF) and in bilateral parietal cortices when attending to the right visual field (RVF). Functional magnetic resonance imaging (fMRI) studies also found enhanced haemodynamic responses in bilateral frontal cortices, parietal cortices and the cingulate cortex in association with visual attention in a variety of covert visual orienting tasks (Nobre *et al*., 1997; Gitelman *et al*., 1999; Kim *et al*., 1999; Yantis *et al*., 2002). Activations in these cortical areas may occur during shifts of attention but before targets are displayed

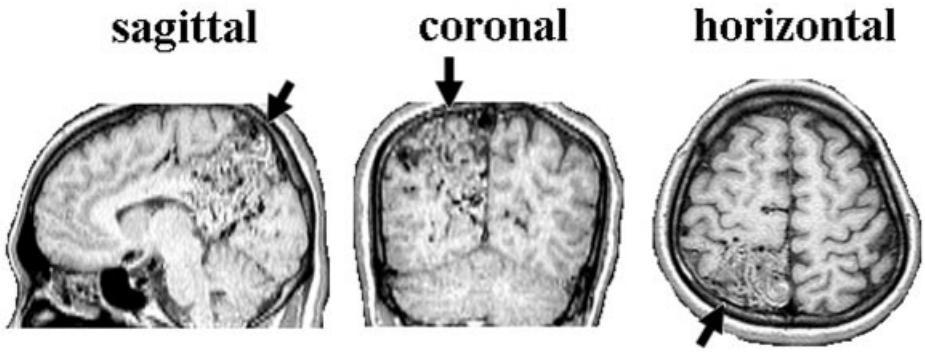

Fig. 1 MRI scans showing the patient's lesion indicated by black arrows. His left superior posterior parietal lobe was occupied by angiomas as indicated by the arrows.

(Corbetta *et al*., 2000; Hopfinger *et al*., 2000; Kastner *et al*., 1999), suggesting that engagement of these areas in guiding spatial attention may take place independently of target processing. Similar patterns of activations were observed in visual search tasks that are assumed to involve spatial attention (Leonards *et al*., 2000). Taken together, the results indicate that a large-scale distributed network involving frontal, parietal and cingulate areas is engaged in guiding visual spatial attention.

On the basis of the prior work, neural network models of attention have been established in which the parietal lobe is proposed to either provide an internal perceptual map of the external word (Mesulam, 1981, 1990) or bring attention to a location in space (Posner and Petersen, 1990; for review, see Corbetta, 1998; Kanwisher and Wojciulik, 2000). However, some issues about the role of the parietal cortex in the attention network remain unresolved. For example, event related potential (ERP) studies have shown that stimuli presented at attended locations elicit a positive wave (P1) with larger amplitudes than do stimuli presented at unattended locations (Mangun and Hillyard, 1991; Gomez Gonzalez *et al*., 1994; Martinez *et al*., 2001). The P1 peaks at ~100 ms after sensory stimulation and has generators in the extrastriate cortex (Gomez Gonzalez *et al*., 1994; Heinze *et al*., 1994; Clark and Hillyard, 1996). Recent fMRI studies also found stronger activities in human striate and extrastriate cortices elicited by attended relative to unattended stimuli (Tootell *et al*., 1998; Gandhi *et al*., 1999; Martinez *et al*., 1999). These observations indicate that activities of the early visual cortex are modulated by spatial attention in a manner of gain control. However, it is undefined to what extent the parietal cortex contributes to the attentional modulation of the occipital activities. In addition, although neuroimaging studies have shown that the frontal cortex and the parietal cortex usually co-activate in spatial attention tasks (Gitelman *et al*., 1999; Hopfinger *et al*., 2000), the relation between the frontal and parietal cortices in the attention network is poorly understood. Do the frontal and parietal lobes work independently or do they depend on each other in guiding spatial attention?

To address these issues, we recorded behavioural performance and haemodynamic responses using fMRI from a patient with focal left parietal damage in association with covert visual orienting tasks. We compared the patient's neural correlates of attention to the LVF or RVF stimuli. Since attention to each hemifield is dominated by the contralateral hemisphere (Luck *et al*., 1989; Corbetta *et al*., 1993) and the patient's right parietal cortex was intact, we would expect, when attending to the LVF stimuli, activations of an attention network involving the parietal and frontal cortex, and attentional modulations of occipital activities showing a similar pattern to those observed in normal subjects. The fMRI results in the condition of attending to the RVF stimuli, however, gave us a chance to examine to what extent the modulation of occipital activities by spatial attention and the engagement of the frontal cortex in the attention network depend upon the normal function of the left parietal cortex.

Methods

The patient

Patient Q.C. was a 17-year-old right-handed male, who was a student in a Chinese high school in September 2002, when he twice suffered epilepsy. An immediate anatomical MRI 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 out of 20) was better than that of the left eye (5 out of 20). All the tests reported here were conducted in October 2002.

To test Q.C.'s ability to recognize shapes, he was presented with overlapping shapes (such as circles, triangles and squares) and asked to report what he saw. Q.C. performed normally (18 out of 18 correct in reporting two or three overlapped shapes). Three tests from the Visual Object and Space Perception Battery (Warrington and James, 1991) were administered to Q.C. to test his spatial ability:

(i) Dot-counting test assessed the spatial scanning abilities: the patient was presented with stimuli consisting of arrays of five, six, seven, eight or nine dots arranged in a random pattern and was asked

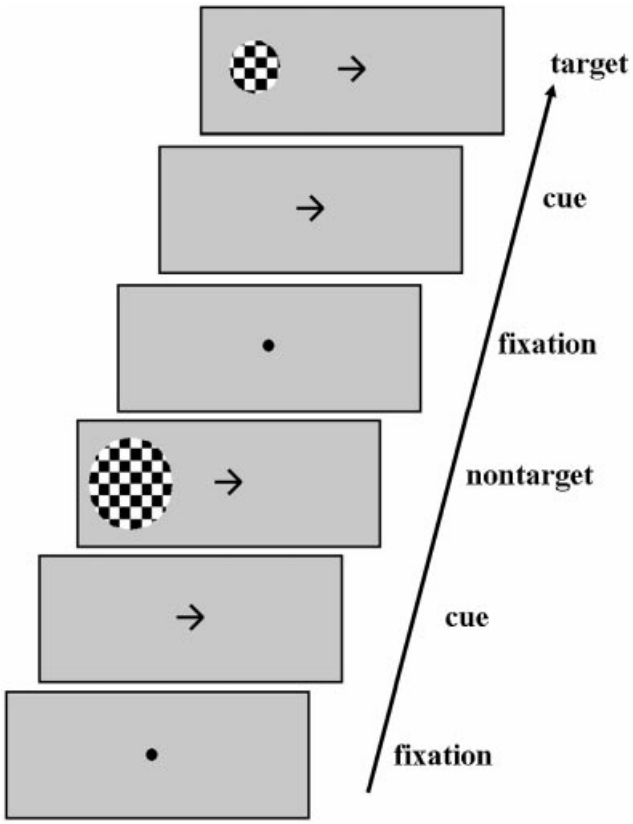

Fig. 2 Illustration of the stimuli and procedure used in the current study. Large (non-target) and small (target) circular checkerboards, which were preceded by arrow cues, were flashed in random order to the LVF and RVF locations. Subjects responded to the presence of targets by pressing a button.

to report the number of dots present. Q.C. performed normally (10 out of 10; normal range 8–10).

(ii) Number location test assessed the ability to locate points spatially: the patient was presented with stimuli consisting of two squares placed one above the other. The top square contained randomly placed digits (1–9) and the bottom square contained a randomly placed dot corresponding to the position of one of the numbers. The patient was asked to identify the number in the same location within the square as the dot in its square. Q.C. performed normally (10 out of 10; normal range 7–10).

(iii) Position discrimination test assessed the ability to discriminate relative spatial positions: the patient was presented with stimuli consisting of two adjacent horizontal squares. In one square, a black dot (5 mm diameter) was printed at the exact centre; in the other square, a black dot was just off-centre. The subject pointed to the centred dot. Q.C. performed normally (20 out of 20; normal range 18–20).

The controls

Four healthy subjects (three male, aged between 18–19 years) participated in the behavioural experiment and six healthy subjects (five male, aged between 18–23 years) participated in the fMRI experiments as controls. All the controls were right-handed and had normal or corrected-to-normal vision. Informed consent was obtained from both the patient and controls. The study was approved by the academic and ethical committee of the Department of Psychology, Peking University.

Covert visual orienting tasks

Stimuli were square-wave modulated black and white checkerboard patterns, circular in overall form and displayed on a grey background. The stimuli were presented on a computer monitor in behavioural studies. In the fMRI experiment, the stimuli were presented through a liquid-crystal display (LCD) projector onto a rear-projection screen located at the subject's feet, which was viewed with an angled mirror positioned on the head-coil. The checks were aligned with the horizontal and vertical axes of the screen. In both behavioural and fMRI experiments, an oddball paradigm was used, in which circular checkerboards appeared randomly to the left or right of the fixation that was located at the centre of the screen (see the illustrations in Fig. 2). The patient and the controls responded to a small percentage of small checkerboard patterns (target) by a key (or button) press, while ignoring large checkerboard patterns (non-target). The centre of both target and non-target stimuli was equally distant from the fixation. The stimuli appeared at either the location indicated by pre-cues (valid condition) or the location opposite to the cue direction (invalid condition).

In the behavioural experiment, target and non-target stimuli subtended visual angles of 2.8° × 2.8° (wide and high) and 4.9° × 4.9°, respectively, at a viewing distance of 50 cm. Each of the black or white checks subtended a visual angle of 0.61° × 0.61° in both target and non-target stimuli. Each trial began with the presentation of small black dot (0.28° × 0.28°) in the centre 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 (0.91° × 0.91°) pointing to the LVF or RVF, serving as a cue to direct subject's attention. Some 1000 ms later, the checkerboard stimuli appeared in the LVF or the RVF with its centre 6.1° away from fixation. 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 50% of the trials by pressing a key on a standard keyboard with the right index finger. To obtain mean reaction times (RTs) from a large number of trials, we used a higher percentage of targets and higher percentage of invalid trials in the behavioural than in the fMRI experiments. Seventy percent of target and non-target stimuli appeared in the hemifield to which the arrow pointed (valid cue condition), whereas 30% of target and non-target stimuli appeared in the hemifield opposite to the direction of the arrow (invalid cue condition). There were 16 practice trials followed by 200 trials in two blocks.

In the fMRI experiments, target and non-target checkerboard stimuli subtended visual angles of 1.9° × 1.9° and 3.4° × 3.4°, respectively, at a viewing distance of 270 cm. As stimuli were presented on computer monitors or a rear-projection screen with different scales, stimulus sizes were different across the experiments. Each of the black or white checks subtended a visual angle of 0.43° × 0.43° in both target and non-target stimuli. The fixation cross and arrow cues subtended a visual angle of 0.63° × 0.63°. The centre of each checkerboard stimuli was 4.3° apart from the fixation. Target and non-target stimuli appeared on 20% and 80% of the trials randomly in the LVF or the RVF. The stimulus displays were

Table 1 *Behavioural results of the patient and normal controls*

Stimulus location	LVF		RVF	
	Valid	Invalid	Valid	Invalid
RT (ms)				
Controls	382	409	379	401
Patient	480	525	512	514
Hit (%)				
Controls	98	100	98	100
Patient	97	100	100	100
False alarm (%)				
Controls	5.0	3.3	4.9	3.3
Patient	0.0	0.0	2.8	0.0

presented for 150 ms. Interstimulus intervals were randomized between 300 and 400 ms. In order to obtain strong fMRI signals, longer stimulus durations and shorter interstimulus intervals were used so that there were more stimuli presented in the same period of time in the fMRI than in the behavioural experiments. There were three conditions in the fMRI experiment similar to those used in prior work (Martinez *et al*., 2001):

(i) Attention to the LVF: an arrow pointing to the LVF was continuously visible in the centre of the screen while the checkerboard stimuli flashed randomly in the LVF or RVF. The patient responded with a button press only to the LVF targets.

(ii) Attention to the RVF: an arrow pointing to the RVF was continuously visible in the centre of the screen while the checkerboard stimuli flashed randomly in the LVF or RVF. The patient responded only to the RVF targets.

(iii) Passive viewing: the fixation cross was continuously visible in the centre of the screen while the checkerboard stimuli flashed randomly in the LVF or RVF. The patient and the controls were asked to fixate at the cross and not to respond to any stimuli.

Six scans of 64 s were obtained. Each scan consisted of three epochs of 40 trials (20 s for each epoch), alternating between the three attention conditions. The first 4 s of each scan were excluded from statistical analysis to obtain a steady baseline.

Examination of the function of the visual cortex

To assess if the function of the patient's visual cortex was affected by the parietal damage, haemodynamic responses were measured with fMRI to flickering checkerboard stimuli presented in the left or the right hemifield reversing contrast at 8.0 Hz. The checkerboard stimulus was a part of a circular form with a radius of 19.0 cm, which covered the left or the right hemifield with the regions along the vertical meridian being spared. Two scans of 196 s were obtained. Each scan consisted of six epochs of 32 s, alternating between the following three conditions: (i) flickering checkerboard stimuli were presented in the LVF; (ii) flickering checkerboard stimuli were presented in the RVF; and (iii) no flickering checkerboard stimuli were displayed. The first 4 s of each scan were excluded from statistical analysis to obtain a steady baseline. In all stimulus conditions, the patient was instructed to maintain fixation at the central cross (0.63° × 0.63°), which was separated from the checkerboard stimuli by 1.0°.

fMRI image acquisition and analysis

Brain imaging was performed using a 1.5 T GE Signa MRI scanner (Milwaukee, Wisconsin, USA) with a custom head coil. Fifteen axial slices of functional images that covered the whole cerebral cortex were acquired using echo-planar imaging [(64 × 64 × 15 matrix with 3.75 × 3.75 × 6 mm spatial resolution; TR (repetition time) = 2000 ms; TE (echo time) = 40 ms; FOV (field of view) = 240 mm; flip angle = 90°]. 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). Subjects' heads were immobilized during the scanning sessions using pieces of foam. SPM99 software (The Wellcome Department of Cognitive Neurology, London, UK) was used for imaging data processing and analysis. The functional images were realigned to the first scan to correct the head movement between scans. The structural image was co-registered with the mean image produced during the process of realignment. All images were normalized to a 2 × 2 × 2 mm^3 Montreal Neurological Institute (MNI) template in Talairach space (Talairach and Tournoux, 1988) using bilinear interpolation. Functional images were spatially smoothed using a Gaussian filter with a full-width at half maximum (FWHM) parameter set to 6 mm. Data were modelled using a box-car function. In the covert visual orienting task, contrasts were defined to compare the effect of attention (attended versus passive viewing) for the LVF and RVF stimuli, respectively. Regions preferentially engaged in attention to the LVF (or RVF) were defined as areas more activated in the condition of responding to the LVF (or RVF) targets relative to the passive viewing condition. In the task to examine the function of the visual cortex, regions activated by the LVF (or RVF) stimuli were defined as areas more activated in the condition of presentation of the LVF (or RVF) stimuli relative to the condition of presentation of only the fixation cross. The patient's image data were estimated using a fixed effect model. Areas of significant activation were identified at the cluster level for values exceeding an uncorrected P value of 0.001 for all comparisons. The image data of each of the controls were first estimated to establish a fixed-effect model. A conjunction contrast analysis was then conducted to make population inference from a relative small number of subjects (Friston *et al*., 1999). A threshold was set at $P < 0.01$ for the group analysis. Clusters of voxels <20 voxels were not displayed. The statistical parametric mapping (SPM) coordinates for standard brain from MNI were converted to Talairach coordinates using a non-linear transform method (http://www.mrc-cbu.cam.ac.uk/Imaging/mnispace.html).

Results

Behavioural experiment

In the behavioural experiment, we measured the patient's behavioural performance in a covert orienting task, in which an arrow cue pointing to the possible stimulus locations in the LVF or RVF was followed by checkerboard stimuli that appeared at the cued or uncued locations. The patient was instructed to respond to the presence of targets as quickly and accurately as possible while keeping his eyes fixated on the fixation. Table 1 shows the behavioural performances of the patient and control subjects. RTs, hit and false alarm rates were subjected to repeated measure analyses of variance

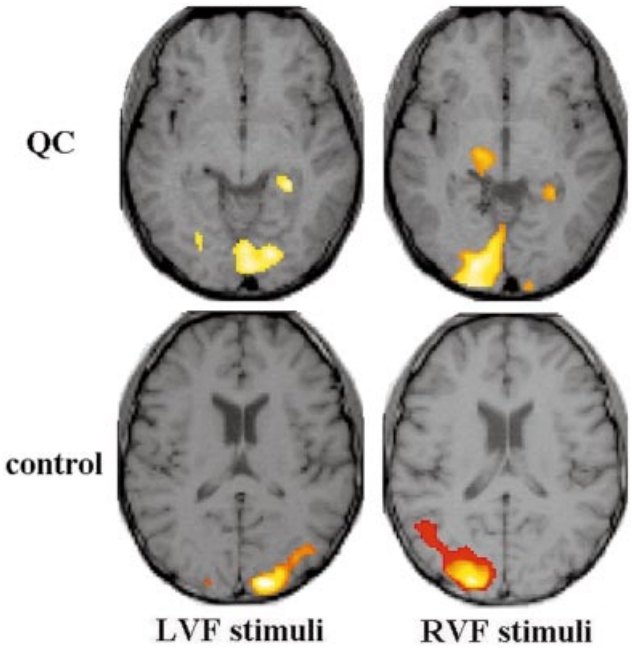

Fig. 3 Visual areas of the patient and one of the controls activated by flickering checkerboard stimuli presented in the LVF and RVF, respectively. The threshold for activation of all clusters was $P < 0.001$ (uncorrected).

(ANOVAs) with Group (patient versus control subjects), Cue Validity (valid versus invalid), and Visual Field (left versus right) as independent variables. The patient responded slower than the controls [$F(1,4) = 13.4$, $P < 0.03$]. There was a significant interaction of Cue Validity × Visual Field [$F(1,4) = 13.4$, $P < 0.03$], suggesting that the effect of cue validity on RTs was larger for the LVF than the RVF stimuli. In addition, the triple interaction of Group × Cue Validity × Visual Field was significant [$F(1,4) = 16.8$, $P < 0.025$] because, relative to the valid cue, the invalid cue slowed the patient's responses to the LVF stimuli whereas his responses to the RVF stimuli did not differ between the valid and invalid conditions. For the controls, however, the invalid cue slowed responses to both LVF and RVF stimuli. No significant effects on hit and false alarm rates were found.

fMRI experiment

We first assessed the function of the patient's visual cortex using flickering checkerboard stimuli presented in the LVF or RVF. Stronger activations were found in the patient's visual cortex contralateral to the stimulated hemifields when checkerboard stimuli were displayed relative to when only fixation was presented (Fig. 3). The activated regions included Brodmann area (BA) 17 and 18 (the Talairach coordinates of the activations were $x, y, z = 6, -88, -12$ for the LVF stimuli and –19, –90, –10 for the RVF stimuli). Activations in the same areas were observed in the controls (illustrated in Fig. 3). The fMRI results indicate that the patient's striate and extrastriate cortices in both hemispheres can be activated by visual stimuli regardless of the damage of the left parietal lobe.

The patient was then given a covert orienting task, in which he was presented with checkerboard stimuli flashed randomly in the LVF or RVF. The experiment employed a box-car design with three conditions: (i) attending to the LVF; (ii) attending to the RVF; or (iii) passive viewing. The results are shown in Fig. 4 and Table 2. Relative to the passive viewing condition, attention to the LVF generated stronger activations in bilateral medial and inferior frontal areas, the right post-central and inferior parietal cortices, right anterior cingulate cortex, right extrastriate cortex and left fusiform gyrus (Fig. 4A). However, no stronger activations were observed in association with attention to the RVF relative to the passive viewing condition at the usual statistical threshold of $P = 0.001$. Lowering the threshold to $P = 0.005$ revealed only one focus of activity in the right medial frontal lobe in association with attention to the RVF (the Talairach coordinates of this activation were $x, y, z = 50, 20, 38$; see Fig. 4B).

The patient's eye movement and behavioural performance in the scanner were not recorded because of equipment limitations. However, the results of the behavioural experiment suggest the patient was able to control his eye movement and detect most targets in the periphery. To strengthen this analysis, contrasts between conditions of passive viewing and responding to the LVF (or RVF) targets were conducted. If the patient moved his fixation to the stimulus location in the LVF when responding to the LVF target, there would be an asymmetry between the visual inputs into his left and right hemispheres, i.e. the RVF stimuli would be projected into his left hemisphere while the LVF stimuli would be projected into both hemispheres. This would induce stronger activation in the right visual cortex in the passive viewing condition relative to the condition of responding to the LVF targets. Similarly, if the patient moved his fixation to the stimulus location in the RVF when responding to the RVF target, we would expect stronger activation in the left visual cortex in the passive viewing condition relative to the condition of responding to the RVF target. However, the contrast between passive viewing and responding to the LVF (or RVF) target did not produce significant activation in either the left or right occipital cortex, indicating that eye movement cannot account for the attentional effect observed in the visual cortex.

The fMRI results in the covert orienting task from the controls are shown in Figs 5 and 6, and Table 3. Relative to the passive viewing condition, attention to the LVF generated stronger activations in bilateral inferior frontal lobules, right medial frontal cortex, left post-central cortex, right superior parietal cortex and right medial occipital cortex. Attention to the RVF resulted in activations in bilateral medial frontal areas, right superior parietal cortex, bilateral precuneus, left superior temporal area and left medial occipital area.

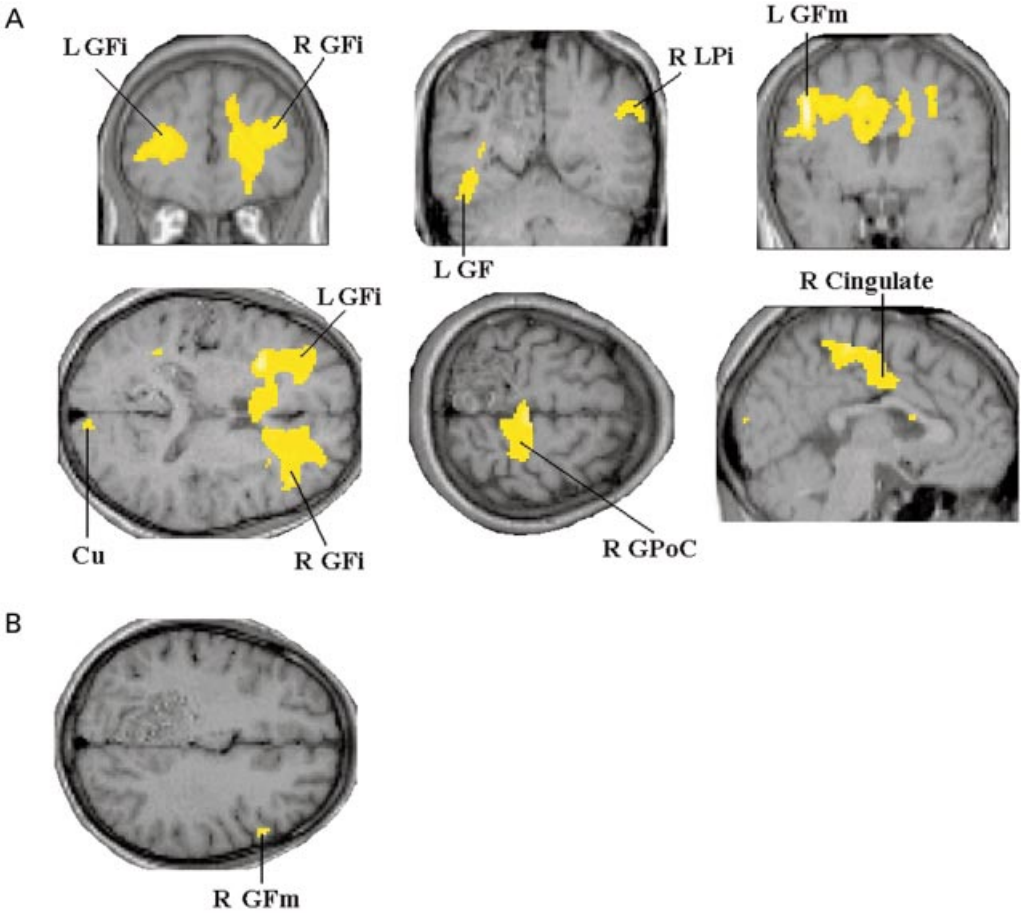

Fig. 4 (**A**) The patient's brain areas showing stronger activations in the condition of attending to the LVF relative to the passive viewing condition. These areas include the bilateral medial and inferior frontal areas, right post-central and inferior parietal cortices, right cingulate cortex, right striate and extrastriate cortices, and left fusiform gyrus. The *Z*-values and Talairach coordinates for the regional maxima are given in Table 2. The threshold for activations of all clusters was $P < 0.001$ (uncorrected). Cu = cuneus; GF = fusiform gyrus; GFi = inferior frontal gyrus; GFm = medial frontal gyrus; GPoC = post-central gyrus; LPi = inferior parietal lobule. (**B**) The patient's brain areas showing stronger activations in the condition of attending to the RVF relative to passive viewing condition. Only an area in the right middle GFm showed activation at the threshold of $P < 0.005$ (uncorrected).

Table 2 *The patient's brain areas activated by attention to the LVF*

Region	BA	Talairach coordinates			*Z* value
		x	*y*	*z*	
Left hemisphere					
Medial frontal	9	–46	2	35	5.01
Inferior frontal	44	–48	5	27	4.73
Fusiform	37	–44	–51	–9	3.90
	37	–44	–39	–11	3.48
Right hemisphere					
Medial frontal	46	46	34	13	3.94
Inferior frontal	10	34	43	–2	4.01
Post-central	1,2	65	–22	27	3.86
Cingulate	32	14	30	26	3.90
Inferior parietal	40	65	–22	27	3.86
	40	50	–47	32	3.44
Occipital	18	8	–91	14	3.27

Uncorrected $P < 0.001$ for all clusters listed.

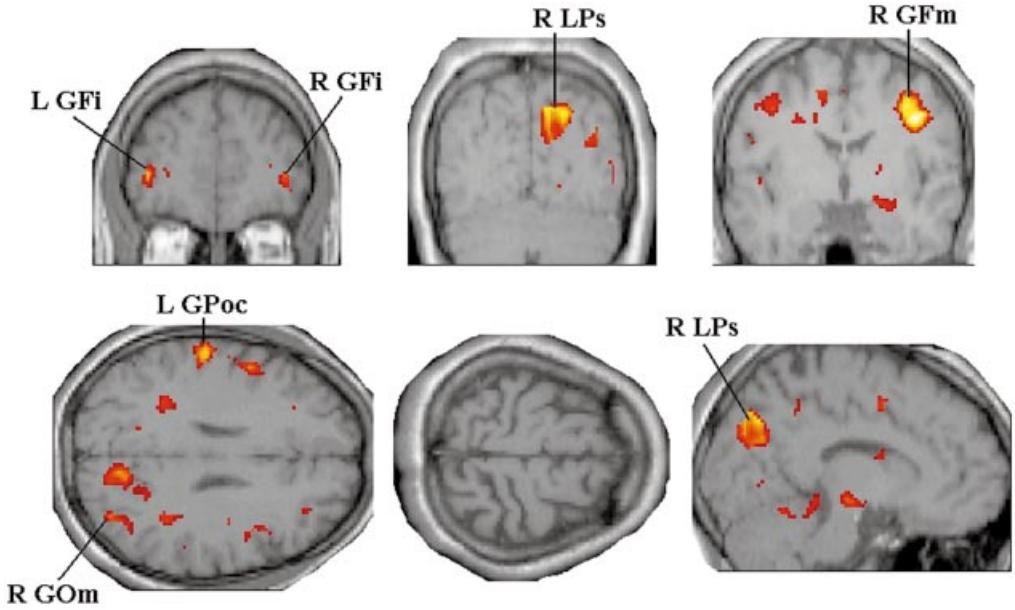

Fig. 5 The controls brain areas showing stronger activations in the condition of attending to the LVF relative to the passive viewing condition. These include bilateral inferior frontal areas, right medial frontal area, left post-central area, right superior parietal area, and right medial occipital area. The *Z*-values and Talairach coordinates for the regional maxima are given in Table 3. The threshold for activations of all clusters was $P < 0.01$ (uncorrected). GOm, medial occipital gyrus; GFi = inferior frontal gyrus; GFm = medial frontal gyrus; GPoC = post-central gyrus; LPs = superior parietal lobule.

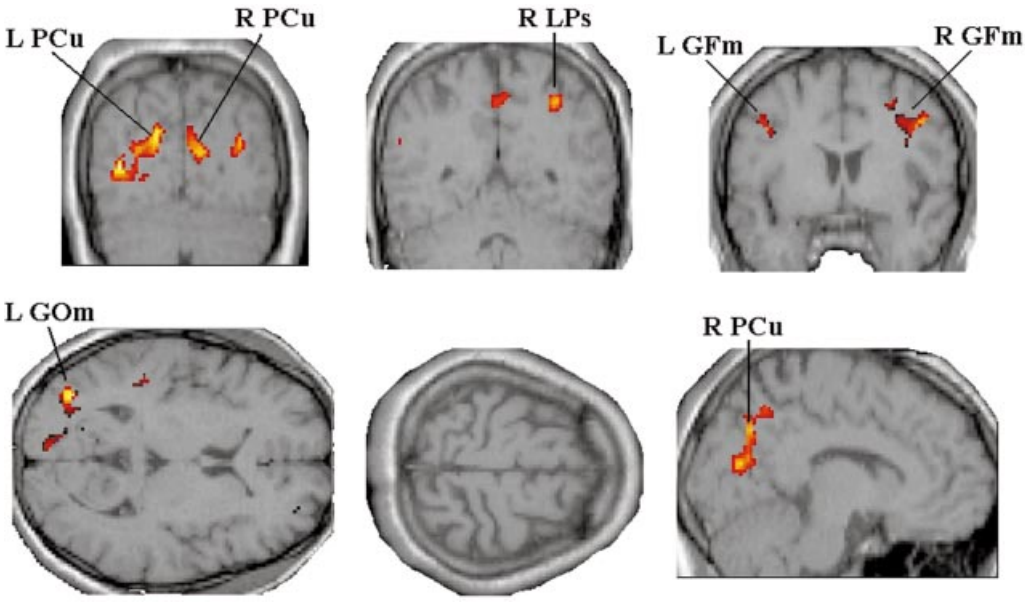

Fig. 6 The controls brain areas showing stronger activations induced by attention to the RVF relative to the passive viewing condition. These include bilateral medial frontal areas, right superior parietal area, bilateral precuneus, left superior temporal area and left medial occipital area. The *Z*-values and Talairach coordinates for the regional maxima are given in Table 3. The threshold for activations of all clusters was $P < 0.01$ (uncorrected). GFm = medial frontal gyrus; GOm = medial occipital gyrus; LPs = superior parietal lobule; PCu = precuneus.

Table 3 *The controls' brain areas activated by attention to the LVF and RVF*

Hemifield/region	BA		Talairach coordinates			*Z* value
			x	*y*	*z*	
Attention to the LVF						
Left hemisphere	Inferior frontal	46	–40	43	2	3.34
	Post-central	1,2	–57	–18	29	3.68
Right hemisphere	Medial frontal	6	36	–2	41	3.92
	Inferior frontal	46	40	47	1	2.82
	Superior parietal	7	18	–72	40	3.76
	Occipital	19	36	–74	28	2.68
Attention to the RVF						
Left hemisphere	Medial frontal	6	–48	6	35	3.16
	Superior temporal	22	–59	–36	17	3.30
	Precuneus	7	–16	–74	26	4.29
	Occipital	19	–36	–75	13	4.81
Right hemisphere	Medial frontal	6	46	8	35	4.34
	Superior parietal	7	34	–48	47	3.10
	Precuneus	7	8	–64	38	3.68

Uncorrected $P < 0.001$ for all clusters listed.

Discussion

To study the role of human parietal cortex in attention networks, we combined behavioural and fMRI measurements of cue validity effects in covert visual orienting tasks in a patient with focal left parietal lesions. The comparisons between the cue validity effects on activities of the occipital and frontal cortices in association with the LVF and RVF stimuli provide implications of the functional role of the left parietal cortex in neural networks guiding spatial attention. Because the left parietal damage did not induce neglect or extinction, the findings reported here are unlikely to result from deficits of other cognitive functions such as visual awareness.

While behavioural performances of the controls showed similar cue validity effects on responses to the LVF and RVF stimuli, the patient's data from the behavioural experiment showed clearly asymmetric cue validity effects on the patient's responses to the LVF and RVF stimuli. Responses to the LVF stimuli were faster in the valid than in the invalid conditions, whereas his responses to the RVF stimuli were not influenced by cue validity. The asymmetric cue validity effects were mainly because the patient's responses were slower to the RVF than the LVF stimuli in the valid condition, whereas his responses to the RVF and LVF stimuli did not differ in the invalid condition. The results suggest a deficit in attentional orienting to the hemifield contralateral to the damaged hemisphere rather than in disengaging attention. This is different from previous lesion studies, which showed that parietal damages usually produce deficits of disengaging attention from a location other than targets, i.e. longer responses to contralateral stimuli in the invalid condition (Posner *et al*., 1984, 1987). Most of the patients in previous studies were elderly adults, and their brain lesions usually resulted from acute stroke and led to neglect or extinction. The patient reported here, however, was young, and his brain lesions resulted from angiomas which possibly developed over a long period and led to reorganization of his attention networks. This may be the reason that the patient's parietal lesion did not generate neglect or extinction, and thus produced a deficit in attentional orienting rather than in disengaging attention.

The fMRI data from the controls showed that spatial attention to the LVF or RVF induced stronger activations in the contralateral lateral occipital cortex. This is consistent with previous reports (Martinez *et al*., 2001) and indicated attentional modulations of the neural activities of the visual cortex contralateral to the stimulated hemifield. Interestingly, for the patient, the LVF stimuli also generated stronger activations in the right striate and extrastriate cortices when being attended rather than being viewed passively. However, this contralateral attention effect in the right visual cortex was weak compared with the large attention effects in the ipsilateral left fusiform gyrus for LVF stimuli. In addition, no activation was observed in the left visual cortex in association with the patient's attention to the RVF. The patient's results indicate that the left parietal lesions weakened attentional modulations of the neural responses of bilateral visual cortex and this effect was particularly salient for the damaged hemisphere. It is possible that, although attention to each hemifield is dominated by the contralateral hemisphere (Corbetta *et al*., 1993; Luck *et al*., 1989), guiding spatial attention to each hemifield requires cooperation between the parietal cortices in both hemispheres (e.g. activations were observed in bilateral parietal lobes in association with attentional cues to either the LVF or RVF; Hopfinger *et al*., 2000). Consequently, the damage to the left parietal cortex impaired both the neural substrates for directing attention to the right hemifield and the coherence between left and right parietal lobes that was important for guiding attention to the left hemifield. This may help to

account for the elimination of the modulation of the left occipital activities associated with the rightward attention and weakness of the modulation of the right occipital activities related to the leftward attention. Whatever the case, our findings suggest that the left parietal cortex, being a part of the interconnected network of cortical areas, contributes to the attentional modulation of neural activities of the visual cortex.

The fMRI data from the controls also showed that attention to the LVF or RVF activated the attention network including both anterior (e.g. bilateral frontal cortices) and posterior (e.g. post-central gyrus, superior parietal cortices, precuneus) brain structures. Thus, both the anterior and posterior parts of the attention network were engaged in guiding spatial attention to each hemifield. This is in agreement with the results of previous experiments (Gitelman *et al*., 1999; Hopfinger *et al*., 2000; Leonards *et al*., 2000). The patient's fMRI data showed activations associated with leftward attention in bilateral medial and inferior frontal lobules, right post-central gyrus, inferior parietal cortices and right cingulate cortex. The pattern of the results was similar to that obtained from the controls. Therefore, we may conclude that the patient's neural network responsible for directing attention to the LVF was intact. For the RVF stimuli, however, no stronger activation in any brain areas (with a standard threshold of *P* values) was found when being attended rather than being passively viewed. It appears that the left parietal damage deteriorated not only the attentional modulation of the activities of the visual cortex but also the engagement of bilateral frontal cortex in guiding spatial attention. One possibility is that spatial representations in the parietal cortex (Colby and Goldberg, 1999) may provide a necessary spatial map for the frontal cortex to control orienting of attention in space. Once the parietal cortex is damaged and the spatial map is destroyed, the frontal cortex loses the substrates through which to control voluntary attention to specific locations in visual space. Based on our results, it may be speculated that, besides executing attentional modulations of activities of the occipital cortex, the left parietal cortex is also important for the engagement of the frontal cortex in the attention network.

Finally, some previous studies found that the cingulate cortex is activated in association with spatial attention tasks (Nobre *et al*., 1997; Gitelman *et al*., 1999; Kim *et al*., 1999). However, the activations are mostly limited to the anterior cingulate cortex, which is assumed to be part of attentional control system (Posner and Petersen, 1990; Gitelman *et al*., 1999). Given previous findings and theoretical assumptions, we suggest that the lesion in the posterior cingulate cortex of our patient contributes little to his deficits of visual attention, or at least, the damage of the posterior cingulate cortex played a much less important role than that of the left superior parietal cortex in producing the results observed in the current study.

In conclusion, the current study provides behavioural and fMRI evidence that focal left parietal damage degraded the patient's ability to covertly orient to the RVF and weakened attentional modulations of activities of the visual cortex and frontal activations during covert visual orienting. These effects were confirmed under the condition that the left visual cortex and bilateral frontal cortex were intact. The findings support the proposition that the parietal cortex plays an important role in attentional modulations of neural responses of the visual cortex. Moreover, our results suggest that the parietal cortex may contribute to the engagement of the frontal cortex in voluntary attentional control.

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