# Voluntary orienting is dissociated from target detection in human posterior parietal cortex

Maurizio Corbetta[1,2,3], J. Michelle Kincade[1,4], John M. Ollinger[2], Marc P. McAvoy[2] and Gordon L. Shulman[1]

[1] *Department of Neurology and Neurological Surgery, Washington University School of Medicine, 4525 Scott Avenue, St. Louis, Missouri 63110, USA*

[2] *Mallinckrodt Institute of Radiology, Washington University School of Medicine, 4525 Scott Avenue, St. Louis, Missouri 63110, USA*

[3] *Department of Anatomy and Neurobiology, Washington University School of Medicine, 4525 Scott Avenue, St. Louis, Missouri 63110, USA*

[4] *Department of Psychology, Washington University School of Medicine, 4525 Scott Avenue, St. Louis, Missouri 63110, USA*

*Correspondence should be addressed to M.C. (mau@npg.wustl.edu)*

**Human ability to attend to visual stimuli based on their spatial locations requires the parietal cortex. One hypothesis maintains that parietal cortex controls the voluntary orienting of attention toward a location of interest. Another hypothesis emphasizes its role in reorienting attention toward visual targets appearing at unattended locations. Here, using event-related functional magnetic resonance (ER-fMRI), we show that distinct parietal regions mediated these different attentional processes. Cortical activation occurred primarily in the intraparietal sulcus when a location was attended before visual-target presentation, but in the right temporoparietal junction when the target was detected, particularly at an unattended location.**



Acute structural damage to the right temporoparietal cortical junction (TPJ; inferior parietal lobule and superior temporal gyrus) in humans produces a complex clinical syndrome characterized by the inability to attend and respond to objects positioned in the left visual field (unilateral visual neglect)[1–3]. Some symptoms of neglect may reflect a deficit in reorienting attention toward new stimuli in the visual field opposite to the lesion (contralesional field)[4–7]. Patients with parietal lesions can detect visual stimuli in the contralesional field when correctly cued to their locations, but they are slow or fail to detect the same stimuli when attending to other locations[4]. This deficit is more severe after right than left parietal lesions[5], and is localized to the TPJ[7]. These findings suggest that the posterior parietal cortex near TPJ may be critical for reorienting the focus of attention toward visual stimuli appearing at unattended locations (reorienting hypothesis).

Other data suggest that posterior parietal cortex near/along the intraparietal sulcus (IPs), which separates the superior from the inferior parietal lobule, is involved in voluntarily directing attention to a spatial location (voluntary orienting hypothesis). Neurons in the IPs increase firing rate when a monkey attends to a location while preparing a response[8–11]. Human functional brain imaging shows activations in the IPs (and superior parietal lobule) when observers voluntarily pay attention to and detect peripheral visual stimuli, with or without concurrent eye movements[12–16]. It is unknown to what extent areas in human and monkey IPs are homologous.

These complementary functional anatomical theories (reorienting to targets in the TPJ and voluntary orienting in the IPs) make specific predictions about which regions should be activated while attending to a spatial location and, subsequently, when detecting a visual target there. If IPs is preferentially involved in voluntary orienting, then it should be activated when an observer attends to a location before presentation/detection of a visual target. If TPJ is necessary for reorienting to a visual target, then its activation should follow the presentation/detection of the target, particularly when it is presented at an unattended location. We tested these predictions using ER-fMRI and an ANOVA-based procedure[17]. This method has two important characteristics: it can separate the responses to events presented within the same cognitive trial, and it is sensitive to differences in both magnitude and timing of responses. Unlike other published ER-fMRI methods[18–21], this method makes no assumptions about the shape of the underlying response function.

## Results

Normal observers were given a cue indicating the most likely location of a subsequent target stimulus they were required to detect, according to a protocol modified from a published procedure[4]. The stimulus display consisted of a central fixation cross flanked on either side by square boxes. The length of each arm of the central fixation cross subtended 16 minutes of visual angle. The boxes (size, 1°) were placed at 3.3° of visual angle to either side of the fixation spot. Accurate fixation of the central cross-hair was emphasized throughout the experiment. At the beginning of a trial, a cue arrow pointing to the left or right box was superimposed on the fixation cross. The arrow indicated the most likely location of a subsequent target stimulus, and leftward or rightward arrows were equally probable. The cue arrow remained on the screen for one MR frame (2360 ms; cue period).

The sequence of events following presentation of the cue arrow depended on the type of trial. On a cue trial (20% of the

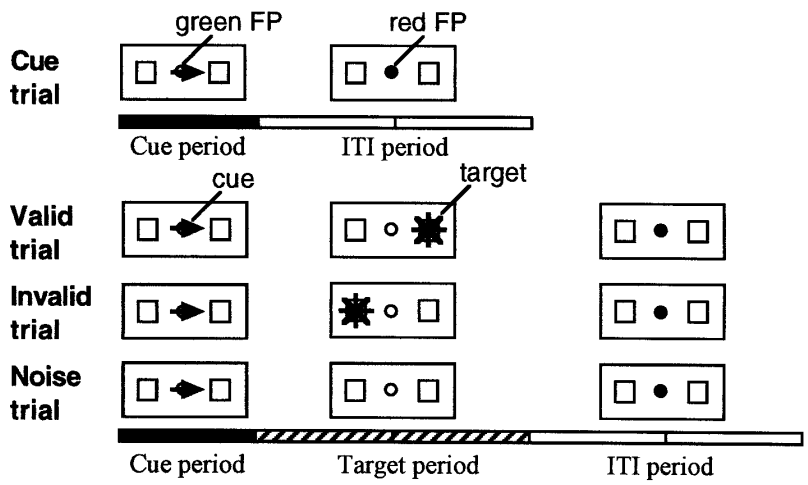


**Fig. 1.** Display, trial types and MR design. Each trial lasted between 4 and 7 MR frames, and each MR frame was 2.36 s long. MR frames are indicated by elongated rectangles below displays. In a cue trial, a cue arrow was presented for 1 MR frame (cue period) at fixation (black rectangle) followed by an intertrial interval (ITI) period signaled by a change in the color of the fixation point (from green to red). The ITI period lasted for 2, 3 or 4 MR frame duration (white rectangles). A two-frame ITI is shown. In a valid trial, the cue period was identical. During the test period (2 MR frames or 4.72 s; crossed rectangles), after a randomly selected time between 1500–3000 ms, a 100-ms target stimulus (asterisk) was flashed in the box cued by the arrow. Subjects indicated target detection with a key press. The ITI period followed. Invalid trial, same as valid trials except that the target was flashed at the uncued box location. Noise trial, same as valid trials, except that no target was flashed during the target period.



trials), the trial ended immediately after cue presentation. The end of the trial was signaled by a change in the color of the fixation cross from green to red. During a cue trial, a subject shifted attention toward the location indicated by the cue and maintained it there until the end of the trial. On a noise trial (20% of the trials), the cue period was followed by a test period lasting 2 MR frames (4720 ms) in which no target was presented. During noise trials, the subject presumably shifted and maintained attention on the cued location for a longer time than during a cue trial (7080 versus 2360 ms; **Fig. 1**). On a valid trial (44% of the trials), the cue period was followed by a test period during which a target appeared at the location indicated by the cue. The target was a white asterisk that appeared in one of the square boxes for 100 ms. On an invalid trial (16% of the trials), a target appeared during the test period at the uncued location. The cue arrow correctly predicted the target location on 73% of the trials in which a target was presented. These four trial types (cue, noise, valid, invalid) were randomly intermixed. Subjects were instructed to press a button as quickly as possible upon detection of the target and to withhold responses on cue or noise trials.

We used this protocol during whole-brain measurements of blood oxygenation level dependent (BOLD) responses on a Siemens Vision 1.5 T magnet. The time course of the BOLD response for each trial type and each trial period (cue period, test period/noise, test period/valid, test period/invalid) was estimated in each subject using linear regression. Pixel-wise and regional ANOVAs were used for appropriate statistical contrasts[17] (see Methods).

## Behavior

Reaction times for target detection were faster on valid than invalid trials (380 ms versus 426 ms, $F_{1,11} = 21.92$, $p = 0.0007$), indicating that subjects used the cue arrow to attend to the location of the target. No responses were recorded during noise and cue trials.

## Imaging

During the cue period, a series of ventral and dorsal visual regions were active; these included bilateral anterior fusiform (Fus; *x*, *y*, *z* atlas coordinates, 35, –57, –20, right; –31, –55, –16, left), lateral occipital (LO;–31, –83, 0, left; 27, –87, 0, right)[22,23],

**Table 1. List of parietal regions during cue and target periods (averaged over valid and invalid targets) and showing significant validity effect.**

| | Cue | | | | Target | | | | Valid – invalid | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Regions | *x* | *y* | *z* | *Z*-score | *x* | *y* | *z* | *Z*-score | *x* | *y* | *z* | *Z*-score |
| L pos IPS | –25 | –67 | 48 | 7.58 | –25 | –65 | 48 | 6.94 | | | | |
| L ant IPs | –25 | –57 | 46 | 7.55 | –25 | –57 | 42 | 7.27 | | | | |
| L vIPS | –23 | –67 | 32 | 7.42 | | | | | | | | |
| “ | –27 | –75 | 26 | 7.23 | –27 | –77 | 20 | 7.16 | | | | |
| R ant IPs | 27 | –59 | 52 | 6.75 | 33 | –51 | 48 | 7.84 | 39 | –47 | 48 | 4.09 |
| R pos IPs | 21 | –65 | 52 | 6.62 | | | | | | | | |
| R vIPS | 29 | –71 | 22 | 6.01 | 27 | –71 | 30 | 6.63 | | | | |
| R IPL | | | | | 53 | –45 | 20 | 8.53 | 53 | –49 | 30 | 5.12 |
| R STG | 51 | –55 | 4 | 5.41 | | | | | 57 | –45 | 12 | 4.44 |
| R PC | | | | | 7 | –73 | 32 | 7.71 | 7 | –75 | 34 | 4.28 |
| L PC | | | | | –9 | –71 | 40 | 7.37 | –5 | –71 | 34 | 4.27 |

See Figs. 2 and 3 for anatomical labels. Coordinates (*x*, *y*, *z*) correspond to the Talairach atlas.

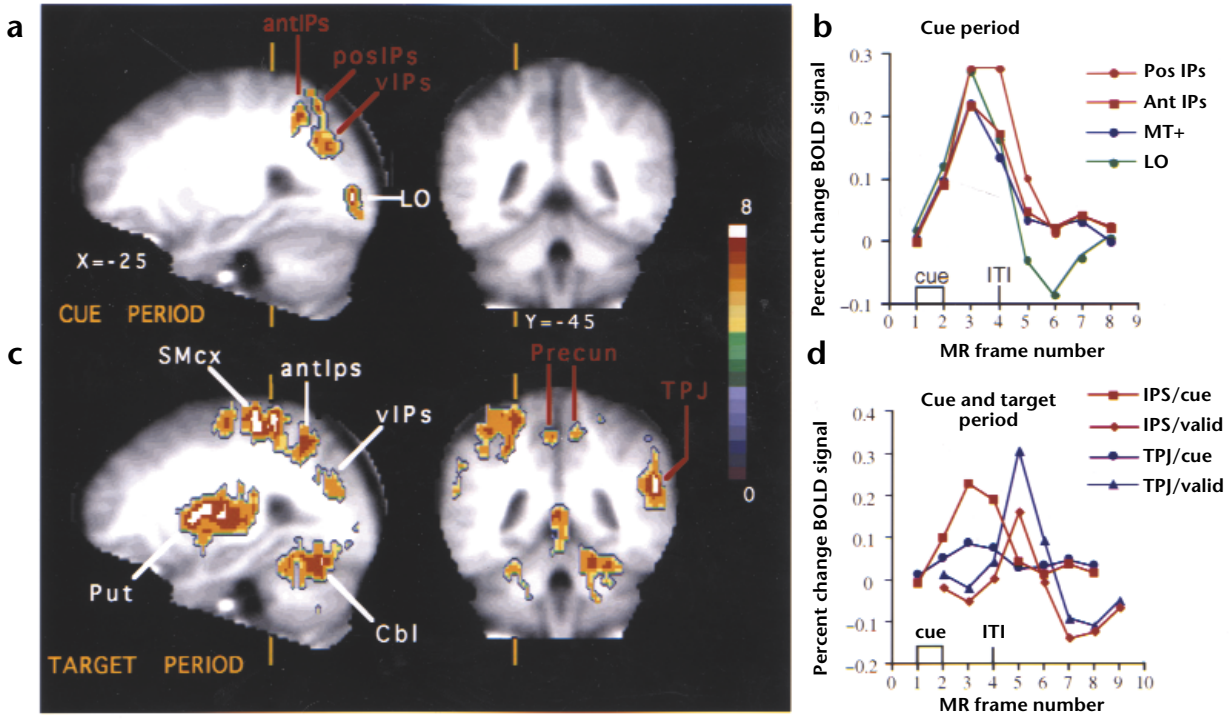


**Fig. 2.** BOLD responses during cue and target periods. (**a**) ANOVA *F* map transformed to *Z* map for the cue period (averaged over subjects, cue direction). Left, sagittal slice 25 mm left of midline. Right, coronal slice 45 mm posterior to center of atlas space. Yellow lines indicate corresponding planes of section. Parietal regions with sustained responses to cues are labeled in red: posIPs, posterior intraparietal sulcus; antIPs, anterior intraparietal sulcus; vIPs, ventral intraparietal sulcus. The lateral occipital region LO (labeled in white) showed a transient response to the cue. (**b**) BOLD time courses in different regions (averaged over subjects, cue direction and hemispheres) during the cue period. Response typically peaked two frames after onset of cue arrow on frame 1. ITI began on frame 4. (**c**) Target period (averaged over subjects, valid and invalid targets). Parietal regions with prevalent responses to targets are shown in red label: TPJ, temporoparietal junction; Precun, precuneus. Several motor-related responses are also shown: SMcx, sensory-motor cortex; Put, putamen; Cbl, cerebellum. (**d**) BOLD time courses in IPs and TPJ (averaged over subjects, cue direction, target field and hemispheres) during cue and target (valid) periods. Target was randomly presented around frame 3. IPs/cue, IPs response during cue period; IPs/valid, IPs response during valid-target period; TPJ/cue, TPJ response during cue period; TPJ/valid, TPJ response during valid-target period.



MT+ (–45, –69, –2, left; 45, –69, –4, right)[24,25] and ventral (vIPs), anterior (ant IPs) and posterior intraparietal sulcus (pos IPs; **Fig. 2a**, left; see **Table 1** for coordinates of parietal foci). No significant activation was detected in the TPJ during the cue period (**Fig. 2a**, right). The IPs activity did not reach the surface of the inferior parietal lobule, but spread into the superior parietal lobule. The vIPs region was located at the junction of dorsal occipital and parietal cortex, just dorsal and anterior to the V3A representation[26].

The time course of the BOLD response during the cue period was more sustained within the intraparietal sulcus (posIPs, antIPs, vIPs) than in occipital regions (Fus, LO, MT+; ANOVA regions × frame, $F_{56,672} = 5.18$, $p = 0.0001$). We compared a transient time course in two occipital regions (LO, MT+) with more sustained time courses in antIPs and posIPs (**Fig. 2b**). The difference in response duration cannot be explained by a difference in the peak magnitude. LO and posIPs, for example, showed similar peak magnitudes on frame 3, but different response duration (ANOVA regions × frames 3 and 4 only, $F_{1,12} = 24.3$, $p = 0.0003$). Similarly, antIPs and MT+ had similar magnitudes, but the response was more sustained in antIPs (ANOVA regions × frames 3 and 4 only, $F_{1,12} = 7.22$, $p = 0.02$).

Whereas transient time courses in occipital regions probably reflect visual processes related to the presentation of the foveal cue, the more sustained time courses in intraparietal cortex may reflect longer times required for processing cues related to orienting toward and maintaining attention at the cued location. To further test this idea, we compared BOLD responses of these regions during the noise period, in which subjects maintained attention at a peripheral location for 4.72 seconds after the offset of the cue arrow. Across all regions active during the noise period, only antIPs and vIPs showed sustained activity (ANOVA regions × frame, $F_{77,924} = 7.80$, $p = 0.0001$), with responses of the left hemisphere more sustained than those of the right hemisphere (Ant IPs, $F_{7,84} = 3.76$ $p = 0.0014$; vIPs, $F_{7,84} = 5.40$, $p = 0.0001$). Hence, after the presentation of the cue arrow, some IPs regions (antIPs, vIPs) showed a sustained BOLD response that was maintained during the noise period, during which subjects attended to the cued location for almost five seconds while waiting for the target stimulus.

During the target period, many visual and motor regions were active for both valid and invalid targets (**Fig. 2c**). All occipital regions that were active during the cue period also responded to target presentation, and these responses were significantly stronger for targets in the contralateral visual field. In parietal cortex, significant responses were recorded in antIPs, posIPs, vIPs, precuneus (Precun) and the TPJ, where the activation was much stronger in the right than the left hemisphere (**Fig. 2c**; **Table 1**). BOLD responses in these parietal regions during the cue and valid-target periods were compared by ANOVAs. Data

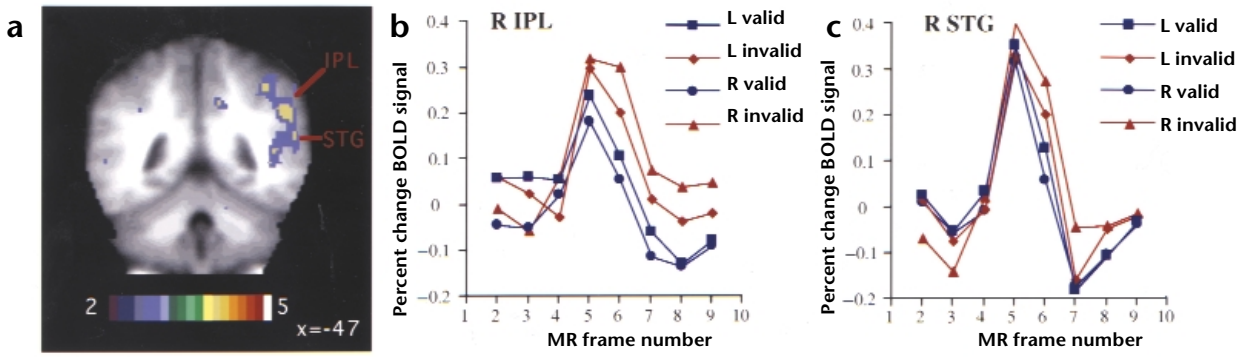


**Fig. 3**. BOLD responses for valid and invalid targets. (**a**) Coronal section through TPJ cortex (~47 mm posterior). ANOVA (validity × frame) *F* map transformed to *Z* map. Voxels that show significant validity effect (different BOLD responses for valid and invalid targets) independent of the visual field of the target. BOLD time courses (averaged over subjects) were estimated in right inferior parietal lobule (R IPL; **b**) and right superior temporal gyrus (R STG; **c**) during the target period for valid and invalid targets in left and right visual field.



for antIPs and posIPs were collapsed, as the borders between the two regions could not be readily identified. Responses of IPs (Ant + Pos) and vIPs regions were stronger during the cue period, whereas those of the TPJ and precuneus region were stronger during the target period ($F_{21,252} = 4.29$ $p = 0.0001$; **Fig. 2d**). These findings indicate that the voluntary orienting and maintenance of attention to a location primarily recruited the cortex within the IPs. In contrast, target detection recruited the TPJ (and precuneus), although a significant target response was also evident in IPs (**Fig. 2d**).

To test whether TPJ was preferentially involved in reorienting attention toward novel unattended stimuli, the time courses of the BOLD responses during the target periods for valid and invalid trials were compared. The strongest validity effect (difference in the BOLD response for valid and invalid trials) across the whole brain was localized in the right TPJ cortex, with separate foci in the inferior parietal lobule (IPL) and superior temporal gyrus (STG; pixel-wise ANOVA *Z*-score = 5.12; **Fig. 3a**; **Table 1**). A regional ANOVA confirmed that the validity effect was significant and independent of the visual field of the target (ANOVA frame × validity, R IPL, $F_{7,84} = 8.13$, $p < 0.0001$; R STG, $F_{7,84} = 7.53$, $p < 0.0001$). The response in right IPL and STG was more sustained for valid than invalid targets in each visual field (**Fig. 3b** and **c**; ANOVA, frames 5 and 6 only × validity, R IPL, $F_{1,12} = 5.212$, $p = 0.041$; R STG, $F_{1,12} = 6.51$, $p < 0.025$). Significant validity effects were also localized bilaterally in the precuneus and near the intersection of the right IPs and postcentral sulcus. This latter region did not overlap with the IPs regions active during the cue period (vector distance, 17 mm; **Table 1**). Significant validity effects not discussed here were also observed in other regions outside of parietal cortex.

## Discussion

We tested two functional-anatomical theories about the role of posterior parietal cortex in visuospatial attention. One theory, supported by studies of neglect patients with parietal lesions, proposes that the TPJ cortex is necessary for reorienting toward visual targets appearing at unattended locations. Another theory, based on single unit and imaging data, proposes that cortex along the IPs is involved in the voluntary orienting of attention toward a location. Our results provide direct confirmation of both views, showing that IPs was active before target presentation when a location was voluntarily attended, independent of processes related to target detection (for instance, visual responses and their attentional modulation or motor responses). In contrast, the right TPJ responded to target presentation more strongly when the target occurred at an unattended location.

### Voluntary orienting of attention

Two findings support a role for the IPs in the voluntary orienting and maintenance of attention to a target location. First, the presentation of a cue arrow indicating the most likely location of a subsequent visual target triggered transient responses in occipital cortex, but more sustained responses in IPs. Transient responses in occipital cortex may reflect the encoding of the cue, which was probably completed within a half second[27]. In contrast, sustained parietal responses may reflect the required shift toward and maintenance of attention at the cued location for the entire cue period (2360 ms). Second, when the delay after the cue offset (noise period) was extended to 4.72 seconds, forcing subjects to maintain attention at the cued location for longer, IPs was the only brain region that showed a sustained response during the delay.

Our results extend findings of increased activity in extrastriate, frontal and IPs cortex without sensory stimulation when subjects attend to a specific object at a specific location during an identification task[20]. Those activations are thought to reflect an attentional signal-biasing activity in visual cortex before stimulus presentation[28]. Here we show, in a simpler detection protocol, that IPs was uniquely active when attention was oriented toward and maintained at a relevant location, suggesting that IPs is the source of spatial biases observed in extrastriate visual cortex.

Cue-related activity in IPs may underlie an attentional signal that 'marks' a location of interest[9,11] or an intentional signal that prepares a response (eye movement or hand reaching) toward that location[10]. However, attending to direction of motion also drives IPs regions before the presentation of any motion target[17]. Activations are found in IPs during shifts in an object feature (for instance, color or shape)[29] or between percepts during binocular rivalry[30]. These results suggest that the intraparietal cortex may be involved in visual selection beyond the selection of locations.

The BOLD response in IPs was time locked to different processes in the course of a trial. Early in the trial (cue and noise

period), the response was controlled by voluntary orienting processes, whereas the response was controlled by detection processes later in the trial (target period; **Fig. 2d**). The coexistence of different processes in human IPs resembled that observed in monkey IPs, where neurons also manifest activity time locked to different components of a task (for instance, visual, attentional, memory, oculomotor or reaching)[9–11,31,32]. Although spatial overlap between regions of activation during different protocols (visual attention versus eye movements) in previous imaging studies was used to examine colocalization of processes[16,33], the enhanced temporal resolution of new ER-fMRI procedures[17,20,34,35] allows a richer and more realistic view of the temporal dynamics of activation in the human brain.

### Target detection

The right TPJ (and precuneus) was specifically engaged during target detection. Unlike other parietal regions that showed both cue and target responses (antIPs, vIPs), right TPJ and precuneus showed little if any response to the cue.

The pattern of activation in the TPJ cortex fits two main features of unilateral spatial neglect. The much stronger activation on the right than the left TPJ agrees with the clinical[2,3,36] and neuropsychological findings[5] that neglect is more severe following right TPJ lesions. The stronger TPJ response following the presentation of a target at an invalid location is also consistent with clinical and experimental[4,7] observations that neglect is worse for objects presented at unattended locations.

Activation of the right TPJ may underlie the process of spatially redirecting the focus of attention toward the location of unattended stimuli. This reorienting was indexed by longer reaction times for invalid targets than for valid targets; thus, the sensitivity of the TPJ activation to target validity implies that the BOLD signal, despite its slow temporal resolution (seconds), can track neuronal processes that yield behavioral differences of a few tens of milliseconds (the difference in reaction time between valid and invalid trials).

Another possibility, however, is that activity in right TPJ cortex is related to spatially nonselective neural processes triggered by reorienting to an unattended target. Interestingly, TPJ damage reduces the amplitude of P300 scalp electrical potentials, which are commonly elicited by the detection of infrequent visual, auditory and somatosensory targets during spatial and nonspatial tasks[37]. The right TPJ is also selectively activated when observers monitor the environment for infrequent targets (for example, vigilance[38]), and is the region most densely innervated by noradrenergic projections from the locus coeruleus[39] that are thought to mediate vigilance and arousal. Damage to the right TPJ can cause vigilance problems in patients with unilateral neglect[40]. Changes in the level of vigilance have a slower time course than shifts of attention[41] and, therefore, might produce stronger and more sustained right TPJ responses.

A dissociation of function between intraparietal and temporoparietal cortex may explain why a verbal cue directing attention toward the contralesional field can transiently reduce neglect in some neglect patients, who typically have damage in the right TPJ region. This effect, extensively used by therapists to ameliorate unilateral visual neglect[42,43], may reflect the preserved activation of the IPs, which mediates the allocation of attention by cognitive cues. Normal orienting, however, can be maintained only for a short time in neglect patients based on voluntary strategies. Typically, orienting involves both cognitive and sensory-driven mechanisms[27]. This study, along with lesion analyses of patients with TPJ damage, indicates that the right TPJ region is critical for visual reorienting, and dissociates this region from voluntary orienting in nearby IPs.

## Methods

**Subjects.** Thirteen subjects (6 female, 7 male; aged 18–38) were recruited from the Washington University community following procedures approved by the local human studies committee. All subjects were strongly right-handed as measured by the Edinburgh handedness inventory[44] and had normal or corrected-to-normal visual acuity and no significant abnormal neurological history. Informed consent was obtained from each subject. Before the MR session, subjects participated in one behavioral session during which they were trained to perform the task while maintaining central fixation. Eye movements were monitored with electro-oculography, and all subjects were able to perform the task without breaks of fixation (resolution, 1.5°). Eye movements were not recorded during the fMRI session. Although we cannot rule out the occurrence of small eye movements during the fMRI session, several arguments diminish the likelihood of this possibility. The visual set-up was identical to the one used in the psychophysical session, in which eye movements were monitored and found negligible. The detection task was not demanding in terms of acuity. Subjects reported no problems in maintaining fixation, in agreement with many studies involving detection tasks[27]. Additionally, there was no activity in the frontal eye field during the noise period, when the tendency to look at the peripheral box was strongest. Reaction times were not collected from one subject because of equipment malfunction.

**Apparatus.** Stimuli were generated by an Apple Power Macintosh computer and projected onto a screen at the head of the bore by a Sharp LCD projector. Subjects viewed the stimuli through a mirror attached to the head coil. Subjects recorded behavioral responses by pressing an MRI-compatible fiber-optic key held in the right hand.

**Data analysis and fMRI scan acquisition.** An asymmetric spin-echo, echoplanar imaging sequence was used to measure blood oxygenation-level-dependent (BOLD) contrast (TR = 2.36 s, TE = 50 ms, flip angle = 90°). Each scan consisted of 128 frames during which 16 contiguous 8 mm axial slices were acquired (3.75 × 3.75 mm in-plane resolution). Anatomical images were acquired using a sagittal MP-RAGE sequence (TR = 97 ms, TE = 4 ms, flip angle = 12°, inversion time T1 = 300 ms). Functional data were realigned within and across runs to correct for head movement and coregistered with anatomical data. Whole-brain normalization was applied to equalize signal intensity across subjects. In each subject, hemodynamic responses (8 frames long) were estimated at the voxel level using the general linear model. The design matrix was defined using impulse-basis functions such that at each frame, the data were modeled as the sum of the overlapping hemodynamic response produced by each task effect and a linear trend. The use of catch trials (trials in which only the cue stimulus was present) made it possible to estimate unique responses for the cue, noise, target-valid, and target-invalid periods even though the cue response overlaps noise and target responses in each full trial[17] (J.M.O. *et al.*, *Soc. Neurosci. Abstr.* **24**, 1178, 1998). A random-effects analysis was performed by entering the individual time points of each hemodynamic response into voxel-level ANOVAs[45]. These ANOVAs had two main effects, time and task. The main effect of time was used to determine which voxels were activated. The resulting *F*-maps were corrected for multiple comparisons using a Gaussian random fields approach[46]. *F*-statistics at each voxel were converted to equivalent *Z*-statistics. These *Z*-maps were used to delineate regions of interest. Separate ANOVAs were then run at the regional level to determine the task effect of cue direction (left, right), noise direction (left, right), visual field of the target (left, right) and target validity (valid, invalid).

## Acknowledgements

*This research was supported by NIH EY00379 and EY12148 (M.C.). We thank Thomas Conturo, Avi Snyder and Erbil Akbudak for technical support.*

Received 27 September 1999; accepted 6 January 2000

1. Mesulam, M. M. A cortical network for directed attention and unilateral neglect. *Ann. Neurol.* **10**, 309–315 (1981).
2. Heilman, K. M., Watson, R. T. & Valenstein, E. in *Clinical Neuropsychology* (eds. Heilman, K. M. & Valenstein, E.) 243–293 (Oxford, New York, 1985).
3. Halligan, P. W. & Marshall, J. C. Toward a principled explanation of unilateral neglect. Special Issue: The cognitive neuropsychology of attention. *Cognit. Neuropsychol.* **11**, 167–206 (1994).
4. Posner, M. I., Walker, J. A., Friedrich, F. J. & Rafal, R. D. Effects of parietal injury on covert orienting of attention. *J. Neurosci.* **4**, 1863–1874 (1984).
5. Morrow, L. A. & Ratcliff, G. The disengagement of covert attention and the neglect syndrome. *Psychobiology* **16**, 261–269 (1988).
6. Di Pellegrino, G. Clock-drawing in a case of left visuospatial neglect: a deficit of disengagement? *Neuropsychologia* **33**, 353–358 (1995).
7. Friedrich, F. J., Egly, R., Rafal, R. D. & Beck, D. Spatial attention deficits in humans: a comparison of superior parietal and temporo-parietal junction lesions. *Neuropsychology* **12**, 193–207 (1998).
8. Bushnell, M. C., Goldberg, M. E. & Robinson, D. L. Behavioral enhancement of visual responses in monkey cerebral cortex. I. Modulation in posterior parietal cortex related to selective attention. *J. Neurophysiol.* **46**, 755–772 (1981).
9. Colby, C. L., Duhamel, J. R. & Goldberg, M. E. Visual, presaccadic, and cognitive activation of single neurons in monkey lateral intraparietal area. *J. Neurophysiol.* **76**, 2841–2852 (1996).
10. Snyder, L. H., Batista, A. P. & Andersen, R. A. Coding of intention in the posterior parietal cortex. *Nature* **386**, 167–170 (1997).
11. Gottlieb, J. P., Kusunoki, M. & Goldberg, M. E. The representation of visual salience in monkey parietal cortex. *Nature* **391**, 481–484 (1998).
12. Corbetta, M., Miezin, F. M., Shulman, G. L. & Petersen, S. E. A PET study of visuospatial attention. *J. Neurosci.* **13**, 1202–1226 (1993).
13. Nobre, A. C. *et al.* Functional localization of the system for visuospatial attention using positron emission tomography. *Brain* **120**, 515–533 (1997).
14. Vandenberghe, R. *et al.* The influence of stimulus location on the brain activation pattern in detection and orientation discrimination-a PET study of visual attention. *Brain* **119**, 1263–1276 (1996).
15. Gitelman, D. R. *et al.* Functional imaging of human right hemispheric activation for exploratory movements. *Ann. Neurol.* **39**, 174–179 (1996).
16. Corbetta, M. *et al.* A common network of functional areas for attention and eye movements. *Neuron* **21**, 761–773 (1998).
17. Shulman, G. L. *et al.* Areas involved in encoding and applying directional expectations to moving objects. *J. Neurosci.* **19**, 9480–9496 (1999).
18. Zarahn, E., Aguirre, G. & D'Esposito, M. A trial-based experimental design for fMRI. *Neuroimage* **6**, 122–138 (1997).
19. Friston, K. J., Josephs, O., Rees, G. & Turner, R. Nonlinear event-related responses in fMRI. *Magn. Reson. Med.* **39**, 41–52 (1998).
20. Kastner, S., Pinsk, M. A., De Weerd, P., Desimone, R. & Ungerleider, L. G. Increased activity in human visual cortex during directed attention in the absence of visual stimulation. *Neuron* **22**, 751–761 (1999).
21. Chawla, D., Reese, G. & Friston, K. J. The physiological basis of attentional modulations in visual areas. *Nat. Neurosci.* **2**, 671–676 (1999).
22. Dupont, P. *et al.* The kinetic occipital region in human visual cortex. *Cereb. Cortex* **7**, 283–292 (1997).
23. Mendola, J. D., Dale, A. M., Fischl, B., Liu, A. K. & Tootell, R. B. The representation of illusory and real contours in human cortical visual areas revealed by functional magnetic resonance imaging. *J. Neurosci.* **19**, 8560–8572 (1999).
24. Watson, J. D. *et al.* Area V5 of the human brain: evidence from a combined study using positron emission tomography and magnetic resonance imaging. *Cereb. Cortex* **3**, 79–94 (1993).
25. Tootell, R. B. H. *et al.* Functional analysis of human MT and related visual cortical areas using magnetic resonance imaging. *J. Neurosci.* **15**, 3215–3230 (1995).
26. Tootell, R. B. H. *et al.* The retinotopy of visual spatial attention. *Neuron* **21**, 1409–1422 (1998).
27. Posner, M. I. Orienting of attention. *Q. J. Exp. Psychol.* **32**, 3–25 (1980).
28. Desimone, R. & Duncan, J. Neural mechanisms of selective visual attention. *Annu. Rev. Neurosci.* **18**, 193–222 (1995).
29. Le, T. H., Pardo, J. V. & Hu, X. 4T-fMRI study of nonspatial shifting of selective attention: cerebellar and parietal contributions. *J. Neurophysiol.* **79**, 1535–1548 (1998).
30. Lumer, E. D., Friston, K. J. & Rees, G. Neural correlates of perceptual rivalry in the human brain. *Science* **280**, 1930–1934 (1998).
31. Colby, C. L., Gattass, R., Olson, C. R. & Gross, C. G. Topographic organization of cortical afferents to extrastriate visual area PO in the macaque: a dual tracer study. *J. Comp. Neurol.* **238**, 1257–1299 (1988).
32. Gnadt, J. W. & Andersen, R. A. Memory related motor planning activity in posterior parietal cortex of macaque. *Exp. Brain Res.* **70**, 216–220 (1988).
33. Culham, J. C. *et al.* Cortical fMRI activation produced by attentive tracking of moving targets. *J. Neurophysiol.* **80**, 2657–2670 (1998).
34. Courtney, S. M., Ungerleider, L. G., Keil, K. & Haxby, J. V. Transient and sustained activity in a distributed neural system for human working memory. *Nature* **386**, 608–611 (1997).
35. Cohen, J. D. *et al.* Temporal dynamics of brain activation during a working memory task. *Nature* **386**, 604–607 (1997).
36. Mesulam, M.-M. Large-scale neurocognitive networks and distributed processing for attention, language, and memory. *Ann. Neurol.* **28**, 597–613 (1990).
37. Knight, R. T. & Scabini, D. Anatomic bases of event-related potentials and their relationship to novelty detection in humans. *J. Clin. Neurophysiol.* **15**, 3–13 (1998).
38. Pardo, J. V., Fox, P. T. & Raichle, M. E. Localization of a human system for sustained attention by positron emission tomography. *Nature* **349**, 61–64 (1991).
39. Foote, S. L. & Morrison, J. H. Extrathalamic modulation of cortical function. *Annu. Rev. of Neurosci.* **10**, 67–96 (1987).
40. Robertson, I. H., Mattingley, J. B., Rorden, C. & Driver, J. Phasic alerting of neglect patients overcomes their spatial deficit in visual awareness. *Nature* **395**, 169–172 (1998).
41. Parasuraman, R., Warm, J. S. & See, J. E. in *The Attentive Brain* (ed. Parasuraman, R.) 221–256 (MIT Press, Cambridge, Massachusetts, 1998).
42. Weinberg, J. *et al.* Visual scanning training effects on reading-related tasks in acquired brain-damage. *Arch. Phys. Med. Rehabil.* **58**, 479–486 (1977).
43. Antonucci, G. *et al.* Effectiveness of neglect rehabilitation in a randomized group study. *J. Clin. Exp. Neuropsychol.* **17**, 383–389 (1995).
44. Raczkowski, D., Kalat, J. W. & Nebes, R. Reliability and validity of some handedness questionnaire items. *Neuropsychologia* **12**, 43–47 (1974).
45. Braver, T. S. *et al.* A parametric study of prefrontal cortex involvement in human working memory. *Neuroimage* **5**, 49–62 (1997).
46. Worsley, K. J. *et al.* A unified statistical approach for detemining significant signals in images of cerebral activation. *Hum. Brain Mapp.* **4**, 58–73 (1995).