Neural Correlates of Change Detection and Change Blindness in a Working Memory Task

Luiz Pessoa[1] and Leslie G. Ungerleider[2]

[1]Department of Psychology, Brown University, Providence, RI, USA and [2]Laboratory of Brain and Cognition, National Institute of Mental Health, Bethesda, MD, USA

Detecting changes in an ever-changing environment is highly advantageous, and this ability may be critical for survival. In the present study, we investigated the neural substrates of change detection in the context of a visual working memory task. Subjects maintained a sample visual stimulus in short-term memory for 6 s, and were asked to indicate whether a subsequent, test stimulus matched or did not match the original sample. To study change detection largely uncontaminated by attentional state, we compared correct change and correct no-change trials at test. Our results revealed that correctly detecting a change was associated with activation of a network comprising parietal and frontal brain regions, as well as activation of the pulvinar, cerebellum, and inferior temporal gyrus. Moreover, incorrectly reporting a change when none occurred led to a very similar pattern of activations. Finally, few regions were differentially activated by trials in which a change occurred but subjects failed to detect it (change blindness). Thus, brain activation was correlated with a subject's report of a change, instead of correlated with the physical change *per se*. We propose that frontal and parietal regions, possibly assisted by the cerebellum and the pulvinar, might be involved in controlling the deployment of attention to the location of a change, thereby allowing further processing of the visual stimulus. Visual processing areas, such as the inferior temporal gyrus, may be the recipients of top-down feedback from fronto-parietal regions that control the reactive deployment of attention, and thus exhibit increased activation when a change is reported (irrespective of whether it occurred or not). Whereas reporting that a change occurred, be it correctly or incorrectly, was associated with strong activation in fronto-parietal sites, change blindness appears to involve very limited territories.

Keywords: attention, fMRI, working memory

Introduction

Detecting changes in an ever-changing environment is highly advantageous, and this ability may be critical for survival. In the real world, changes are often accompanied by transients of some sort, such as motion signals that attract attention to their location (Remington *et al.*, 1992). Evidence indicates that when an item is seen to change, attention is drawn to the location of that item to facilitate visual processing. For example, Thornton and Fernandez-Duque (2000) showed that subjects are faster to discriminate a subsequent target at the location of a change than at a distant location (see also Smilek *et al.*, 2000; Rensink, 2002), indicating that a change can function as an orienting cue. However, changes may occur in the absence of accompanying transients, such as those occurring during saccades, blinks, or flicker. Under these circumstances, changes can be quite difficult to detect, and even large changes may go unnoticed in the absence of focused attention (Rensink *et al.*, 1997; Rensink, 2002). In this view, attention must be directed to the region of space in which a change occurs at the time the change takes place. When this is not the case because of, say, saccades or flicker, 'change blindness' will ensue (e.g. an engine repeatedly appearing and disappearing from the photograph of an airplane may go unnoticed). In most instances, however, changes in the environment are in fact accompanied by transients. Thus, change detection is associated with two related, but distinct, events, namely, a reactive deployment of attention that can have an orienting function, and a goal-directed allocation of attention that may be instrumental in allowing changes to be perceived in demanding situations.

In a recent functional magnetic resonance imaging (fMRI) study, Beck *et al.* (2001) investigated the neural substrates of change detection in an experiment in which subjects indicated whether there was a change or not in stimuli containing either faces or houses. They showed that change detection (i.e. detected vs undetected changes) was associated with enhanced activation in bilateral superior parietal lobule and right middle frontal gyrus. They also observed change-related activation in the fusiform gyrus, a region likely involved in the processing of the stimuli employed. From these results, it was concluded that a fronto-parietal network of regions is involved in the awareness of a change. However, because the contrast of detected versus undetected changes involved correct (detected) and incorrect (undetected) trials, the activations were possibly contaminated by variations in the subject's attentional state (cf. Ress *et al.*, 2000; Pessoa *et al.*, 2002). Fluctuations in attention are a reason for concern because spatially directed attention strongly modulates processing in sensory areas, such as the fusiform gyrus (Wojciulik *et al.*, 1998), and relies on a set of regions that includes those in parietal and frontal cortex observed by Beck *et al.* (for a review, see Kastner and Ungerleider, 2000). We therefore thought it would be important to re-examine change detection under conditions that are more closely matched for attentional state. In this manner, contributions from 'goal-directed attention' would be closely matched, thereby revealing the neural correlates of 'reactive attention' during the detection of visual change.

In the present study, we investigated the neural correlates of change detection in a working memory (WM) task. Subjects maintained a sample visual stimulus in short-term memory for 6 s, and were asked to indicate whether a subsequent, test stimulus matched or did not match the original sample. To study change detection largely uncontaminated by attentional state, we compared correct change and correct no-change trials. Because we were interested in the question of change detection, all statistical analyses were performed on the test phase of the WM task, i.e. at the time of change detection.

Cerebral Cortex May 2004;14:511–520; DOI: 10.1093/cercor/bhh013

Analyses related to activations during encoding and memory maintenance of stimuli have been reported elsewhere (Pessoa *et al.*, 2002). The design of our task enabled us not only to compare correct change and no-change trials, but also to probe activations during false alarm trials in which no physical change occurred but subjects reported perceiving one. By analyzing such trials it was possible to determine whether brain activations were associated with a subject's *report* of a target change. Based on findings in the monkey brain (e.g. Thompson and Schall, 1999, 2000), we hypothesized that brain activation would follow the subject's reporting of a change and not the physical presence of a change. Thus, we predicted that reporting a change when none occurred would be virtually equivalent, at the neural level, to reporting a change when it in fact had occurred.

Materials and Methods

Subjects

Nine healthy subjects (five women, 22–36 years old) participated in the study, which was approved by the National Institute of Mental Health (NIMH) Institutional Review Board. All subjects were in good health with no past history of psychiatric or neurological disease and gave informed written consent. Subjects had normal or corrected-to-normal (with contact lenses) visual acuity.

Visual Task

There were three experimental conditions: working memory (WM), fixation control (FC), and detection (DT; results related to the latter condition are not presented in this paper and will not be discussed further). Each run comprised 24 trials in random order (16 WM, 4 FC and 4 DT) with each trial lasting 14 s. The stimuli employed in WM trials consisted of a fixation spot (0.2°) and eight bars (1°) positioned around fixation. The orientation of the bars was vertical, horizontal, and oblique (+45° or –45°), chosen randomly for each display. In each run, half of the WM trials (8/16) involved a single change in the visual display (i.e. on these non-match trials, one of the bars in the test display changed orientation compared with the sample display), and half did not involve a change (i.e. the sample and test displays were identical). Subjects participated in 8–10 runs, each lasting 5 min 36 s (with a 1–2 min rest period between runs).

The temporal structure of the trials is indicated in Figure 1. In WM trials, after a 1 s fixation, a sample visual display was presented for 0.5 s, followed by a 6 s fixation, and a test display for 0.5 s. Subjects were then prompted by a display with the letter 'm' (for memory) to indicate 'same' or 'different' by using two hand-held buttons (right and left hand, respectively). 'Same' meant the test matched the sample, and 'different' meant it did not match. Subjects also indicated the confidence level of their response by indicating 'high' or 'low' (right and left hand, respectively) when 'c' appeared on the display. Each of the two response periods lasted 2 s. Finally, a blank screen terminated the trial, which lasted 2 s (inter-trial interval). Subjects were instructed to maintain fixation for those displays with a fixation spot. FC trials did not have any maintenance demands. On these trials, subjects were instructed to maintain fixation and press both buttons in both response periods. Before the actual scan session, subjects underwent a practice session in which they performed 5–6 runs in order to become familiar with the task.

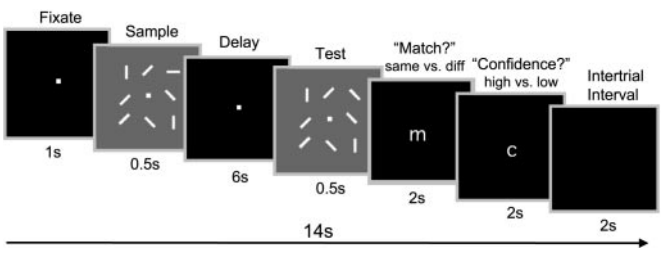

Figure 1. Experimental design. In WM trials, subjects indicated whether the sample and test displays were the same or different (note that the bar orientation on the upper right changed in the present case). They also indicated the confidence of their response (high vs low).

MRI Data Acquisition

Images were acquired with a 3.0 T GE Signa scanner (Milwaukee, WI) using a custom-made head coil (IGC-Medical Advances, Milwaukee, WI). Subjects were tested in a scanning session that lasted ~2 h. Functional images were taken with a gradient echo echo-planar imaging sequence (T_R = 2 s; T_E = 30 ms; flip angle = 90°; 64 × 64 matrix; FOV = 24 cm). Whole-brain coverage was obtained with 32 sagittal slices (thickness, 5 mm; in-plane resolution, 3.75 × 3.75 mm). Echo-planar images were co-registered to a high-resolution anatomical scan of the same subject's brain taken in the same session (3D SPGR, T_R = 15 ms, T_E = 5.4 ms, flip angle = 45°, 256 × 256 matrix, FOV = 24 cm, 124 sagittal slices, thickness, 1.2 mm).

Visual stimuli were rear-projected onto a translucent screen placed outside the bore of the magnet. Stimuli were viewed from inside the magnet via a mirror system attached to the head coil. The first scanner pulse of each functional run synchronized MR acquisition with visual presentation.

Data Analysis

WM trials were sorted according to whether they were correct or incorrect, and whether subjects reported high or low confidence. Trials in which the subjects did not respond at both response periods were discarded in subsequent analyses. All of the analyses reported here employed high-confidence trials only. In this fashion the contribution from guessing was minimized. There were two types of correct trials, one without a change in the display (correct match trials, also called 'hits') and the other involving a change (correct non-match trials, also called 'correct rejects'). There were also two types of incorrect trials, one without a change in the display (incorrect match trials, also called 'false alarms') and the other involving a change (incorrect non-match trials, also called 'misses').

Functional data were smoothed with an isotropic 8 mm Gaussian kernel (FWHM) and analyzed with multiple regression (Friston *et al.*, 1995). For the analyses, a set of regressors were defined and convolved with a canonical hemodynamic impulse response function in order to account for response lag and dispersion (Cohen, 1997). Such regressors modeled the effects of encoding, maintenance, and test. Here, we mainly present analyses related to change detection for which the statistical contrasts pertain to the test phase of the WM task. Analyses related to the encoding and delay phases of the task, independent of performance, are presented in order to allow for the evaluation of the overlap between change-related activations and those during encoding and delay. Performance-related analyses during encoding, memory maintenance, as well as the test phase, have been published elsewhere (Pessoa *et al.*, 2002).

Two types of analysis were performed on correct trials: fixed-effects and random-effects; both were carried out in AFNI (Cox, 1996). The fixed-effects analysis is useful for the inspection of the regions that were differentially activated, for instance, in the form of voxel-wise maps. At the same time, the random-effects analysis is important to determine activations that are robust and reliable.

The fixed-effects analysis employed standard multiple regression methods (Friston *et al.*, 1995). The linear models included a constant term and a linear term (for every run) that served as covariates of no interest (these terms controlled for drifts of MR signal across and within runs). F-maps of the contrasts of interest were generated for each individual. Fixed-effects statistical group maps were then obtained by converting each individual's F-map into a Z-map and then combining these into a composite final Z-map. For that purpose, each individual's brain was transformed with AFNI into the standard coordinate space of Talairach and Tournoux (1988). These transformed maps were then combined (averaged together and multiplied by the square root of the number of subjects). Because this analysis is uncorrected for multiple comparisons, a P value of 0.001 was chosen for statistical significance.

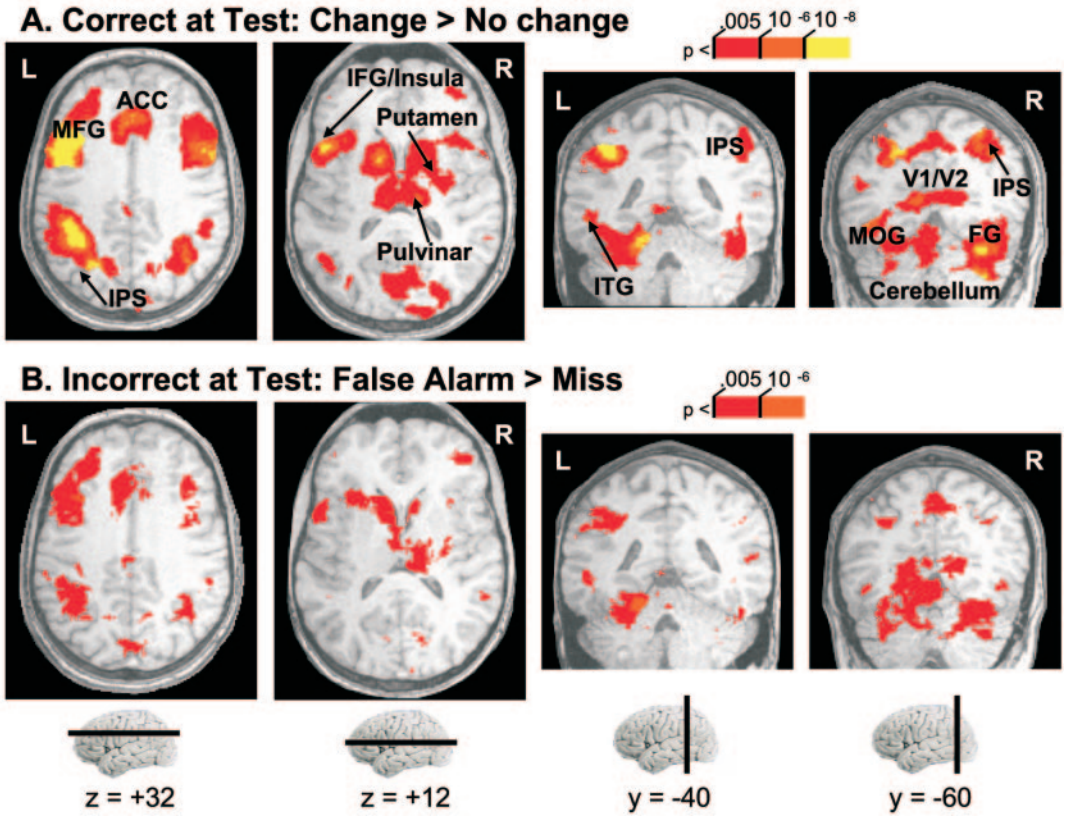

Figure 2. Similar brain activations occur on correct change and false alarm trials. (*A*) Functional group maps showing regions activated at test on correct change (non-match) compared with no-change (match) trials. (*B*) Functional group maps showing regions activated at test on incorrect no-change (false alarms) compared with incorrect change (miss) trials, at the same slice levels. Although the number of high-confidence, incorrect trials was small and the associated activations weaker, comparing the two patterns of activation revealed a great deal of overlap. Statistical group maps are shown overlaid on structural scans from a representative individual. The level of the axial and coronal sections is indicated on the small whole-brain insets. The color bar indicates *P* values (uncorrected).

For correct trials, an additional random-effects analysis was performed. First, the parameter estimates (beta weights) provided by multiple regression were obtained for regressors modeling correct reject (change) trials and hit (no change) trials. Then, we performed a paired *t*-test comparing the magnitudes of the parameter estimates for correct reject and hit trials at each voxel. Because this analysis is very conservative, a *P* value of 0.05 was considered for statistical significance.

For incorrect trials, few trials were available for the comparison of false alarms versus misses, and the associated statistical maps would have been relatively noisy. Thus, we initially determined the voxels with a significant response during the test phase of the task irrespective of trial type by applying a *t*-test to assess whether beta weights associated with the encoding phase were significantly greater than zero. Because, at the group level, these responses were very robust, we employed a threshold of 10^{-10} for significance (note that the specific threshold does not affect the results in any major way). The significant voxels were then used to create a mask, such that the results of the contrast of false alarms versus misses could be shown for locations within the pre-defined mask (Fig. 2*B*). For comparison, the results of the contrast of correct change versus correct no-change trials are shown in the same manner (Fig. 2*A*). Thus, the two contrasts shown in Figure 2 employ identical thresholding and color schemes.

To generate the summary time series shown in Figure 4, we employed the peak Talairach coordinate from the random-effects analysis (see Table 1) and extracted the associated time series for each individual. Signals were averaged according to the experimental condition in question, and then averaged across subjects to obtain the final values. Results are expressed in terms of per cent signal increase relative to fixation trials.

Results

Behavioral Results

Invalid trials, in which subjects did not respond at either response period were extremely rare (less than 3% overall), and were not included in the analysis. Mean performance across subjects was 71.4% correct for high-confidence trials (high-confidence trials comprised 53% of the total number of trials). No significant difference in reaction time (RT) was observed for correct and incorrect trials (mean ± SD: correct: 899 ± 138 s; incorrect: 936 ± 181 s; $P > 0.05$, *t*-test). For the low-confidence trials, mean performance dropped to 60.8% correct, indicating that indeed guessing came into play on these trials. All fMRI analyses reported here employed high-confidence trials only. There were two types of correct trials, one without a change in the display (correct match trials, also called 'hits') and the other involving a change (correct non-match trials, also called 'correct rejects'). Per cent correct for hits and correct rejects were 74% and 67%, respectively, which did not differ significantly ($P > 0.5$). Behaviorally, no significant difference in RT was observed for these two types of correct trials (mean ± SD: match: 826.39 ± 158.97 s; non-match: 906 ± 162 s; $P > 0.05$, *t*-test). There were also two types of incorrect trials, one without a change in the display (incorrect match trials, also called 'false alarms') and the other involving a change (incorrect non-match trials, also called 'misses'). Behaviorally, no significant difference in RT was observed for these two types of incorrect trials (mean ± SD: match: 981.89 ± 178.07 s; non-match: 919.94 ± 273.43 s; $P > 0.05$, *t*-test).

Table 1
Change detection: correct change trials > correct no-change trials

Region	Hemisphere	Talairach coordinates			Brodmann area	*Z* score
		X	*Y*	*Z*		
ITG	L	–48	–46	–13	37	4.7
	R	59	–37	–13	20/21	4.0
Anterior IPS	L	–37	–46	35	40	7.0
	R	33	–40	35	40	6.0
Precuneus	R	29	–77	34	19	3.7
Central sulcus	R	36	–28	47	4	10.0
SFG	L	–5	9	55	6/8	6.3
MFG	L	–41	15	31	9	8.0
	R	46	12	33	9	6.0
IFG/Anterior insula	L	–45	11	12	44/45	5.7
ACC	L	–8	23	30	32	4.0
Putamen	R	20	9	10		4.3
Pulvinar	R	13	–23	6		6.0
Cerebellum	R	26	–63	–46		6.0

ACC: anterior cingulate cortex; IFG: inferior frontal gyrus; IPS: intraparietal sulcus; ITG: inferior temporal gyrus; MFG: middle frontal gyrus; SFG: superior frontal gyrus.

Table 2
Incorrect at test: false alarm > miss

Region	Hemisphere	Talairach coordinates			Brodmann area	*Z* score
		X	*Y*	*Z*		
ITG	L	–51	–41	–13	20/37	3.7
	R	58	–38	1	21	4.0
Anterior IPS	L	–40	–44	35	40	3.3
	R	30	–40	47	40	4.7
Precuneus	R	20	–77	30	19	3.7
Central sulcus	R	36	–28	47	4	3.3
SFG	L	–5	11	55	6/8	3.7
MFG	L	–27	15	31	8/9	5.3
	R	35	12	27	9	3.3
IFG/Anterior insula	L	–53	13	12	44	4.3
ACC	L	–8	26	30	32	4.3
Subgenual ACC	L	–7	27	4	24	6.0
Putamen	L	–13	9	3		3.7
Pulvinar	R	13	–22	6		4.3
Cerebellum	R	26	–63	–34		3.3

ACC: anterior cingulate cortex; IFG: inferior frontal gyrus; IPS: intraparietal sulcus; ITG: inferior temporal gyrus; MFG: middle frontal gyrus; SFG: superior frontal gyrus.

Activity Related to Change Detection: Correct Trials Only

To investigate brain regions engaged by change detection while minimizing potential contributions from varying attentional states, we compared only *correct* change (correct reject) versus *correct* no-change (hit) trials at the test phase of the WM task. Note that this comparison was uncontaminated by differences in RT, which did not differ for these two types of trials. Greater activation for change trials was observed across a wide network of brain regions (Table 1; Fig. 2). Frontal regions included the superior frontal gyrus (SFG, BA 6/8), the middle frontal gyrus (MFG, BA 9), the anterior cingulate cortex (ACC, BA 32), and the inferior frontal gyrus (IFG)/anterior insula (BA 44/45). Parietal regions included the anterior intraparietal sulcus (IPS, BA 40) and, medially, the precuneus (BA 19). Visual processing regions included the inferior temporal gyrus (ITG, BA 37 on the left, BA 20/21 on the right) and, with a less exacting fixed-effects analysis, the cortex within and around the calcarine fissure (BA 17/18). At the subcortical level, the right putamen, the cerebellum and pulvinar, both mainly on the right, showed greater activation on correct trials for the change compared with no-change contrast. Because the cerebellar activation was bilateral, it could not be attributed solely to button presses.

Correct change and no-change trials involved making responses with the left and right hands, respectively. Thus, as expected, we observed a large activation within and surrounding the right central sulcus (i.e. within motor cortex) for the contrast of correct change > correct no-change trials. Other activations reported for the contrast of these conditions could, in theory, also reflect contributions of hand-specific motor responses or motor response preparation. We find this possibility unlikely, however, because the reverse contrast, namely, correct no-change trials > correct change trials, revealed no differential activation aside from the left central sulcus, whose activation was related to a right button press on no-change trials, i.e. no hand-specific motor-related responses outside of motor cortex were observed.

Activity Related to False Alarms

Were these same areas also more active when the subjects reported a change but none had actually occurred? If this were true, then the pattern of activation for false alarms (on incorrect match trials) compared to misses (on incorrect non-match trials) should be similar to the one observed for rejects (on correct non-match trials) compared to hits (on correct match trials). Note that for false alarm trials, no physical change occurred, but subjects reported perceiving a change with high confidence; for miss trials, a physical change occurred, but subjects reported that no change occurred, again with high confidence.

The comparison of false alarm and miss trials elicited a pattern of activation very similar to that observed in the comparison between correct change and no-change trials (Fig. 2; see Methods). In fact, all the regions listed in Table 1 were differentially activated for false alarm compared to miss trials (see Table 2; note, however, that the putamen site switched from the right to the left hemisphere). It thus appears that reporting a change when none has occurred is virtually equivalent, in regional brain activations, to reporting a change when it has in fact occurred. These results are especially noteworthy given that only a limited number of false alarm trials were available for analysis; overall ~30% of the trials were incorrect and there were even fewer trials in which subjects reported a change when none had occurred.

What is the origin of a false alarm? One possibility is that increased activity in visual processing areas provides a signal that is used by the subjects to indicate, albeit incorrectly, that a change occurred. In our study, dorsal occipital cortex, the ITG, and the posterior calcarine fissure appeared to be

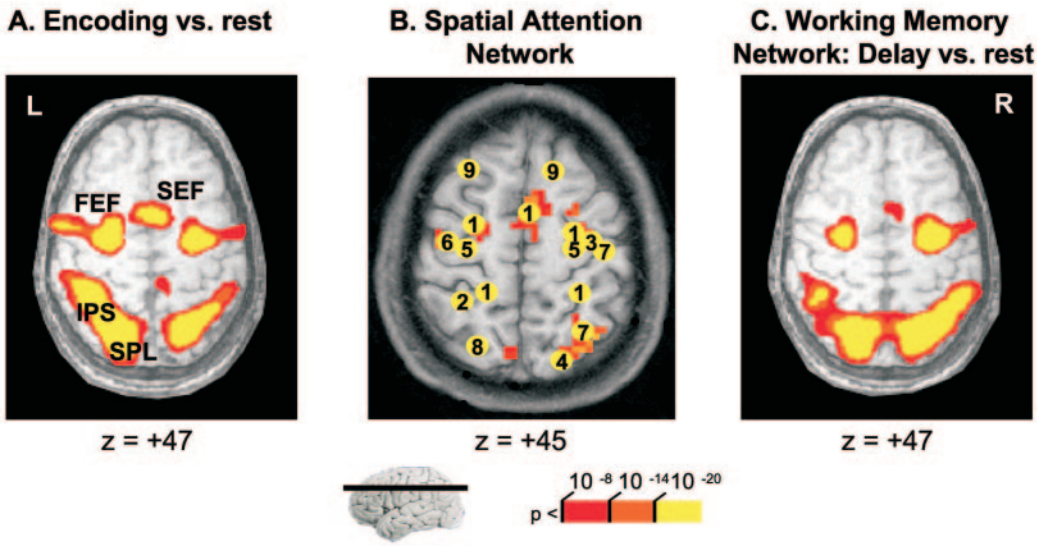

Figure 3. Regions involved in visual spatial attention and visual WM. (*A*) Encoding versus rest. (*B*) Regions in the spatial attention network as determined by a meta-analysis of imaging data by Kastner and Ungerleider (2000). (*C*) Working memory network revealed by the contrast of WM delay versus rest. The statistical group map is shown overlaid on a structural scan of a representative individual. The level of the axial section is indicated on the small whole-brain inset.

involved in the visual processing of the stimulus display, as these regions generated transient activity at encoding and at test (Pessoa *et al.*, 2002). When contrasting false alarm to miss trials, among these visual regions responding transiently to our stimuli, the strongest sites of activation were in the left and right ITG (BA 20/37 on the left, BA 21 on the right), sometimes extending into the adjacent fusiform gyrus. We thus tested whether an increase in activity in the left or right ITG made it more likely that the subjects would report a change by performing a logistic regression analysis. The results showed that, for the right ITG, elevated activity at 10 s after the trial began was indeed associated with a higher likelihood that the subject would report a change that did not occur ($P < 0.05$). For a 1% increase in fMRI signal amplitude, the probability of incorrectly reporting a change was 67%. For the left ITG, as well as posterior calcarine and dorsal occipital cortex, the logistical regressions were not statistically significant. Because the test stimulus is presented for half a second starting at 7.5 s from trial onset, if one assumes roughly a 2 s lag for the initial rise of the hemodynamic response (Cohen *et al.*, 1997; Bandettini, 1999), then activity evoked by the test stimulus should start to rise at ~9.5 s. As the ITG did not exhibit sustained activation during the delay (Pessoa *et al.*, 2002), we suggest that activity ~10 s largely reflects activity due to the test stimulus or, conceivably, just prior to its presentation. Thus, it appears that elevated activity in the right ITG around the time of the test stimulus might contribute to the subject's reporting that a change occurred, even when none was physically present.

Change Blindness: Activity Related to Miss Trials

We also determined voxels more strongly activated by trials in which a change occurred but subjects did not detect it (miss trials) compared to trials in which a change did not occur but subjects reported seeing it (false alarm trials). This comparison is interesting because it reveals activation associated with a physical change in the stimulus independent of the subject's subjective experience of detecting it, namely *change blindness*. In contrast to the explicit report of change detection, we found activations during change blindness in few sites (Fig. 3; Table 3), which included the right parahippocampal gyrus (BA 20), the posterior IPS bilaterally (BA 7), the posterior occipital cortex (cuneus, BA 18), the left central sulcus (BA 4), the left precentral gyrus (BA 6), the supplementary eye field (SEF, BA 6/32), the right IFG/anterior insula (BA 44), and right cerebellum. Activations in both the left central sulcus and left precentral gyrus likely are related to right-hand button presses when subjects incorrectly indicate that no change occurred.

Table 3
Change blindness: incorrect at test: miss > false alarm

Region	Hemisphere	Talairach coordinates			Brodmann area	*Z* score
		X	*Y*	*Z*		
Parahippocampal gyrus	R	25	–37	–22	20	3.9
Posterior IPS	L	–23	–66	27	7	5.1
	R	21	–66	40	7	3.9
Cuneus		0	–79	15	18	3.5
Central sulcus	L	–33	–27	53	4	5.7
Precentral gyrus	L	–32	–10	45	6	5.5
SEF		0	5	48	6/32	3.9
IFG/Anterior insula	R	47	12	11	44	3.7
Cerebellum	R	3	–65	–33		3.6

IFG: inferior frontal gyrus; IPS: intraparietal sulcus; SEF: supplementary eye field.

Comparison Between Networks for Change Detection, Spatial Attention, and Working Memory

As stated in the Introduction, we assume that activations evoked by the contrast of correct change versus correct no-change trials reflect the neural correlates of 'reactive attention', akin to the phenomenon of 'exogenously' driven attention. This was because 'goal-directed' attention was largely controlled for by comparing only correct trials. It was there-

fore instructive to compare the activations in the present study to activations due to goal-oriented attention. We pursued this in two ways. First we compared the present activations (i.e. at test) to the ones revealed by the contrast of the encoding phase of the trial relative to a similar period during control trials in which subjects simply viewed a blank screen. We reasoned that the latter contrast would reveal regions involved in goal-directed attention because, behaviorally, a key component of successfully performing the task involved directing attention to the to-be-encoded stimulus array (Pessoa *et al.*, 2002). The most robust activations revealed by this contrast, shown in Table 4 (which lists only the regions that survived the random-effects analysis at a P value of 0.05), involved a fronto-parietal network of regions consisting of the superior parietal lobule (SPL, BA 7), the anterior IPS (BA 40), the precuneus (BA 18/19), the precentral gyrus (BA 6), the frontal eye field (FEF, BA 6), the SEF (BA 6/32), the dorsolateral portion of the MFG (DLPFC, BA 46), and the IFG/anterior insula (BA 44). As indicated above, the key fronto-parietal sites revealed by the contrast of correct change versus correct no-change trials included the anterior IPS (BA 40), the precuneus (BA 19), the SFG (BA 6/8), the MFG (BA 9), the ACC (BA 32), and the IFG/anterior insula (BA 44). Thus, although there was overlap between the location of activations found during encoding and during change detection at test, such as the anterior IPS (see regions marked with footnote symbols in Table 4), a conspicuous feature of the comparison was the ***lack*** of overlap at more dorsal brain sites. In particular, the SPL, FEF and SEF were not strongly driven by the detection of a change; as shown in Table 1, none of these sites were revealed by the random-effects analysis. Conversely, regions strongly recruited during the detection of a change that were not recruited at encoding included the IFG (BA 44/45), the pulvinar and the cerebellum.

We also compared the activations in the present study to activations reported in a recent meta-analysis of spatial attention by Kastner and Ungerleider (2000). A common feature among the visuospatial tasks in the meta-analysis is that subjects were asked to maintain fixation at a central point and to direct attention covertly to peripheral target locations in order to detect a stimulus, to discriminate it, or to track its movement. In other words, these tasks involved endogenously driven, goal-directed attention rather than reactive attention. The meta-analysis revealed a fronto-parietal network of regions (Fig. 3*B*) consisting of areas in the SPL (BA 7), the IPS (BA 40), the FEF (BA 6/8), and the SEF (BA 8). In addition, but less consistently, activations in the lateral prefrontal cortex in the region of the MFG (BA 9/46) and the ACC (BA 32) have been reported. These activations closely resemble the pattern observed during encoding (compare Fig. 3*A* and *B*), supporting the idea that the latter reflect, to a large extent, goal-directed attention. Like the comparison above, the overlap between the location of change-related activations and goal-directed attention, as indicated by the meta-analysis, was quite limited.

Because the present task involved WM maintenance, our study provides the unique opportunity to compare activations due to attentional processes engaged by change detection and those engaged by WM maintenance in the same task and in the same subjects. Therefore, we compared the activations during the delay period of WM relative to rest to the activations evoked by the contrast of correct change versus correct no-change trials. WM delay-related activations were observed in several brain regions, including the SPL (BA 7), anterior IPS (BA 40), SEF (BA 6), and dorsolateral portions (DLPFC) of the MFG (BA 9/46) (Table 5), with the strongest activations observed in the SPL and SEF at z = +47 (Fig. 3*C*; note that the FEF was also strongly activated, though it did not survive the random-effects analysis). At the same time, the strongest focus of frontal and parietal activation associated with change detection was at z = +32. Of all the regions involved in change detection, only the anterior IPS also exhibited strong delay-related activation. Importantly, although delay-related and change detection acti-

Table 4
Working memory encoding > rest

Region	Hemisphere	Talairach coordinates			Brodmann area	*Z* score
		X	*Y*	*Z*		
MOG/ITG	L	–40	–65	2	37	10.3
	R	48	–56	–14	20/37	12.3
SPL	L	–21	–62	47	7	15.7
	R	17	–64	49	7	19.6
Anterior IPS[a]	L	–39	–46	41	40	10.6
	R	34	–46	52	40	12.9
Precuneus[a]	L	–18	–76	28	18/19	12.5
	R	26	–77	28	19	17.7
Precentral gyrus	L	–42	–3	31	6	14.0
	R	48	–3	40	6	17.8
FEF	L	–24	–8	47	6	15.0
	R	24	–9	45	6	16.3
SEF		0	1	50	6/32	12.5
MFG (DLPFC)	R	40	30	23	46	8.6
IFG/Anterior insula[a]	L	–32	13	12	44	6.6
	R	29	15	12	44	3.3
Putamen	L	–22	10	7		4.8
	R	19	5	10		4.7

DLPFC: dorsolateral prefrontal cortex; FEF: frontal eye field; IFG: inferior frontal gyrus; IPS: intraparietal sulcus; MFG: middle frontal gyrus; MOG: middle occipital gyrus; SEF: supplementary eye field; SPL: superior parietal lobule.
*Fronto-parietal areas in which activations overlapped with those for contrast of correct change > correct no change trials (see Table 1).

Table 5
Working memory delay > rest

Region	Hemisphere	Talairach coordinates			Brodmann area	*Z* score
		X	*Y*	*Z*		
SPL	L	–23	–60	47	7	19.5
	R	21	–58	47	7	13.5
Anterior IPS	L	–39	–41	40	40	13.8
	R	37	–36	40	40	16.0
SEF	L	–5	2	50	6	7.3
MFG (DLPFC)	L	–39	26	30	9/46	8.2
	R	45	32	26	9/46	7.8

DLPFC: dorsolateral prefrontal cortex; IPS: intraparietal sulcus; MFG: middle frontal gyrus; SEF: supplementary eye field; SPL: superior parietal lobule.

vation were both observed in the MFG, the former was located in a more anterior site (for the left and right, respectively, y = 26 and 32 for delay-related activity and y = 15 and 12 for change-related activity; see Tables 5 and 1, respectively). Thus, the most conspicuous feature of the networks activated by change detection and WM maintenance was that they were largely non-overlapping, with the notable exception of the anterior IPS, which was shared by both. To illustrate dynamic differences in the more dorsal and more ventral fronto-parietal networks, Figure 4 shows the responses of the left FEF and left MFG for correct change detection trials. The FEF exhibited sustained activation throughout the delay interval, consistent with its strong differential activation in the contrast of WM delay versus fixation trials. Such sustained behavior was also observed in other more dorsal regions, such as the SPL. On the other hand, the MFG showed no sustained response during the delay, but instead exhibited activity that appeared to be evoked around the time of the test stimulus. Again, a similar behavior was also observed in other more ventral regions, such as the IFG/anterior insula. Thus, it appears that attentional processes due to change detection recruit more ventral cortical territories in both frontal and parietal cortex than those engaged by WM maintenance.

We also compared the WM network (as obtained by the contrast of WM and fixation trials during maintenance) to the activations revealed by the meta-analysis of Kastner and Ungerleider (2000). Interestingly, the WM network strongly resembled the spatial attention network, with key activations in the FEF, SPL and IPS (Fig. 3*B* and *C*).

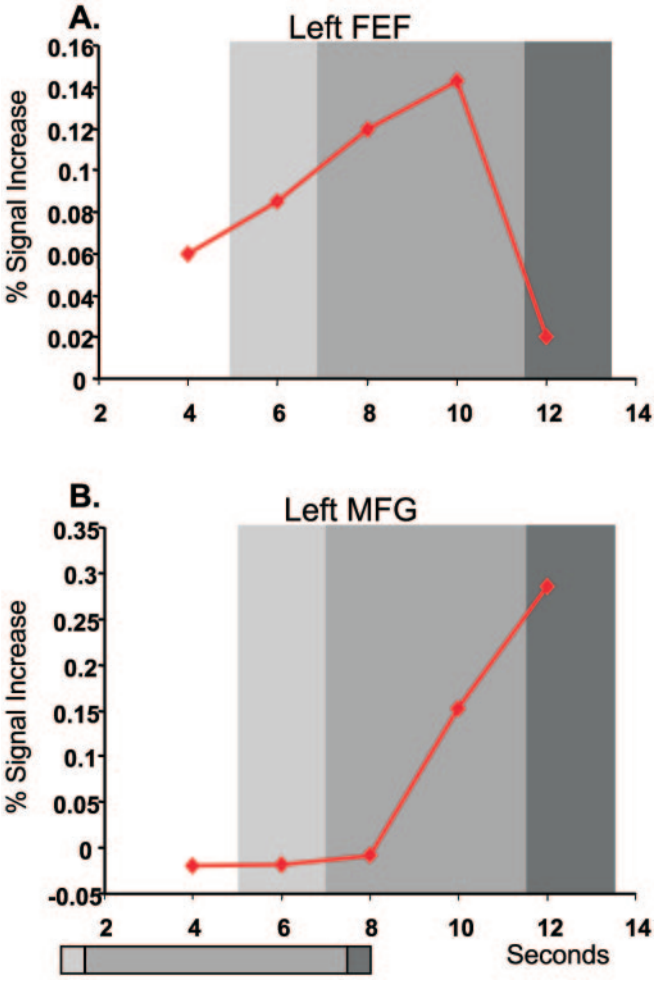

Figure 4. Time course of responses during correct change detection trials. (*A*) Left frontal eye field (FEF). (*B*) Left middle frontal gyrus (MFG). The bar below the *x* axis codes the periods when the sample stimulus (light gray), the delay (intermediate gray), and the test stimulus (dark gray) occurred during the task. The vertical gray bars for encoding and test are centered 5 s after the stimulus presentation; the gray bar for delay was centered between the latter two bars. The FEF showed a sustained response during the delay period, whereas the MFG increased its activity to peak at around the time of the test stimulus.

Discussion

In the present study, we investigated the neural substrates of change detection. Our results revealed that correctly detecting a change was associated with activation of a network comprising parietal and frontal brain regions, as well as activation of the pulvinar, cerebellum and ITG. Moreover, activations associated with incorrectly reporting a change when none occurred led to a very similar pattern of activation.

Neural Substrates of Change Detection

In a recent study, Beck *et al.* (2001) investigated the neural correlates of change detection and change blindness. They showed that change detection (i.e. detected vs undetected changes) was associated with enhanced activity in bilateral SPL (BA 7), right MFG (BA 46 at y = 30), as well as the fusiform gyrus, a region that encompassed sites responsive to the stimuli employed. Because the contrast of detected versus undetected changes involved correct (detected) and incorrect (undetected) trials, the activations possibly included contributions due to variations in the subject's attentional state. For example, Ress *et al.* (2000) attributed fluctuations in activity in V1 to trial-to-trial fluctuations in attention, which they suggested accounted for the variability in behavioral performance on a target detection task. Fluctuations in attention are a reason for concern because spatially directed attention strongly modulates activity in sensory processing areas, such as V1/V2 and the fusiform gyrus, and relies on a set of regions that includes those observed by Beck *et al.* (for reviews, see Desimone and Duncan, 1995; Kastner and Ungerleider, 2000).

To investigate the neural correlates of change detection while minimizing fluctuations of attention as a potential confound, we compared only high-confidence correct change versus correct no-change trials. This contrast revealed fronto-parietal sites that included the SFG, the MFG, the ACC, the IFG/anterior insula, the anterior IPS, and the precuneus. Interestingly, at the fronto-parietal sites reported by Beck *et al.* (2001), we encountered only weak activation for change trials, and these did not survive our random-effects analysis. In interpreting the results of the present study, we propose that change detection activates frontal and parietal regions via bottom-up mechanisms, thereby triggering attentional mechanisms located in these regions, which then function via top-down feedback to deploy attention to the location of a change, enabling further, more elaborate processing of the stimulus. This proposal is consistent with psychophysical studies that show that stimuli presented at the location of a change are processed more effectively than those presented at other locations (Smilek *et al.*, 2000; Thornton and Fernandez-Duque, 2000). At the same time, we propose that the activations reported by Beck *et al.* (2001) more closely reflect attentional

processes of goal-directed attention (see below for further discussion).

In our study, it is not possible to determine which fronto-parietal regions are the target of bottom-up inputs driven by change detection and which are the source of top-down control. However, neuroimaging studies of attentional control have demonstrated that some of the sites exhibiting change-related activation, namely the IPS and the ACC, can be activated in the absence of visual stimuli during the time in which subjects expect the occurrence of a stimulus (Kastner *et al.*, 1999; see also Shulman *et al.*, 1999; Hopfinger *et al.*, 2000). This suggests that both the IPS and the ACC (as well as other fronto-parietal regions) are capable of top-down control in the absence of bottom-up inputs. Studies employing techniques such as magnetoencephalography, which has better temporal resolution than fMRI, should help to determine the time course of the effects we observed and determine the flow of information associated with change detection.

We compared for the WM task the activations associated with change detection at test with those occurring at encoding, as well as those from a recent meta-analysis of imaging data of the network subserving spatial attention (Kastner and Ungerleider, 2000; Fig. 3*B*). The studies included in this meta-analysis comprised endogenous attention tasks. Important cortical nodes revealed by both the analysis of the encoding phase of the WM task and the meta-analysis of spatial attention included the SPL, the FEF and the SEF. These areas were only weakly activated during the detection of change in our study and, notably, did not survive the random-effects analysis. Instead, robust activations were found in more inferior sites within the MFG, the IFG and the anterior IPS. Recently, Corbetta and Shulman (2002) have proposed that there exist two anatomically segregated, but interacting, networks for spatial attention. According to their scheme, a *dorsal* fronto-parietal system is involved in the generation of attentional sets associated with goal-directed stimulus–response selection (endogenous attention). Key nodes within this largely bilateral network would include the SPL, IPS and the FEF. A second, more *ventral* system, which is strongly lateralized to the right hemisphere, is proposed to detect behaviorally relevant stimuli and to work as an alerting mechanism for the first system when these stimuli are detected outside the focus of processing (exogenous attention). Although our task was not a standard exogenous attention task in which attention is captured by a peripheral event (e.g. a flashing stimulus), the detection of a change likely involved the deployment of attention toward the location of the change, thereby engaging brain areas more involved in exogenous attention.

Regions activated by the detection of a change in our study did not greatly overlap with regions involved in maintenance processes in the WM task. Instead, delay-related activations more closely matched those of the meta-analysis of Kastner and Ungerleider (2000) of spatially directed attention. We suggest that the goal-directed aspect of these tasks, such as maintaining a focus of attention, engages neural mechanisms that are more closely tied to WM processes. Thus, the observed spatial overlap in activation would reflect such shared mechanisms (Mesulam, 1990; Desimone and Duncan, 1995; Awh and Jonides, 2001). It is noteworthy that WM-related activations were similar to the ones observed in the change-detection study by Beck *et al.* (2001), consistent with the idea that the latter reflect directed attention processes that allow the perception of a change.

Incorrectly Reporting a Change Triggers Virtually the Same Regions As Correctly Detecting a Change

In the present study, we were interested in the differences in activation between trials that had the same physical parameters in which subjects generated *opposite* reports. For this purpose, we contrasted the activity between the two types of incorrect trials at test, one in which subjects reported a change when none occurred in the display (false alarms), the other in which subjects missed a change that had in fact occurred (misses). Although the number of high-confidence, incorrect trials was small and the associated activations were generally weaker, this contrast revealed a network of activations with a striking degree of overlap with the network observed when subjects correctly detected a change. Thus, we suggest that these two types of events are virtually equivalent.

An analogous dissociation between physical parameters and perceptual reports has been observed in the visual cortex of monkeys observing threshold or ambiguous stimuli. For example, Bradley *et al.* (1998) showed that the responses of MT neurons to bi-stable, rotating cylinders defined by structure-from-motion cues were linked to the perception of which surface was perceived in front. This was also true for error trials in which the monkey's behavioral response reflected neuronal responses (the cell's preferred depth) rather than the physical cues of the stimulus. In another single-cell study, Thompson and Schall (1999, 2000) probed the neural substrates of target detection and showed that neural responses in the FEF to a target stimulus were greater when the target was detected than when it was missed. Moreover, neural responses were greater on false alarm trials than on trials in which the target was absent. Thus, our fMRI study reveals similar neural correlates of perceptual decisions in the human brain.

We also tested whether increased visual activation just prior to the presentation of the test stimulus would render subjects more likely to report that they had seen a change, even when a change had not actually occurred. Consistent with this idea, in the right ITG, a visual region that responded transiently to the stimulus and exhibited strong false alarm-related activation, elevated activity at 10 s was associated with a greater likelihood that the subject would report a change that did not occur ($P < 0.05$). For a 1% fMRI signal increase, the probability of reporting a change was 67%. Thus, it appears that elevated activity in the right ITG at the time of, or just preceding, the test stimulus might contribute to the subject's reporting that a change occurred, even when none was physically present. No other visual region was found to show this relationship between fMRI signal strength and false alarm reports.

Pulvinar

In the present study, correctly detecting a change was associated with pulvinar activation, especially in the right hemisphere (Fig. 2*A*), which was among the strongest and most consistently activated regions across the sites observed in this study. Right pulvinar activation was also observed during false alarm trials (Fig. 2*B*), consistent with our suggestion that correctly detecting a change and incorrectly reporting a change are very similar at the neural level.

Single-cell studies in monkeys reveal that the pulvinar nucleus of the thalamus has an important role in selective

attention processes (Chalupa, 1977; Petersen *et al.*, 1985). As summarized by Robinson and Petersen (1992), pulvinar cells generate signals related to the salience of visual objects and are involved in the selection of salient targets and the filtering of non-salient distracters. In monkeys, pulvinar lesions lead to impairments in active visual scanning (Ungerleider and Christensen, 1979), and inactivation of the pulvinar produces a slowing down of attention shifts (Petersen *et al.*, 1987). In humans, the right pulvinar is the principal site in the thalamus associated with spatial neglect (Karnath *et al.*, 2002). Imaging studies with humans also have obtained evidence of pulvinar involvement in attentional processes (LaBerge and Buchsbaum, 1990; Corbetta *et al.*, 1991), although not consistently. One possibility is that the pulvinar is a small structure that may be difficult to image with standard 1.5 T scanners (see Ugurbil *et al.*, 1999). In the present experiment, which was performed at 3 T, we found robust and consistent pulvinar activation, which we suggest was involved in the deployment of spatial attention to the location of the change.

Cerebellum

Like the pulvinar, the cerebellar cortex was strongly activated when subjects correctly detected a change, as well as when they incorrectly reported a change. Both change-related conditions were associated with a left-hand press. Although some of the activation ipsilateral to the hand press might have been related to motor processes, such processes are an unlikely explanation of the right cerebellar activation. There is now evidence that the cerebellum has functions beyond those of motor processing (Middleton and Strick, 1994; Fiez, 1996; Schmahmann, 1996; Thach, 1996). In particular, based on studies of patients with cerebellar lesions, it has been proposed that the cerebellum mediates rapid shifts in attention (Akshoomoff and Courchesne, 1992, 1994). In an fMRI study to test cerebellar involvement in attentional processes, Le *et al.* (1998) compared a condition of shifting attention to a condition of sustained attention, which revealed lateral cerebellar activation (see also Allen *et al.*, 1997). Thus, the present study adds additional evidence to the idea that the cerebellum is involved in attentional processes in general, and in the detection of change in particular.

Change Blindness

The suggestion that greater activation evoked during false alarm compared to miss trials reflects the subjective report of detecting a change is reinforced by our finding that voxels revealed by the reverse comparison (miss > false alarm) did not overlap with regions revealed during correctly detecting a change (correct reject > miss). Regions more strongly activated during miss compared to false alarm trials may potentially reveal implicit mechanisms triggered by the unreported occurrence of a physical change in the stimulus (i.e. change blindness). We found significant activations for this contrast in very few sites (Table 3), which included the parahippocampal gyrus (BA 20), the posterior IPS (BA 7), the SEF (BA 6/32), the IFG/anterior insula (BA 44), and a posterior visual region (cuneus, BA 18).

Correlates of change blindness have also been reported by Beck *et al.* (2001), which in their study involved regions in the lingual and fusiform gyrus that were activated by their visual stimuli, as well as the IFG. We also found activations in this latter region on the right, although our site was 21 mm more anterior and included the anterior insula in BA 44. Overall, in our study, few regions were differentially activated by miss trials, and, of these, only the SEF overlapped with change-related activations. Thus, whereas reporting that a change occurred, be it correctly or incorrectly, is associated with strong activation in fronto-parietal sites, change blindness appears to involve more limited territories.

Conclusions

In summary, the present study provides evidence that change detection is associated with a distributed set of brain regions, including visual processing areas, a number of frontal and parietal regions, as well as the pulvinar and cerebellum. In general, two types of attentional processes are involved in change detection, goal-directed mechanisms that allow the perception of change, and reactive deployment mechanisms that may be important for further processing the change. By controlling for fluctuations in goal-directed attention (by contrasting correct trials only), the present study allowed us to investigate the neural correlates of 'reactive attention' during the detection of visual change. We propose that frontal and parietal regions might be involved in controlling the deployment of attention to the location of a change, thereby allowing further processing of the visual stimulus. The fronto-parietal regions are possibly assisted by the cerebellum and the pulvinar, which were also involved in the detection of a change. Finally, visual processing areas, such as the ITG, may be recipients of top-down feedback from the fronto-parietal regions that control the reactive deployment of attention, and thus exhibit increased activation when a change is reported. Overall, our results are consistent with the proposal by Corbetta and Shulman (2002) of segregated, yet interacting, dorsal and ventral attention systems, which are thought to be involved in endogenous and exogenous attention, respectively.

Notes

We thank Eva Gutierrez and David Sturman for assistance with data collection, analysis, and for assistance in the preparation of the manuscript. This study was supported by the National Institute of Mental Health – Intramural Research Program.

Address correspondence to Dr Luiz Pessoa, Department of Psychology, 89 Waterman Street, Brown University, Providence, RI 02912, USA. Email: pessoa@brown.edu.

References

Akshoomoff NA, Courchesne E (1992) A new role for the cerebellum in cognitive operations. Behav Neurosci 106:731–738.

Akshoomoff NA, Courchesne E (1994) ERP evidence for a shifting attention deficit in patients with damage to the cerebellum. J Cogn Neurosci 6:388–399.

Allen G, Buxton RB, Wong EC, Courschesne E (1997) Attentional activation of the cerebellum independent of motor movement. Science 275:1940–1943.

Awh E, Jonides J (2001) Overlapping mechanisms of attention and spatial working memory. Trends Cogn Sci 5:119–126.

Bandettini PB (1999) The temporal resolution of the functional MRI. In: Functional MRI (Moonen CTW, Bandettini PB, eds), pp. 205–220. Berlin: Springer-Verlag.

Beck DM, Rees G, Frith CD, Lavie N (2001) Neural correlates of change detection and change blindness. Nat Neurosci 4:645–650.

Bradley DC, Chang GC, Andersen RA (1998) Encoding of three-dimensional structure-from-motion by primate area MT neurons. Nature 392:714–717.

Chalupa LM (1977) A review of cat and monkey studies implicating the pulvinar in visual function. Behav Biol 20:149–167.

Cohen JD, Perlstein WM, Braver TS, Nystrom LE, Noll DC, Jonides J, Smith EE (1997) Temporal dynamics of brain activation during a working memory task. Nature 386: 604–608.
Cohen MS (1997) Parametric analysis of fMRI data using linear systems methods. Neuroimage 6:93–103.
Corbetta M, Shulman GL (2002) Control of goal-directed and stimulus-driven attention in the brain. Nat Rev Neurosci 3:201–215.
Corbetta M, Miezin FM, Dobmeyer S, Shulman GL, Petersen SE (1991) Selective and divided attention during visual discriminations of shape, color, and speed: functional anatomy by positron emission tomography. J Neurosci 11:2382–2402.
Cox RW (1996) AFNI: Software for analysis and visualization of functional magnetic resonance neuroimages. Comput Biomed Res 29:162–173.
Desimone R, Duncan J (1995) Neural mechanisms of selective attention. Annu Rev Neurosci 18:193–222.
Fiez JA (1996) Cerebellar contributions to cognition. Neuron 16:13–15.
Friston KJ, Holmes AP, Worsley KJ, Poline J-P, Heather JD, Frackowiak RS (1995) Statistical parametric maps in functional imaging: a general linear approach. Hum Brain Mapp 3:165–189.
Hopfinger JB, Buonocore MH, Mangun GR (2000) The neural mechanisms of top-down attentional control. Nat Neurosci 3:284–291.
Karnath HO, Himmelbach M, Rorden C (2002) The subcortical anatomy of human spatial neglect: putamen, caudate nucleus and pulvinar. Brain 125:350–360.
Kastner S, Ungerleider LG (2000) Mechanisms of visual attention in the human cortex. Annu Rev Neurosci 23:315–341.
Kastner S, Pinsk MA, De Weerd P, Desimone R, Ungerleider LG (1999) Increased activity in human visual cortex during directed attention in the absence of visual stimulation. Neuron 22:751–761.
LaBerge D, Buchsbaum MS (1990) Positron emission tomographic measurements of pulvinar activity during an attention task. J Neurosci 10:613–619.
Le TH, Pardo JV, Hu X (1998) 4 T-fMRI study of nonspatial shifting of selective attention: cerebellar and parietal contributions. J Neurophysiol 79:1535–1548.
Mesulam MM (1990) Large-scale neurocognitive networks and distributed processing for attention, language, and memory. Ann Neurol 28:597–613.
Middleton FA, Strick PL (1994) Anatomical evidence for cerebellar and basal ganglia involvement in higher cognitive function. Science 266:458–461.
Pessoa L, Gutierrez E, Bandettini PB, Ungerleider LG (2002) Neural correlates of visual working memory: fMRI amplitude predicts task performance. Neuron 35:975–987.
Petersen SE, Robinson DL, Keys W (1985) Pulvinar nuclei of the behaving rhesus monkey: visual responses and their modulation. J Neurophysiol 54:867–886.
Petersen SE, Robinson DL, Morris JD (1987) Contributions of the pulvinar to visual spatial attention. Neuropsychologia 25:97–105.
Remington RW, Johnston JC, Yantis S (1992) Involuntary attentional capture by abrupt onset. Percept Psychophys 51:279–290.
Rensink RA (2002) Change detection. Annu Rev Psychol 53:245–277.
Rensink RA, O'Regan JK, Clark JJ (1997) To see or not to see: the need for attention to perceive changes in scenes. Psychol Sci 8:368–373.
Ress D, Backus BT, Heeger DJ (2000) Activity in primary visual cortex predicts performance in a visual detection task. Nat Neurosci 3:940–945.
Robinson DL, Petersen SE (1992) The pulvinar and visual salience. Trends Neurosci 15:127–132.
Schmahmann JD (1996) From movement to thought: anatomical substrates of the cerebellar contribution to cognitive processing. Hum Brain Mapp 4:174–198.
Shulman GL, Ollinger JA, Akbudak E, Conturo TE, Snyder AZ, Petersen SE, Corbetta M (1999) Areas involved in encoding and applying directional expectations to moving objects. J Neurosci 21:9480–9496.
Smilek D, Eastwood JD, Merikle PM (2000) Does unattended information facilitate change detection? J Exp Psychol Hum Percept Perform 26:480–487.
Talairach J, Tournoux P (1988) Co-planar stereotaxis atlas of the human brain. New York: Thieme Medical.
Thach WT (1996) On the specific role of the cerebellum in motor learning and cognition: clues from PET activation and lesion studies in man. Behav Brain Sci 19:411–431.
Thompson KG, Schall JD (1999) The detection of visual signals by macaque frontal eye field during masking. Nat Neurosci 2:283–288.
Thompson KG, Schall JD (2000) Antecedents and correlates of visual detection and awareness in macaque prefrontal cortex. Vision Res 40:1523–1538.
Thornton IM, Fernandez-Duque D (2000) An implicit measure of undetected change. Spat Vis 14:21–44.
Ugurbil K, Hu X, Chen W, Zhu XH, Kim SG, Georgopoulos A (1999) Functional mapping in the human brain using high magnetic fields. Phil Trans R Soc Lond 354:1195–1213.
Ungerleider LG, Christensen CA (1979) Pulvinar lesions in monkeys produce abnormal scanning of a complex visual array. Neuropsychologia 17:493–501.
Wojciulik E, Kanwisher N, Driver J (1998) Covert visual attention modulates face-specific activity in the human fusiform gyrus: fMRI study. J Neurophysiol 79:1574–1578.