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Neural correlates of conscious perception in the attentional blink

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If attending to a target in a rapid stream of visual stimuli within the next 400 ms or so, a second target in the stream is frequently not detected by an observer. This so-called attentional blink can provide a comparison of neural signals elicited by identical stimuli that, in one condition, reach conscious awareness and, in the other, fail to be selected for awareness. In the present study, using event-related functional magnetic resonance imaging (fMRI), differences of neural activation were studied in an attentional blink experiment in order to identify brain regions putatively involved in controlling the access of information to consciousness. Subjects viewed a rapid stream of black letters in which the second target (T2) was either presented within or outside the attentional blink period, or not at all. We observed an increase in activation for detected as compared to missed T2 presented during the attentional blink in frontal and parietal cortices. In contrast, in occipitotemporal regions activation was increased for missed as compared to detected T2. Furthermore, in several frontal and parietal areas, missed targets were associated with increased activity if compared to the condition in which no second target was presented. Finally, a selective decrease in activation for detected T2 presented during the attentional blink was observed in areas associated with emotional and predominantly automatic processing. While activations in occipitotemporal regions might mainly reflect duration of attentive search, the frontoparietal areas seem to be involved in a highly distributed network controlling visual awareness.

Keywords: Consciousness; Perception; Brain

Introduction

In recent years, it has become possible to study neural correlates of consciousness (NCC) with methods of cognitive neuroimaging. The search for NCC is an empirical investigation that remains, at least at present, neutral with respect to philosophical issues of mind–brain relationship or causality. Instead, this search concentrates on identifying and characterizing neural activity patterns that specifically co-vary with conscious experience, rather than with unconscious perception or action (Crick and Koch, 1990; Engel and Singer, 2001; Rees et al., 2002). Due to convergent results from studies using neuroimaging techniques in normal human subjects, invasive recordings in patients, as well as microelectrode approaches in animals, some progress has been made in recent years, both with respect to identifying mechanisms that may be involved in controlling access to consciousness and with respect to studying activity patterns that correlate with specific contents of conscious mental states. What emerges from these studies is that conscious awareness presupposes a complex set of intertwined functions, including sensory preprocessing, attention, and working memory (Crick and Koch, 1990, 2003; Rees et al., 2002). The NCC, thus, is likely to involve a highly distributed set of brain areas subserving these functions. This network engages, via large-scale dynamic interactions, in globally coherent states (Dehaene et al., 2003; Engel and Singer, 2001; Engel et al., 2001; Varela et al., 2001) that seem required for the establishment of a global workspace carrying the contents of awareness (Newman and Baars, 1993). What is still largely unclear is which areas exactly are involved in the network controlling the selection of information through cooperative interaction and, moreover, what exactly the constraints and mechanisms are that underlie the selection of sensory signals for conscious awareness.

Sensory paradigms suited for the study of NCC allow the comparison of brain activation in response to physically identical stimuli that are selected for conscious perception in one experimental condition but excluded from conscious perception in a control condition. A paradigm that meets this criterion particularly

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well is the attentional blink paradigm. The "attentional blink" (Raymond et al., 1992) can be described as a transient reduction of attention which occurs if more than one target has to be processed in a series of stimuli that rapidly succeed one another—a phenomenon first observed in the mid-1980s (Broadbent and Broadbent, 1987; Reeves and Sperling, 1986; Weichselgartner and Sperling, 1987). In a typical visual attentional blink experiment, a series of up to about 20 stimuli is displayed at the same location with a frequency of about 10 per second. The series of stimuli contains two predefined targets (T1, T2) occurring with variable temporal lag relative to each other. If both targets have to be attentively processed, detection accuracy for the second target (T2) is strongly impaired when it follows the first by about 200–400 ms. This latency range is typically regarded as the attentional blink time window, as task performance for T2 is often found to be better if it is presented immediately following T1 or with a delay of about 500 ms or more.

Previous neuroimaging studies (Marois et al., 2000) have focused on psychophysical findings that emphasize the relevance of processing of the T1 stimulus for the magnitude of the attentional blink (Chun and Potter, 1995; Jolicoeur and Dell'Acqua, 1998; Raymond et al., 1992). The neuroimaging experiments were focused on the perceptual interference between T1 and subsequent distractor stimuli, comparing conditions of high and low interference between target and distractors. These measurements revealed differential activation for regions in the right intraparietal sulcus, and in anterior cingulate and lateral prefrontal areas (Marois et al., 2000). Results were interpreted in the context of a neural network related to visuospatial and nonspatial attention (Corbetta et al., 1998; Coull and Frith, 1998; Coull and Nobre, 1998). However, due to the design chosen, the experiments do not allow to distinguish between effects of masking on target identification and the attentional blink phenomenon as such, since performance on T2 was only tested in behavioral experiments, but not during the fMRI runs.

An important characteristic of the attentional blink is the obvious difference in performance on T2 when presented within or after the attentional blink window. T2 processing was addressed in two recent fMRI studies (Marcantoni et al., 2003; Marois et al., 2004). Marcantoni et al. (2003) observed increased activation in inferotemporal, lateral frontal, left posterior parietal, and occipital cortex for T2 stimuli presented during the attentional blink window. The authors concluded that these regions seem to be involved in resolving the dual-task interference in the attentional blink. The event-related study by Marois et al. (2004) differs from their earlier work in that they now explicitly distinguished the effects of consciously perceived and non-perceived T2 stimuli. In this study, faces (T1), scenes (T2), and scrambled scenes (distractors) served as stimulus material. In perceptual areas known to preferentially respond to scenes activation was found to be largest for perceived T2, intermediate for non-perceived T2 and smallest for trials in which no T2 had been presented. Furthermore, it was found that in lateral frontal cortex, activation strongly depended on whether the target was explicitly reported: The hemodynamic response was enhanced for detected, correctly identified T2 as compared to missed T2, and as compared to the control condition (no T2 present). In contrast to their earlier work (Marois et al., 2000), significant differences in activation were not found for parietal cortex. Results were interpreted to reflect the predominant role of frontal cortex in selecting consciously perceived items.

To better understand the attentional blink phenomenon, it is most crucial to address the question why in some trials the attentional blink is evident, and in other trials, it is not. In other words, what causes the difference between cases in which a second target cannot be reported from those in which it is consciously perceived (Dehaene et al., 2003). By comparing the event-related potentials (ERP) evoked by detected and missed targets presented in the attentional blink interval, we recently demonstrated that detected T2 evoke a P3 component, whereas missed targets do not (Kranczioch et al., 2003). We concluded from this study that detected targets presented during the attentional blink window do indeed reach working memory and, therefore, enter awareness. This suggests that subjects did not simply guess when indicating that they did perceive the second target. This finding is in accordance with a semantic priming experiment (Rolke et al., 2001) showing that detected as well as missed words presented in an attentional blink interval similarly affected the N400 evoked by a probe word presented later, thereby replicating comparable results of Luck et al. (1996). Luck et al. (1996) did not separate trials in which the second target was missed and in which it was detected in their analysis though. Taken together, the ERP studies on the attentional blink suggest that targets presented during the attentional blink are perceptually analyzed, and processed up to a semantic level. However, it remains an open question why only for a fraction of the trials the sensory information reaches awareness.

In the present study, we have investigated the neural correlates of conscious target detection in an attentional blink context using an event-related fMRI approach. Our specific goal was to identify the network of areas that respond differentially during T2 processing, depending on whether T2-related signals reach conscious awareness or not. In the experimental approach chosen, the temporal lag between the second and the first target was varied to achieve different degrees of target interference. Data analysis then focused on differences in activation between the behaviorally derived conditions "T2 detected" and "T2 missed". In addition, possible differences between conditions with different presentation lags of the second target were investigated, and activations were compared for T2 present and T2 absent conditions.

Previous fMRI studies of the attentional blink have suggested that interference between target and mask (Marois et al., 2000) or between targets (Marcantoni et al., 2003) is associated with increased activation in a frontoparietal network, including lateral frontal, anterior cingulate and intraparietal areas. Since in the present study perceptual interference between targets and masks could be assumed to be equal in all conditions yet target interference varied, we expected that activation in the frontoparietal network should be largest for the condition *T2 detected (lag 1, 2),* smallest for the *no T2* condition, and intermediate for the *T2 detected (lag 7)* condition. Furthermore, if this network represents the attentional bottleneck to perceptual awareness (Marois et al., 2004), activation in the *T2 missed (lag 1, 2)* condition should be comparable or only slightly above that seen in the *no T2* condition. With regard to visual areas, it was expected that detection of T2 would be associated with increased activation, but that also the mere presence of T2 should result in an increase in activation if compared to the *no T2* condition (Marois et al., 2004).

Materials and methods

Stimulus paradigm

The rapid serial visual presentation (RSVP) sequence consisted of 20 capital black letters and one capital green letter (T1) shown for 100 ms with no inter-stimulus interval. Letter stimuli were 3.5° wide and 4.0° high, and were presented at fixation on a white background. T1 could appear at serial positions 4 to 7. For T1, the vowels A, E, O, U, and all consonants were used, with the exception of F, K, Q, and Y. In 75% of the trials, T1 was followed by a black capital X defined as T2. T2 could appear either immediately (lag 1), as the second letter (lag 2), or as the seventh letter after T1 (lag 7) (Kranczioch et al., 2003). These three lags were applied with equal probability. As distractors, all consonants were used except F, K, Q, and Y. Within a trial, a distractor could appear repeatedly, albeit not at two successive positions.

The layout of a trial is depicted in Fig. 1. At the start of a trial, the black fixation cross shown between two trials turned into red for 1000 ms. Then, the RSVP sequence was run for 2100 ms. Following the RSVP sequence, the screen remained blank for 500 ms, and afterwards, responses were requested for T1 and T2 via response screens. Participants were first asked whether T1 had been a vowel, then they had to indicate whether an X had been present. In either case, 'Yes' or 'No' responses were given with the left (Yes-response) or the right (No-response) index finger by using two key pads that the subjects held in the respective hand. Maximal response time was limited to 5400 ms. After this time had elapsed, the black fixation cross was shown for either 3, 5, or 7 s, until the start of the next trial was indicated by the black cross turning red again. Overall, trial duration was either 12, 14, or 16 s.

Prior to the fMRI experiment, participants were provided with task instructions. Subjects were instructed to search the RSVP stream for the green letter (T1), to decide whether it was a vowel and, in addition, to search for a black X (T2). They were informed that the black X—if present—would be presented always after the green letter. All subjects practiced the task outside the scanner for a total of 20 trials. The fMRI experiment consisted of six runs with 36 trials per run, resulting in a total of 54 trials for each of the four conditions (no T2, T2 at lag 1, T2 at lag 2, and T2 at lag 7). Trial sequence was randomized within runs.

Participants

Twelve participants were tested in the fMRI experiment. They were required to be free of current or past neurological or psychiatric disorders, had normal or corrected-to-normal visual acuity, normal color vision, and were right-handed. All subjects where paid for participation and informed consent was obtained prior to the start of the experiment. All subjects also participated in an EEG experiment on the AB, the data of which will be presented elsewhere. Half of the subjects started with the fMRI experiment, the other half with the EEG experiment. Experiments were at least 14 days apart.

Based on their behavioral data five subjects (age 19–34; one male) were selected for further data analysis. Selection criterion was to have at least 10 trials for each of the conditions *T2 detected (lag 1)*, *T2 missed (lag 1)*, *T2 detected (lag 2)* and *T2 missed (lag 2)* in order to allow the detected-missed comparison. The criterion could not be applied to condition *T2 (lag 7)* because in this condition no subject had 10 or more *T2 missed (lag 7)* trials. This condition was therefore not included in contrast computations. The number of trials contributing to each condition varied as a result of subjects' performance. Trials were only included into data analysis if T1 had been correctly identified. The total number of trials in the six conditions was 110 for *T2 detected (lag 1)*, 80 for *T2 detected (lag 2)*, 155 for *T2 missed (lag 1)*, 181 for *T2 missed (lag 2)*, 239 *T2 detected (lag 7)*, and 254 for *no T2* (T2 correctly rejected), respectively.

Image acquisition

Echoplanar images were collected on a 3-T whole body MRI system (Siemens Magnetom Trio, Siemens, Erlangen, Germany) using the standard head coil. We used a gradient echoplanar sequence (TR = 2000; TE = 35; FA = 90°) to visualize changes of blood oxygen level-dependent (BOLD) contrast (FOV = 224 × 224 mm^2; slice thickness = 4.5 mm; imaging matrix = 64 × 64; resulting voxel size = 3.5 × 3.5 × 4.5 mm^3). Images were acquired interleaved in 25 contiguous axial slices. To ensure time locking of image acquisition to trial presentation, trial duration always was a multiple of TR. The first trial started after the tenth volume was acquired. Experimental runs had a duration 9:20 min. A T1–weighted 3-D magnetization prepared rapid acquisition gradient echo sequence (MP RAGE) scan (voxel size = 1 × 1 × 1

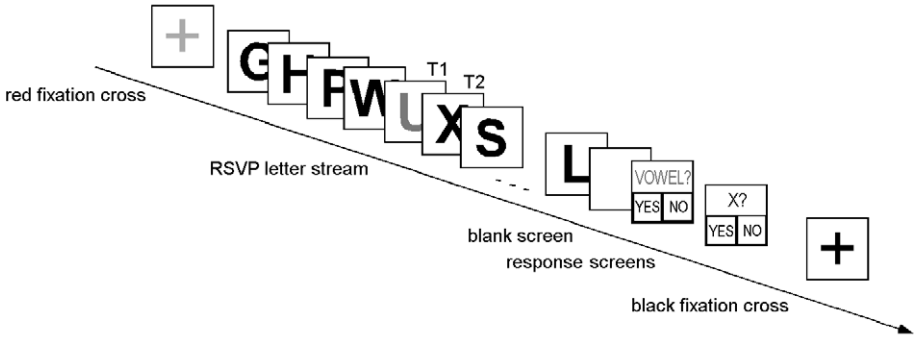

Fig. 1. Illustration of the paradigm used in the present study. In the trial shown here, T2 is presented at lag 1. Each trial started with a red fixation cross followed by the RSVP stream. After a blank screen was presented briefly, two consecutive response screens prompted subjects to give their responses regarding T1 and T2. Finally, a black fixation cross was presented for 3, 5, or 7 s.

mm^3, TR = 2300 ms, TE = 3.93 ms, TI = 1100 ms) lasting 9:50 min was recorded after the third experimental run.

fMRI data analysis

Data were analyzed using BrainVoyager 2000 4.9 and BrainVoyager QX 1.0 software (Brain Innovation B.V., Maastricht, The Netherlands). The first two scans were excluded from data analysis. The following preprocessing steps were performed: slice scan time correction (using sinc interpolation), linear trend removal, temporal high-pass filtering to remove low-frequency non-linear drifts of 3 or fewer cycles per time course, and 3D motion correction to detect and correct for small head movements by spatial alignment of all volumes to the first volume by rigid body transformations. Estimated translation and rotation parameters were inspected and never exceeded 3 mm. Co-registration of functional and 3-D structural measurements was computed by relating T2*-weighted images and the T1-weighted 3-D MP RAGE measurement, which yields a 4-D functional data set. Structural 3-D and functional 4-D data sets were transformed into Talairach space (Talairach and Tournaux, 1988).

In order to statistically evaluate the differences between the experimental conditions a multiple regression approach was applied. The stimulation protocol included predictors for the following conditions: *T2 detected (lag 1)*, *T2 missed (lag 1), T2 detected (lag 2)*, *T2 missed (lag 2)*, *T2 detected (lag 7)*, *T2 missed (lag 7)*, and *no T2*. In addition, the 3-, 5-, or 7-s intervals between two trials during which the fixation cross was black served as a baseline condition. For each subject, six stimulation protocols were compiled reflecting individual performance and trial order for each experimental run. These protocols served to derive appropriate reference functions reflecting experimental and baseline conditions, which were convoluted with a hemodynamic response function (Boynton et al., 1996) to account for the expected delay and generic shape of the BOLD signal. These reference functions served as independent predictors for a fixed-effects general linear model (GLM). In order to correct for multiple comparisons, the false discovery rate (FDR) controlling procedure was applied on the resulting p values for all voxels. The value of q specifying the maximum FDR tolerated on average was set to 0.05. With this value, a single-voxel threshold is chosen by the FDR procedure which ensures that from all voxels shown as active, only 5% or less are false-positives (Benjamini and Hochberg, 1995; Genovese et al., 2002).

In a voxel-based approach, contrast maps were computed for the predictors *T2 detected* (*lag 1, 2*) vs. *no T2* and for the predictors *T2 detected (lag 1, 2)* vs. *T2 missed (lag 1, 2)*. Significantly activated clusters of 50 voxels or more [q(FDR) $\leq$ 0.05] were selected for a more sensitive region of interest (ROI) analysis. For all ROI time courses, additional fixed-effects GLM and appropriately weighted linear contrasts were computed. Contrast definitions are summarized in Table 1. The region time courses were standardized, so that beta weights of predictors reflect the BOLD response amplitude of one condition relative to the variability of the signal. Furthermore, the event-related average of the BOLD signal change was computed for the ROIs.

Results

Behavior

In the fMRI experiment, only dual-task conditions were tested, that is, subjects were always required to identify both T1 and T2. As expected, detection accuracy for the second target (T2) varied as a function of lag relative to the first target (T1). Only trials in which T1 had been correctly identified were selected for further analysis. Fig. 2 shows the T2 detection rate as a function of lag (lag 1 = 100 ms, lag 2 = 200 ms, lag 7 = 700 ms). Detection rate at both lags 1 and 2 was reduced as compared to lag 7 (lag 1: $t(4) = 6.2$, P (one-tailed) $\leq$ 0.0015, lag 2: $t(4) = 15.00$, P (one-tailed) $\leq$ 0.0001), but at lag 1 it was slightly better than at lag 2 ($t(4) = 2.43$, P (one-tailed) $\leq$ 0.035).

Performance on the T1 task was analyzed separately for *T2 detected*, *T2 missed*, and *no T2* trials. For lag 1 trials, T1 was correctly detected in 96.5% of trials in which T2 was also correctly detected, and in 99% of trials in which T2 was missed. For lag 2 trials, T1 was correctly detected in 98.5% of trials in which T2 was also correctly detected, and in 96% of trials in which T2 was missed. For lag 7 trials, T1 was correctly detected in 97.5% of trials in which T2 was also correctly detected as well as in trials in which T2 was missed. Finally, for trials in which no T2 was presented, T1 was correctly detected in 97% of trials in which T2 was correctly rejected, and in 100% of trials in which T2 was falsely detected (9 of 270 trials were false alarms). Statistical comparison of these values did not reveal significant differences in T1 performance in relation to T2 detection in any of the four conditions (all $P > 0.1$).

Independent of performance on the T1 task, the response criterion beta was computed for T2 for all subjects to reveal

Table 1
Summary of contrasts computed for regions of interest (ROI)

Contrast	Lag 1		Lag 2		T2 detected *(lag 7)*	No T2 (correct rejection)
	T2 detected	T2 missed	T2 detected	T2 missed		
T2 detected (lag 1, 2) vs. no T2	+		+			– –
T2 missed (lag 1, 2) vs. no T2		+		+		– –
T2 detected (lag 1, 2) vs. T2 missed (lag 1, 2)	+	–	+	–		
Lag 1 vs. Lag 2	+	+	–	–		
Interaction Lag vs. T2 detected/missed	+	–	–	+		
T2 detected (lag 7) vs. no T2					+	–
T2 detected (lag 7) vs. T2 detected (lag 1, 2)	–		–		+ +	
T2 detected (lag 7) vs. T2 missed (lag 1, 2)		–		–	+ +	

The table indicates the direction of the contrasts by plus and minus signs for the relevant conditions. Two signs indicate that a given contrast was balanced to account for an unequal number of conditions contributing to a contrast.

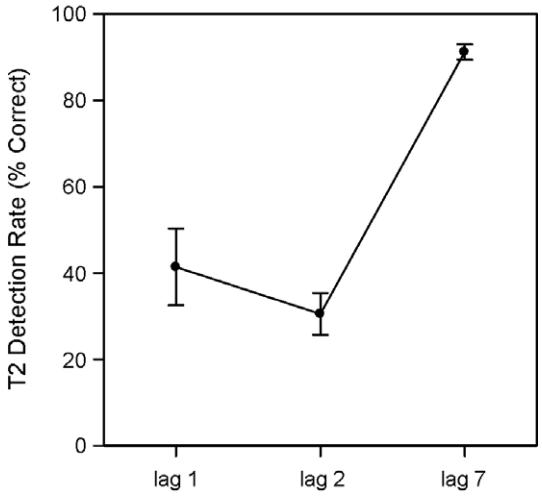

Fig. 2. Mean T2 detection rate ±SE for T2 at lags 1, 2, and 7 (n = 5).

whether subjects were biased in their answers. All beta values were 2.5 or higher, indicating that subjects' response criterion was rather conservative, that is, there were few correctly detected T2 stimuli due to false rejections but also only few wrongly detected ones (false alarms).

fMRI data: voxel-based ROI specification

In a first analysis step, a voxel-based approach was applied to reveal brain regions for detailed analyses (regions of interest, ROIs). The contrasts *lag 1* vs. *lag 2* and *interaction lag 1/lag 2* vs. *T2 detected/T2 missed* did not show any significant activation. Thus, activation was not different in the lag 1 and 2 conditions. Hence, in all further contrasts conditions were considered together for the two lags, but separate for detected and missed T2, that is, *T2 detected (lag 1, 2)* and *T2 missed (lag 1, 2)*.

The *T2 detected (lag 1, 2)* condition was compared to the *no T2* condition, the latter comprising trials in which T2 was correctly rejected by the subjects. This comparison was performed to reveal activation differences between trials in which T2 was objectively absent and objectively present, respectively, both being correctly perceived by the subjects. A number of regions showed significantly different activation [q(FDR) ≤ 0.05; P ≤ 0.00054] for these two conditions (Fig. 3A; Table 2). These included clusters in the right and especially the left inferior parietal lobules (IPL), in the left inferior frontal gyrus (IFG), and the left superior frontal gyrus/anterior cingulate cortex (SFG/ACC). In addition, increased activation of the right precentral gyrus (PCG) was observed, reflecting motor activity related to the button press (left hand button press for 'Yes' response). This activation was complementary to a relative decrease in activation in the left PCG. Table 2 summarizes the Talairach coordinates of the center of mass together with the number of voxels in each cluster.

The critical test for investigating correlates of visual awareness in the context of the attentional blink paradigm is to compare activation for trials in which T2 has been detected with trials where T2 was missed albeit physically present. To this end, the contrast *T2 detected (lag 1, 2)* and *T2 missed (lag 1, 2)* is of primary interest. Computing this contrast for all voxels revealed differential activation [q(FDR) ≤ 0.05, P ≤ 0.0004] in a number of regions, including clusters in the left lateral frontal cortex (LFC), left IPL, left and right lateral occipital complex (LOC), left and right fusiform gyrus (FFG), and left amygdala (Fig. 3B, Table 3). The contrast was negative for clusters in the occipital lobes (LOC, FFG) and the amygdala. As in the *T2 detected (lag 1, 2)–no T2* contrast motor-response related activity was observed in left and

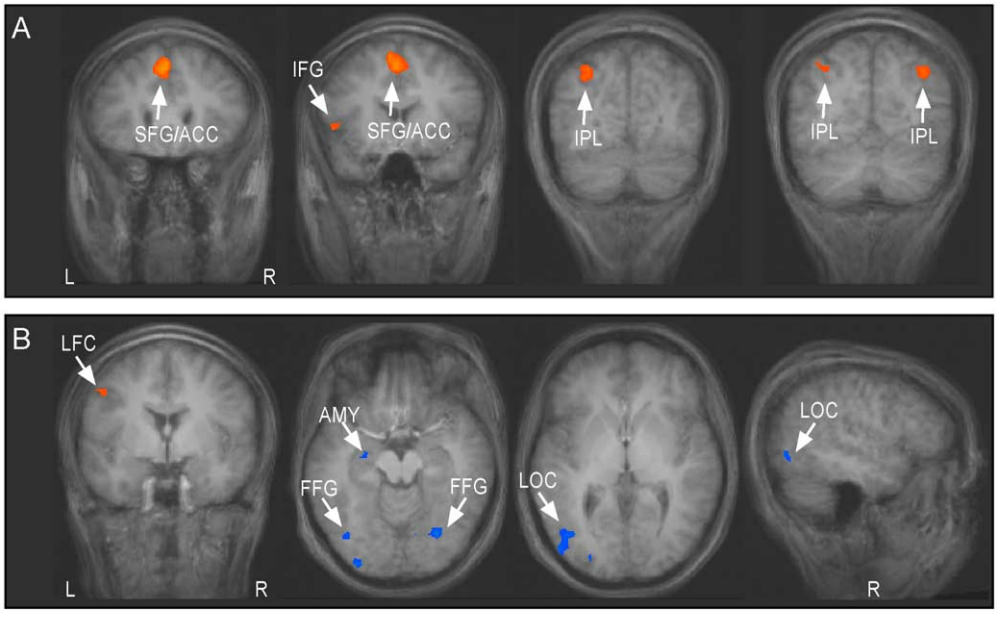

Fig. 3. Brain regions activated differently in the voxel based contrasts *T2 detected (lag 1, 2)-no T2* (A) and *T2 detected (lag 1, 2)-T2 missed (lag 1, 2)* (B). For a complete list of voxel clusters showing significant differences, see Tables 2 and 3. The activations were thresholded at q(FDR) ≤ 0.05, color coded, and superimposed on the average of the individual T1-weighted structural images. Abbreviations: AMY—amygdala, FFG—fusiform gyrus (x, y, z = −36, −69, −12 and −26, −66, −11), IFG—inferior frontal gyrus, IPL—inferior parietal lobe (x, y, z = −29, −67, 36 and 37, −58, 38), LFC—lateral frontal cortex, LOC—lateral occipital complex (x, y, z = −41, −70, 3 and 47, −66, −5), SFG/ACC—superior frontal gyrus/anterior cingulate cortex.

Table 2
Coordinates of voxel clusters significantly more active in the *T2 detected (lag 1, 2)* as compared the *no T2* condition

Area		Voxels in cluster	Talairach coordinates		
			x	*y*	*z*
Left	SFG/ACC	2723	−2	19	47
	IFG	151	−43	13	5
	IPL anterior	158	−40	−50	39
	IPL posterior	1361	−29	−67	36
	PCG	2019	−35	−26	53
Right	IPL anterior	83	46	−43	45
	IPL posterior	338	37	−58	38
	PCG	3109	34	55	−25

Coordinates are shown for the center of mass together with the number of active voxels in each cluster.

Abbreviations: IFG—inferior frontal gyrus, IPL—inferior parietal lobule, PCG—precentral gyrus, SFG/ACC—superior frontal gyrus/anterior cingulate cortex.

right PCG. Talairach coordinates of the center of mass together with the number of voxels in each cluster except PCG are summarized in Table 3.

fMRI: analyses of activation within ROIs

The voxel based approach was followed by the computation of GLM contrasts and event-related averages of the hemodynamic response for occipitotemporal, parietal, and frontal areas that turned out to be differentially modulated by task performance and T2 presence. By this approach, the NCCs in the context of the attentional blink paradigm could be investigated in greater detail.

Occipitotemporal cortex

As shown by the event-related averages of the hemodynamic response (cf. Figs. 4A and B, upper panels), FFG and LOC reached their maximal activation earlier than frontoparietal areas. Event-related analysis revealed furthermore that at short T2 lags, perceptual analysis in occipitotemporal areas differed for detected as compared to missed targets. Activation was largest for *T2 missed (lag 1, 2)* trials and smallest for *T2 detected (lag 1, 2)* trials (Fig. 4A, Table 4). Intermediate activation was observed for the *no T2* and the *T2 detected (lag 7)* conditions. While activation in these two conditions did not differ significantly, it was found to be significantly larger than in the *T2 detected (lag 1, 2)* and significantly smaller than in the *T2 missed (lag 1, 2)* condition for some of the regions analyzed in detail (Table 4).

Amygdala

Interestingly, a negative contrast was found for the left amygdala, resulting from a deactivation relative to baseline for the *T2 detected (lag 1, 2)* condition. This deactivation was significantly different from activity in the *T2 missed (lag 1, 2)*, *no T2*, and *T2 detected (lag 7)* conditions (Fig. 4A, Table 4). The maximum of the deactivation was reached at the same time as the maximal activation in the occipitotemporal areas (Fig. 4A).

Frontoparietal cortex

Fig. 4B and Table 4 summarize our data for the frontoparietal ROIs. In superior and inferior frontal areas, especially in a SFG/ACC cluster (Fig. 4B, Table 4), activation was largest for *T2 detected (lag 1, 2)* trials, and smallest for the *no T2* condition, and intermediate for the condition *T2 detected (lag 7)*. Activation in the *T2 missed (lag 1, 2)* condition was yet significantly higher than in the *no T2* condition. ROIs in the inferior parietal lobules (IPL posterior left and right, Fig. 4B) showed a very similar activation pattern, with the exception that a higher BOLD increase was observed in the *T2 detected (lag 7)* condition that was comparable to the activity in the *T2 detected (lag 1, 2)* condition. The left frontal cortex (LFC) also showed significantly higher activation for the two conditions where T2 was detected, as compared to the *T2 missed (lag 1, 2)* and the *no T2* condition (Fig. 4B, Table 4). In this case, the latter two did not differ significantly. Event-related analysis of fMRI responses yielded yet another interesting observation with regard to the time course of activation. In all areas of the frontoparietal network, activation differed between detected and missed T2 stimuli already for the second volume, whereas such a difference is observed only later in the occipitotemporal areas.

Discussion

In the present study, neural correlates of visual awareness were investigated by comparing conditions differing with regard to the physical presence of a second target, the temporal distance between the first (T1) and the second target (T2), and the conscious perception of T2. Activation in occipitotemporal areas specialized to process visual stimulus materials was found to be negatively correlated with the detection of T2 presented at short lags. In contrast, activation of frontal and parietal areas seems to reflect the explicit perception of T2, since BOLD increase was consistently stronger for conditions where T2 had been detected. However, for the majority of these areas activation differed between the *T2 missed* and the *no T2* conditions, suggesting that the absence of an explicit percept in the two conditions does not reflect the same process. While in the *no T2* conditions, the occipitotemporal areas simply supply no T2-related visual information, the *T2 missed* condition may imply incomplete processing of target-related

Table 3
Coordinates of voxel clusters significantly different between the *T2 detected (lag 1, 2)* and the *T2 missed (lag 1, 2)* condition

Area		Voxels in cluster	Talairach coordinates		
			x	*y*	*z*
Left	AMY	68	−24	−11	−12
	LFC	141	−43	−1	39
	LOC	274	−41	−70	3
		57	−38	−84	1
		83	−24	−83	5
		57	−19	−89	9
	FFG	233	−36	−69	−12
		192	−28	−87	−9
	IPL posterior	152	−28	−71	35
Right	LOC	52	47	−66	5
	FFG	307	26	−66	−11
		132	36	−60	−14

Coordinates are shown for the center of mass together with the number of active voxels in each cluster.

Abbreviations: AMY—amygdala, FFG—fusiform gyrus, IPL—inferior parietal lobule, LFC—lateral frontal cortex, LOC—lateral occipital complex.

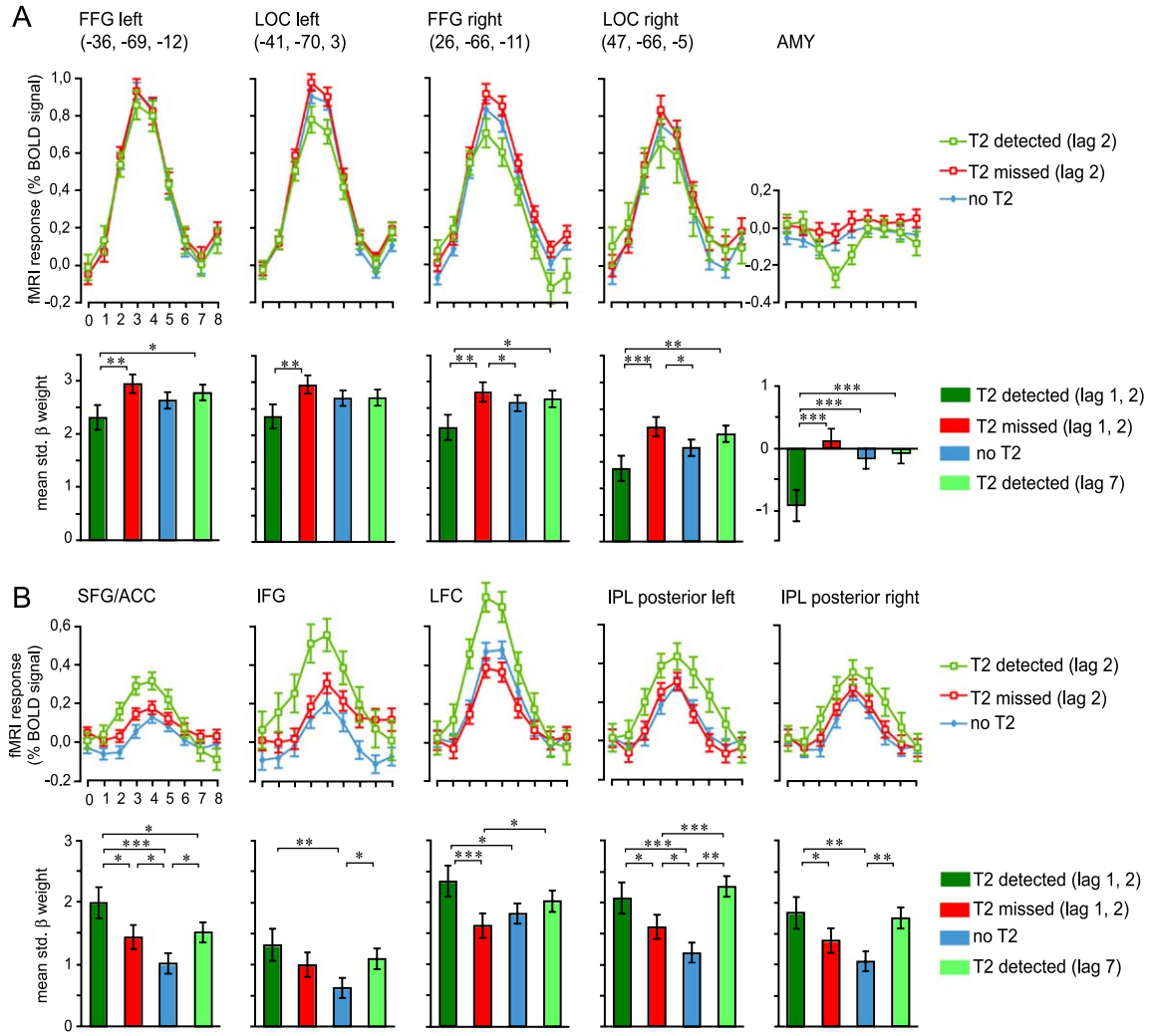

Fig. 4. Results of a detailed region of interest (ROI) analysis. (A) ROIs in occipitotemporal cortex and amygdala. (B) ROIs in parietal and frontal cortex. The upper panels show event-related averages of the hemodynamic response. Percent signal change, error bars: ±SE. In the lower panels, mean standardized beta weights of predictors as revealed by fixed-effects GLM are plotted. Error bars: ±SE. Significant GLM contrasts are indicated by asterisks: $*P \leq 0.05$, $**P \leq 0.01$, $***P \leq 0.0001$. See Table 4 for a complete list of GLM contrasts. Abbreviations: AMY—amygdala, FFG—fusiform gyrus, IFG—inferior frontal gyrus, IPL—inferior parietal lobe, LFC—lateral frontal cortex, LOC—lateral occipital complex, SFG/ACC—superior frontal gyrus/anterior cingulate cortex.

information in the selection process carried out by the frontoparietal network.

Occipitotemporal cortex

Both the FFG and LOC have been related to processing of letter stimuli (Goebel et al., 1998; Joseph et al., 2003; Polk et al., 2002). As expected from the role of these areas in perceptual analysis of the stimulus material, in the present study, these regions reached their maximal activation earlier than frontoparietal areas. However, activity in these areas was significantly larger when T2 was missed than when it was detected, which is contrary to what might be expected from previous work (Marois et al., 2004). Activation was intermediate between the two other conditions when T2 was presented at lag 7 or was not present at all. The most likely account for this pattern of activation seems that the attentive search for a target in the letter stream was aborted early in *T2 detected (lag 1, 2)* trials, whereas for the other conditions, the stream was searched until its end. By this interpretation, it could also be explained why activation in occipitotemporal areas did not follow the pattern observed by Marois et al. (2004), who found activation in parahippocampal place area to be largest for hits, intermediate for misses, and smallest for correct rejections. In their study, essentially only one T2 lag was investigated and the RSVP sequence ended after the T2+1 item in all conditions. This did not allow effects of active termination of search to emerge, which are likely to be seen in the present findings.

If the interpretation regarding termination of search holds true, activation should be comparable for trials in which T2 was missed and in which it was not presented. However, at least for some ROIs, the two conditions were found to differ significantly (Fig. 4B, Table 4), indicating that early termination of search might only be part of the puzzle. One option might be that activation in occipitotemporal areas also reflects differences in T1 processing. Yet, as for the present study, the design was chosen to be as comparable as possible to the wealth of previous attentional blink studies, at this point, it remains an open question whether such an interpretation is valid. Because in our study both T1 and T2 were

Table 4
Results of ROI GLM contrasts

ROI		Talairach coordinates			Contrasts					
		x	*y*	*z*	T2 det. (lag 1, 2) – no T2	T2 miss (lag 1, 2) – no T2	T2 det. (lag 1, 2) – T2 miss. (lag 1, 2)	T2 det. (lag 7) – no T2	T2 det. (lag 7) – T2 det. (lag 1, 2)	T2 det. (lag 7) – T2 miss. (lag 1, 2)
Left	Frontal									
	SFG/ACC	−2	19	47	4.28***	2.14*	2.53*	2.37*	−2.05*	0.41
	IFG	−43	13	5	3.04**	1.91	1.47	2.23*	−0.96	0.49
	LFC	−43	−1	39	2.33*	−0.98	3.31***	0.97	−1.4	2.00*
	Parietal									
	IPL anterior	−40	−50	39	2.50*	2.43*	0.43	3.20**	0.46	1.01
	IPL posterior	−29	−67	36	3.90***	2.13*	2.14*	5.09***	0.81	3.29***
	Occipital									
	LOC	−41	−70	3	−1.75	1.45	−3.12**	0.05	1.78	−1.37
		−38	−84	1	−1.16	1.10	−2.19*	−0.11	1.04	1.20
		−24	−83	5	−0.82	2.42*	−3.01**	0.11	0.91	−2.26*
		−19	−89	9	−0.34	2.05*	−2.18*	0.53	0.82	−1.45
	FFG	−36	−69	−12	−1.60	1.75	−3.22**	0.76	2.27*	−0.91
		−28	−87	−9	−1.58	0.69	−2.26*	0.31	1.84	−0.36
	Amygdala									
	AMY	−24	−11	−12	−3.44***	1.48	−4.90***	0.42	3.78***	−1.01
Right	Parietal									
	IPL anterior	46	−43	45	2.57*	1.62	1.23	2.12*	−0.60	0.66
	IPL posterior	37	−58	38	3.43***	1.71	2.03*	3.29***	−0.37	1.80
	Occipital									
	LOC	47	−66	5	−1.86	2.23*	−3.93***	1.36	3.10**	−0.74
	FFG	26	−66	−11	−2.21*	1.10	−3.28**	0.33	2.50*	−0.73
		36	−60	−14	−1.95	1.70	−3.55***	1.02	2.86**	−0.59

t values and significance of paired *t* tests.
Abbreviations: AMY—amygdala, FFG—fusiform gyrus, IFG—inferior frontal gyrus, IPL—inferior parietal lobule, LOC—lateral occipital complex, SFG/ACC—superior frontal gyrus/anterior cingulate cortex. T2 det.—T2 detected.
* $P \leq 0.05$.
** $P \leq 0.01$.
*** $P \leq 0.0001$.

letters, activation related to processing of either cannot be separated clearly. In order to test this hypothesis, the use of target categories known to be processed in different visual areas such as scenes, faces, or letters would be required (Epstein and Kanwisher, 1998; Goebel et al., 1998; Joseph et al., 2003; Kanwisher et al., 1997; Marois et al., 2004). The resulting activation levels in these areas could then be analyzed as a function of T2 detection.

Frontoparietal cortex

A network of lateral frontal, anterior cingulate, and intraparietal areas previously implicated in directing visual attention (Corbetta et al., 1998; Coull and Frith, 1998; Coull and Nobre, 1998; Kanwisher and Wojciulik, 2000) was postulated to represent the attentional bottleneck to perceptual awareness (Marois et al., 2000, 2004). In overall agreement with these earlier studies, our data also suggest that parietal and frontal areas are involved in the attentional blink. Specifically, activation of lateral frontal and parietal areas seems to reflect the explicit perception of T2, since signal increases were consistently stronger for conditions where T2 had been detected.

For short T2 lags, lateral frontal activation depended on whether T2 was detected, and there was no significant difference between conditions in which subjects failed to see T2 and in which T2 was physically absent (Fig. 4B). Interestingly, comparable ROIs were found to be differentially active in all previous neuroimaging studies of the attentional blink (Marcantoni et al., 2003; Marois et al., 2000, 2004), which might indicate a specific role of lateral frontal cortex in the attentional blink. Marois et al. (2004) suggest that lateral frontal activation is associated with consolidation and maintenance of targets in working memory for later report (Courtney et al., 1998a,b). In line with a working memory related interpretation of LFC activation, ERP studies of the attentional blink show that the P3 component, assumed to specifically indicate working memory processes (Donchin and Coles, 1988; Verleger, 1988), is impaired during the attentional blink (Kranczioch et al., 2003; Rolke et al., 2001; Vogel and Luck, 2002; Vogel et al., 1998). However, in our *T2 detected (lag 7)* condition, activation in LFC did not rise significantly above activation in the *no T2* condition, which is inconsistent with an interpretation mainly applying to working memory processes. On the other hand, Marcantoni et al. (2003) propose that lateral frontal cortex might be involved in resolving dual task interference. As dual task interference can be assumed to be rather small if T2 is presented at lag 7, our results might thus indicate that lateral frontal cortex activation reflects a combination of both, that is, working memory processes, but also to some degree, the interference between items.

For the other areas of the frontoparietal network, activation profiles were generally similar to LFC. However, in superior frontal and inferior frontal regions, as well as in the parietal ROIs,

activation differed between the *T2 missed* and the *no T2* conditions, suggesting that the absence of an explicit percept in the two conditions is due to different processes. We hypothesize that the slightly larger activity in the *T2 missed* condition reflects that in these areas target-related information is processed, yet only incompletely. A comparable account has been made recently for data indicating that missed target stimuli were processed in various frontal and temporoparietal areas (Shulman et al., 2003). It cannot be fully ruled out however that it is not processing of unconscious information that is reflected in the activations associated with missed T2. Rather, processing of information derived from trials in which subjects could get some information regarding T2, yet were too conservative in their decision criterion might have led to activations in parietal and frontal areas. This is a potential objection to all studies using a subjective rather than objective criterion for distinguishing conscious and unconscious processing (Palmer, 1999).

The activation pattern in inferior frontal cortex (IFG) was comparable to that of superior frontal/anterior cingulate cortex (SFG/ACC). Similar to lateral frontal areas, inferior frontal cortex has been related to working memory, specifically object working memory (Courtney et al., 1997, 1998a,b). On the other hand, inferior frontal regions have been observed to be active in a number of neuroimaging studies with interfering response alternatives or interfering tasks, frequently with concurrent activation in anterior cingulate/superior frontal cortex (Braver et al., 2003; Dove et al., 2000; Schubert and Szameitat, 2003). Areas in the anterior cingulate sulcus and superior frontal gyrus (SMA, pre-SMA) have been related to motor functions (Paus, 2001; Picard and Strick, 1996), and it has been suggested that the increased activation in anterior cingulate cortex mainly observed for detected T2 might be response related (Marois et al., 2004), maybe reflecting indecision or conflict monitoring processes (Botvinick et al., 1999; Carter et al., 1998; Paus, 2001). In line with this response-related account, no activation in anterior cingulate cortex was found in an attentional blink study in which no motor response was required (Marcantoni et al., 2003).

Parietal cortex, on the other hand, has been related to controlling the distribution of attentional resources among visual events (Coull and Nobre, 1998; Marois et al., 2004; Wojciulik and Kanwisher, 1999). It remains open why, in contrast to our findings, no differences in parietal activations were observed in the Marois et al. (2004) study. Besides the different stimulus materials used, the second major difference between studies is that while in the present study, T2 was followed by at least seven distractor stimuli, in the study of Marois et al. T2 was always the second-to-last stimulus. It seems unlikely that parietal activations observed in the present but also previous attentional blink studies (cf. Marois et al., 2000, 2003) are related to the use of alpha-numeric stimulus material. Accordingly, differences might be related to the second issue: with only one distractor following T2, the need to reorient attentional resources away from distractors and to the target might be largely reduced, resulting in only small differences between T2 detected and T2 missed trials.

Generally, we observed activation of the frontoparietal network to be larger in the left hemisphere. The left inferior and superior parietal cortex has been found to be selectively activated in passive viewing and silent naming of letters (Joseph et al., 2003). A left-hemispheric bias of parietal activation was also observed in RSVP tasks using letter stimuli (Marcantoni et al., 2003; Wojciulik and Kanwisher, 1999) Thus, the left-hemispheric bias in activation found might be due to the stimulus material used. Yet as the stimulus material was equal in all conditions, this alone cannot explain the differences in activation. Similar to spatial orienting of attention, orienting attention in time has been related to a frontoparietal network involving inferior parietal areas but with a left-hemispheric bias (Coull and Nobre, 1998). Even though temporal attention or attentional orienting in time is not to equate with the attentional blink (Coull and Nobre, 1998), in attentional blink experiments, items are nevertheless presented at different points in time and, therefore, attention has to be redistributed across time to allow successful processing of both T1 and T2. Thus, even though the predominant left hemispheric activation in the present study might be related to the stimulus material, it is likely to reflect the reallocation of attention to T2.

Limbic regions

Interestingly, we found the left amygdala to be deactivated relative to baseline for *T2 detected (lag 1, 2)* trials. It has been suggested that activation in ventromedial frontal cortex and amygdala produced by emotional arousal favors automatic processing and interferes with performance in non-automatic tasks. To prevent interference, this activation might be inhibited during non-automatic processing (Drevets and Raichle, 1998; Shulman et al., 1997). In line with these suggestions, exploratory analysis of a ROI in ventromedial frontal cortex (x, y, z = 2, 41, 6) also revealed a significant deactivation for *T2 detected (lag 1, 2)* as compared to *T2 missed (lag 1, 2)* trials [q(FDR) $\leq$ 0.08, $P \leq$ 0.001]. Thus, it might be that to some degree, the deactivations for the *T2 detected (lag 1, 2)* condition observed in our study also reflect inhibition of automatic processing, which becomes specifically evident in this condition of high demand on processing resources. Clearly, however, at this point, this interpretation is highly speculative, and further research is needed to test this hypothesis.

Implications for theories of the attentional blink

Most models of the attentional blink assume two stages of processing. In the first stage, stimuli are identified automatically, but to be reportable stimuli need to be consolidated in a second, capacity-limited stage also equated with working memory (Brehaut et al., 1999; Chun and Potter, 1995; Jolicoeur and Dell'Acqua, 1998; Shapiro et al., 1994; Vogel et al., 1998). While in the interference model (Isaak et al., 1999; Shapiro et al., 1994), it is further suggested that potential targets are passed on to the second stage where they compete for selection, the two stage model (Chun and Potter, 1995; Potter et al., 2002) postulates that T2 does not enter the second stage as long as this is occupied by T1. The response pattern that we have observed in areas of the frontoparietal network is not in line with the assumption of the interference model that both targets reach working memory. Rather, our findings support the two-stage model indicating that T2 frequently fails to reach working memory.

However, our finding of increased activation for *T2 missed* as compared to *no T2* trials in inferior frontal, parietal, and superior frontal/anterior cingulate cortex also strongly suggests that targets that eventually do not reach awareness are processed beyond a first stage of perceptual identification (but see also discussion of frontoparietal cortex). In a hybrid model of the

attentional blink (Vogel et al., 1998) combining the interference and the two-stage models, it has been suggested that after being identified in the first stage, potential target items are initially stored in a conceptual short-term memory (CSTM) buffer, where they are prone to decay and to replacement by other stimuli. Attentional resources for the transfer of potential targets into a more durable and reportable form (or into visual working memory) are limited, and thus T2 cannot be consolidated as long as T1 is transferred. This is assumed to result in errors in the report of T2 for a subset of trials. Thus, assuming that the neural substrate of the first stage resides in visual cortex and the neural substrate of working memory consolidation is in lateral frontal cortex (Marois et al., 2004), activation in inferior frontal, superior frontal/anterior cingulate, and parietal areas might reflect processing in the CSTM buffer. Thus, our results suggest that models of the attentional blink might be expanded by including a third processing stage prior to working memory consolidation, as has been proposed by Vogel et al. (1998).

Implications for visual awareness

Robust evidence suggests that activation of neural representations within visual cortex is not sufficient for access of visual stimuli to awareness. Rather, additional contributions from parietal and frontal areas seem a necessity (Beck et al., 2001; Dehaene et al., 2003; Lumer and Rees, 1999; Marois et al., 2004; Portas et al., 2000; Rees et al., 2002). Our data suggest that intraparietal regions, anterior cingulate cortex, as well as regions in inferior frontal gyrus and lateral frontal cortex may be part of such a selection network. These areas may exert top-down control over processing in sensory cortices, providing 'bias signals' that can modulate the selection of stimuli in a context-dependent fashion (Engel et al., 2001; Leopold and Logothetis, 1999; Miller, 2000). However, unambiguous correlates of processing in sensory cortices specifically related to either T1 or T2 selection could not be provided in the context of the present study.

Selection of sensory signals for access to awareness has also been studied in the context of other paradigms like, for example, binocular rivalry (Blake and Logothetis, 2002; Engel and Singer, 2001; Fries et al., 2002; Leopold and Logothetis, 1999). Animal studies employing this paradigm have revealed that synchronization among cortical neurons as measured by intracranial recordings is likely to be important for selection (Engel et al., 2001; Fries et al., 2002). Interestingly, the changes are particularly prominent in the so-called gamma-band distinguished by synchronized activity at frequencies above 30 Hz (Fries et al., 2002). Likewise, numerous studies in humans (Debener et al., 2003; Fell et al., 2003) and animals (Fries et al., 2001; Steinmetz et al., 2000) show that attentional selection is associated with an increase of cooperativity in the gamma-band.

Taken together, the picture that emerges is that conscious awareness presupposes several interrelated processes, including sensory preprocessing by modality-specific cortical circuits, attentional selection by frontoparietal networks, and transfer of the selection results into working memory (Crick and Koch, 1990, 2003; Rees et al., 2002). If, as discussed above, gamma-band synchrony is indeed critically involved in this process, it may be predicted that higher gamma-band responses should be observed for detected T2 targets as compared to missed T2 stimuli. This prediction remains to be tested in future attentional blink experiments.

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