

Primary visual cortex reflects behavioral performance in the attentional blink

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When two masked targets are presented in a rapid sequence, attentional limitations are reflected in reduced identification accuracy for the second target (T2). We used functional magnetic resonance imaging to disentangle the distinct neural substrates of T2 processing during this attentional blink phenomenon. Spatially separating the two targets allows the retinotopic localization of the different stimuli's encoding sites in primary visual cortex (V1)

and thus enables activation elicited by each target to be differentially measured in V1. The encoding location of the second target mirrored T2 identification accuracy in a retinotopically specific manner. These results are the first evidence for effects of behavioral performance on hemodynamic responses in V1 under conditions of the attentional blink. *NeuroReport* 19:1277–1281 © 2008 Wolters Kluwer Health | Lippincott Williams & Wilkins.

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Introduction

When two masked targets have to be identified in a rapid serial visual presentation (RSVP) of distractor items, attentional limitations are reflected in reduced identification accuracy for the second target, given both targets are separated by short temporal lags. In analogy to eye blinks, this phenomenon has been termed as an 'attentional blink' (AB) [1] and is thought to reflect the limited capacity of temporal attentional processes [1,2]. Similar results have been obtained when participants were required to report two spatially separated targets (T1 and T2) appearing at varying stimulus onset asynchronies (SOAs), followed by pattern masks [3–6]. The time course of interference resulting from attention to T1 on the identification of T2 during this measurement of 'attentional dwell time' [3] is comparable to the AB. Correct report of T2 is poorest at short SOAs of about 200–400 ms between the two target items, and it improves with increasing lags up to about 700–800 ms. As performance is virtually identical for spatially separated masked targets and masked targets presented at fixation, interference effects revealed by the attentional dwell time paradigm and the AB paradigm are thought to represent the same limits of temporal attention [6].

Until recently, functional magnetic resonance imaging (fMRI) studies investigating the AB by displaying RSVP streams had to focus on the summed activation of both T1 and T2 because of the relatively coarse temporal resolution

of fMRI [7,8] or investigated brain activation under artificial conditions not reflecting the actual AB paradigm by excluding the second target and assessing hemodynamic activity to T1 only [9,10]. Only by choosing targets that activate highly specialized areas in extrastriate cortex such as the parahippocampal place area and the fusiform face area, activity of each target has been assessed separately [11–13]. Given a growing body of literature demonstrating a tight link between primary visual cortex (V1) activity and conscious perception (e.g. Ref. [14]), this study sought to elucidate the neural underpinnings of the AB at the earliest stage of visual cortical processing, that is, in V1.

Owing to the retinotopic organization of V1, spatially separating T1 and T2 allows the retinotopic localization of the different stimuli's encoding sites in V1 [15]. Crucially, this approach permits the measurement of T2-related activity uncontaminated by T1 processing. Adapting the attentional dwell time paradigm by Duncan *et al.* [3] in which two spatially separated targets appearing at varying SOAs are displayed and immediately followed by pattern masks, we used fMRI to compare activity elicited by T2 items in trials in which T2 was correctly reported to trials in which it was incorrectly reported within the precise encoding locations of the T2 stimuli in V1. In the light of converging evidence from single cell recordings, fMRI and lesion studies suggesting that V1 activity is crucial for conscious vision [14], we expected the T2 encoding locations

in V1 to be strongly activated in T2 correct trials opposed to T2 incorrect trials.

Methods

Participants

Three healthy right-handed participants (two females, ages 24, 25, and 28 years) were extensively tested in several behavioral testing sessions, fMRI sessions, and in an eye tracking session. The participants had normal or corrected to normal vision and no history of neurological disorder. In accordance with the Declaration of Helsinki, informed consent was obtained and observers were paid for their participation.

Stimuli and procedure

Participants could see the stimuli projected on a screen (spatial resolution 1024×768 pixels, refresh rate 60 Hz) placed outside the scanner through a mirror mounted on the head coil. The fixation display, subtending $3.75 \times 3.75^\circ$ at a viewing distance of 70 cm, was present throughout the trial and comprised a central fixation cross ($0.20 \times 0.20^\circ$) drawn in light gray and four gray frames indicating possible stimulus locations ($1.00 \times 1.00^\circ$). These frames consisted of four small squares arranged in a quadratic shape. On each trial, T1 (digit 1, 2, 3, or 4) appeared in one of the vertical positions of the display (either the top or the bottom frame), and T2 (letter A, B, C, or D) appeared in one of the horizontal positions (the left or the right frame; Fig. 1a). T1 and T2 were separated by an SOA of either 200 or 800 ms. Target characters were drawn in white Courier New font ($0.40 \times 0.50^\circ$), and were presented for 167 ms (T1) or 83 ms (T2). Each character was immediately followed by one of four randomly selected pattern masks ($0.60 \times 0.60^\circ$), which were made up of white line segments. The masks were displayed until the end of the stimulus presentation sequence.

After presenting the fixation display for 1000 ms, the two characters appeared as described above, the stimulus positions and the SOA varying randomly. Each stimulus presentation sequence lasted for 1 s; differences in SOAs were compensated by adjusting the duration of the presentation of the display after T2 presentation, which contained solely the two pattern masks. Subsequently,

participants were prompted to recall T1 and consecutively T2. In at least 896 practice trials, participants had been trained to enter their T1 and T2 responses in a time frame of 1117 ms, respectively. Responses were made with the right hand. Feedback was given immediately after the response had been executed by changing the fixation cross for 183 ms into blue (correct response) or red (incorrect response). The session consisted of four runs containing 256 trials each. Given the unpredictability of target locations, the short stimulus durations, and the requirement to fixate the eyes on the fixation point, it is unlikely that eye movements could play an important role here. This was confirmed in a separate eye tracking experiment outside the scanner (EyeLink 1000, SR Research, Osgoode, Canada) in which each participant conducted 320 trials. Eye movements as defined by deviations of more than 1.50° from the fixation cross were present in only 3% of all trials and these were solely because of slow drifts, neither saccades nor stimulus-related eye movements were recorded.

Functional MRI parameters

Images were acquired on a 1.5-T whole-body MRI scanner (Siemens Sonata Maestro; Siemens, Erlangen, Germany) using a 40 mT/m gradient system and a high-resolution eight-channel head coil (MRI Devices, Gainesville, Florida, USA). The session started with a localizer scan with seven sagittal slices, which was used to place 21 transversally oriented slices (voxel size $3 \times 3 \times 3$ mm, slice thickness 3 mm) to the caudal part of the brain of each participant, covering at least the occipital, parietal, and temporal lobes. There were 768 volumes per run (repetition time 2000 ms, echo time 50 ms, flip angle 85° , field of view 192 mm, interleaved acquisition). A T1-weighted magnetization-prepared rapid acquisition gradient echo scan (voxel size $1 \times 1 \times 1$ mm) was recorded after the retinotopic localization run at the end of the session.

Localization of T2 encoding positions in V1

Brain clusters encoding the target stimuli during the attentional blink experiment were retinotopically localized by presenting one flickering dartboard (0.70° in size,

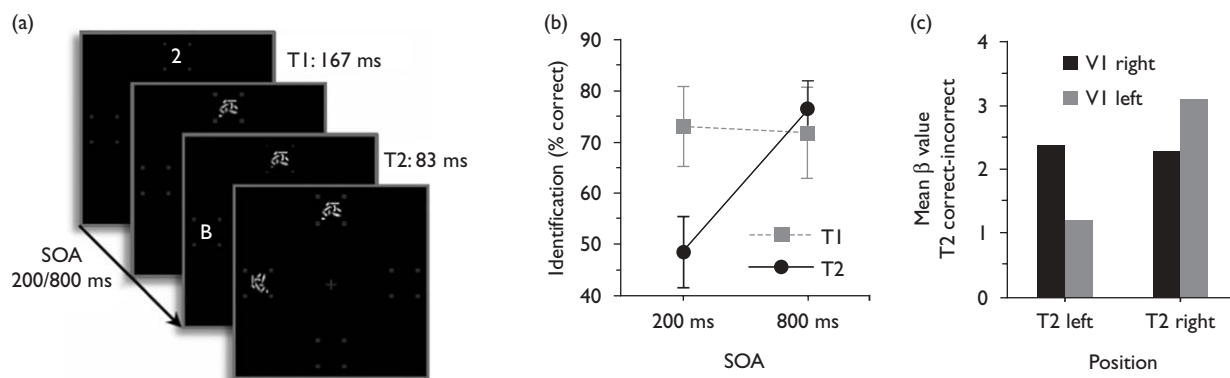


Fig. 1 (a) Schematic representation of a typical trial. First target (T1) (digit 1, 2, 3, or 4) was presented for 167 ms in one of the two vertical positions and second target (T2) (letter A, B, C, or D) was shown for 83 ms in one of the horizontal positions. Both targets were separated by a stimulus onset asynchrony of 200 or 800 ms and immediately followed by pattern masks. (b) Mean percentage of accurate T1 and T2 report for both stimulus onset asynchronies. T2 accuracy was analyzed contingently on correct T1 report. Error bars represent the standard errors of the means. (c) Results of the region of interest (ROI) analysis. Depicted are β weight differences between T2 correct and T2 incorrect (contingent on T1 correct) trials separately for T2s presented at the left and the right positions, respectively, and for both ROIs representing the two distinct T2 encoding positions in V1.

maximum contrast, 8Hz flicker rate) in each of the four locations in which the T1/T2 target stimuli were presented in the main experiment. Participants had to fixate a red cross in the middle of the screen throughout the run. Each single position was presented five times for a period of 30 s in a block design (scanning parameters identical to the main experiment, except repetition time 3000 ms, echo time 75 ms) such that alternating hemispheres were stimulated in the following order: left, right, top, and bottom. After each block displaying the flickering dartboard a fixation cross was presented for 30 s.

Data analysis

The data were analyzed using standard linear regression techniques implemented in SPM5 (<http://fil.ion.ucl.ac.uk/spm>). The imaging series were time corrected, realigned, and coregistered against a high-resolution magnetization-prepared rapid acquisition gradient echo T1 image, normalized to a standard template from the Montreal Institute of Neurology (MNI152T1), and smoothed with an isotropic 6-mm Gaussian kernel. The onset vectors for T2s correctly reported and T2s incorrectly reported were convolved with a canonical hemodynamic response function (HRF), mean corrected and regressed upon the data with standard linear regression. In addition, data were high-pass filtered to remove subject-specific drifts in signal, and corrected for intrinsic serial autocorrelations. The full-volume group analysis was performed according to the random effects procedure, using the single-subject contrast images as input. Group statistical parametric maps were generated using a one-sample *t*-test with a voxel threshold of $P < 0.005$ and an extent threshold of 20 voxels.

Images gathered during the retinotopic localization run were preprocessed identically to the images from the experimental runs. The onset vector for each dartboard representing the T1/T2 positions was convolved with a canonical HRF and mean corrected to set up four regressors for the SPM analysis. *T*-test contrast vectors were defined for both regressors corresponding to the two T2 locations against all other regressors to reveal voxels responding solely to one particular T2 position. For each participant, two regions of interest (ROIs) covering a sphere of 10 mm each were defined according to the peak activations corresponding to the left and right dartboard presentation, respectively. ROI data analysis was supplied by the ROI toolbox MARSBAR (<http://marsbar.sourceforge.net>). Four onset vectors according to T2 position and participants' T2 performance were convolved with a canonical HRF, mean corrected and regressed upon the data from each of the two ROIs with standard linear regression. Beta values for T2 correct and T2 incorrect (separately for both sides of T2 presentation) were derived from each ROI and contrasted with a one-tailed *t*-test.

Results

Behavioral results

Analyses of T2 performance and fMRI analyses were based solely on trials in which T1 was accurately reported as in T1 incorrect trials the source of error is unknown (e.g. Ref. [2]). The present paradigm successfully induced an AB as demonstrated by the difference in T2 accuracy between long and short SOAs [27.93%; $t(2)=3.39$, $P < 0.05$, one-tailed].

Table 1 Results of the region of interest analysis

	T2 left correct > incorrect	T2 right correct > incorrect
V1 right	$t(2)=4.01$, $P < 0.05$	$t(2)=1.55$, NS
V1 left	$t(2)=1.68$, NS	$t(2)=3.53$, $P < 0.05$

One-tailed *t*-tests were used to compare β weight differences for correctly and incorrectly reported T2s separately for both T2 positions and region of interests.

T2, second target; V1, primary visual cortex.

Figure 1b shows the percentages of correct T1 and T2 identification for both SOAs.

T2 encoding clusters in V1

Figure 1c and Table 1 depict the results of the ROI analysis. Beta weight differences estimated for T2 correct and T2 incorrect trials were calculated separately for T2s appearing in the left and right hemifield, respectively, and for both ROIs representing the two T2-encoding positions in V1. Activity in both ROIs was higher for correctly reported T2s as compared with incorrectly reported T2s (Fig. 1c). One-tailed *t*-tests comparing β weight differences for correctly and incorrectly reported T2s separately for both T2 positions and both ROIs (Table 1) demonstrated that these effects were retinotopically specific, that is, significant differences between correct and incorrect T2 trials were only revealed in V1 ROIs corresponding to the specific hemifield in which T2s were presented.

Full-volume analysis

Brain clusters exhibiting stronger activation during T2 correct compared with T2 incorrect trials, regardless of the T2 position, are shown in Fig. 2. Right-lateralized occipital areas corresponding to BAs 18 and 19 were significantly activated. At a less conservative threshold, these clusters were similarly present in the left hemisphere. Additionally, bilateral foci in the precuneus and the posterior parietal cortex including inferior parietal lobules, angular gyri, and right supramarginal gyrus exhibited stronger activation during T2 correct trials. Finally, left cingulate gyrus was significantly activated. For the reverse contrast (T2 incorrect > T2 correct), no significantly activated clusters were obtained.

Discussion

This study provides evidence that activity in V1 reflects the observers' correct T2 report during the AB. Moreover, stronger V1 activity for accurately identified T2s was shown to be retinotopically specific. Further, the full-volume analysis revealed that activation of posterior parietal cortex, precuneus, and middle occipital gyri correlated with correct T2 identification.

To the best of our knowledge, this is the first study demonstrating stronger V1 activation for correct T2 identification in the AB. Earlier studies using RSVP streams to investigate hemodynamic responses to correctly identified targets in the AB reported the somewhat counterintuitive finding of higher activity in extrastriate cortex during conditions of reduced T2 performance [7,8,11,13]. In contrast, Marois *et al.* [9] found activation of parahippo-

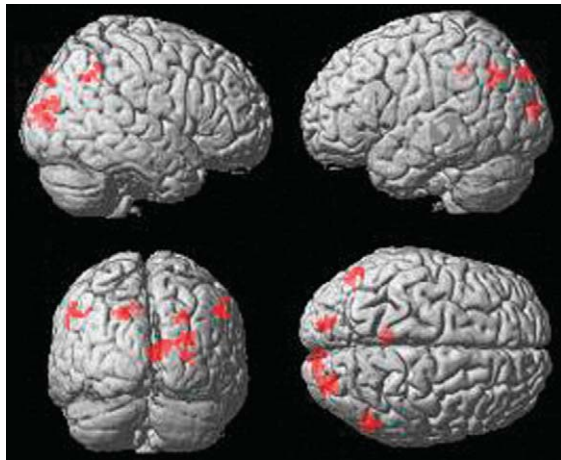


Fig. 2 Results of the full-volume random effects analysis comparing second target (T2) correct versus T2 incorrect trials (contingent on T1 correct), regardless of the T2 position ($P < 0.005$, extent threshold 20 voxels).

campal place area to mirror observers' perception of a T2 scene target. In these studies, the investigation of primary visual cortex activity has been neglected, probably because theories on the AB typically assume the second target deficit to arise from a capacity-limited higher processing stage (e.g. Refs [2,5]).

However, both cognitive perspectives on the AB [16] and evidence from fMRI studies investigating the relationship between V1 activity and conscious perception [14] have led us to the assumption that V1 is likely to be involved in target detection during the AB. Revealing the paradoxical result that unidentified T2s showed semantic priming effects for related words, but no repetition priming in a perceptual threshold task, Martens *et al.* [16] speculated that a corrupted feedback cycle from high-level presentations to the preceding lower level representations in V1 (for a review, see Ref. [17]) might be responsible for their finding. Concurrently, it has been shown that V1 activity closely resembles conscious perception (e.g. Ref. [18]) and visual attention (e.g. Ref. [19]). This study extends these findings to the domain of temporal attention as assessed by the AB paradigm.

The parietal foci of activation yielded in the full-volume analysis corroborate earlier studies that reported differential activity of posterior parietal cortex in the AB [7–10], although only Kranczoch *et al.* [7] found parietal cortex to reflect correct T2 identification. Given that patients with right parietal lesions suffering from unilateral visual neglect [20] and even nonneglect patients with lesions in the inferior parietal lobe [21] show impoverished T2 identification accuracy, posterior parietal cortex seems to play a critical role in the deployment of visuotemporal attention. Notwithstanding the restrictions posed by the sluggishness of the hemodynamic response that disallow definite conclusions about functional relationships over time, it is possible that conscious perception in the AB is mediated by reciprocal connections between posterior parietal and visual cortex (see Refs [17,22,23]).

Although it is not entirely clear which possible sources of error (e.g. mental distraction or guessing) contributed to a correct or incorrect T2 report in this study, it seems plausible that the present results reflect the importance of recurrent

processing involving higher-level areas like posterior parietal cortex and lower-level areas like V1 for correct T2 performance in the AB.

Conclusion

The present experiment suggests that conscious perception as defined by correct T2 identification during the attentional blink is reflected by retinotopically specific primary visual cortex activity. Possibly, recurrent connections between V1 and higher-order areas in the posterior parietal cortex are critical for conscious perception under conditions of high temporal attentional load.

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