Temporal Dynamics of the Attentional Spotlight: Neuronal Correlates of Attentional Capture and Inhibition of Return in Early Visual Cortex

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Abstract

■ A stimulus that suddenly appears in the corner of the eye inevitably captures our attention, and this in turn leads to faster detection of a second stimulus presented at the same position shortly thereafter. After about 250 msec, however, this effect reverses and the second stimulus is detected faster when it appears far away from the first. Here, we report a potential physiological correlate of this time-dependent attentional facilitation and inhibition. We measured the activity in visual cortex representations of the second (target) stimulus' location depending on the stimulus onset asynchrony (SOA) and spatial distance that separated the target from the preceding cue stimulus. At an SOA of 100 msec, the target yielded larger responses when it was presented near to than far away from the cue. At an SOA of 850 msec, however, the response to the target was more pronounced when it appeared far away from the cue. Our data show how the neural substrate of visual orienting is guided by immediately preceding sensory experience and how a fast-reacting brain system modulates sensory processing by briefly increasing and subsequently decreasing responsiveness in parts of the visual cortex. We propose these activity modulations as the neural correlate of the sequence of perceptual facilitation and inhibition after attentional capture. ■

INTRODUCTION

Processing of a target stimulus is influenced by a preceding cue stimulus that has entered the visual system shortly before. This interaction was demonstrated in a now classical series of experiments by Posner and colleagues in which temporal and spatial relations between cue and target determined whether target processing was facilitated or inhibited (Posner, 1985; Posner & Cohen, 1984). The experiments revealed a double dissociation between stimulus onset asynchrony (SOA) and spatial congruency of cue and target: At short SOAs, detection of the target was faster in valid trials, that is, when the target appeared at or close to the cue's location than somewhere else, whereas at SOAs exceeding some 250 msec, the effect reversed with targets reported faster in invalid trials, where the target appeared far away from the cue. This effect was explained in a model of transient attention whereby a cue popping out in the periphery would first attract attention to its location, whereas later the focus of attention would be redirected to the center and at the same time inhibited to return to the formerly visited location. The inhibitory aftereffect was labeled ''inhibition of return'' (IOR) and was proposed to support visual search behavior by preventing attention to return to the same location over and over again.

On a physiological basis, functional imaging in humans has demonstrated enhanced activity in visual areas coding the region to which attention is covertly directed and this has been taken as a correlate for superior behavioral performance for stimuli presented at the attended location (e.g., Brefczynski & DeYoe, 1999). Apart from one recent study (Liu, Pestilli, & Carrasco, 2005), however, this attention effect on visual cortex activity has been studied with functional imaging only in situations in which a symbolic cue (e.g., an arrow at the center of the screen) instructed subjects to direct attention voluntarily to the periphery. This situation is quite different from the reflexive attention shifts induced by salient peripheral stimuli and, in fact, lacks the inhibitory aftereffect. Hence, functional imaging results obtained with symbolic cues cannot be taken to generalize and reveal the neural mechanisms associated with attentional capture by peripheral cueing.

The reason why functional imaging studies have preferred symbolic cues over peripheral ones is simple: A peripheral cue is processed in the same retinotopic visual cortical regions as the subsequent target. Hence, when measuring hemodynamic responses that are smeared in relation to neural activity, it would appear

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impossible to disentangle attentional modulation of target processing from sensory cue–target interactions.

Here, we aimed at circumventing this pitfall by making use of the fact that the left and right visual hemifield are represented by the early visual cortices of separate hemispheres and of the fact that the attentional modulation operates the sensory retinotopic representation on a coarser grain. We presented cues and targets next to each other but on opposite sides of the vertical meridian. This approach left the aforementioned behavioral effects intact but allowed us to access activity in the visual cortex that represented the target locations only and hence should not be involved in sensory processing of the cues.

METHODS

The crucial question in our experiment was whether activity in retinotopic cortex representing the target location would be modulated as a function of delay and spatial proximity to a preceding noninformative peripheral cue. Our design exploited the fact that sensory receptive fields are more tightly spatially tuned than attentional fields (Müller & Kleinschmidt, 2004). In other words, the expanse of the visual field and, thus, the extent of cortical surface that benefits from attentional enhancement are larger than the central portion of this field that responds to sensory stimulation. This allowed us to present cues and targets next to each other but at slightly offset positions. Still, it would usually remain difficult to dissociate the sensory responses to cues and targets at these distances by functional neuroimaging given hemodynamic point spread. However, by placing cue and target positions onto opposite sides of the vertical meridian we could use a situation where closely neighboring visual field positions are represented at cortical locations on separate hemispheres. With these modifications, we could use functional neuroimaging to investigate the neural underpinnings of the behavioral effects described by Posner and colleagues some 20 years ago (Posner, 1985; Posner & Cohen, 1984).

Subjects

Ten healthy subjects (age range, 21–28 years; 7 women) with normal vision were paid to take part in the study, which was conducted in conformity with the Declaration of Helsinki and approved by the local ethics committee.

Behavioral Procedure

A gray fixation cross at the center of the black screen and two gray placeholder square frames (side length, 1.5°) placed 1.5° left of the vertical and 7.5° above and below the horizontal meridian, respectively, were presented throughout the whole experiment (see Figure 1). Subjects were instructed to fixate the central cross at all

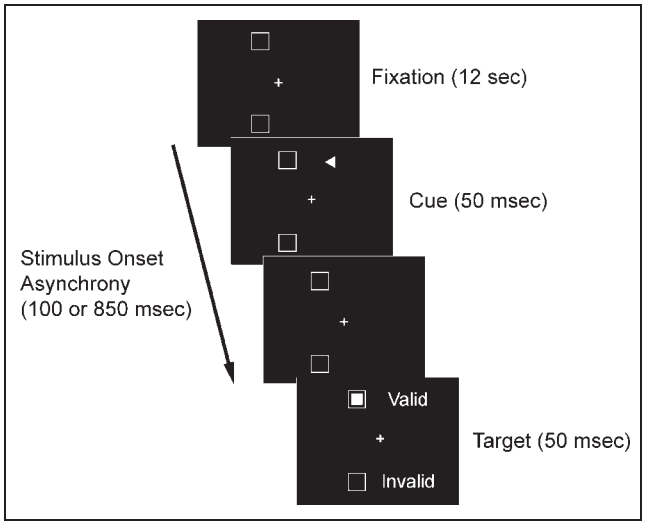

Figure 1. Schematic illustration of the paradigm.

times. Each trial began with a fixation period of 12 sec, then a cue stimulus was presented in the upper or lower visual hemifield for 50 msec. Cues were triangles pointing to the left that were placed opposite the frames, that is, 1.5° right to the vertical meridian in the upper or lower visual field. After offset of the cue, the screen apart from the fixation cross and the frames remained blank for 50 or 800 msec. Then the target, a filled square (1° side length, gray color) was presented either within the upper or lower frame for 50 msec, to which subjects were instructed to respond as fast as possible with a button press. In 43.75% of trials, the target was presented at the location predicted by the cue; in the same amount of trials, however, it appeared at the opposite position. In 12.5% of trials, no target appeared (catch trials) in order to avoid automatic responding to the cue. Subjects had 1000 msec to respond, and then the next trial started. Valid, invalid, and catch trials, and trials with different SOAs were presented in randomized order. Altogether, 384 trials per subject were acquired in four functional runs on two consecutive days. Before scanning, subjects completed ~30 training trials with shorter fixation periods (3 sec) to become acquainted with the task.

Stimulus presentation was controlled by a personal computer running the ERTS software package (Experimental Run Time System; Berisoft, Frankfurt, Germany) that was triggered by the scanner. For better temporal resolution of blood oxygenation level dependent (BOLD) responses the offset between trigger and stimulus presentation varied within a range of 0 to 1500 msec in 500-msec steps.

Functional Magnetic Resonance Imaging Procedure

Functional magnetic resonance imaging (fMRI) data were acquired with a 3-T Allegra scanner (Siemens, Erlangen, Germany) equipped with a standard head coil. Stimuli

were presented through MR-compatible video goggles (MRVision 2000 Ultra; Resonance Technology, Northridge, CA; resolution, 180,000 pixels; field of view, 30°). A custom-built fiber-optic response box was used for reporting. In each session and from each subject, we obtained BOLD contrast (T* weighted) echo-planar image volumes (repetition time [TR] = 2000 msec; echo time [TE] = 30 msec; flip angle = 60°; 32 axial slices; voxel size $3.3 \times 3.3 \times 3.3$ mm^3) and T1-weighted three-dimensional (3-D) structural scans (magnetization-prepared rapid gradient-echo sequence: TR = 2250 msec; TE = 2.6 msec; flip angle = 9°; inversion time (TI) = 900 msec; 176 sagittal slices; voxel size = 1 mm^3).

Eye Movement Control

Using a digital infrared eye tracker (Ober 2; Permobil Meditech, Timra, Sweden), we recorded the subjects' eye movements during training of the experiment outside the scanner in order to ensure that they followed our instructions and kept central fixation throughout the experiment. All subjects, most of whom had participated in other studies requiring central fixation before, produced saccades in less than 1% of trials and hence were considered suitable for the MR sessions.

Data Analysis

Behavioral Data

Mean reaction times (RTs) for correct answers were entered in a repeated measures analysis of variance (ANOVA) with the factors Validity and SOA.

fMRI: Preprocessing

Brainvoyager QX software (BrainInnovation, Maastricht, the Netherlands) was used for all fMRI analyses. The first two volumes of each functional run were discarded; the remaining were corrected for slice scan time differences within a volume, motion-corrected, and temporally high-pass filtered (three cycles per run). The functional data were coregistered with the 3-D MPRAGE data sets obtained in the same session that were then 3-D–3-D aligned to the MPRAGE data set acquired in the first session and transformed into stereotactic space (Talairach & Tournoux, 1988) such that volume–time courses from different sessions could be compared.

Cortical Surface Reconstruction

The cortical surface of each subject was reconstructed from the 3-D data set. The white matter was segmented by use of a grow-region function; then a sphere was covered smoothly around the segmented region, and the reconstructed white matter was expanded into the gray matter. After separation of the hemispheres, the sulci were smoothed by a cortical inflation procedure.

Retinotopic Mapping and Determination of Regions of Interest

BOLD responses to the targets were measured in regions of interest (ROIs) in retinotopic visual areas, similar to previous work (Müller, Bartelt, Donner, Villringer, & Brandt, 2003; Saenz, Buracas, & Boynton, 2002; Ress, Backus, & Heeger, 2000). ROIs were separately mapped in sessions where subjects, while keeping central fixation, passively watched checkerboard stimuli reversing at 8 Hz (see Figure 2). The checkerboard stimuli exactly matched the size and location of the cues and targets used in the experimental task. These ROIs were then subdivided according to the retinotopic boundaries of areas V1/2 and V3/V4, respectively, which were separately mapped by checkerboard stimulation along the horizontal and vertical meridians (Sereno et al., 1995). As the ROIs represented locations close to the vertical meridian that marks the borders of visual areas V1/V2 and V3/V4, respectively, we refrained from attempting to separate these areas further, as they appeared as continuous activated foci on the reconstructed surfaces. Note that for area V4 only a ventral representation exists; that is, all data for V4 were collected from the locations in the upper visual field.

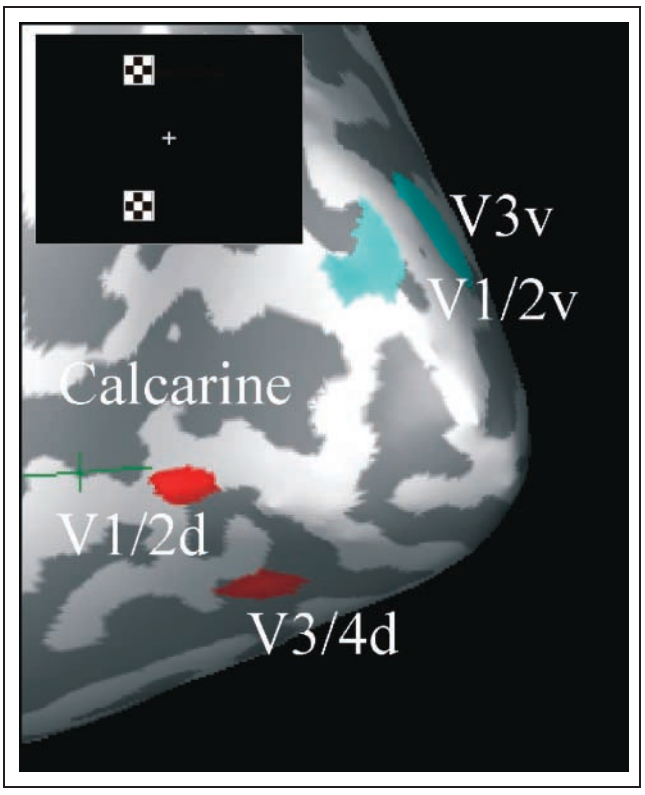

Figure 2. ROIs determined by passive stimulation at the upper (red) and lower (blue) target location.

Cueing Task: ROI Analysis

The BOLD response to the target was averaged across voxels of the ROIs representing the respective target location.[1] Valid and invalid trials and trials with short and long SOAs were analyzed separately. The 2 sec preceding the target served as a baseline. From the event-related time courses, the value at 6 sec after target stimulus that approximately represented the peak of the BOLD response was extracted. Because behavioral data showed no systematic differences between performance for targets in the upper and lower visual field, $F(1,9) = 0.23$, the peak values for the lower and upper target locations were collapsed. With these values, a repeated measures ANOVA with the factors Validity (valid, invalid), SOA (short, long), and Area (V1/V2, V3/V4) was calculated.

Cueing Task: Whole-brain Analysis

In order to test whether our version of the exogenous cueing task involved similar brain areas as reported in previous fMRI studies using the classic IOR paradigm (e.g., (Lepsien & Pollmann, 2002), we performed a whole-brain analysis of our functional data. After z transformation, a fixed-effects general linear model was used to compute statistical maps for the group average. In order to identify brain areas associated with IOR, the main effect of SOA was analyzed; that is, valid and invalid trials with long SOAs were contrasted with valid and invalid trials involving the short SOA. The effects were thresholded at correlation coefficients corresponding to $p < 10^{-5}$, uncorrected.

RESULTS

The behavioral results are summarized in Figure 3. At the short SOA, subjects were faster in valid than in invalid trials (412 vs. 422 msec, $t = -2.18$, $p < .03$), whereas at the long SOA, RTs were faster in invalid trials (378 vs. 412 msec, $t = 7.37$, $p < .001$). In other words, for valid trials, RTs were the same at short and long SOAs. These findings are reflected in the ANOVA results revealing a main effect for validity, $F(1,9) = 20.8$, $p < .001$; a trend for SOA, $F(1,9) = 3.4$, $p < .09$; and, most crucially, a Validity × SOA interaction with $F(1,9) = 32.4$, $p < .0001$. Hence, we found the usual facilitation for targets presented near the cued location at short SOAs and inhibition for these targets at long SOAs.

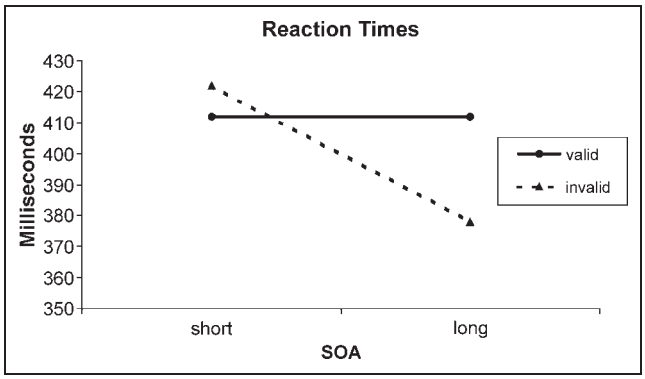

Figure 3. Reaction time data showing the typical IOR effect.

As accuracy reached near-ceiling levels (97% correct trials for valid trials with short SOAs, 95% correct trials for all other conditions) we renounced a further analysis of these data.

The BOLD signals in the visual cortex mirrored the behavioral data (see Figure 4). In all ROIs assessed, the BOLD responses to targets presented shortly after the cue were more pronounced in valid than in invalid trials ($t = 1.8$, $p < .05$), whereas at the long SOA, targets at invalid locations yielded the larger BOLD response ($t = -2.57$, $p < .02$). Hence, there was a Validity × SOA interaction with $F(1,9) = 5.9$, $p < .038$. Overall, the signal was stronger at the long SOA, $F(1,9) = 5.8$, $p < .039$), and in early visual areas, $F(1,9) = 7.5$, $p < .023$ for area. No other main effects or interactions were significant.

In the whole-brain analysis (see Figure 5) correlates of IOR were assessed by contrasting long-SOA versus short-SOA trials. The following brain areas were revealed: multiple regions in the lateral prefrontal cortex along the precentral sulcus, among them—in the latitude of the caudalmost part of the superior frontal sulcus—the frontal eye field (FEF). On the medial frontal cortex, activations were observed in the (pre)supplementary motor area (SMA) and nearby cingulate cortex. Other clusters of activation were located in the posterior parietal cortex (PPC) and in the vicinity of the temporoparietal junction (TPJ) and in the lateral occipital cortex (LOC).

Subcortical activity was seen in the thalamus bilaterally and in the superior colliculi (SC). Finally, IOR activated subareas of the cerebellum.

DISCUSSION

This study provides a potential physiological correlate for the facilitated versus inhibited detection of a peripheral stimulus, which is determined by its temporal and spatial relation to a preceding stimulus.

As our study design differed somewhat from the classical IOR paradigm in an attempt to avoid confounds from sensory cue–target interaction, it was crucial to compare both behavioral and imaging results to previous work employing the classic design. Behaviorally, we observed the typical results, that is, speeded responses at the short SOA in valid trials, where the second stimulus appeared close to the cued location, but faster responses at the long SOA in invalid trials, where the target was presented far away from the cue in the opposite hemifield. That is, the small spatial offsets, which

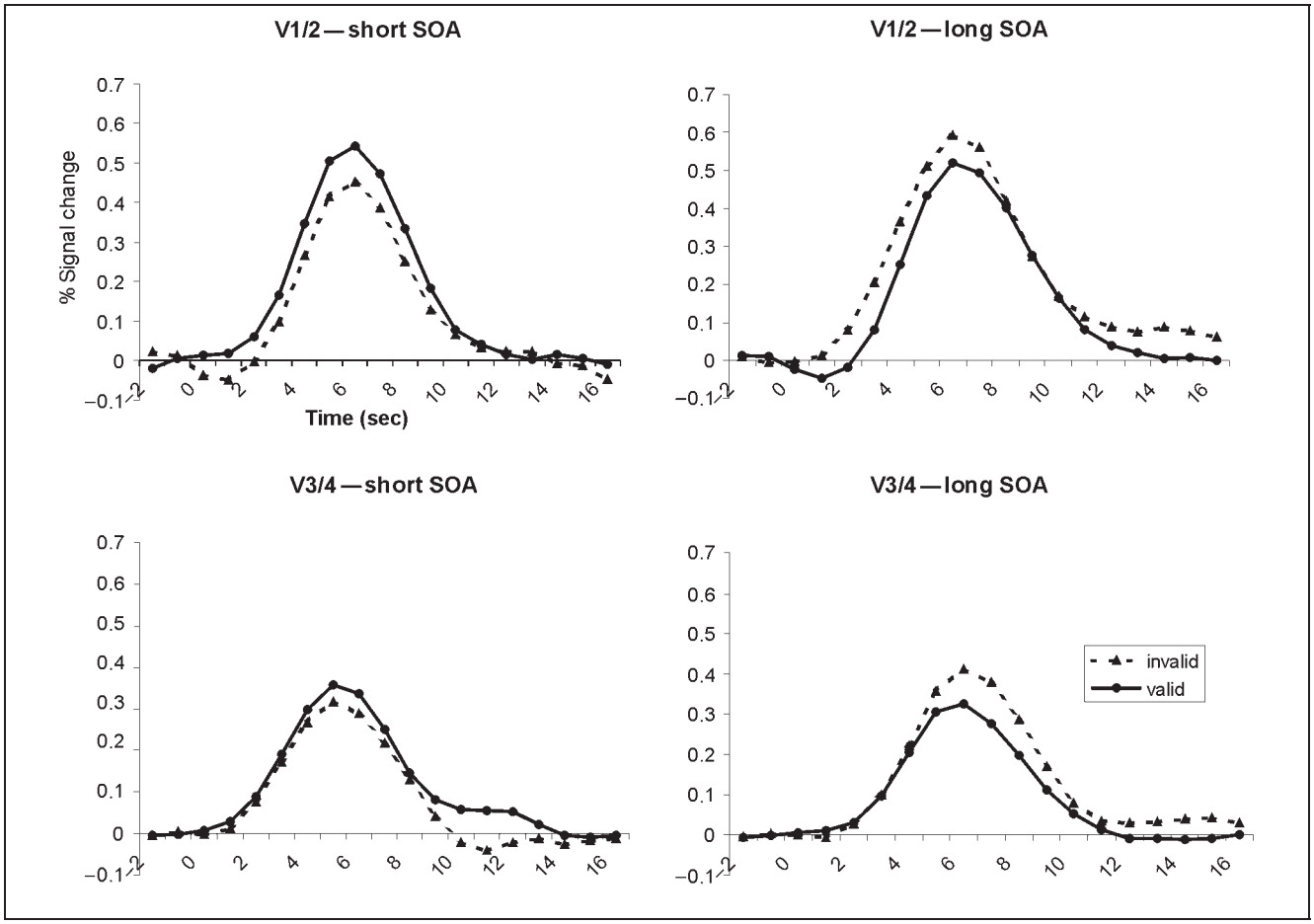

Figure 4. BOLD responses from regions of interests in visual areas. Note larger BOLD responses for targets presented close to the cue at the short SOA and for targets presented far away from the cue at the long SOA, respectively.

we had introduced to allow for activation of different visual subareas, had no significant effect on behavior. This finding complements earlier studies that demonstrated that IOR was not limited to the cued location but spreads to nearby locations (Bennett & Pratt, 2001; Maylor & Hockey, 1985), suggesting that IOR operates in rather coarse spatial coordinates.

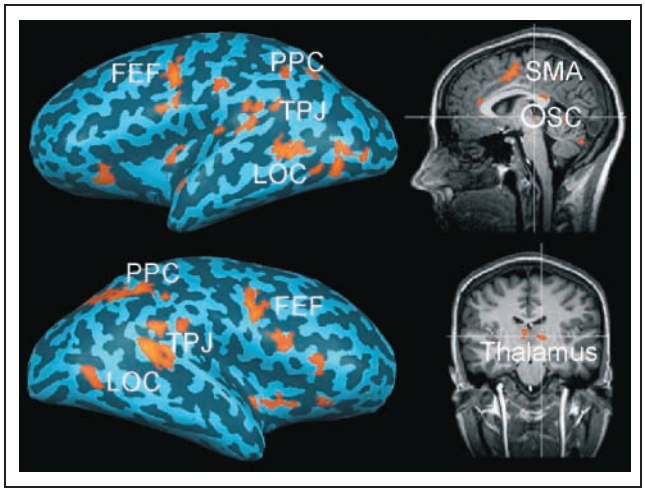

Figure 5. Whole-brain group analysis. Left: activation in lateral cortex. Right: activation in medial and subcortical structures.

Our whole-brain analysis revealed neural correlates of IOR that closely resemble the activation pattern of a previous fMRI study using the classic paradigm (Lepsien & Pollmann, 2002). Most importantly, the long SOA condition revealed stronger activation in areas belonging to the oculomotor control system: the FEF, SMA, SC, and PPC. These areas, especially the SC, have been proposed to play a crucial role in generation of IOR (Ro, Farne, & Chang, 2003; Dorris, Klein, Everling, & Munoz, 2002; Danziger, Fendrich, & Rafal, 1997), in line with the hypothesis that the attentional and oculomotor systems largely overlap (Corbetta et al., 1998). Hence, the whole-brain analysis, like the behavioral data, supports the finding that our paradigm activated the same brain areas as found in typical IOR tasks (see below for a more detailed discussion of the neural substrates of IOR).

The behavioral data were complemented by the fMRI data collected from the ROIs in the visual cortex: The target stimulus yielded a stronger transient signal increase in its retinotopic representations across all visual areas assessed in valid than in invalid trials at the short

SOA, an effect that reversed at the long SOA in which targets at the uncued location evoked the larger signal.

Sensory interaction between cue and target is an unlikely explanation for the observed effects in the visual cortex. First, targets and cues were presented in the left and right hemifield, respectively, for which even V4 has separate cortical representations. Second, even if the areas representing the targets were also involved in sensory processing of the cues this may have explained larger responses to targets in valid trials, in which the cue appeared close to the target. However, due to the sluggishness of the BOLD response, such a summation of sensory processes should evoke the same BOLD signal across different SOAs; yet, at the long SOA, the spatially adjacent stimuli of the valid trials yielded a weaker response than those of the invalid trials that were separated by a large distance. Third, a recently published fMRI study using a similar paradigm (Liu et al., 2005) reported increased transient activation in visual representations of the target only when the target was immediately preceded by the nearby cue but not when the same cue was presented after the target, although this temporal manipulation left the overall sensory input unchanged. From this, the authors concluded that transient deployment of attention to the cued location, but not sensory summation, accounted for the increased signal to the validly cued targets.

We likewise propose that the present effects in the visual cortex were driven by attention. Yet, we provide evidence for an additional time-dependent component that strongly modulates the response to a salient peripheral stimulus. A sudden-onset peripheral stimulus first leads to a transient increase in neural activity in visual subareas representing its approximate location. However, this initial effect is then followed by a transient activity reduction that creates a bias in favor of subareas coding the remaining visual field. Hence, the observed activity pattern closely follows the initial proposal by Posner and colleagues who suggested a biphasic deployment of attention whereby attention is first directed to the peripheral stimulus, but then disengaged from there and inhibited to return to it (Posner, 1985; Posner & Cohen, 1984).

Apart from the fMRI study mentioned above (Liu et al., 2005), modulation of sensory activity in response to peripheral cues has only been measured in electrophysiological studies, which, however, lack the possibility to assess retinotopically defined visual subareas. In these studies, the P1 component, which is believed to originate from the extrastriate cortex, showed modulations in line with the results of the present study. At short cue–target SOAs, associated with speeded responses for validly cued targets, the P1 was larger for validly than invalidly cued targets, whereas at long SOAs, associated with slowed responses for valid targets, the P1 was reduced for validly compared with invalidly cued targets (Prime & Ward, 2004; McDonald, Ward, & Kiehl, 1999; Hopfinger & Mangun, 1998; Anllo-Vento, 1995; Eimer, 1994; Hillyard, Luck, & Mangun, 1994). Prime and Ward (2004) concluded that ''IOR must arise at least in part from changes in perceptual processes, and, at least when measured with manual key presses, IOR does not arise from inhibition of motor processes'' (p. 275).

The authors refer to a long-lasting debate regarding the neuronal correlates of IOR (Lepsien & Pollmann, 2002; Klein, 2000; Sapir, Soroker, Berger, & Henik, 1999; Posner, 1985). The most common assumption is that the brain system that controls saccadic eye movements, namely, the SC, the FEFs, and the parietal eye fields within the PPC, plays a crucial role in stimulus-driven transient attention shifts during both initial facilitation and subsequent inhibition. As opposed to the slower frontoparietal cortical network that is involved in voluntary and sustained attentional control (Müller, Donner, et al., 2003; Corbetta & Shulman, 2002; Hopfinger, Buonocore, & Mangun, 2000), this system is held to be fast enough to control the transient, more automatic shifts induced by stimuli that pop out in the periphery. These areas, therefore, might be part of a feed-forward (i.e., stimulus-driven) fast attention system that enhances activity in retinotopic visual cortex immediately after a salient stimulus has been presented in the periphery in order to process this stimulus more effectively. After this has been accomplished, however, the fast attention system drains processing resources from this location to give priority to representations of other regions of the visual field that may potentially reveal new stimuli. Indeed, in our whole-brain analysis when comparing trials with long SOAs (in which IOR was initiated) to trials with short SOAs, the named areas were found to be activated. However, activation clearly went beyond the oculomotor system, including multiple regions in the lateral prefrontal cortex and the PPC corresponding to the so-called frontoparietal attention network. This network exerts top-down attentional (i.e., endogenous) control on sensory processing (Hopfinger et al., 2000). Although our experiment was explicitly designed to activate the exogenous attention system, it is conceivable that the presentation of salient stimuli led to an increase in unspecific attentional arousal and/or attentional focusing on the stimuli. Indeed, it has been shown that parietal cortex involvement generalizes over a wide range of attention tasks (Wojciulik & Kanwisher, 1999). The observation that activity in the parietal cortex was more pronounced in the long-SOA trials may then simply be due to the fact that this system is rather slow, so that with short SOAs the attentional level returned back to baseline soon. Note, however, that although the stronger frontoparietal activation at long SOAs may have driven a general increase in visual cortex activity in this condition, it hardly can explain the much more complex pattern we observed where enhancement in subregions of the visual cortex depended on SOA duration and spatial relation to the cue.

In sum and in conjunction with our previous results (Müller & Kleinschmidt, 2004), it is becoming increasingly clear that even detailed and dynamic spatiotemporal features of the attentional spotlight model are accurately reflected in activity variations in the early visual cortex. It is also becoming increasingly clear that gain control is a highly efficient cortical mechanism and that it involves suppression to an equivalent extent as facilitation.

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Note

1. Note that we do not report data from ROIs representing the location at which no target was shown in the respective trial in order to leave sensory input the same.

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