Inhibition of Return Impairs Phosphene Detection

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Abstract

■ Efficient visual exploration requires the ability to select possible target locations via spatial attention and to deselect previously inspected locations via inhibition of return (IOR). Although a great deal is known about the effects of spatial attention on processing in visual cortex, much less is known about the effects of IOR on early visual areas. One possibility is that IOR acts in an opposite way to spatial attention, such that, whereas spatial attention enhances target related neural signals in visual cortex, IOR suppress target-related signals. Using a novel dual-coil TMS protocol, we found that IOR reduced the probability of detecting a TMS-induced phosphene in extrastriate cortex (V5). Specifically, a nonpredictive spatial precue presented 500 or 800 msec before stimulation significantly reduced the probability of detecting a phosphene when the precue appeared contralaterally to the site of stimulation (i.e., ipsilaterally to the potential location of the phosphene), compared with ipsilaterally or centrally presented cues. This result demonstrates that IOR facilitates visual exploration by directly affecting the strength of target-related signals in extrastriate visual cortex. This result is consistent with neurophysiological models of attention, which postulate that IOR modulates perception by biasing competition between sensory representations. ■

INTRODUCTION

Salient locations in the environment attract attention and become the goal of eye movements. This bottom–up mechanism ensures that potentially important visual information is rapidly processed. However, in many cases, the most salient item in a scene is not the most behaviorally relevant item in the scene (e.g., looking for keys on a cluttered desk), and it is necessary to orient to locations of lesser salience. This situation could potentially be problematic for an orienting mechanism that relies primarily on bottom–up information about salience. To address this problem, the visual system has an inhibitory mechanism that biases exploration away from salient locations, known as inhibition of return (IOR; Posner, Rafal, Choate, & Vaughan, 1985).

In the laboratory, IOR can be operationalized in cue–target tasks. Here, a peripheral cue is used to create a location of high salience. For a brief period (~150 msec), stimuli occurring at this cued location are processed more efficiently than stimuli at other locations. However, at longer intervals, stimuli that appear at the once-salient location are processed less efficiently. IOR affects performance on a number of measures including manual RT (Posner et al., 1985), saccadic RT (ReuterLorenz, Jha, & Rosenquist, 1996), stimulus discrimination (Lupianez, Milan, Tornay, Madrid, & Tudela, 1997), and change detection (Smith & Schenk, 2010).

The neural mechanisms underlying the generation of IOR have been the subject of considerable debate, but recent evidence suggests the existence of two distinct forms of IOR: a perceptual inhibition that affects non-goal-directed responses (e.g., button-press responses), which is generated in the visual system, and a saccadic IOR that affects goal directed responses (e.g., eye movements), which is generated in the oculomotor system (Bourgeois, Chica, Migliaccio, de Schotten, & Bartolomeo, 2012; Sumner, Nachev, Vora, Husain, & Kennard, 2004; Taylor & Klein, 2000). These two mechanisms have been dissociated experimentally, such that perceptual IOR can be observed in the absence of oculomotor activation (Smith, Rorden, & Schenk, 2012; Smith, Jackson, & Rorden, 2009; Sumner et al., 2004) and in neuropsychological patients, such that patients with hemispatial neglect have no perceptual IOR but intact saccadic IOR (Bourgeois et al., 2012).

There is compelling evidence that saccadic IOR occurs as the consequence of a reduction in the strength of target-related signals in the oculomotor system (Anderson & Rees, 2011; Fecteau & Munoz, 2005; Fecteau, Bell, & Munoz, 2004). However, the mechanism by which perceptual IOR produces changes in visual processing remains contentious. One possibility is that perceptual IOR operates by reducing the strength of target-related neural representations in visual cortex. This suggestion is consistent with neurophysiological evidence from nonhuman primates that IOR arises at early stages of visual processing (Ikeda, Yoshida, & Isa, 2011). In humans, perceptual IOR is associated with changes in CBF in human visual cortex (V1–V4), such that targets at inhibited locations elicit smaller BOLD responses than targets appearing at uncued locations (Muller & Kleinschmidt, 2007). One interpretation of these observations is that IOR supresses the neural signal associated with stimuli at the cued location.

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However, it is important to note that these studies did not use neutral trials (e.g., a cue at fixation), so it is impossible to know whether the relatively lower BOLD signal at the cued location was caused by suppression of neural response at the cued location or enhancement of response at the uncued location.

Other evidence that IOR influences the strength of target-related neural representations comes from psychophysiological studies using ERPs. A number of studies have shown that IOR is associated with a reduction in the amplitude of ERPs generated by stimuli at inhibited locations (Prime & Jolicoeur, 2009; Prime & Ward, 2004, 2006; Wascher & Tipper, 2004; McDonald, Ward, & Kiehl, 1999), and the magnitude of this amplitude change is modulated by factors that also effect the magnitude of the IOR effect, such as the presence of a central reorienting event (Prime & Jolicoeur, 2009). These data would seem to suggest that IOR is associated with the modulation of signals in early visual cortex. However, as with the previous imaging studies, neutral trials were typically absent, making it difficult to know the source of the difference between the wave forms. Furthermore, the evidence from ERPs is not entirely consistent: Some studies show changes in ERP wave form but no behavioral IOR (Wascher & Tipper, 2004; Hopfinger & Mangun, 1998; Eimer, 1994 Experiment 2), whereas others show behavioral IOR but unreliable or absent changes in ERP (Prime & Ward, 2006; Hopfinger & Mangun, 2001; Experiment 2). In addition, ERPs have poor spatial resolution, making it difficult to draw firm conclusions regarding the cortical source of the signal change.

Given the ambiguity in the existing evidence, the suggestion that perceptual IOR operates by reducing the strength of neural representations in visual cortex remains contentious. A more direct way to test this hypothesis is to evaluate the effect of IOR on the strength of neural signals in human observers. One technique for assessing the strength of neural signals in visual cortex is to measure the ease with which magnetic stimulation can elicit illusory visual phenomena known as phosphenes (Walsh & Pascual-Leone, 2003). This technique has previously been successfully used to demonstrate that endogenous spatial attention enhances the neural signals at attended locations by demonstrating that phosphene thresholds are lower at attended locations (Bestmann, Ruff, Blakemore, Driver, & Thilo, 2007; Silvanto, Lavie, & Walsh, 2006). Using TMS-induced phosphenes to investigate perceptual IOR has the additional advantage that these visual percepts are purely cortical; that is, the tectopulvinar visual pathway is not activated via stimulation of the retina by light. This is important as it means that any change in performance can be unambiguously attributed to modulation of sensory processing in visual cortex rather than modulation of sensory processing in the oculomotor system.

To test the prediction that perceptual IOR reduces the strength of neural signals in visual cortex, we used nonpredictive peripheral precues to generate IOR and then delivered single-pulse TMS over left or right V5. V5 was chosen as the strength of neural signals in this area are known to be influenced by attention (e.g., Silvanto et al., 2006; Smith, Jackson, & Rorden, 2005) and it is sufficiently lateralized to allow the simultaneous placement of coils over left and right hemispheres. This manipulation was important as it meant that participants were unable to use either the location of the precue or the location of the stimulator coil to predict the location of the phosphene. The hypothesis that IOR reduces the strength of sensory representations in early visual cortex leads to a clear and testable prediction: Participants should detect fewer phosphenes on trials where the precue and phosphene were spatially congruent compared with trials where the precue and phosphene were spatially separated.

METHODS

Participants

Nine participants were recruited from the University of Durham (five men). Ages ranged from 23 to 52 years (mean = 28 years). Six participants were right-handed. All participants had normal or corrected-to-normal vision. Participants gave signed informed consent in accordance with the Declaration of Helsinki and with the approval of Durham University Ethics Advisory Committee.

Apparatus and Stimuli

Magnetic stimulation was delivered using two Magstim 200 monopulse stimulators (Magstim, Whitland, Carmarthenshire, UK) via 70-mm figure-of-eight coils. Stimuli were generated using E-Prime v1.1 (Psychology Software Tools, Inc., Pittsburgh, PA) and displayed on an RM VL700 CRT colour monitor with a 60 Hz refresh rate. E-Prime was also used to trigger TMS. Responses were collected using a custom built response box with two buttons. The fixation point was a 0.2° pixel dim gray square presented in the center of the screen. The attentional cue was a dim gray ring with a width of 2 pixels and a diameter of 2°. The cue used to re-orient attention to the fixation point was an unfilled 0.4° × 0.4° dim gray square presented around the fixation point. Phosphene localization and experimental trials were conducted in the dark.

Phosphene Localization

V5 was functionally localized in each hemisphere. Participants sat in a dark room with their head supported by a chinrest. The viewing distance was 57 cm. Participants were asked to fixate a dim gray cross (0.5°) presented in the center of a black screen. Coils were initially placed 3 cm above the mastoid-inion line and 5 cm lateral to the midline in the sagittal plane. A win-stay/lose-move paradigm was used to identify locations where phosphenes could be elicited. Participants verbally reported the detection of a phosphene after each pulse. Participants were specifically

asked to report the perception of moving phosphenes. Before beginning the testing session participants were given some examples of the type of movement they might see, based on the descriptions provided by Cowey and Walsh (2000) This hunting procedure began with TMS set at 65% of the maximum machine output (i.e., 1.3 T). If phosphenes could not be elicited at this intensity, the output was increased by 5 percentage points and the hunting procedure repeated until a site could be localized. When a suitable stimulation site had been identified, participants were given 3 further pulses to confirm the visibility of the phosphene. If phosphenes were visible on all 3 trials the participant indicated the location of the phosphene by mouse-clicking the corresponding location on the monitor. If the participant did not give 3/3 positive responses the hunting procedure was started again. This localization procedure was carried out separately for each hemisphere. There was some variability in the intensity of stimulation required to elicit phosphenes (range 65–80%, median 65%). The coil was tangential to the skull, with the handle perpendicular to the surface of the head. The coil was held in place by the researcher allowing for precise control of the coil position. The average scalp position of the coil relative to the inion was 2.4 cm up and 5.1 cm lateral for the left coil and 2.3 cm up and 5.2 cm lateral for the right coil.

Procedure

Participants were seated with their head in a chinrest 57 cm away from the monitor. Two coils were placed on the head, one over each of the V5 stimulation sites. Trials began with the presentation of a central fixation point for 1000 msec. The peripheral cue was then presented at one of the peripheral locations that overlapped with the phosphene or around the fixation point for 100 msec. Fifty milliseconds after the offset of the attentional cue the fixation point was cued for 50 msec. There was then a delay of either 350 or 550 msec. After this delay, a single TMS pulse was delivered via one of the coils. Stimulator intensity was set individually for each participant, such that the same level of intensity required to elicit phosphenes from that individual during the localization phase was used during the experiment (range 65–80%, median 65%). On 33% of trials the TMS pulse was delivered via the coil sited contralaterally to the location of the cue. In this case the phosphene would appear in the same spatial location as the peripheral cue (Valid trials). On 33% of trials the TMS pulse was delivered via the coil sited ipsilaterally to the location of the cue. In this case the phosphene would appear in the opposite hemifield (Invalid trials). On the remaining 33% of trials the cue was presented centrally and the pulse was delivered via the left or right hemisphere coil with equal probability (Neutral trials). The total SOA could be either 500 msec or 800 msec. Figure 1 illustrates the procedure. The protocol made it impossible for participants to accurately predict the site of stimulation based on the location of the cue or the location of the stimulator coil. Participants were asked to make an unspeeded discrimination judgment about whether or not they had perceived a phosphene. Each participant completed two blocks of 48 trials

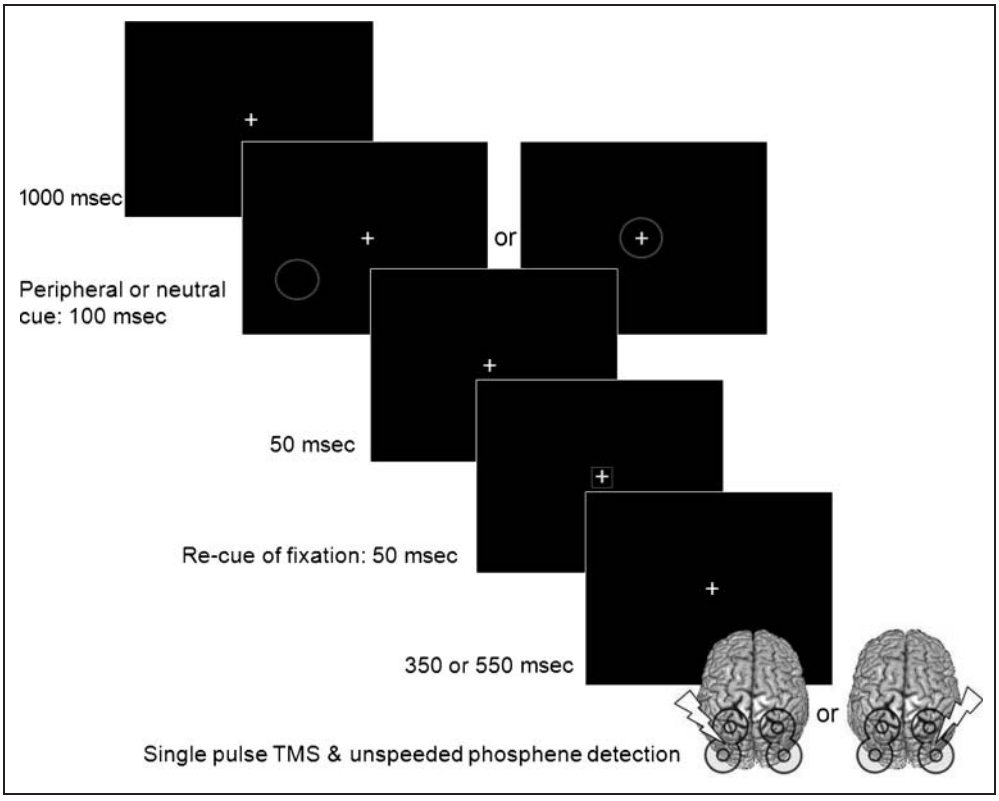

Figure 1. Schematic showing the time course of a typical trial. Peripheral cues appeared on the left or right; the neutral cue appeared around fixation. Cues appeared in the three locations with equal frequency. TMS was delivered contralaterally to the cue on 50% of peripheral cue trials (i.e., 50% of peripheral cue trials were valid, such that the phosphene could appear at the cued location).

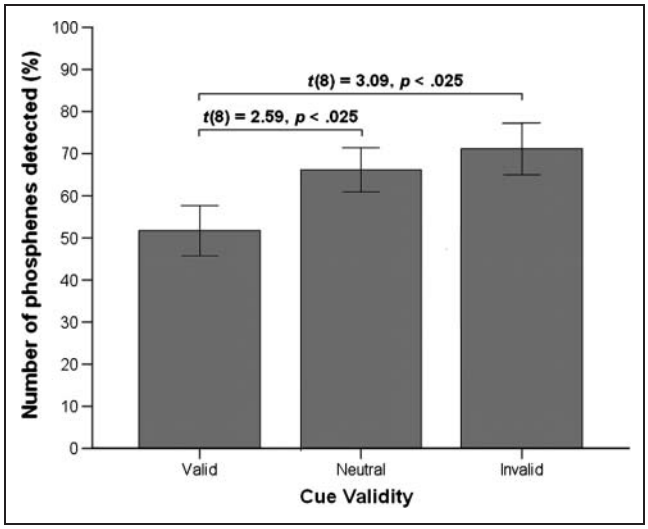

Figure 2. Probability of phosphene detection (%) during Valid, Neutral, and Invalid trials. Detection rates were significantly reduced on Valid trials relative to Invalid and Neutral trials. Error bars show ±1 *SEM*.

(16 Valid, 16 Invalid, and 16 Neutral trials at each SOA). Participants were instructed to fixate the central of the display throughout each trial but we did not monitor eye movements as previous studies have shown that participants spontaneously suppress eye movements during luminance detection tasks (Posner, Nissen, & Ogden, 1977).

RESULTS

The hit rates (i.e., the proportion of trials in which a phosphene was detected) were subjected to a 3 × 2 repeated-measures ANOVA with factors of Validity (Valid, Invalid, Neutral) and SOA (500 msec and 800 msec). The ANOVA revealed a main effect of Validity ($F(2, 16) = 7.77, p < .05$). There was no main effect of SOA and no SOA × Validity interaction. Inspection of Figure 2 suggests that the main effect of Validity was driven by lower hit rates for Valid trials compared with Invalid trials and Neutral Trials. Planned comparisons (1-tailed paired samples *t* tests) confirmed that hit rates were significantly lower on Valid trials compared with Invalid trials ($t(8) = 3.09, p < .025$) and Neutral trials ($t(8) = 2.59, p < .025$).

DISCUSSION

The aim of this experiment was to test the hypothesis that IOR reduces strength of target related representations in visual cortex. Consistent with this hypothesis hit rates for detecting TMS induced phosphenes were significantly reduced when phosphenes appeared at a cued location, compared with trials where phosphenes were spatially separate from the cued location. These data are consistent with previous reports that IOR is associated with changes in the strength of signals in early visual cortex (e.g., Anderson & Rees, 2011; Prime & Jolicoeur, 2009; Muller & Kleinschmidt, 2007; Prime & Ward, 2004, 2006; Wascher & Tipper, 2004; McDonald et al., 1999; Eimer, 1994). However, the results extend these previous findings in two important ways. Firstly, they establish that the changes in neural activation are causally related to the impaired perceptual processing observed in IOR. Secondly, performance at the cued location was also worse than performance following a neutral cue. This result shows that the relatively poorer phosphene detection at the cued location was likely to be because of suppression of neural signal at the cued location rather than facilitation of the signal at the uncued location. In other words, IOR suppressed performance at the cued location below baseline, rather than driving performance at the uncued location above baseline. This result is worthy of note, as it rules out the possibility that the IOR effect was caused by an attentional bias directed to the uncued location. If participants had an attentional bias toward the uncued location one would expect facilitation of phosphene detection at the uncued location relative to neutral trials (Bestmann et al., 2007). However, we observed no such effect.

Superficially, the suggestion that IOR acts by suppressing target-related signals in visual cortex may appear inconsistent with previous data from Danziger, Fendrich, and Rafal (1997) showing that cues presented to the blind field of a patient with hemianopia (i.e., no visual cortex) were able to elicit IOR (e.g., Anderson & Rees, 2011). However, it is important to note that in this experiment although the cue was presented to the blind hemifield, after cue presentation the patient made a saccade into the blind field which brought the cued location into the sighted field. The target was then presented to the sighted hemifield. In other words, IOR was observed in the sighted field, not the blind field. The experiment therefore shows that visual cortex is not required to generate IOR, but it does not speak to the impact this inhibitory signal has on processing in visual cortex.

The results of this experiment confirm previous claims that IOR operates at an early stage of visual processing and acts to suppress the neural representation of targets. A recent neurophysiological account of IOR argues that saccadic and perceptual IOR arise from the same underlying mechanism (Fecteau & Munoz, 2005). Specifically, it is argued that poorer perceptual performance and slower eye movements as seen for targets which have been preceded by pre-cues are the result of weak target related activation in a neural "priority map". As activation in this map is used to guide both attention and eye movements, weak target-related signals in this map would produce both poorer perceptual performance and slower overt orienting. Our current results indicate that the weak target-related activity in priority maps described by Fecteau & Munoz is driven by a reduction in the strength of the target-related activity in the visual areas that provide the bottom–up input for the priority map.

However, the neurophysiological mechanism underlying this reduction in signal strength is not clear. One

straightforward possibility is that IOR reduces baseline activity in visual areas such as V5, thus making it more difficult for incoming sensory signals to push the cells above threshold. This reduced baseline activity would result in the activation of fewer cells by the TMS pulse, thus reducing the strength of the target-related neural signal. In this view, perceptual IOR operates in exactly the opposite direction to endogenous attention, which has been claimed to enhance the excitability of visual cortex (Bestmann et al., 2007). One problematic issue for this explanation is that previous work in humans indicates that the effects of TMS over visual cortex are state dependent, such that TMS has a greater effect on neurons with lower levels of baseline activation. For example, in a series of experiments Silvanto and colleagues examined the properties of visual phosphenes elicited before and after adaptation to color or motion (see Silvanto, Muggleton, & Walsh, 2008). When TMS was delivered after adaptation, phosphenes tended to share properties of the adapted stimulus (i.e., the stimulus that to which neural responses had been attenuated by adaptation), not the unadapted stimulus. If IOR causes a suppression of activation and TMS preferentially activates suppressed neurons, one would actually predict facilitated phosphene detection at cued locations, which is the opposite of what was observed in this experiment. Furthermore, the suggestion that IOR reduces cortical excitability is inconsistent with previous work showing that IOR is associated with heightened levels of baseline activity but lower target-specific activity in the primate superior colliculus (Dorris, Klein, Everling, & Munoz, 2002). However, it is important to be aware that these results were obtained for saccadic, not perceptual IOR.

An alternative explanation is that perceptual IOR acts to increase the baseline activation of cells at the cued location, and this increased activation lowers the signal to noise ratio of incoming signals, thus making them harder to detect. This account is consistent with the biased competition model of attention, for example, (Desimone, 1998), which holds that attention emerges from competitive interactions in the early visual system. More specifically, it is proposed that sensory representations compete until the signals with the highest physical salience dominate and the representations associated with other stimuli are suppressed. This competition can be biased by task context (i.e., the goal of the observer), such that signals from task-relevant stimuli are enhanced. The winner of the biased competition is attended, in the sense that those representations are then analyzed for semantic content and response selection. In this view, raised baseline activation makes it more difficult for the incoming sensory signal to be discriminated from the noise (i.e., the signal faces more competition in the visual system). The consequence is that it takes longer for the incoming signal to win the competition, thus retarding RTs or in some cases (where the incoming signal is weak) the signal fails to win the competition and goes undetected (e.g., Smith & Schenk, 2010). Although speculative, this account is consistent with fMRI data showing that perceptual IOR is associated with enhanced BOLD signal in V5/MT+ (Mayer, Seidenberg, Dorflinger, & Rao, 2004) and is also consistent with the current data set.

There is technically no way to present no-target trials in the current paradigm, so we cannot definitively rule out the possibility that our results are influenced by a shift in criterion, such that participants adopt a looser criterion when the phosphene appears at the uncued location rather than changes in neural processing. However, we believe this explanation is unlikely to account for our data for three reasons. First, a looser criterion at uncued locations should be reflected in the hit rate at uncued locations (it should be higher than in the neutral condition), but this is not what we observed. Second, there seems to be no apriori reason for participants to systematically adopt a looser criterion for phosphenes that appear at the uncued location than those that appear at the cued location. Finally, a reduced signal strength interpretation is consistent with previous neurophysiological data demonstrating that IOR is associated with changes in BOLD signal and ERP amplitude (e.g., Prime & Jolicoeur, 2009; Muller & Kleinschmidt, 2007). These neurophysiological effects are not predicted by a criterion shift explanation. Rather, we argue that the impairment of phosphene detection at the cued location reflects reduced signal strength.

In summary, it has been shown that perceptual IOR makes it more difficult to elicit phosphenes when stimulating V5. This result confirms previous observations that perceptual IOR modulates the strength of target-related signal in extrastriate cortex and provides direct evidence that this reduction in signal strength is causally related to impaired visual perception. Theoretically, these data are consistent with the view that IOR operates at an early stage of visual selection via the process of biased competition. We believe our data are consistent with the view that the neurophysiological basis of IOR is an increase in the excitability of extrastriate neurons, which adds noise to the visual system, thus lowering the signal-to-noise ratio of the incoming visual signal.

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