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Research article

Is saccade preparation required for inhibition of return (IOR)?

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ABSTRACT

The effect of slower responses to validly than invalidly cued targets is known as inhibition of return (IOR). Opposing accounts of IOR have been proposed: one postulates a singular phenomenon explained by oculomotor mechanisms alone, while the other, more diverse account postulates both perceptual-cognitive and motor factors. In our research we considered the relation between motor programming and IOR. In an extended replication of an earlier study, using an eye abduction technique we restricted eye movement in the temporal half-space; this resulted in IOR attenuation in that area, compared to the unrestricted, nasal part of the visual field. Our results contradict the earlier result and demonstrate that IOR does depend on preparation of eye movement, as predicted by the *oculomotor priming hypothesis*.

1. Introduction

In a cuing task, reactions to target stimuli tend to be generally faster at spatial locations previously signaled by a cue (so called *valid trials*); however, Posner and Cohen [29] found this to be true only for some cue–target intervals (called *Stimulus Onset Asynchrony*, SOA). At SOAs longer than 300 ms, the effect reverses and, paradoxically, invalid cues speed up responses to targets more than valid cues. The authors proposed that this negative cuing effect, which they called inhibition of return (IOR), reflected suppression of orienting towards previously attended locations. Because IOR can also be produced by eye movements alone [30], it is usually defined broadly as a slower reaction time to targets presented at a previously cued or inspected location (see [20] for a review).

There are two major views on the causes of the IOR effect. One postulates distinct sensory and motor mechanisms (*input based IOR* and *output based IOR*) [17,19,22]. The other regards IOR as a unitary, motor phenomenon related to the preparation of eye movements (*oculomotor priming hypothesis*) [31,33].

According to the first view, output-based IOR occurs when the task requires preparation or execution of saccades [17], but when no eye movement is necessary it originates from input factors, i.e. perceptual sensitivity reduction at cued locations *(short-term sensory adaptation)* [10] or other non-oculomotor inhibition [17,22,29]. The other view assumes an exclusively oculomotor nature of all observed IOR effects. The alleged input IOR is explained by automatic preparation of eye movements toward the cued locations [31,33].

The one-factor oculomotor explanation is supported by a significant body of research [4,15,21,23,32,37,46], but a short review merits mentioning but a few. The microsaccades have been shown to get drawn away temporarily from the cue just when IOR effects for manual responses begin to emerge [15]. Saccadic eye movements tend to target the midpoint of several concurrent visual stimuli [12,27]. An analogous "center of gravity" effect is also observed for IOR, both with manual and with saccadic responses [4,21,23].

The IOR is larger in the temporal than the nasal part of the visual field [24,32,33,40,46]. This might be related to the characteristics of the *superior colliculus* (SC) [38], which receives more input from temporal than nasal parts [9,32,33]. The SC has been shown to play a crucial role in generating reflexive saccades and their activity diminishes during IOR [10]. In patients with a damaged SC, the normal pattern of IOR is also disturbed [30,37]. Although many studies have implicated eye movement programming as the most likely cause of IOR, some researchers argue that the effect cannot be explained by oculomotor processes alone [14,17,22,43,44].

Sumner et al. [44] demonstrated that stimuli processed by S-type cones (thought to be effectively invisible to superior colliculi involved in the programming of eye movement) generate IOR for manual but not for saccadic responses. This seems to show that a cortical mechanism [45] associated with attentional control could generate IOR with no involvement of motor processes. However, this claim has been weakened by White et al. [49] and, more recently, by the data of Hall & Colby [16], which suggests that the SC might in fact not be blind to shortwave stimuli.

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Smith, Schenk and Rorden [43] observed the IOR effect in parts of the visual field to which no eye movement could be executed due to the extreme eye position required. The authors employed a typical exogenous cueing task, with a modification borrowed from Craighero, Nascimben and Fadiga [7]: one of the participants' eyes was blindfolded, and the freedom of movement of the other was restricted by sitting the participant at an angle relative to the computer screen; the angle was adjusted so that in order to focus on the center of the screen, the participant had to rotate her or his eye to the temporal extreme, beyond which no further eye movement was possible; this constricted eye movements to the nasal part of the visual field, preventing saccades toward stimuli displayed in the temporal part [7,41]. With an abducted eye, the facilitatory effect of the cue at short SOA (exogenous attentional capture) was restricted to locations within the scope of eye movement capacity, i.e. to the nasal but not the temporal part of the visual field. Unlike attentional capture, which was eliminated by the manipulation, IOR was present in both parts of the visual field and remained unaffected by eye rotation. The authors concluded this to be strong evidence of the effect's non-motor origin.

The eye abduction technique produces no visual acuity deficits [7] and allows for non-invasive control of eye movement capability in healthy individuals. As such it is particularly well suited to testing whether eye saccade motor programming is necessary for IOR. According to the oculomotor priming hypothesis [31,33], if the IOR effect is caused by eye movement preparation, then eye abduction should result in IOR decrease at locations that are unattainable for saccades. To our knowledge, the study of Smith et al. is the only attempt at testing the oculomotor hypothesis employing the eye abduction technique. Therefore, we decided to reexamine their findings due to their significant implications. We used a larger sample to improve statistical power, and diminished the eye rotation angle from 40 to 37 ° because our earlier unpublished attempts showed the former to be very strenuous, and even painful for many participants.

2. Method

2.1. Participants

30 university students (20 female, 10 male[2]; aged 19–24) volunteered in the present experiment for course credits. The participants were familiarized with the procedure before giving their consent. They were informed that the study concerned perceptual processes, but other than that they were naive to the purpose of the study (however, they were debriefed on completion). All had normal or corrected-to-normal vision. They were all right-handed. Their eye dominance was determined with the Porta procedure [35].

2.2. Setup

Fig. 1A depicts the experimental setup. The participants were seated 60 cm from the screen with one eye patched. In the central condition they were facing the screen, while in the rotated condition their seats were rotated 37° in the direction of their occluded eyes. Compensatory head movements were prevented by means of orthopedic collars. In both conditions the subjects were asked to fixate on the center of the screen and this required eye abduction in the rotated condition. The abduction allowed for full visual acuity in both visual fields, at the same time preventing eye movement in the temporal visual field.

[2] Because of possible differences in attention between men and women [11,28], our sample reflected the sex ratio of the study of Smith et al. for easier comparison. However, it should be noted that this decision might potentially bias the results.

2.3. Stimuli

The experiment employed a peripheral cueing paradigm. All stimuli were displayed in white on black on a 19" CRT monitor of a Pentium PC running a DMDX [13] script. A 0.2° × 0.2° fixation point was displayed at the centre of the screen. The cues (white 3 × 3° frames drawn with a 0.2° thick line) were presented 6° to the left or to the right of the fixation box, and then centrally (cue back to fixation). The targets (1.5° × 1.5° squares) were displayed at the fixation point in the center of the screen on neutral trials, at a lateral cue position on valid trials, and contralaterally to the cued positions on invalid trials.

2.4. Procedure

An example trial is outlined in Fig. 1B. A randomized cue–target interval (stimulus onset asynchrony, SOA) of 300–500 ms was used to minimize anticipation effects. At a random interval of 50–120 ms after cue offset, a cue back was presented for 100 ms at the fixation point. The participants responded by pressing a key on a PC gameport response pad. They were asked to be fast, but to also avoid errors. Every participant took two experimental sessions, separated by a 30 min break, in balanced order, one with the non-dominant, and one with the dominant eye occluded. Each session consisted of 472 trials, organized in 8 blocks of 59 trials. The 59 trials of each block were subdivided into 32 (54%) valid trials in which targets were presented at cued locations, and 18 (31%) invalid trials in which the targets were displayed at locations opposite to the cued ones. Additionally, 3 (5%) neutral trials with cues and targets presented at the fixation point, and 6 (10%) catch trials with no target were introduced to maximize cueing effectiveness [see 50]. The eye position (central or abducted) was changed every four blocks and counterbalanced across participants.

2.5. Data analysis

The data was first submitted to a descriptive analysis. No participant had more than 3 false alarms in 24 catch trials, most had none. 2.6% of responses were rejected as anticipatory (faster than 120 ms) or outlying (> 3 SD). Mean RTs were calculated for the remaining correct RTs. Subsequently, all participants' mean RTs were analyzed with a 2 × 2 x 2 × 2 repeated-measure ANOVA with Validity (valid, invalid), Visual Field (nasal, temporal), Eye dominance (dominant, non-dominant) and Eye abduction (central, abducted) as four independent variables. Because the introductory analysis showed no significant sex differences, this factor was not included in the ANOVA. The IOR effect was calculated as the difference between reaction times in valid trials (target appears in the same location as cue) and invalid trials (target appears in the opposite location than cue).

3. Results

The ANOVA showed statistical significance for main effects of validity and visual field. Validity interacted significantly with eye position and eye dominance; there was also a significant three-way interaction of validity, eye position and eye dominance. The effects for individual factors are detailed below.

3.1. Validity (IOR effect)

The procedure succeeded in producing a clear IOR effect: responses to targets preceded by valid cues were on average 14 ms slower than those invalidly cued (valid 297 ms, invalid 283 ms, 95% CI for the difference [9.7, 18.0], $F(1, 29) = 46.60$, $p < 0.001$, $\eta_p^2 = 0.61$, Cohen $d = 0.4$).