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Microsaccadic response during inhibition of return in a target–target paradigm

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Abstract

This study examined the relationship between inhibition of return (IOR) in covert orienting and microsaccade statistics. Unlike a previous study [Galfano, G., Betta, E., & Turatto, M. (2004)], IOR was assessed by means of a target–target paradigm, and microsaccade dynamics were monitored as a function of both the first and the second visual event. In line with what has been reported with a cue-target paradigm, a significant directional modulation was observed opposite to the first visual event. Because participants were to respond to any stimulus, this rules out the possibility that the modulation resulted from a generic motor inhibition, showing instead that it is peculiarly coupled to the oculomotor system. Importantly, after the second visual event, a different response was observed in microsaccade orientation, whose direction critically depended of whether the second visual event appeared at the same location as the first visual event. The results are consistent with the notion that IOR is composed of both attentional and oculomotor components, and challenge the view that covert orienting paradigms engage the attentional component in isolation.

Keywords: Microsaccades; Inhibition of return; Visual attention; Fixation; Oculomotor programming

1. Introduction

Microsaccades are tiny eye movements executed without awareness during prolonged fixation periods of stationary objects (Ditchburn & Ginsborg, 1953; Ratliff & Riggs, 1950). Results from recent spatial cueing studies indicate that microsaccade direction, instead of being randomly distributed as previously thought (e.g., Steinman, Haddad, Skavenski, & Wyman, 1973), can be correlated with the direction of covert attention shifts, suggesting that microsaccades may, to some extent, be coupled to covert orienting (Engbert & Kliegl, 2003; Hafed & Clark, 2002).

The idea that microsaccades may bridge the gap between covert attention shifting and oculomotor programming is appealing and would add nicely to the great bulk of neurophysiological evidence testifying a robust relationship between the two processes (e.g., Cavanaugh & Wurtz, 2004; Moore & Fallah, 2001; Müller, Philiastides, & Newsome, 2005). However, the data reported so far are not always unequivocally straightforward, although it should also be pointed out that the use of very different experimental paradigms may largely account for the apparently inconsistent results (cf., Rolfs, Engbert, & Kliegl, 2004; Tse, Sheinberg, & Logothetis, 2004).

Engbert and Kliegl (2003) measured microsaccades in a Posner-like spatial cueing paradigm in which a central cue was highly predictive (80%) of the target location, and found that, about 300 milliseconds (ms) after cue onset, microsaccade direction was congruent with attention shifts. In agreement with this notion, Deaner and Platt (2003) have recently shown that non-informative eye-gaze cues presented at fixation not only elicit a corresponding covert

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attention shift (e.g., Friesen & Kingstone, 1998), but also result in a spatial bias of microsaccades towards the location where the gaze is directed. Although these studies using central cues (i.e., cues presented at fixation) reported a microsaccade directional bias congruent with the cued direction, other works have shown a microsaccade bias *opposite* to the direction suggested by highly predictive peripheral visual or auditory cues (Laubrock, Engbert, & Kliegl, 2005; Rolfs, Engbert, & Kliegl, 2005). This pattern has been interpreted as the outcome of increased endogenous oculomotor control aimed to counteract the automatic oculomotor capture evoked by the peripheral predictive cue in a situation in which observers are required to maintain fixation (Rolfs et al., 2004). Galfano, Betta, and Turatto (2004) extended this finding to the case of a peripheral non-informative cue, and argued that the microsaccade bias opposite to a peripheral abrupt onset was consistent with inhibition of return (IOR, Posner & Cohen, 1984), a phenomenon which is thought to reflect an adaptive mechanism that favors search behavior. On the behavioral side, the presence of IOR is inferred when an increase in response time (RT) is observed on trials in which the target is spatially congruent, compared to when it is incongruent, to a prior non-informative, peripheral cue. IOR has been thought as generated by a mechanism that either biases attention from visiting previously attended objects or locations (e.g., Maylor, 1985) or inhibits motor responses towards locations that had previously evoked motor programs with a congruent spatial vector (e.g., Klein, Munoz, Dorris, & Taylor, 2001).

In the experimental context examined by Galfano et al. (2004), both interpretations of IOR may prove correct. In addition, as regards the view of IOR as a motor phenomenon, observers might have exerted either a *specific oculomotor* inhibition to avoid oculomotor capture by the cue, or a *general motor* inhibition to avoid manually responding to the cue (e.g., Poliakoff, Spence, O'Boyle, McGlone, & Cody, 2002; Tassinari, Campara, Benedetti, & Berlucchi, 2002). Hence, the question arises whether the microsaccade bias opposite to peripheral cues is merely the result of a (general or ocular) motor inhibition or it is also suggestive of attentional shifts. The results reported by Rolfs et al. (2004, 2005; also see Laubrock et al., 2005) seem to indicate that a motor component may indeed be present. In those studies, the authors found microsaccades opposite to a peripheral cue in paradigms in which the cue was 80% predictive of target location, a situation in which no attentional IOR should be expected since attention is likely to be endogenously maintained at the cued location. Following this line of reasoning, one should interpret the microsaccadic bias opposite to the cue as a purely (oculo)motor phenomenon. Nonetheless, since microsaccade direction seems indeed to be affected by covert attention shifts (Deaner & Platt, 2003; Engbert & Kliegl, 2003), it is likely that an attentional component is operating also in IOR paradigms. This latter position is supported by recent evidence suggesting that no oculomotor component should contribute to IOR in *covert orienting* tasks. Specifically, Hunt and Kingstone (2003) have tried to dissociate attentional and (oculo)motor components underlying IOR by using manual and saccadic responses and by adopting an experimental rationale based on the logic of the Additive Factor Method (e.g., Sternberg, 2001), according to which two given phenomena are thought to occur in distinct processing stages when their effects combine additively, whereas an interaction is taken to indicate that they share a common processing stage. Hunt and Kingstone (2003) used a classic IOR paradigm and manipulated target luminance and fixation offset, under the assumption that these variables tap selectively onto attentional and oculomotor mechanisms, respectively. The results revealed a double dissociation: IOR interacted with target luminance for manual but not saccadic responses, whereas an opposite interaction emerged for fixation offset. Under the assumption that target luminance selectively affects attentional processes, Hunt and Kingstone concluded that a significant interaction with IOR for manual responses only was evidence that IOR in *overt orienting* (i.e., requiring a saccadic response) tasks reflects the *(oculo)motor component* in isolation. In the same fashion, under the assumption that fixation offset selectively modulates oculomotor processes, Hunt and Kingstone concluded that a significant interaction with IOR for saccadic responses only was evidence that IOR in *covert orienting* tasks purely reflects an *attentional component*.

In the present study, we investigated the contributions of general and specific inhibitory motor control and covert attentional orienting to the directional bias of microsaccades opposite to a peripheral stimulus in an IOR paradigm. Importantly, unlike Galfano et al. (2004), a target–target paradigm was used to rule out the possibility that the microsaccade bias after cue onset merely reflected a general motor inhibition (see Harvey, 1980). In this paradigm, introduced by Maylor and Hockey (1985, 1987), two identical stimuli are presented in sequence at the same or different location and observers have to manually respond to both. Pratt, Kingstone, and Khoe (1997) have argued that the target–target paradigm may be less than ideal to assess IOR, as it may trigger facilitatory effects related to repetition priming that may overlap with (and mask) independent inhibitory effects related to IOR. However, it has been repeatedly shown that this paradigm has the advantage of not being contaminated by general motor inhibition of manual response (e.g., Poliakoff et al., 2002; Roggeveen, Prime, & Ward, 2005; Spence & Driver, 1998; Tassinari et al., 2002), which was one of the topics we wanted to address. If the microsaccadic bias opposite to the cue reported by Galfano et al. (2004) was due to a general motor inhibition, then no such modulation was expected in the present study. Such a scenario may also reconcile Galfano et al.'s data with the proposal that no oculomotor IOR should be observed during covert orienting tasks (Hunt & Kingstone, 2003; see Sumner, Nachev, Vora, Husain, & Kennard, 2004 for a related position). If, however, the microsaccadic bias were replicated in the present study,

then the view that during covert orienting tasks only the attention-based component of IOR should be visible, ought to be revised.

All the previous studies that investigated microsaccades in spatial orienting paradigms monitored the microsaccadic response to the first visual event only (the cue). Therefore, a second aim of the present study was to investigate the microsaccadic response after the second visual event (the second target in our paradigm) and, in particular, to look for any effect of target congruency on temporal and spatial dynamics of microsaccades after the onset of the second target.

Finally, it should also be noted that microsaccades have been shown to help to correct fixation errors (Engbert & Kliegl, 2004). Hence, possible displacements in eye position might also represent a source of bias in microsaccade direction. No predictions were made about the contribution of eye displacements to microsaccade direction, because eye displacements, being produced by drifts as well as microsaccades, can not be anticipated. However, in post-hoc analyses we did investigate whether eye displacements were associated with microsaccade directions and could therefore account (at least in part) for the observed directional biases in microsaccades.

2. Methods

2.1. Participants, apparatus, stimuli, and procedure

Twenty-six adults (age range: 19–31 years, eight females) took part in the study after providing informed consent. All were naïve as to the purpose of the study and reported normal or corrected visual acuity and normal color vision. The study was approved by the local ethical committee at the University of Trento.

Participants sat with their head on a chinrest at about 72 cm in front of a Dell CRT 19" (1024 × 768, 75 Hz) monitor. Presentation of the stimuli was controlled by a custom-made C program running under Windows 2000 on a Pentium IV Dell PC. Eye movements were recorded by an Eyelink II system (SR Research, Ontario, Canada) with a sampling rate of 500 Hz, a spatial resolution of 0.01°, and a calibration error of the absolute gaze position of about 0.5° of visual angle. Microsaccades were monitored monocularly, since the greater part of microsaccades is conjugated (e.g., Møller, Laursen, Tygesen, & Sjølie, 2002; also see Abadi & Gowen, 2004).

A standard nine-point-grid calibration was performed at the beginning of the experiment. The stimuli were white on a black background. At the beginning of each trial participants were shown a central fixation spot (0.73° in diameter) which lasted throughout the trial, along with two white boxes (whose side measured 3°) aligned on the horizontal meridian and equidistant from fixation (12.4° on the left and the right. See Fig. 1). A warning tone (800 Hz, 50 ms) signaled participants that the fixation check was about to begin, in which they had to maintain their gaze within an imaginary central square (3° of visual angle) for 500 ms. When this was achieved, a drift correction based on the average eye position during fixation was performed. If this could not be detected after 800 ms, the display went black and the experimenter could recalibrate the tracker and restart the trial.

Fixation check was followed by a 1000-ms pre-cue baseline phase, with no changes taking place in the display. Then two targets were sequentially displayed with a stimulus onset asynchrony (SOA) that varied randomly between 900 and 1300 ms. SOA values were chosen in order to tap onto IOR. Previous studies with similar experimental settings have proved that SOAs in the range we adopted are optimal to observe IOR in simple detection tasks (e.g., Khatoon, Briand, & Sereno, 2002; Riggio, Scaramuzza, & Umiltà, 2000; also see Samuel & Kat, 2003, for a meta-analysis). In addition, these long values should have resulted in maximizing the possibility of observing microsaccades, since it is well known that microsaccades take place mostly during periods of protracted fixation and that display changes result in a robust suppression of microsaccades (see, e.g., Tse, Sheinberg, & Logothetis, 2002). Each target consisted of one of the two boxes becoming thicker and brighter for 50 ms. Participants were allowed 800 ms to respond to each target by pressing a button on a joypad (i.e., they were to perform a simple detection task). RTs were registered from target onset. The trial ended 1000 ms after the second target onset. The intertrial interval was 2000 ms during which the display went black and no eye movement recording took place. Each participant underwent 128 trials (4 blocks of 32 trials each). 16 (12.5%) of these were catch-trials, in which one of the two target did not appear. In the remaining trials (112) the two target appeared at the same location (congruent trials) or at opposite locations (incongruent trials) with equal probability. Participants were explicitly informed that the first target location was not informative of the location at which the second target would appear. They were instructed to maintain fixation throughout a trial, and to press the button as fast as possible on target-present trials and to withdraw from responding on catch trials. The feedback for misses and catch trial responses was a 1000-Hz tone delivered for 500 ms.

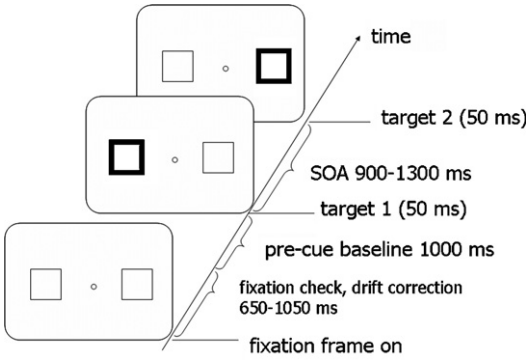

Fig. 1. Sequence of events in a trial. The stimuli were white on a black background. Each target consisted of one of the two boxes becoming thicker and brighter for 50 ms, and could appear on the right or left with equal probability.

2.2. Analysis of microsaccades

2.2.1. Microsaccade detection

Microsaccades were identified with the algorithm first described by Engbert and Kliegl (2003), and adapted for the 500 Hz sampling rate. Microsaccades were defined as intervals (12 ms or longer) in which the eye velocity computed by a 9-point moving average exceeded an elliptic threshold defined by six median standard deviations of the horizontal and vertical velocity. The algorithm was applied from 1000 ms before the first to 1000 ms after the second target onset.

2.2.2. Microsaccade rate

The time course of microsaccade rate was computed for each participant by a window of 100 ms moving in 2 ms steps. The data before the second target onset were time-locked with respect to the first target onset, whereas the data following the second target were time locked to the second target.

2.2.3. Microsaccade direction

Directional biases were measured through the *directional rate* given by the difference between the frequencies of microsaccades with horizontal component towards and opposite to the target (see Hafed & Clark, 2002). The average time course of the directional rate was compared with the null hypothesis that no directional bias exists. The time course of the

directional rate under the no-bias hypothesis can be simulated by randomly re-assigning the direction of each microsaccade in the original data set. A 95% confidence band for the no-bias hypothesis was defined as 1.96 standard deviations of a set of 1000 such simulated rate curves. The time windows in which the curve of directional rate exceeded the no-bias confidence band were selected, and *t*-tests on the mean directional rate were run on these windows. Only windows longer that 20 ms were considered, to minimize the chance of false positives. Paired *t*-tests were run to compare congruent vs. incongruent trials.

2.2.4. Starting position

As microsaccades can help reducing fixation errors (Engbert & Kliegl, 2004), the presence of a bias in the starting position of microsaccades would index the eye displacement as a possible source of directional bias. Therefore, we measured what may be called *positional rate*, i.e., the difference between the rate of microsaccades starting on the target side and that on the opposite side. The positional rate was analyzed following the same logic as for directional rate (i.e., looking for windows where the observed time course exceeded the no-bias confidence band, and performing *t*-tests on the positional rate in those windows).

2.2.5. FDR control

To control type-I errors in multiple inferences, we used the procedure developed by Benjamini and Hochberg (1995) to control the false discovery rate (FDR, i.e., the expected proportion of the rejected null-hypotheses that are erroneously rejected). We applied the algorithm to the whole set of 13 *t*-tests that were carried out in the analysis. The *p*-values reported here are per-comparison *p*-values; however, the adjusted α value produced by the control algorithm (0.03548) was used as criterion.

3. Results

3.1. Manual RTs

Responses on catch-trials were rare (< 5%) and were not analyzed. Responses to the second target were analyzed to check whether the classic behavioral signature of IOR was present. A *t*-test with type of trial (congruent vs. incongruent) as factor revealed that participants responded slower on congruent ($M = 299$ ms, SD = 39) than on incongruent trials ($M = 285$ ms, SD = 39), $t(25) = -4.277$, $p < 0.001$, thus showing a reliable IOR.

3.2. Microsaccade rate

First, we checked that the eye movements classified as microsaccades by the detection algorithm satisfied the linear relation between amplitude and peak velocity (the "main sequence", Zuber, Stark, & Cook, 1965) which is typical of all saccades. Twelve thousand and two microsaccades were detected in the study. The correlation coefficient between amplitude and peak velocity was 0.82, and the regression slope was $(63.5 \pm 0.4)\,s^{-1}$, a value perfectly in line with the data reported by Møller et al. (2002). The mean microsaccade rate was $1.4\,s^{-1}$ (min 0.57, max 2.54, SD 0.49). Fig. 2 shows the time course of microsaccade rate during the trial. The characteristic modulation of microsaccade rate (suppression and enhancement) occurred after each target onset (also see, e.g., Engbert & Kliegl, 2003; Rolfs et al., 2005). The shape of the rate modulation after the second target appears to be independent of target congruency. This indicates that such oculomotor response to display changes is independent of the location of the changes (see also Laubrock et al., 2005).

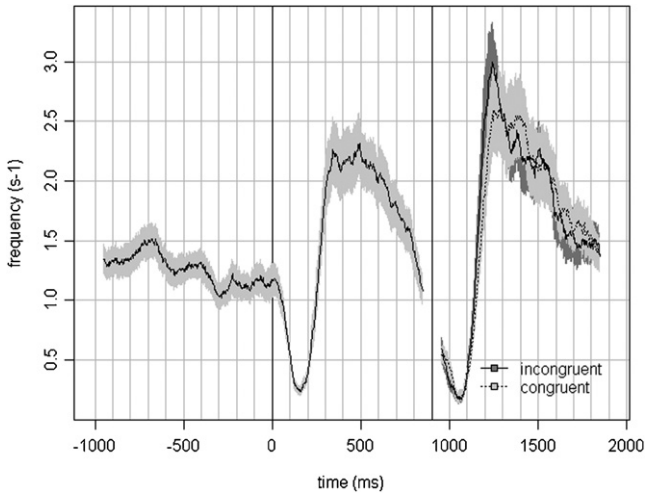

Fig. 2. Evolution of microsaccadic frequency, computed by a moving window of 100 ms. This is the mean of 26 participants. The error band is the standard error of the mean. Black vertical lines indicate target onsets. Pre-target 2 data are time-locked to target 1. Post-target 2 data are time-locked to target 2. Pre-target 2 data are duplicated in panels a and b (congruent and incongruent trials are collapsed).

3.3. Microsaccade direction

Fig. 3 shows the time course of the directional rate of microsaccades relative to the target. In time windows J (206–512 ms) and H (1092–1302 ms) on incongruent trials, and K (1632–1738 ms) on congruent trials, the curve of directional rate exceeded the 95% confidence band corresponding to the hypothesis that there is no directional bias. One sample *t*-tests against zero conducted on the mean directional rate of microsaccades in these time windows confirmed that microsaccades were biased in the opposite direction to the first target in window J, $t(25) = -5.047$, $p < 0.00003$. On incongruent trials, microsaccades were biased opposite to the second target in window H, $t(25) = -3.802$, $p < 0.0008$, whereas on congruent trials they were biased toward the second target in window K, $t(25) = 2.988$, $p < 0.006$. Thus, microsaccades were biased away from the first target about 350 ms after target onset. However, a similar opposite bias following the second target occurred only on incongruent trials. On congruent trials, microsaccades were apparently biased *towards* the second target about 750 ms after its onset. The directional rate in window H was significantly different between congruent and incongruent trials, paired $t(25) = -3.497$, $p < 0.0017$, whereas in window K the difference was not significant, $t(25) = -1.132$, $p = 0.268$.

3.4. Starting position of microsaccades

Overall, 71% of the microsaccades went toward the center of the screen, i.e., microsaccades starting on the left were

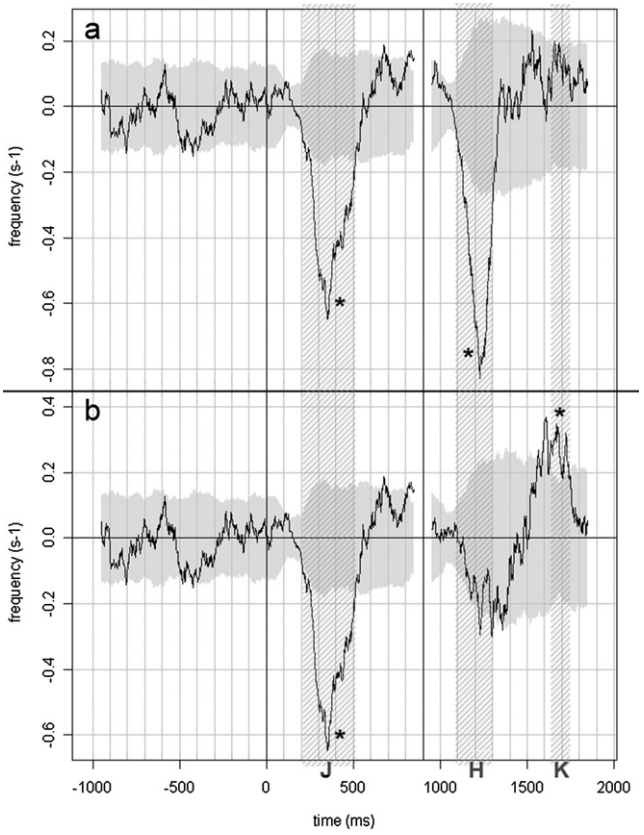

Fig. 3. Evolution of the directional rate of microsaccades, i.e., frequency toward the target minus frequency opposite to the target. Black vertical lines indicate target onsets. Pre-target 2 data are time-locked to target 1. Post-target 2 data are time-locked to target 2. Pre-target 2 data are duplicated in panels a and b (congruent and incongruent trials are collapsed). The curve is the mean of 26 participants. The gray band across the *x*-axis is the 95% confidence interval for the no-bias hypothesis. In time windows J, H, and K the mean directional rate was tested; asterisks mark significant biases. (a) Incongruent trials, (b) congruent trials.

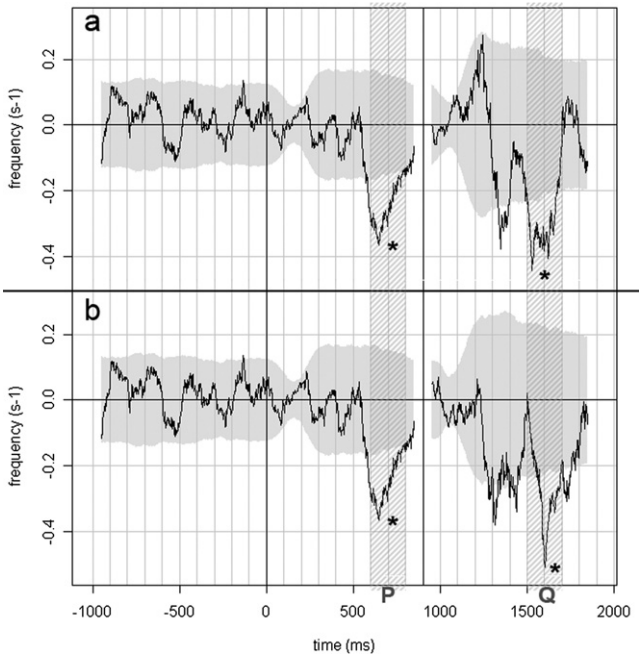

Fig. 4. Evolution of the positional rate of microsaccades, i.e. frequency of microsaccade starting on the target side minus frequency of microsaccades starting on the opposite side. Black vertical lines indicate target onsets. Pre-target 2 data are time-locked to target 1. Post-target 2 data are time-locked to target 2. Pre-target 2 data are duplicated in panels a and b (congruent and incongruent trials are collapsed). The curve is the mean of 26 participants. The gray band across the *x*-axis is the 95% confidence interval for the no-bias hypothesis. In time windows P and Q the mean positional rate was tested; asterisks mark significant biases. (a) Incongruent trials, (b) congruent trials.

mostly oriented rightward and vice versa, so it seems that gaze position did influence microsaccade direction, resulting in corrective microsaccades. Fig. 4 shows the positional rate of microsaccades relative to the target. In time windows P (600–800 ms) and Q (1500–1700 ms) the curve of positional rate exceeded the 95% confidence band corresponding to the hypothesis that there is no bias. This occurred on both congruent and incongruent trials. *T*-tests against zero on the positional rate of microsaccades in these time windows confirmed that in window P microsaccades started mostly from a position opposite to the cue, $t(25) = -3.481$, $p < 0.0018$. The same was true in window Q on both incongruent, $t(25) = -2.905$, $p = 0.0075$, and congruent trials, $t(25) = -2.622$, $p = 0.0146$.

4. Discussion

The relationship between attention and fixational eye movements has recently received much interest in visual neuroscience (see Martinez-Conde, Macknik, & Hubel, 2004). Specifically, several studies have addressed whether microsaccades can be modulated by visual attention (e.g., Engbert & Kliegl, 2003; Hafed & Clark, 2002; Laubrock et al., 2005; Rolfs et al., 2005) and by manual response preparation (Betta & Turatto, 2006). In a recent IOR study, Galfano et al. (2004) reported a microsaccadic spatial bias consistent with the direction of attention shifts after the occurrence of a non informative peripheral cue. However, because Galfano et al. used a cue-target paradigm, a general motor component could not be ruled out as the major determinant for the IOR-related pattern (e.g., Poliakoff et al., 2002; Spence & Driver, 1998; Tassinari et al., 2002). Hence, one of the purposes of the present study was to address the microsaccadic response by adopting a paradigm that eliminates a general manual motor component in IOR. To this aim, we used a target–target paradigm, in which participants have to respond to any visual change in the display (e.g., Maylor & Hockey, 1985). This rules out the possibility that IOR, if any, can emerge as a result of inhibition of manual response (see Harvey, 1980). In the present study, we observed IOR in classic behavioral measures, and also replicated the directional bias opposite to the first peripheral non informative visual onset in microsaccade statistics observed by Galfano et al. (2004). In addition, in the present study, the spatial and temporal dynamics of microsaccades were monitored also after the onset of the second visual onset, as a function of whether

this occurred in a congruent or incongruent spatial location relative to the first visual onset. Interestingly, the microsaccadic response to the second target differed depending on whether the latter appeared at the same location as the first target or not. When the second target appeared at a location opposite to the first, microsaccades were biased opposite to the second target about 350 ms after its onset. However, when the two visual onsets shared the same location the microsaccadic bias opposite to the second visual onset was much less evident if not completely absent. In addition, a late directional bias *towards* the second target emerged about 750 ms after its onset.

Two hypotheses may account, at least partially, for the observed pattern of data. On the one hand, one could interpret the data as consistent with the assumed path of attention during IOR. That is, after the first visual event occurs, say in the left box (see Fig. 5), attention would be first shifted leftwards, and then rightwards. On the oculomotor side, the first attention shift would not be visible because of a general inhibition of microsaccades following display changes (e.g., Rolfs et al., 2004; Tse et al., 2002). By contrast, microsaccade direction would be biased according to the following rightward attention shift due to IOR. The withdrawal of attention from the left box would cause an inhibitory tag to be linked to the corresponding location (as if to say that particular location has been inspected already). Therefore, when the second target appears on the right box performance would benefit from attention being there, or having to move to a location that has not yet been attended; this is indexed by the fact that the manual response to the second target is slower on congruent than incongruent trials. As depicted in Fig. 5, attention would then shift back to the left for the same IOR mechanism just described, and microsaccades would be biased accordingly. However, if the second target appears on the left (i.e., in the box that had been previously attended) it would trigger a less efficient shift of attention to the left, because of the residual inhibitory tag at that location. Thus, the subsequent rightward shift would also be weaker, and so would the associated microsaccade bias. This latter situation is consistent with the reduced, if any, bias opposite the second target during congruent trials, and possibly for the late bias towards the second target that may reflect the carry-over aftermath of reorienting of attention to an inhibited location. One may note that, contrary to Galfano et al. (2004) and the present study, Rolfs et al. (2004, 2005) reported a microsaccadic bias opposite a visual or auditory onset that was highly predictive of the location of the target in a spatial cueing paradigm. This may be interpreted as evidence inconsistent with an attentional hypothesis, because attention was likely to be maintained at the cued location in Rolfs et al.'s studies. However, it should also be noted that IOR seem to be independent of endogenous orienting, since it can be observed even when a target appears at an endogenously attended location (Berger, Henik, & Rafal, 2005; Lupiáñez et al., 2004). Thus, it is still possible that the microsaccadic bias observed by Rolfs et al. (2004, 2005) could still reflect IOR elicited by the onset of the peripheral (although predictive) visual stimulus.

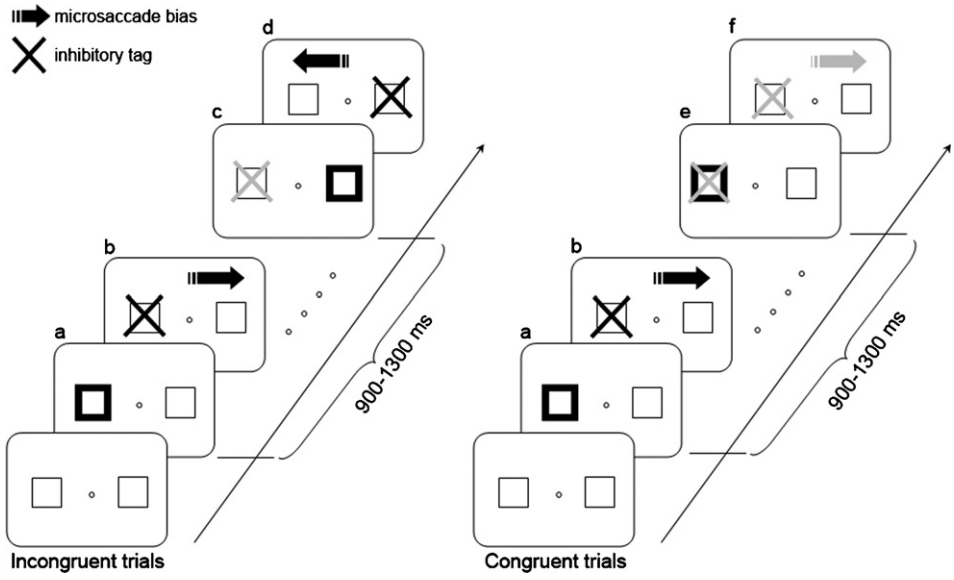

Fig. 5. Attentional account of the observed microsaccade biases. Grey levels indicate the strength of inhibitory tags and microsaccade biases. (a) When the first target appears on the left, attention quickly shifts to the left. This is not reflected in microsaccades because of a general microsaccade inhibition following display changes. (b) Then, because of IOR attention shifts to the right, and microsaccades are biased accordingly. An inhibitory tag is linked to the left box. (c) When the second target appears on the right box (incongruent trial) attention is already there, or moves to that location. In the meantime, the inhibitory tag on the left box is dissipating. (d) Attention shifts back to the left, again because of IOR, and microsaccades are biased accordingly. (e) When the second target appears on the left (congruent trial) it triggers a shift of attention to the corresponding location. This shift, however, would be weaker because of the residual inhibitory tag at that location. Thus, (f) the subsequent rightward IOR shift would also be weaker, and so would the associated microsaccade bias.

On the other hand, the present pattern of microsaccadic response could also be explained without invoking any causal role of attention shifts, but rather by referring to the pattern of activation at the oculomotor level following a peripheral onset when fixation has to be maintained. We call this the "*saccadic inhibitory hypothesis*" (also see Rolfs et al., 2004 for a similar interpretation on microsaccades, and Tassinari, Biscaldi, Marzi, & Berlucchi, 1989, for a related view on IOR). According to this standpoint, when a peripheral *task-relevant* visual transient is presented, the oculomotor system tends to deliver a saccadic response towards the corresponding location. In the present task, however, because participants received clear instruction to maintain fixation, the saccadic activity towards the first visual onset had to be inhibited. This was probably accomplished via an inhibitory signal descending from the Frontal Eye Fields to the Superior Colliculus (e.g., Munoz & Fecteau, 2002; Segraves & Goldberg, 1987; also see Gowen & Abadi, 2005; Ro, Farnè, & Chang, 2003), which resulted in a general inhibition of all the oculomotor activity (saccadic and microsaccadic) that had a vector congruent with the location of the peripheral onset. This imbalance in the neural activity linked to microsaccades might have produced a majority of microsaccades in the direction opposite to the peripheral onset. This might explain why a microsaccadic bias opposite to the first visual onset was found in the present experiment and was also reported by Galfano et al. (2004). The saccadic inhibitory hypothesis would also be consistent with the findings reported by Rolfs et al. (2004, 2005). And the fact that microsaccades are biased away from a visual onset is consistent with the finding that saccades in response to an imperative stimulus deviate away from the onset location (e.g., Doyle & Walker, 2001; Rizzolatti, Riggio, & Sheliga, 1994). Furthermore, recent results by Horowitz, Fencsik, Yurgenson, and Wolfe (in press) would support the view that the microsaccade response to cues reflects activity in the oculomotor system, rather than covert attention.

The saccadic inhibitory hypothesis might provide an answer also for the microsaccadic bias following the second onset. When the second target appeared opposite to the first transient (i.e., incongruent trials), the mechanism described above would again produce a microsaccadic bias in the opposite direction. By contrast, when the second target occurred at the same location as the first visual event (i.e., congruent trials), the results showed a reduced bias in the opposite direction. Such reduced bias might be interpreted as due to a difficulty to inhibit twice the same location in a short time window, a sort of refractory period or neural fatigue in delivering, for the same spatial location, two inhibitory signals one after the other.

In any case, we acknowledge that both the attentional and the saccadic inhibitory interpretation cannot easily account for the late oculomotor bias towards the second stimulus apparent in the congruent condition. Such outcome could still be imputed to Type I error. Alternatively, the late bias towards the second target might be accounted for by a mechanism that corrects eye displacements caused by prior biased microsaccades. Engbert and Kliegl (2004) suggested that on a short time scale microsaccades enhance perception by increasing fixation errors, whereas on a long time scale they reduce fixation errors and binocular disparity. In the present study, microsaccades in the 600–800 ms interval started mainly opposite to the previous target (Fig. 4a,b); however, they were not directionally biased (Fig. 3a,b). On the other hand, microsaccades in the 1500–1700 ms interval, which also started mainly opposite to the previous target, were directionally biased, but only when the two target had been congruent, i.e., they had caused a more sustained eye displacement. This is compatible with the idea that the late bias might be the result of a mechanism that generates corrective microsaccades and that is active only on a long time scale. In this view, after the first target microsaccades go in the opposite direction, producing an eye displacement which is visible in the starting position of later microsaccades (Fig. 4, window P). After the second target, on incongruent trials microsaccades go back toward the first target location, reversing the eye displacement. However, on congruent trials the eyes are displaced again in the same direction, and this sustained displacement finally triggers corrective microsaccades toward the second target (Fig. 3b, window K). The corrective nature of the late directional bias toward congruent second targets is apparent in Fig. 6, where "corrective" microsaccades were filtered out. These were defined as microsaccades that moved the x-coordinate of the gaze closer to the center of the screen, and amounted to 48% of the total. After removing these microsaccades, negative biases are still apparent following each target onset, in time windows J′ (230–638 ms), $t(25) = -5.956$, $p < 0.00001$, and H′ (1100–1418). The latter is significant not only on incongruent ($t(25) = -4.96$, $p < 0.00005$), but also on congruent trials ($t(25) = -3.544$, $p < 0.0016$). However, a significant difference remains between congruent and incongruent trials in window H′, $t(25) = -2.223$, $p < 0.035$. Importantly, the late positive bias on congruent trials is below significance level. Thus, a gaze-corrective mechanism seems indeed to be responsible, at least in part, for the late bias toward second congruent targets.[1]

Following the recent suggestion (Hunt & Kingstone, 2003) that an (oculo)motor component of IOR should only develop under overt orienting conditions (i.e., when a saccadic response is required), we should not have expected any modulation of microsaccade direction as a function of the presence of IOR in the present paradigm. The present findings replicate and extend those reported by Galfano et al. (2004) and are difficult to reconcile with Hunt and

[1] We thank Todd Horowitz who suggested to correct the directional data by filtering out the corrective microsaccades. Error bands and statistical tests for these data were performed with the same procedure and criteria used for the "uncorrected" directional rate, and described in the Methods; the resulting p-values were entered the FDR-control algorithm together with the tests reported in the Sec. 3.

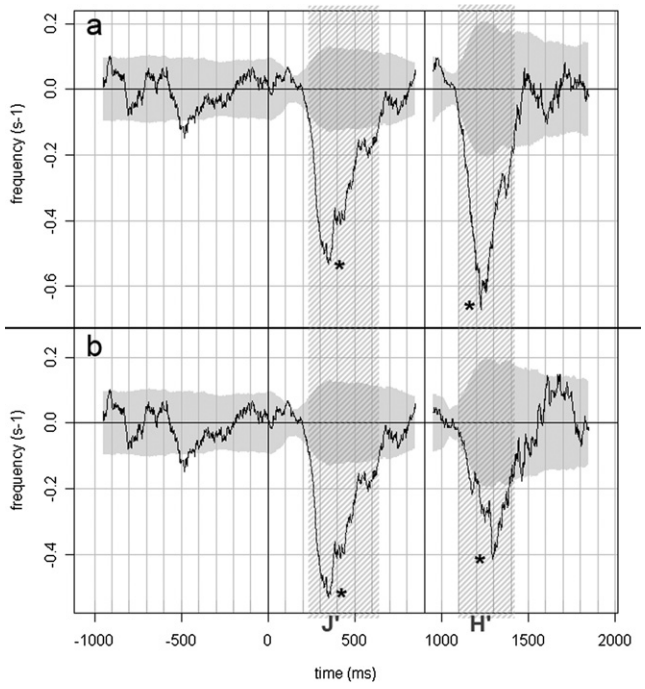

Fig. 6. Evolution of the corrected directional rate of microsaccades. This is similar to Fig. 3, except that "corrective" microsaccades, namely those microsaccades that moved the *x*-coordinate of the gaze closer to the center of the screen, have been filtered out. Black vertical lines indicate target onsets. Pre-target 2 data are time-locked to target 1. Post-target 2 data are time-locked to target 2. Pre-target 2 data are duplicated in panels a and b (congruent and incongruent trials are collapsed). The curve is the mean of 26 participants. The gray band across the *x*-axis is the 95% confidence interval for the no-bias hypothesis. In time windows J′ and H′ the mean directional rate was tested; asterisks mark significant biases. (a) Incongruent trials, (b) congruent trials.

Kingstone's proposal. The data clearly suggest that *an oculomotor component can be observed during IOR also in covert orienting paradigms* and confirm that both oculomotor processes and spatial attention dynamics contribute to the generation of IOR (e.g., Godijn & Theeuwes, 2002; Kingstone & Pratt, 1999; Klein et al., 2001).

In conclusion, the present results indicate that microsaccades in an IOR paradigm are spatially biased even when IOR does not depend from a manual motor inhibition. Salient events, like for example onsets, in the periphery of the visual field produce a microsaccade bias in the opposite direction, no matter if the visual event is informative or not. Finally, the microsaccade bias away from the second onset is weaker, if any, when the second onset is spatially congruent with the first one. This latter result can be explained either by an attentional account of microsaccade direction, or in a purely oculomotor view, in which the need to suppress a reflexive saccade elicited by the peripheral onset results in an opposite microsaccadic bias.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.visres.2006.09.010.

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