



Unconscious cueing effects in saccadic eye movements – Facilitation and inhibition in temporal and nasal hemifield

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ABSTRACT

The current study investigated whether subliminal spatial cues can affect the oculomotor system. In addition, we performed the experiment under monocular viewing conditions. By limiting participants to monocular viewing conditions, we can examine behavioral temporal–nasal hemifield asymmetries. These behavioral asymmetries may arise from an anatomical asymmetry in the retinotectal pathway. The results show that even though our spatial cues were not consciously perceived they did affect the oculomotor system: relative to the neutral condition, saccade latencies to the validly cued location were shorter and saccade latencies to the invalidly cued location were longer. Although we did not observe an overall inhibition of return effect, there was a reliable effect of hemifield on IOR for those observers who showed an overall IOR effect. More specifically, consistent with the notion that processing via the retinotectal pathway is stronger in the temporal hemifield than in the nasal hemifield we found an IOR effect for cues presented in the temporal hemifield but not for cues presented in the nasal hemifield. We conclude that unconsciously processed spatial cues can affect the oculomotor system. In addition, the observed behavioral temporal–nasal hemifield asymmetry is consistent with retinotectal mediation.

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1. Introduction

Attentional processing in the absence of awareness has been shown in studies with hemianopic patients (e.g., Danziger, Fendrich, & Rafal, 1997; Dodds, Machado, Rafal, & Ro, 2002; Kentridge, Heywood, & Weiskrantz, 1999; Rafal, Smith, Krantz, Cohen, & Brennan, 1990) and in studies involving subliminal visual stimuli (e.g., Ivanoff & Klein, 2003; Lambert, Naikar, McLachlan, & Aitken, 1999; McCormick, 1997; Mulckhuysen, Talsma, & Theeuwes, 2007; Woodman & Luck, 2003). McCormick (1997) was the first to show spatial attentional cueing effects without conscious perception of peripheral cues. Typically, in a spatial cueing task, observers fixate their eyes in the middle of the screen while an uninformative cue is flashed in the periphery (Posner, 1980). Subsequently, a target is presented either at the location where the uninformative flash was presented (validly cued) or at the opposite location where no flash was presented (invalidly cued). When the target immediately follows the cue (short SOA), processing of the target at the validly cued location is facilitated; Reaction Times (RTs) to validly cued targets are faster compared to RTs to invalidly cued targets. In contrast, when the target follows the cue after a relatively long interval (long SOA), processing of the target at the validly cued location is impoverished;

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RTs to validly cued targets are slower compared to RTs to invalidly cued targets (Posner & Cohen, 1984). This last phenomenon is called inhibition of return (IOR) and is believed to occur only when attention is captured automatically or when a saccade is endogenously prepared (Rafal, Calabresi, Brennan, & Sciolto, 1989). Note that this latter occurrence indicates the strong relation between the oculomotor system and automatic capture of attention (for review on IOR see Klein, 2000). In McCormick's study, a facilitation effect at the validly cued location was found at the short SOA when observers were unaware of the cue. However, at the long SOA McCormick did not observe IOR. The lack of IOR was attributed to a strategic effect of the observers as a result of the instruction to detect the cue in each trial (see also Ivanoff & Klein, 2003). Presumably, attention lingered longer at the cued location because observers had to search for the cue even though they were unaware of its presence. Nonetheless, subsequent subliminal spatial cueing studies found facilitation at the short SOA as well as inhibition at the long SOA at validly cued locations (e.g., Ivanoff & Klein, 2003; Mulckhuysen et al., 2007). Note that in these studies manual responses were measured. Since attention, IOR and the oculomotor system are strongly related, the current study was designed to determine whether the oculomotor system would be affected by unconscious attentional processing. More specifically we wanted to determine the effect of subliminal cueing on saccade latencies.

One of the interpretations why unconsciously processed visual stimuli can induce attentional orienting involves the retinotectal

or extrageniculate pathway of visual information processing. In contrast to the cortical geniculate pathway, which projects from the retina to the lateral geniculate nucleus (LGN) of the thalamus to the striate cortex, the subcortical retinotectal pathway processes visual information from the retina via the superior colliculus (SC) and the pulvinar of the thalamus to the parietal cortex, which are important structures for spatial attention and eye movements (e.g., Bell, Fecteau, & Munoz, 2004; Dorris, Klein, Everling, & Munoz, 2002; Fecteau & Munoz, 2005; Lamme & Roelfsema, 2000; Shipp, 2004). Hemianopic patient studies seem to corroborate the role of the retinotectal pathway in unconscious attentional and oculomotor processes (Danziger et al., 1997; Van der Stigchel, van Zoest, Theeuwes, & Barton, 2008). Hemianopic patients are unaware of visual stimuli in their blind visual field due to a lesion of the retinogeniculostriate pathway or the striate cortex. However, there is evidence of visual processing in the scotoma (the blind area) in for example blindsight (Weiskrantz, 1986). Blindsight refers to the ability of hemianopic patients to correctly report stimuli presented in the scotoma when asked. In a study by Kentridge et al. (1999), unconscious spatial attentional processing was observed in a hemianopic patient with blindsight. The patient responded faster to validly cued targets in his blind field than to invalidly cued targets in his blind field. The same patient was scanned in an fMRI study by Sahraie and colleagues (1997). They found that subcortical structures and in particular the SC were activated in trials in which the patient reported no awareness of a visual event although his discrimination performance of this visual event was above chance.

To test retinotectal processing in healthy human observers, Ro, Shelton, Lee, and Chang (2004) used transcranial magnetic stimulation (TMS) to mimic a lesion of the retinogeniculostriate pathway. The delivered TMS at striate cortex (V1) interfered with cortical processing and induced a scotoma near fixation. Observers had to make a saccade to a target in the periphery while a distractor was presented in the scotoma. Normally, the presence of a visible remote distractor increases saccade latencies to a target (e.g., Walker, Deubel, Schneider, & Findlay, 1997). In Ro's study, the distractor was presented in the TMS induced scotoma and therefore observers were unaware of the distractor. However, saccade latencies to the target were still increased. Nonetheless, the delay in responding was only observed for saccadic responses. When observers had to make a manual response to the target, the distractor had no effect on reaction time. Ro et al. concluded that the selective disruptive effect of a distractor on saccade latencies and not on manual responses indicated that this process was mediated by the retinotectal pathway. In particular, they reasoned that this selective disruptive effect signified the important role of the SC in this pathway (see also Boyer, Harrison, & Ro, 2005). This selective disruptive effect on saccade latencies is consistent with studies that used a particular type of color contrast (S-cone stimuli), which are not processed via the retinotectal pathway (Sumner, 2006; Sumner, Nachev, Vora, Husain, & Kennard, 2004). In several studies, Sumner et al. (2004) and Sumner (2006) showed that cueing with S-cone stimuli resulted in IOR when a manual response was required but not when a saccadic response was required. They concluded that oculomotor IOR relies more on processes in the retinotectal pathway with an essential role for the SC while 'manual' IOR relies more processes in other collicular pathways. As already mentioned the SC receives direct input from the retina via the retinotectal pathway (Munoz, 2002) and is essential for IOR (Dorris et al., 2002; Rafal, Posner, Friedman, Inhoff, & Bernstein, 1988).

A secondary line of evidence for an important role of the retinotectal pathway and the SC in unconscious processing comes from the temporal–nasal asymmetry effect in attentional orienting: visual stimuli in the temporal hemifield have stronger attentional effects than visual stimuli in the nasal hemifield (e.g., Ansorge, 2003;

Dodds et al., 2002; Posner & Cohen, 1980; Rafal, Henik, & Smith, 1991; Rafal et al., 1990; Simion, Valenza, Umiltà, & Dallabarba, 1995). It has been suggested that these behavioral asymmetry effects arise from an anatomical asymmetry in the retinotectal pathway. The retinotectal pathway is essentially monocular and has more connections from the nasal hemiretina (corresponding to the temporal hemifield) to the contralateral superior colliculus than from the temporal hemiretina (corresponding to the nasal hemifield) to the contralateral superior colliculus. This anatomical asymmetry has been found in cats (Sherman, 1974; Sprague, 1966) and in monkeys (Perry & Cowey, 1984) but is however less clear in humans (Williams, Azzopardi, & Cowey, 1995). Nonetheless, an fMRI study with humans showed that stimulus processing in the SC differed between stimuli presented in the temporal and in the nasal hemifield while this effect was not evident in the LGN or visual cortex (Sylvester, Josephs, Driver, & Rees, 2007). Consistent with the behavioral asymmetry, Sylvester et al. found enhanced activity in the SC for stimuli presented in the temporal hemifield compared to stimuli in the nasal hemifield. However, note that behavior was not measured in this study.

Behavioral studies with hemianopic patients under monocular viewing conditions indicate that unconscious processing is stronger in the temporal hemifield than in the nasal hemifield. In a study by Dodds et al. (2002), a hemianopic patient was tested in a forced choice localization task with targets presented either in the temporal blind hemifield or in the nasal blind hemifield. The results showed that performance was highly accurate in the temporal hemifield and at chance level in the nasal hemifield. Furthermore, in a study by Rafal et al. (1990) hemianopic patients showed an asymmetry effect of a remote distractor in a saccade task: when the distractor was presented in the temporal (blind) hemifield saccade latency increased relative to a no distractor condition, whereas a distractor in the nasal (blind) hemifield did not increase saccade latency.

However, the results from Rafal et al. (1990) were not replicated in a different study with hemianopic patients (Walker, Mannan, Maurer, Pambakian, & Kennard, 2000). Although this study reported a small temporal–nasal asymmetry distractor effect in healthy humans the effect was not present in hemianopic patients. Walker et al. suggested that other cortical processes rather than the retinotectal pathway are responsible for the behavioral asymmetry effect. This conclusion was corroborated in a recent study by Bompas, Sterling, Rafal, and Sumner (2008). In this study they used 'normal luminance' stimuli, which are processed via the retinotectal pathway and S-cone stimuli, which are not processed via the retinotectal pathway. The task they used was the same saccade choice task employed by Posner and Cohen (1980). In this task, observers choose to make a saccade either to a stimulus presented in the temporal hemifield or to a stimulus presented in the nasal hemifield. Observers showed a preference to saccade to the stimulus in the temporal hemifield irrespective of stimulus type, i.e., whether the stimuli were processed by the retinotectal pathway (normal luminance stimuli) or not (S-cone stimuli). The authors concluded that the temporal–nasal asymmetry cannot be the result of processing via the retinotectal pathway but rather is the result of higher cognitive processes. Note that in the Bompas et al. study, participants were aware of the presentation of the cues. Therefore, it is difficult to compare this study with studies in which participants do not consciously process visual stimuli, either because of a lesion or because of the way of stimulus presentation. In addition, although an anatomical asymmetry for cortical visual processing in the temporal and nasal hemifield has been shown in striate cortex in monkeys (LeVay, Connolly, Houde, & Van Essen, 1985), the behavioral asymmetry effect only manifests itself for stimuli beyond the blind spot ($>20^\circ$; Fahle & Schmid, 1987; Paradiso & Carney, 1988).

In the current experiment, our aims were twofold. First, we wanted to investigate whether attentional effects such as facilitation and IOR are manifested by saccade latencies to subliminally cued locations. To avoid strategy effects (Ivanoff & Klein, 2003; McCormick, 1997), observers were not informed that a subliminal cue was presented. Second, we wanted to investigate whether unconscious attentional processing could give rise to a temporal–nasal asymmetry effect. Therefore, we conducted the experiment under monocular viewing conditions. Because involvement of the SC is stronger in oculomotor IOR than in manual IOR, we used saccade latencies as the dependent variable. The stimuli were presented at eccentricities of no more than 8°. We expected to find a facilitation effect at the short SOA and IOR at the long SOA with stronger effects in the temporal hemifield relative to the nasal hemifield.

2. Methods

2.1. Participants

Twenty-four paid volunteers (aged 18–24) participated in the experiment. All participants had normal or corrected to normal vision.

2.2. Apparatus and design

A Pentium IV computer with a processor speed of 2.3 GHz controlled the timing of the events. Displays were presented on an Iiyama 21" SVGA monitor with a resolution of 1024 × 768 pixels and a 60 Hz refresh rate. A second computer controlled the registration of eye movement's data on-line. Eye movements were registered by means of a video-based eye tracker (SR Research Ltd., Canada). The Eyelink 1000 Tower Mount system has a 1000 Hz temporal resolution and a gaze resolution of <0.01° (noise limited) and a gaze position accuracy of <0.5°. Data from the left and the right eye was monitored and analyzed. The distance between monitor and chin rest was 75 cm. The experiment was conducted in a sound-attenuated and dimly lit room.

The experiment consisted of two sessions. In the first session participants had to perform the spatial cueing task. They were not informed of the subliminal cue to avoid that they would start searching for it. In the second session, participants had to perform the cue localization task to examine whether they were able to perceive the subliminal cue. All sessions were performed under monocular viewing. The order of eye patching was counterbalanced between participants.

In the first session (spatial cueing task), each eye was tested in two blocks of 200 trials of which 40% were validly cued trials (20% temporal and 20% nasal hemifield), 40% invalidly cued trials (20% temporal and 20% nasal hemifield) and 20% neutral trials (both hemifields cued). In addition, half of the trials had a short SOA and the other half had a long SOA. All trials were randomly distributed throughout a block. The second session (cue localization task) consisted of two blocks of 24 trials without neutral trials. Again, the cue localization task was also performed under monocular viewing; each eye was tested in one block. On half of the trials the cue was presented on the left and in the other half on the right of fixation point. In addition, half of the trials had a short SOA and the other half had a long SOA. All trials were randomly distributed throughout a block.

All stimuli were presented on a gray background ($x = 0.282$, $y = 0.313$, 6 cd/m^2). Target locations were indicated by two placeholders on the horizontal meridian at a distance of 6.5° left and right from fixation point. The placeholders were two gray ($x = 0.287$, $y = 0.319$, 14 cd/m^2) filled circles of 0.4° in diameter.

Next to the placeholders, at a distance of 8° left and right from fixation point, two larger filled circles of 1.4° in diameter (the mask) were presented. These circles had the same color and luminance as the placeholders. One of these two larger circles next to the placeholders was presented 16 ms earlier. This circle, with a lead time of one refresh rate, served as a cue. The target consisted of a color change of one of the placeholders to black. The distance between cue and target was 1.5° to avoid summation of luminance and lateral forward masking. At the same time, the cued location was close enough to induce attentional processing at the target location (Maylor & Hockey, 1985; Posner & Cohen, 1984).

Fig. 1 shows an example of a trial sequence in which the target was validly cued. Each trial began with a bold fixation point. After 500 ms, the placeholders were presented for 1400 ms with an additional random jitter between 16 ms and 300 ms. The fixation point was dimmed 200 ms before cue onset to disengage the visual fixation system (Munoz, Dorris, Pare, & Everling, 2000). The cue (left, right or both) was presented 16 ms before the mask onset. In the short SOA trials, the target was presented simultaneously with the mask. In the long SOA trials, the target was presented 1000 ms after mask onset.

2.3. Procedure

2.3.1. Session one

Before the experiment started the Eyelink 1000 system was calibrated. Participants had to fixate nine calibration targets that were presented randomly in a 3 × 3 grid across the monitor. On each trial in the experiment, participants were instructed to fixate the centre fixation point and to press the space bar in order to recalibrate the position of the eye. The fixation point then changed into a plus sign as an indication that the position of the eye was recalibrated.

Participants were told to make a speeded saccade to the saccadic target (the black circle). To avoid anticipation saccades a warning beep was presented when participants responded before 80 ms. The warning beep was also presented when participants responded after 800 ms. Participants started the experiment with a practice block of 20 trials. Participants were not informed that cues were subliminally presented.

2.3.2. Session two

Participants were asked to report which of the two outer circles appeared first, the left or the right. To be sure that all participants understood the task, they were shown pictures of a sequence of a trial on paper. Participants responded by pressing the 1 or 3 on the numeric keyboard. The locations on the keyboard corresponded with the locations on the monitor. Each trial started with the same procedure as in the first session, but they did not have to make an eye movement to the saccadic target.

3. Results

3.1. Cue localization task

A one-tailed binomial test for each participants revealed that none of the participants scored significantly above chance level. Mean performance in the cue localization task was 51% (min. 40% and max. 60%) and a one-sample *t*-test showed that this was not significantly above chance level ($p = 0.52$). Because accuracy does not reveal a potential response bias, we also calculated sensitivity (d') and response criterion (C) for 2AFC response data for each participant (Macmillan & Creelman, 2005). A low d' with an extreme C value would indicate that participants scored at chance level not because they were unaware of the cue but rather because

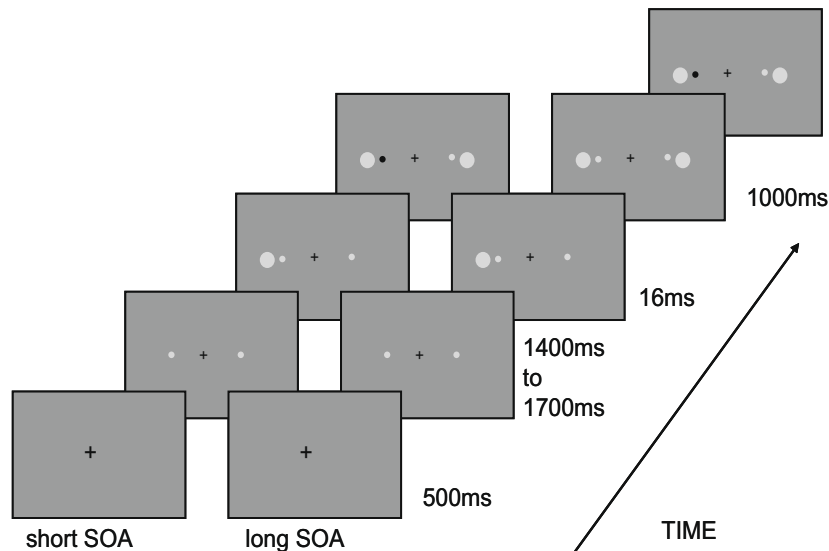


Fig. 1. Example of a trial sequence. Initially, observers fixated a fixation cross for 500 ms after which two placeholders indicated the possible saccade target locations. After a variable interval between 1400 and 1700 ms, a large circle was presented near one of the placeholders. After 16 ms the other large circle was added to the display. The circle with a lead time of 16 ms served as the subliminal spatial cue (in a separate task observers could not tell at which side the cue was presented). Then one of the placeholders turned black indicating to the observer that a saccade had to be made to that location. The placeholder turned black either immediately following the presentation of the cue (left side) or after an interval of 1000 ms (right side). In this example, a trial is displayed in which the cue happened to be valid (cue and target on same side).

they did not search for the cue. The analysis revealed that one of the participants showed an extreme C value (-2.45). This participant was excluded from further analyses. The mean d' of the remaining participants was 0.03 (min. d' -0.43 and max d' 0.54) and the mean C was -0.06 (min. -0.72 and max. 0.69). Both did not differ significantly from zero ($ps > 0.5$).

Furthermore, to exclude the possibility that localization performance was at chance level because performance was averaged out, we performed several additional tests on percentage correct. To test whether performance was above chance level in one hemifield and below chance level in the other hemifield, we performed a two-related Wilcoxon test on percentage correct between cues presented in the temporal hemifield (mean 49%) and cues presented in the nasal hemifield (mean 53%). This test revealed no difference in localization performance between the two hemifields ($p = 0.27$). Furthermore, we examined whether the location of the color change of the saccadic target would have influenced localization performance. A two-related Wilcoxon test revealed no difference ($p = 0.88$) between cue localization performance for cues presented in the same hemifield as the color change (mean 51%) and cues presented in the opposite hemifield of the color change (mean 51%).

3.2. Saccade task

If the endpoint of the initial saccade was within 3° of the centre of the target position and the saccade latency was between 80 ms and 600 ms, the saccade was defined as a correct saccade. Based on these criteria, one participant was excluded because of too many errors (36.5%). Of the remaining 22 participants, 7% of all trials were excluded from analyses.

3.3. Overall effects

To determine the overall effect of the cueing conditions on saccade latency, we collapsed the data from the separate hemifields and performed a 3×2 repeated measures ANOVA with cue validity (valid, neutral and invalid) and SOA (short and long) as factors. The results revealed a main effect of cue validity ($F(2, 42) = 6.55$,

$p < 0.01$, with Greenhouse–Geisser correction), a main effect of SOA ($F(1, 21) = 67.04$, $p < 0.01$) and an interaction effect ($F(2, 42) = 16.73$, $p < 0.01$). Planned comparisons revealed that at the short SOA, mean saccade latency in the valid condition was significantly shorter (311 ms, SE 7.9 ms) than mean saccade latency in the neutral condition (317 ms, SE 8.4 ms; $t(21) = 2.70$, $p < 0.05$). In addition, the mean saccade latency in the neutral condition was significantly shorter than in the invalidly cued condition (324 ms, SE 8.2 ms; $t(21) = 3.08$, $p < 0.01$). This result indicates that the cue captured attention even though observers were unaware of the location of the cue. However, none of the mean saccade latencies for the long SOA differed significantly from each other (see Fig. 2).

3.4. Effect of target per hemifield

To examine whether the targets in the different hemifields would affect saccade latency, we tested the difference in the neutral condition between mean saccade latency in the temporal hemifield and mean saccade latency in the nasal hemifield. Mean

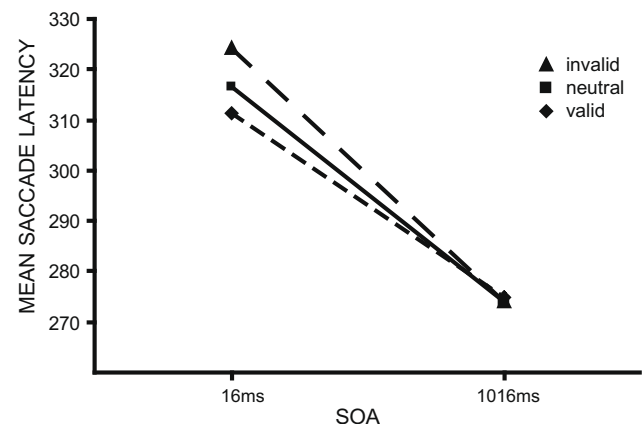


Fig. 2. Mean saccade latency for the validly (filled diamonds), the neutral (filled squares) and the invalidly (filled triangles) cued locations for the short and the long SOA collapsed across hemifields.

saccade latencies to targets in the temporal and nasal hemifields differed at neither short nor long SOAs ($ps = .27$ and $.11$ respectively), suggesting that there were no differences in making a saccade to the temporal or to the nasal hemifield.

3.5. Effect of cue per hemifield

Planned comparisons revealed that at the short SOA, mean saccade latency in the valid temporal hemifield condition was significantly shorter (311 ms, SE 8.2 ms) than mean saccade latency in the invalid temporal hemifield condition (321 ms, SE 8.5 ms; $t(21) = 3.18, p < 0.01$). In addition, the mean latency in the valid nasal hemifield condition was significantly shorter (312 ms, SE 8 ms) than mean saccade latency in the invalid nasal hemifield condition (327 ms, SE 8.3 ms; $t(21) = 4.84, p < 0.01$). These results suggest that attention was captured by the subliminal cue irrespective of hemifield presentation. However, at the long SOA, the valid and invalid condition did not differ for the temporal hemifield or for the nasal hemifield (see Fig. 3).

3.6. Post-hoc analyses

The individual data showed that not all observers demonstrated an inhibition effect at the long SOA when data from both hemifields were collapsed. Previous research showed that in contrast to the facilitation effect, IOR is prone to large individual differences (Avila, 1995). Therefore, we wanted to determine whether an asymmetry effect would occur only for those observers who showed an IOR effect when data from both hemifields were collapsed. To determine an effect of hemifield given that observers showed IOR, only those observers were included that showed a higher mean saccade latency in the validly cued condition compared to the invalidly cued condition at the long SOA when the data was collapsed over both hemifields. This analysis involved 13 participants. The remaining nine participants were observers who did not show an overall IOR effect.

3.7. Cue in nasal hemifield IOR participants

The left panel of Fig. 4 shows the mean reaction times for the IOR participants when the cue was presented in the nasal hemifield. A

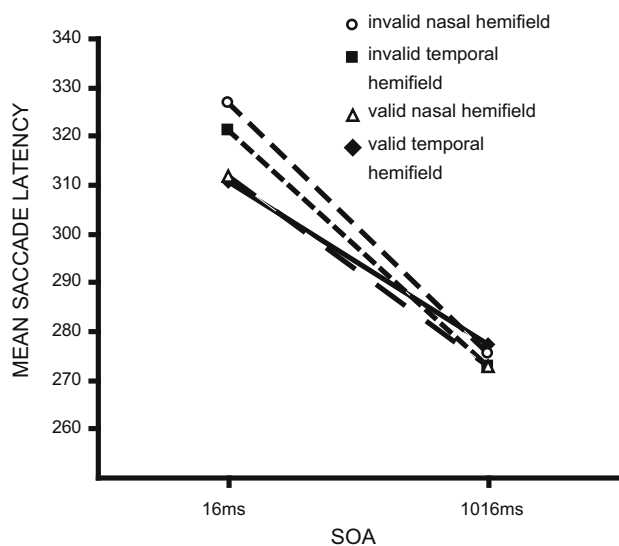


Fig. 3. Mean saccade latency for the validly (diamonds) and the invalidly (squares) cued locations in the temporal hemifield (closed symbols) and for the validly (triangles) and the invalidly (circles) cued locations the nasal hemifield (open symbols) for the short and the long SOA.

repeated measures ANOVA with cue validity (valid and invalid) and SOA (short and long) showed a main effect of SOA ($F(1, 12) = 61.79, p < 0.01$), a main effect of cue validity ($F(1, 12) = 7.92, p < 0.05$), and a significant interaction ($F(1, 12) = 12.36, p < 0.01$). Planned comparisons revealed that, at the short SOA mean saccade latency to the validly cued location in the nasal hemifield was significantly shorter (315 ms, SE 11.3 ms) than mean saccade latency to the invalidly cued location in the nasal hemifield (333 ms, SE 12.9 ms; $t(12) = 4.08, p < 0.01$). However, at the long SOA saccade latencies did not differ between the validly and the invalidly cued location ($p = 0.8$).

3.8. Cue in temporal hemifield IOR participants

The right panel of Fig. 4 shows the mean reaction times for the IOR participants when the cue was presented in the temporal hemifield. A repeated measures ANOVA with cue validity (valid and invalid) and SOA (short and long) showed a main effect of SOA ($F(1, 12) = 35.52, p < 0.01$), and a significant interaction ($F(1, 12) = 17.02, p < 0.01$). Planned comparisons showed that the mean saccade latency at the short SOA to the validly cued location in the temporal hemifield was significantly shorter (317 ms, SE 11.9 ms) than mean saccade latency to the invalidly cued location in the temporal hemifield (326 ms, SE 12.4 ms; $t(12) = 1.98, p < 0.05$, one-tailed). In addition, at the long SOA, an inhibition effect for the validly cued condition was observed in the temporal hemifield. The difference between the valid (285 ms, SE 11 ms) and invalid (274 ms, SE 11 ms) condition was significant $t(12) = 2.44, p < 0.05$.

3.9. Cue in nasal hemifield non-IOR participants

The left panel of Fig. 5 shows the mean reaction times for the non-IOR participants when the cue was presented in the nasal hemifield. A repeated measures ANOVA with cue validity (valid and invalid) and SOA (short and long) showed a main effect of SOA ($F(1, 8) = 25.44, p < 0.01$), and a significant main effect of cue validity ($F(1, 8) = 9.23, p < 0.05$), but no interaction ($F < 1$).

3.10. Cue in temporal hemifield non-IOR participants

The right panel of Fig. 5 shows the mean reaction times for the non-IOR participants when the cue was presented in the temporal hemifield. A repeated measures ANOVA with cue validity (valid and invalid) and SOA (short and long) showed a main effect of SOA ($F(1, 8) = 13.08, p < 0.01$), a marginal significant main effect of cue validity ($F(1, 8) = 4.13, p = 0.08$), and a marginal significant interaction ($F(1, 8) = 4.26, p = 0.07$). Planned comparisons showed that the mean saccade latency at the short SOA to the validly cued location in the temporal hemifield was significantly shorter (301 ms, SE 10 ms) than mean saccade latency to the invalidly cued location in the temporal hemifield (314 ms, SE 11 ms; $t(8) = 2.56, p < 0.05$). But at the long SOA, saccade latencies did not differ between the validly and the invalidly cued location ($p = 0.35$).

3.11. Cue localization task for IOR versus non-IOR participants

It is possible that the mean performance in cue localization differed between the IOR and the non-IOR group. Therefore, we performed a two-independent Mann–Whitney test between cue localization performance for the IOR group and the non-IOR group. The results revealed no significant difference between the two groups; mean performance in the IOR group was 51% and mean performance in the non-IOR group was 50% ($p = 0.6$). In addition, there was no difference between the two groups for cues presented in the temporal hemifield and cues presented in the nasal hemifield ($p = .5$ and $p = .12$ respectively). Finally, we also examined

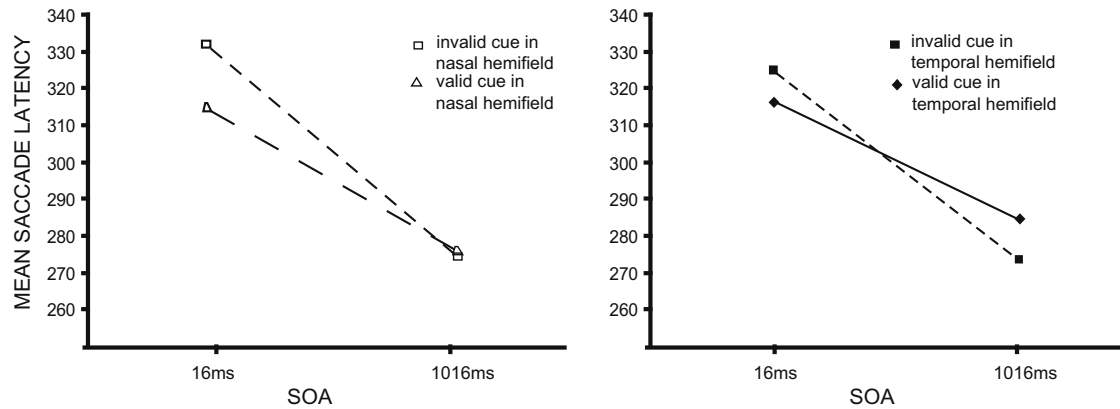


Fig. 4. Mean saccade latency for observers who showed an overall IOR effect. On the left panel the mean saccade latencies for the validly (triangles) and the invalidly (squares) cued locations in the nasal hemifield (open symbols) and on the right panel the mean saccade latencies for the validly (diamonds) and the invalidly (squares) cued locations in the temporal hemifield (closed symbols), both for the short and the long SOA.

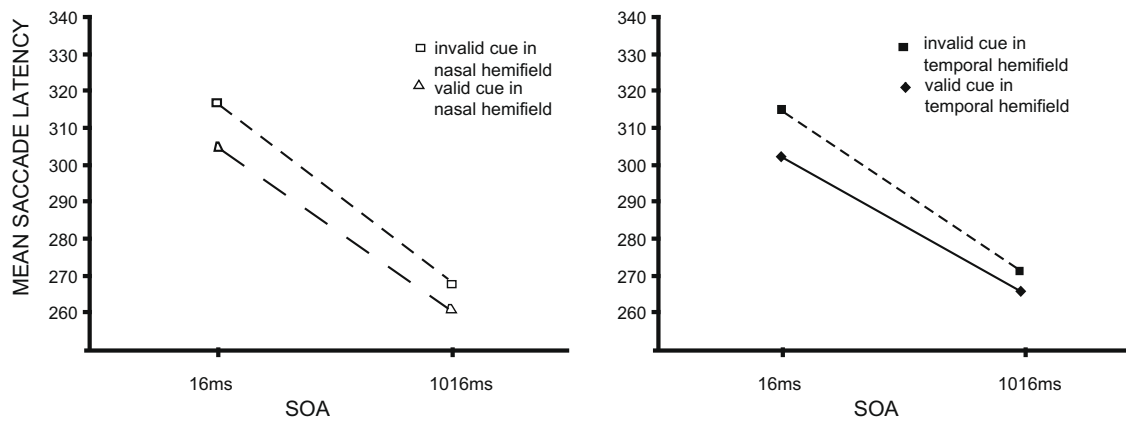


Fig. 5. Mean saccade latency for observers who did not show an overall IOR effect. On the left panel the mean saccade latencies for the validly (triangles) and the invalidly (squares) cued locations in the nasal hemifield (open symbols) and on the right panel the mean saccade latencies for the validly (diamonds) and the invalidly (squares) cued locations in the temporal hemifield (closed symbols), both for the short and the long SOA.

whether there was a difference between the two groups for the different SOAs. As well as for the short SOA as for the long SOA there were no significant differences between the two groups ($p = .2$ and $p = .79$ respectively).

4. Discussion

Results from the cue localization task showed that participants actively searched for the cue but performed at chance level in spite of this, indicating that these participants were unaware of the location of the cue. Even though they were unaware of the cue, the results showed that the subliminal spatial cue triggered attentional orienting to its location. Whereas previous subliminal spatial cueing studies showed the effect of unconscious attentional processing with manual responses (Ivanoff & Klein, 2003; Lambert et al., 1999; McCormick, 1997; Mulckhuyse et al., 2007), we show the effect of unconscious attentional processing on the oculomotor system. At the short SOA, saccade latencies in the validly cued condition were shorter relative to the neutral cued condition, and, in addition, saccade latencies in the invalidly cued condition were longer relative to the neutral cued condition. This result is consistent with spatial cueing studies with visible peripheral cues (Posner, 1980).

As noted, the current experiment was performed under monocular viewing to investigate whether unconscious attentional

processing could give rise to a temporal–nasal asymmetry effect. At the short SOA we did not find a temporal–nasal asymmetry effect but at the long SOA we found an asymmetry effect: IOR at the validly cued location in the temporal hemifield but not in the nasal hemifield. However, the asymmetry effect was only shown for those observers who showed IOR when data from both hemifields were collapsed. The asymmetry effect at the long SOA is consistent with the results of Rafal et al. (1989), although in their study observers did not make a saccadic but a manual response. Consistent with the retinotectal mediation account, Rafal et al. argued that initial covert orienting of attention is less dependent on retinotectal mediation and SC involvement than IOR is. Our results support this notion. However, in Rafal's study the cues were visible. Therefore, it is difficult to compare these studies.

The occurrence of the temporal–nasal asymmetry effect in IOR could signify the role of the SC in the retinotectal pathway. The SC receives direct input from the retina via the retinotectal pathway (Munoz, 2002) and is essential for IOR (Rafal et al., 1988). A neurophysiology study involving macaques provided direct evidence for the role of the SC in oculomotor IOR (Dorris et al., 2002). Neurons in the SC were recorded while the monkeys performed a saccadic spatial cueing task. The results showed a close correspondence between behavior and neural activity: when saccade latencies to the cued location exhibited IOR, target related activity at the cued location in the SC was attenuated (see also Fecteau & Munoz, 2005). Although the correlation between IOR and

neural responses in the SC may provide evidence that the SC is involved in IOR, the authors failed to replicate the pattern of behavior with electrical micro stimulation of the SC. Therefore, they concluded that not the SC itself is inhibited but the SC receives inhibitory signals from cortical areas, possibly the posterior parietal cortex. Danziger et al. (1997) came to a similar conclusion in an IOR study with hemianopic patients. They investigated whether the retinotectal pathway mediates IOR and found conflicting results. Whereas one hemianopic patient showed an IOR effect in the blind field, the other patient did not. Because this latter patient also had a pulvinar lesion, they suggested that the pulvinar, which projects to the parietal cortex, plays the important role in the mediation of IOR. However, a more recent study with patients with a unilateral pulvinar lesion showed that the pulvinar is important in attentional orienting, but damage to it does not prevent IOR (Sapir, Rafal, & Henik, 2002).

One could argue that the cueing effect we found not so much indicate attentional capture but rather visual priming of the oculomotor system. That is, if the cue and target appear at the same location, the cue simply could have primed the oculomotor system to make a saccade to that location. Consequently, saccade latencies are faster to validly cued locations than to invalidly cued locations. This explanation would be consistent with Anson (2003) who argued that the retinotectal pathway is not restricted to attentional processing but also mediates sensori-motor priming. In this study, Anson presented masked primes that could either be congruent or incongruent with the response to the target. In addition, the primes were presented in either the temporal or nasal hemifield. The results from this study showed stronger congruency/incongruency effect with masked primes in the temporal hemifield compared to the nasal hemifield. In our study, the facilitation effect at the short SOA could then be explained as a positive congruency effect (PCE) and the inhibitory effect at the long SOA as a negative congruency effect (NCE), although inhibition is not always found for stimuli presented in the periphery (Schlaghecken & Eimer, 2000, but see Lingnau & Vorberg, 2005). In addition, the explanation for the negative congruency effect as a motor-inhibition account has been subject to debate (see for extensive review Sumner, 2007).

Taken together, our results do not exclude the idea that other pathways or processes than the retinotectal pathway are responsible for unconscious attentional processing (e.g., Bompas et al., 2008; Walker et al., 2000). For example, the orienting response to the subliminal cues could have been the result of feedforward processing via cortical projections (Fahrenfort, Scholte, & Lamme, 2007). However, our results are consistent with the view that unconscious attentional orienting is mediated by the retinotectal pathway (see also Boyer et al., 2005; Dodds et al., 2002; Rafal et al., 1989, 1990; Ro, 2008; Ro et al., 2004).

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