Inhibition of return, gap effect and saccadic reaction time to a visual target

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Abstract

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Simple manual reaction time (MRT) to a visual target (S2) is shortened
when a non-informative cue (S1) is flashed at the S2 location shortly
before the onset of S2 (early facilitation). Afterwards, MRT to S2
appearing at the S1 location is lengthened (inhibition of return - IOR).
Similar results have been obtained for saccadic reaction time (SRT).
Moreover, when there is a temporal gap between offset of the fixation
point (FP) and onset of a target (gap paradigm), SRT is shorter than
SRT in an overlap paradigm (FP remains on). In the present study, we
determined SRT to S2 (10º) after presenting S1 at the same eccentric-
ity (10º) or at a parafoveal position (2º) in the same or in the opposite
hemifield. In addition, we employed both gap and overlap paradigms.
Twelve subjects were asked not to respond to S1 (2º or 10º) to the right
or to the left of FP, but to respond by making a saccadic movement in
response to S2. We obtained the following results: 1) a 40-ms gap
effect, 2) an interaction between gap effect and IOR, 3) a 39-ms delay
(IOR) when S2 appeared at the cued (S1) position, and 4) a smaller (17
ms) but significant inhibition when S1 occurred at 2º in the ipsilateral
hemifield. Thus, a parafoveal (2º) S1 elicits an inhibition of SRT
towards ipsilateral peripheral targets. Since an inhibition of the ipsilat-
eral hemifield by a 1º eccentric cue has been reported to occur when
manual responses are employed, we suggest that the postulated func-
tional link between covert and overt orienting of attention is also valid
for parafoveal cues.

Manual reaction time (MRT) to a periph-
eral visual target is shortened when a non-
informative cue is flashed at the target loca-
tion 100-150 ms before the target onset (early
facilitation). Afterwards, MRT to targets ap-
pearing at the cue position is lengthened
(inhibition of return - IOR) (1). The spatial
distribution of IOR is one of its conspicuous
properties. Several investigators have found
that IOR is maximal at cue position, but the
inhibition spreads over a large extension of
the ipsilateral hemifield (2-8). For instance,
Berlucchi et al. (2) have reported that a cue
located at 1º from the fixation point (FP)
inhibits the manual response to an ipsilateral
target located at a 30º eccentric position (a
29º cue-target distance), but does not inhibit
the response to a contralateral target located

at 1° from the FP (a 2° cue-target distance). These findings are symmetrical with those found in covert voluntary orienting of attention experiments in which the MRT to a target occurring at the attended point is shorter than to targets occurring at other positions (9-11). Moreover, the benefit arising from the orienting of attention decreases with distance between the attended position and target position but does not cross the meridians (3,12). To explain the difference between attended and unattended positions, Rizzolatti and colleagues (11) proposed that the motor programs for eye movement control both overt and covert orienting of attention, so that, "to pay attention" to a peripheral position corresponds to programming an eye movement that will direct the eyes to the attended position if the eye movements are not blocked voluntarily (premotor theory of visual attention). Moreover, the program specifies the direction of eye movement and the exact amplitude to be covered. So, when the stimulus occurs at the attended position, an ocular or manual response will be emitted without further delay. However, when the stimulus occurs at an unexpected position, a time-consuming change in the ocular program is required, involving a cost whose magnitude depends on the movement feature that must be modified. The cost will be greater if the attended and stimulus positions are located on opposite sides of the vertical or horizontal meridian and will be smaller if they are located at different positions inside the same hemifield. On the other hand, the inhibitory effect of a non-informative cue may arise from the veto of overt responses (eye movement) towards the cue, delaying all responses to targets located in the cue hemifield (3,4).

Similar effects of voluntary orienting of attention on saccadic eye movements have also been observed (13). Moreover, inhibition has been found for saccadic eye reaction time when the visual target is presented at the same position as the cue (8,14,15) or in another peripheral position in the same hemifield (12,14,15). In short, the covert voluntary orienting of attention elicits maximal facilitation at the attended position and from there a decreasing facilitation in the ipsilateral hemifield which stops at the main meridians. On the other hand, a non-informative cue elicits an inhibition that follows similar but opposite spatial distribution rules (maximal inhibition at a cue position which decreases with distance and stops at the main meridians) (2-4).

For saccadic reaction time (SRT), it has also been reported that introducing a temporal gap between the offset of the FP and the onset of a peripheral target (gap paradigm) shortens the latency of saccadic eye movements (gap effect) in comparison to a condition in which FP remains on (overlap paradigm) (16). Moreover, Abrams and Dobkin (17) have reported that the magnitude of the gap effect for saccades is greater in the non-cued hemifield (34 ms - experiment 2, 36 ms - experiment 3) than in the previously attended location (28 ms - experiment 2, 19 ms - experiment 3). However, this result may be due to the difference in IOR between the overlap and gap conditions. Indeed, the cited investigators found a small IOR (4 ms in experiment 2, and 2 ms in experiment 3) in the overlap condition and a larger IOR (10 ms in experiment 2 and 19 ms in experiment 3) in the gap condition. Although there are no definitive data excluding the possibility that IOR could be due to a facilitation of the contralateral hemifield, the dominant view is that IOR is a phenomenon which involves inhibition of the ipsilateral hemifield. According to this view, Abrams and Dobkin's results should be interpreted as a reduction of the gap effect in the cued location due to the presence of IOR.

Many studies have suggested that the superior colliculus (SC) is involved in covert and overt orienting of attention as well as in the gap effect (for reviews, see Refs. 18 and 19). However, the SC might not be sufficient

for IOR to be observed (19). Single-unit recording has shown that when the target was presented at a previously cued location the response of collicular neurons was attenuated and the magnitude of this response was correlated with subsequent SRT. However, this reduction in collicular activity was not caused by active inhibition of these neurons due to the cue. Indeed, the neuronal activity increases after the presentation of the cue in its response field and the electrical microstimulation of the SC elicits faster saccades when the cued location is stimulated. This implies that the primate SC participates in the expression of IOR. However, the SC is not the site of inhibition. Instead, the reduced response of collicular neurons reflects a signal reduction elicited by the cue that has occurred upstream, probably in the posterior parietal cortex (19).

The present investigation addresses two questions related to the mechanisms involved in covert and overt orienting of attention as well as in ocular fixation and eye movements. The first one is if there is an interaction between the gap effect and the inhibition of return. The second one is if a parafoveal (2º) cue which is represented in the fixation region in the rostral pole of the SC lengthens the SRT to an ipsilateral peripheral (10º) target.

Twelve right-handed young adults (2 males and 10 females) with normal or corrected visual acuity volunteered to take part in the experiment. All were naive in reaction time tasks and were unaware of the purpose of the experiment. The experiment took place in a sound-attenuated dark room. The subject sat in front of a screen to which green light-emitting diodes were attached. A Pentium microcomputer timed the stimuli and recorded the responses (saccadic movements). We used an infrared scleral reflectance device (Applied Science Laboratories, model 210, Bedford, MA, USA) to record eye movements. Recorded data were sent to a personal computer for storage and analysis

using National Instruments cards (Lab-PC+ and AT-AO-6) and software (Austin, TX, USA). A velocity criterion of 30º/s was employed to define the beginning of the ocular saccade. The head was positioned on a head-and-chin rest so that the distance between the eyes and the screen was maintained at 57 cm. Each subject participated in 5 experimental sessions on separate days or, sometimes, on the same day separated by a rest of at least 1 h.

A session consisted of 320 trials divided into 4 blocks separated by rest periods of a few minutes. The blocks differed according to S1 eccentricity (2 or 10º) and fixation condition (gap or overlap). Each trial began with the presentation of an FP in the center of the screen (Figure 1). Nine hundred milliseconds later, a non-informative cue (S1) appeared at 2º (or 10º) to the right or the left of the FP and remained on for 100 ms. Seven
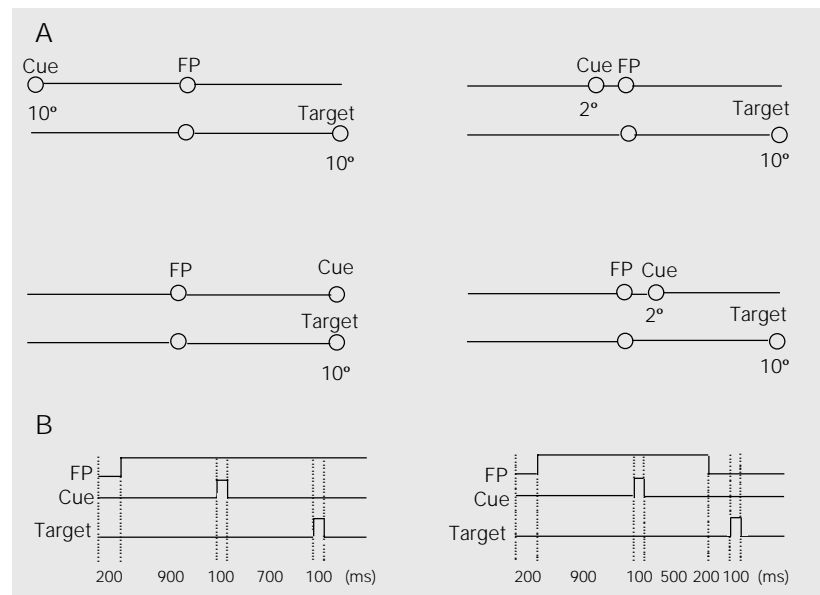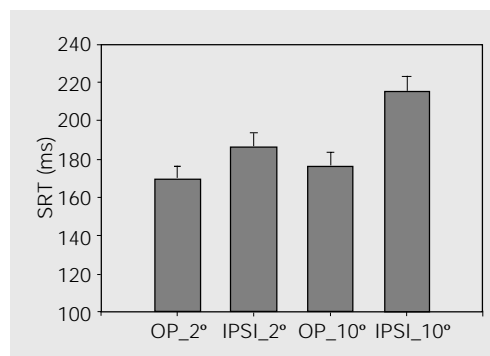

Figure 1. Schematic diagram and temporal sequence of events during trials. A, Relative positions of the fixation point (FP) when the cue occurred: 1, 10º on the left and the target was presented 10º on the right (cue and target in opposite hemifields), 2, 2º on the left and the target was presented 10º on the right (cue and target in opposite hemifields), 3, 10º on the right and the target was presented 10º on the right (cue and target at the same position), 4, 2º on the right and the target was presented 10º at right (cue and target in the same hemifield, but in different locations). B, Temporal sequence of events: the overlap paradigm in which the FP remains on throughout the trial is illustrated on the left and the gap paradigm in which the FP is turned off 200 ms before the appearance of the target is illustrated on the right.

hundred milliseconds after its offset, a target (S2) was flashed for 100 ms 10º to the right or to the left of the FP. The interval between the onset of S1 and of S2 (stimulus onset asynchrony) was 800 ms. In the overlap paradigm, the FP was on when the target appeared and remained on until the end of trial. In the gap paradigm, the FP was offset 200 ms before the appearance of the target. The subjects were instructed to look at FP, not to respond to S1 and to respond as quickly as possible to the onset of S2 by making a target-directed saccade. SRT was measured from the onset of S2 to the beginning of the saccade. SRT shorter than 80 ms or longer than 699 ms were considered to be errors and these trials were not included in the analyses. The first session was used for practice and its data were discarded. The data of the additional 4 sessions were pooled. At the end of these sessions, 16 correct mean SRT values were calculated for each subject. These means were entered into a four-way repeated measure analysis of variance (ANOVA) with the following factors: fixation condition (overlap or gap), S1 eccentricity (2º or 10º), S2 hemifield (left or right), S1-S2 spatial relation (ipsi- or contralateral condition). The pairwise comparisons described below were made using the Newman-Keuls method and the level of significance was always set at $P < 0.05$.

Three main factors had significant effects on reaction time: fixation condition, S1-S2 spatial relation and S1 eccentricity. SRT was longer (207 ms) in the overlap condition ($F(1,11) = 30.122$, $P = 0.000$) than in the gap condition (167 ms). When S2 appeared in the cued hemifield, SRT (201 ms) was longer ($F(1,11) = 70.000$, $P = 0.000$) than when S1 and S2 occurred in opposite hemifields (173 ms). Finally, when S1 occurred at a peripheral (10º) position, SRT (196 ms) was longer ($F(1,11) = 23.884$, $P = 0.000$) than when S1 occurred at a parafoveal (2º) location (178 ms).

In addition, there were two significant interactions. The first was between the fixation condition and the S1-S2 spatial relationship ($F(1,11) = 9.797$, $P = 0.010$). For the gap paradigm, SRT in the ipsilateral hemifield (184 ms) was longer than SRT in the contralateral hemifield (151 ms, IOR = 33 ms). For the overlap paradigm, SRT in the ipsilateral hemifield (219 ms) was also longer than SRT in the contralateral hemifield (195 ms), but IOR (24 ms) was smaller than in the gap paradigm. The gap effect for ipsilateral and contralateral targets was found to be greater when the target and the cue occurred in opposite hemifields (44 ms) than when they occurred in the same hemifield (33 ms), confirming data reported by Abrams and Dobkin (17). The second significant interaction was between the S1-S2 spatial relationship and S1 eccentricity ($F(1,11) = 16.848$, $P = 0.002$) and is illustrated in Figure 2.

SRT obtained when S2 occurred at the S1 position (SRT = 216 ms) was 39 ms longer than when S1 and S2 occurred at 10º in opposite hemifields (177 ms, IOR = 39 ms). A smaller (17 ms) but significant inhibition was elicited when the S1 appeared at 2º from the FP in the ipsilateral hemifield. The SRT (187 ms) when a parafoveal (2º) S1 occurred before a peripheral (10º) S2 in the same hemifield was longer ($P > 0.05$) than the SRT observed when a parafoveal S1 and a peripheral S2 occurred in opposite hemifields

Figure 2. Saccadic reaction time (SRT) to targets presented in the same (IPSI) or in the opposite hemifield (OP) of the cue. The cue appeared at 2º or 10º from the fixation point and the target always appeared at 10º from the fixation point. There was a significant difference between the SRT observed when cue and target appeared at the same position (IPSI_10º) and all other SRT. There was also a significant difference between the SRT observed when the cue appeared at 2º in the opposite hemifield (OP_2º) and at 2º in ipsilateral hemifield (IPSI_2º). On the other hand, there was no significant difference between the conditions in which the cue appeared at 2º or 10º in the contralateral hemifield. Pairwise comparisons were made with the Newman-Keuls test ($P < 0.05$).

(170 ms). On the other hand, there was no significant difference (P > 0.05) between the conditions in which S1 appeared at 2º or 10º in the contralateral hemifield (170 and 177 ms, respectively).

Five general results are striking: a) when the FP was kept on (overlap-paradigm), SRT (207 ms) was longer than when the FP was turned off 200 ms before the target stimulus appeared (gap-paradigm, SRT = 167 ms), a 40-ms gap effect; b) the gap effect was greater when the target and the cue occurred in opposite hemifields (44 ms) than when they occurred in the same hemifield (33 ms); c) the IOR was greater in the gap paradigm (33 ms) than in the overlap paradigm (24 ms); d) S1 elicited a 39-ms delay (IOR) if it occurred at the S2 position, and e) a smaller (17 ms) but significant inhibition was elicited when the S1 appeared at 2º from the FP in the ipsilateral hemifield.

As described before, IOR was discovered using manual responses and covert orienting of the attention paradigm (1). Since then, several investigators have described the spatial distribution of the inhibitory effects of a non-informative cue on manual and ocular responses to a visual target (2,3, 5,7,8,11,14,15). Usually, there is no discrepancy between the results found using covert (without eye movements) and overt (with eye movements) responses, in agreement with the premotor theory of attention (11). However, until know, the non-informative cue in IOR ocular studies was always presented at an eccentric position, that is, outside the fixation region which is represented by the rostral pole of the SC (for reviews, see Refs. 19 and 20). The role of the rostral colliculus in eye fixation and the reciprocal inhibitory connection between the fixation region and the saccadic region in the caudal colliculus (for a review, see Ref. 20) may elicit a dissociation between the IOR for manual responses and the IOR for ocular response. However, our results show that a parafoveal (2º) cue elicited an inhibition of the saccades directed towards peripheral positions in the ipsilateral hemifield that was similar to the inhibition elicited by a 1º eccentric cue on manual responses to targets occurring over a large extension of the ipsilateral hemifield (2). These results suggest that a parafoveal cue has similar effects on covert and overt orientation of attention.

Our main result is that a parafoveal (2º) non-informative cue elicits an inhibition of saccadic eye movements towards an ipsilateral peripheral (10º) target, as observed for manual responses (2). This is an unexpected result because the rostral colliculus is involved in ocular fixation (20). However, a recent electrophysiological study (19) has shown that the primate SC participates in the expression of IOR, but is not the site of inhibition. Instead, the reduced response of collicular neurons reflects a signal reduction elicited by the cue that has occurred upstream, probably in the posterior parietal cortex (19). The possible participation of cortical structures in IOR may explain the similarities between the ocular and manual expression of IOR.

Acknowledgments

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