Saccadic inhibition underlies the remote distractor effect

Antimo Buonocore · Robert D. McIntosh

Abstract  The remote distractor effect is a robust finding whereby a saccade to a lateralised visual target is delayed by the simultaneous, or near simultaneous, onset of a distractor in the opposite hemifield. Saccadic inhibition is a more recently discovered phenomenon whereby a transient change to the scene during a visual task induces a depression in saccadic frequency beginning within 70 ms, and maximal around 90–100 ms. We assessed whether saccadic inhibition is responsible for the increase in saccadic latency induced by remote distractors. Participants performed a simple saccadic task in which the delay between target and distractor was varied between 0, 25, 50, 100 and 150 ms. Examination of the distributions of saccadic latencies showed that each distractor produced a discrete dip in saccadic frequency, time-locked to distractor onset, conforming closely to the character of saccadic inhibition. We conclude that saccadic inhibition underlies the remote distractor effect.

Keywords  Oculomotor · Saccadic inhibition · Remote distractor effect · Vision

Introduction

As we visually explore the world around us, changes may occur at locations remote from our next intended fixation. The problem of multiple potential targets was first investigated experimentally by Lévy-Schoen (1969), who presented two targets simultaneously, either within a hemifield or bilaterally. Participants were biased to look towards the more proximal target, but were 30–40 ms slower on average to begin moving under bilateral conditions. This saccadic reaction time (SRT) cost was interpreted as the additional time required to choose which direction to look in. Subsequent work, however, established that the cognitive element of choice is not critical, since comparable increases of SRT were induced by the onset of a stimulus at fixation, 50–150 ms following the onset of a single lateralized target (Ross and Ross 1980, 1981). Moreover, Braun and Breitmeyer (1990) obtained similar interference from stimuli at fixation, or in the contralateral field, when target location was predefined and constant (see also Weber and Fischer 1994). In such tasks, the additional stimulus can be conceived of as a distractor rather than as a competing target.

This phenomenon, now known as the remote distractor effect (RDE), has been explored most extensively in two papers by Walker et al. (1995, 1997). The first of these confirmed that the RDE arises even when target location is predictable, and further examined the consequences of manipulating target-distractor asynchrony for a contralateral distractor. Walker et al. (1995) observed reliable increases of SRT when the distractor was simultaneous with, or within 40 ms after the target, with a diminishing influence thereafter. Numerically, the effect was greatest (~18 ms) with a simultaneous distractor. Comparing the SRT frequency distribution in the simultaneous-distractor condition with that for the target alone, Walker et al. suggested that the RDE results from a reduction in the frequency of “fast-regular” saccades relative to “slow-regular” saccades.
More recently, another finding has emerged within the eye movement literature that may have important implications for understanding the RDE. Reingold and Stampe (1999, 2000, 2003, 2004) noted that large transient visual changes during text reading or visual search tasks produce “saccadic inhibition”: a characteristic dip in saccade frequency, visible as early as 60–70 ms after the change, with its nadir around 90–100 ms, and returning to normal levels within 120–130 ms. They subsequently demonstrated the same effect in target-elicited saccadic tasks (Reingold and Stampe 2002). Participants responded to a lateralized visual target, and online analysis of the eye-tracking data from each trial enabled a running computation of the median SRT. On “flash” trials, a dramatic but brief visual change was applied, (on average) 100 ms in advance of the current median SRT, with the top and bottom thirds of the screen turning from grey to white for 33 ms. As in reading and visual search, there was a pronounced dip in saccade frequency beginning 60–70 ms after the flash, with its nadir around 90 ms. The dip was time-locked to the flash, and independent of the asynchrony between target and flash, showing a comparable character across wide variations in baseline saccadic latencies associated with gap, overlap, prosaccade and antisaccade tasks.

In discussing these findings, Reingold and Stampe (2002) hypothesised that saccadic inhibition might cause the slowing of SRTs observed in the RDE. The RDE is usually elicited by a small, localised visual change, whilst saccadic inhibition has been studied using a large flash, but this superficial difference might belie common mechanisms of influence. The “saccadic inhibition hypothesis” (SIH) implies that the key factor governing the RDE would not be the temporal relationship between target and distractor, but that between the distractor and the planned saccade. Of course, to the extent that the timing of saccades depends upon the timing of target presentation, there will be a relationship between RDE magnitude and the target-distractor asynchrony. However, this relationship would be indirect and task-specific, determined dually by the SRT distribution for the baseline task and the time-course of saccadic inhibition. As noted by Reingold and Stampe (2002): “If saccadic inhibition caused by the presentation of the visual change... is responsible for the observed slowing of SRTs, then whether or not such an effect is predicted depends on two important factors: the latency between the onset of the target and the onset of the distractor... and the characteristics of the SRT histogram obtained when a distractor is not presented” (p. 380).

Under this interpretation, the fact that different authors have found the RDE to be maximal at different target-distractor asynchronies might be due to differences in the SRT distributions for the baseline (no-distractor) tasks. Ross and Ross (1980) found the RDE to be maximal when the distractor followed the target by 100 ms, whereas Walker et al. (1995) obtained their maximum effect with a simultaneous distractor. Consistent with the SIH, the former observation was made in the context of a relatively late baseline SRT distribution (mean ~290 ms); the latter with a much earlier distribution (mean ~168 ms). Moreover, we can speculate that the selective reduction in fast regular saccades that Walker et al. (1995) identified might just reflect the portion of their baseline distribution most visibly affected by the dip induced by a simultaneous distractor, rather than the RDE being characterised universally by the attrition of fast-regular saccades.

The SIH predicts that different portions of the baseline SRT distribution should be impacted by distractors presented at different times, with the dip time-locked to distractor onset. Our study was designed to test this critical prediction, using a task based upon that of Walker et al. (1995), in which the asynchrony between target and distractor is varied. As expected, the data show a robust RDE for distractors presented simultaneously with, or shortly after the target. However, our main concern is with the character of the changes to the baseline SRT distribution associated with the different distractor onsets. The SIH predicts that the RDE will invariably be accompanied by a discrete dip in the frequency distribution, developing within ~70 ms of distractor onset. Our data provide unequivocal support for this prediction, suggesting that saccadic inhibition does indeed underlie the RDE.

**Methods**

**Participants**

Fourteen volunteers from the University of Edinburgh aged between 18 and 24 years participated. All participants were free from neurological and visual impairments. This experiment was conducted in accordance with the 1964 Declaration of Helsinki, with the approval of the Ethics Committee of the School of Philosophy, Psychology and Language Sciences at the University of Edinburgh. All participants gave informed consent prior to testing.

**Apparatus and stimuli**

Stimuli were white on black, presented on a 19 inch CTR monitor (1,024 × 768 pixels) driven by a Pentium IV processor at 120 Hz. Participants were seated in front of the monitor, with their head immobilised in a chin rest and their eyes horizontally and vertically aligned with the centre of the screen at a viewing distance of 90 cm. Eye movements were recorded with the EyeLink II head-mounted system, running in pupil-tracking mode, at a sampling frequency of 500 Hz.
A 0.50° fixation cross occupied the centre of the screen throughout each trial (overlap design). The saccadic stimulus was a circle of 0.57° diameter, presented at 4.5° eccentricity in the right visual field. The target appeared after a fixation interval that varied randomly between 500 and 1,000 ms. The target was presented alone or accompanied by a mirror-image distractor on the left. Distractor onset was simultaneous with target onset, or delayed by 25, 50, 100, or 150 ms. The target was presented for 400 ms followed by a blank screen for 100 ms. Distractors were presented for 100 ms.

Procedure

Each trial began with drift correction and a tone signalling the onset of the fixation cross. Participants were required to fixate the cross and to move their eyes to the target as soon as it appeared on the right side of the screen. The target could be presented alone (T) or with a distractor (D), presented under five delay conditions (D0, D25, D50, D100, D150). Each participant completed 1,440 trials split into three sessions comprising ten blocks of 48 trials each. Within each block, eight trials for each of the six conditions were shuffled randomly. A three point-calibration on the horizontal axis was run at the beginning of each session and after three consecutive blocks; additional calibrations were run if the participant moved their head from the chinrest. After each session the headset was removed and the participant took a short break. The experiment lasted ~90 min in total per participant.

Data screening

Saccades to the left (1.55%), saccades of less than 1° amplitude (1.68%), and saccades with a latency under 70 ms (2.39%) or over 400 ms (1.51%) were excluded.

Results

RDE analysis

For each participant, the median SRT was taken as the measure of central tendency for each condition and entered into a repeated-measures ANOVA by condition (T, D0, D25, D50, D100, D150). The means of these medians are displayed in Fig. 1, showing a robust elevation of SRT for distractors presented simultaneously with, or within 50 ms after the target. The overall effect of condition was highly reliable ($F_{5,65} = 13.170; P < 0.0001$). Planned contrasts comparing each distractor condition against the baseline (T) confirmed a reliable RDE for the first three delay conditions (D0: $F_{1,13} = 9.283; P < 0.009$; D25: $F_{1,13} = 27.598$;

$$P < 0.0001; D50: F_{1,13} = 35.110; P < 0.0001), \text{ but not for the last two (D100: } F_{1,13} = 3.497; P = 0.084; D150: F_{1,13} = 0.801; P = 0.387). \text{ Numerically, the maximum RDE arose with a distractor delay of 50 ms.}$$

Saccadic inhibition analysis

The presence of saccadic inhibition was assessed via the character of the changes in the baseline SRT histogram induced by distractor presentation. Saccadic inhibition manifests as a clear dip in saccadic frequency, occurring at a relatively constant interval after the responsible visual change. In a prosaccade task with fixation overlap, as used here, Reingold and Stampe (2002) estimated the peak of inhibition to occur at 91.3 ms.

Our analysis had several stages. For each participant, for each condition, a percentage frequency histogram of SRTs was created, with a bin-width of 2 ms (the maximum temporal resolution of EyeLink II). A nine-point moving-window-average smoothing function was then applied. The smoothed histograms were then averaged, bin-by-bin, across participants, for each condition. The mean SRT histogram for the baseline (T) condition is shown in the top panel of Fig. 2. The five lower panels show the difference histograms for each of the distractor conditions, computed via the bin-by-bin subtraction of the baseline histogram from the mean histogram for that condition. For each difference histogram, the interval between the distractor onset and the histogram minimum is reported below the grey shaded area.

A discrete dip is present in each difference histogram, occurring later as the distractor onset is progressively delayed. The dip is thus roughly time-locked to the distrac-
tor onset: the estimated interval to maximum inhibition ranges between 87 and 113 ms. In reality, the timing of the dip is even more stable than this. Because the difference histograms in Fig. 2 represent absolute differences, a bias arises, whereby the nadir of each dip is pulled towards the peak of the baseline distribution. This causes an overestimation of the latency of dips arising before the peak of the baseline distribution, and an underestimation of those arising after. If the plotted differences are normalised as percentages of the baseline, the estimated latencies all lie between 88 and 101 ms. In the D50 condition, in which the maximal RDE was observed, the normalised dip represents a 35% reduction in the baseline frequency.

The patterns in Fig. 2 provide compelling evidence that saccadic inhibition is responsible for the RDE. However, the difference histograms per condition are somewhat noisy. To better characterise the timing of the dip, the difference histograms for each condition were temporally aligned to distractor onset, then averaged bin-by-bin to create a grand mean difference histogram. This histogram, shown as Fig. 3, bears the hallmarks of saccadic inhibition: saccadic frequency drops reliably below baseline levels by 67 ms after distractor onset, reaching its nadir at 93 ms, and persisting below baseline levels until 125 ms, with a phase of relative elevation thereafter.

Discussion

The present study firstly replicates the RDE, confirming that the onset of a distractor increases SRTs compared to unilateral target presentation (cf. Braun and Breitmeyer 1990; Ross and Ross 1980, 1981; Walker et al. 1995; Weber and Fischer 1994). The size of the RDE was related to the timing of the distractor, being pronounced when the distractor was presented simultaneously with, or within 50 ms after target onset, but not reliable when the distractor was delayed by 100 ms or more. Our results correspond well to those of Walker et al. (1995), with a minor difference being that our RDE was numerically greatest for a distractor presented with 50 ms delay, rather than at simultaneity. This pattern falls between that of Walker et al. (1995) and Ross and Ross (1980, 1981), since the latter authors found that the RDE was still large with a distractor delay of 100 ms.

Our main purpose, however, was to assess whether the effect of remote distractors is consistent with the character of saccadic inhibition. As predicted by the SIH, a clear dip relative to the baseline SRT distribution was present in all five distractor conditions, emerging within ~70 ms of distractor onset and returning to baseline levels within 125 ms. Our best estimate for the latency to the peak of inhibition arises, whereby the nadir of each dip is pulled towards the peak of the baseline distribution. This causes an overestimation of the latency of dips arising before the peak of the baseline distribution, and an underestimation of those arising after. If the plotted differences are normalised as percentages of the baseline, the estimated latencies all lie between 88 and 101 ms. In the D50 condition, in which the maximal RDE was observed, the normalised dip represents a 35% reduction in the baseline frequency.

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was 93 ms, closely comparable to the 91.3 ms estimated by Reingold and Stampe (2002), using a similar baseline task (prosaccade, overlap fixation). This temporal equivalence arose despite large differences in the responsible visual events, which in their task was a flash of two-thirds of the screen, and in ours was the onset of a small dot in the unattended field. The relative saliences of the events, however, may have been reflected in the degree of inhibition, which was 89% of the baseline frequency for Reingold and Stampe’s flash, but only 35% of baseline for our D50 condition, in which the RDE was maximal.

One major implication of these findings is that the proximal factor governing the RDE is not the temporal relationship between target and distractor (Ross and Ross 1980, 1981; Walker et al. 1995), but the more complex relationship between the distractor onset and the baseline SRT distribution. The characteristics of baseline SRT distributions may thus be key to understanding differences between RDE results from different studies. As noted in the Introduction, Walker et al. (1995) found a maximal RDE with a simultaneous distractor in the context of a relatively early baseline SRT distribution, whilst Ross and Ross (1980) observed large effects with a distractor delay of 100 ms in the context of a relatively late baseline distribution. In our study, the RDE was maximal with a distractor delayed by 50 ms, presumably because the maximum saccadic inhibition, following this distractor by 103 ms, coincided with the peak of the baseline SRT around 150 ms (see Fig. 2). To extrapolate from our data, one would expect that the earliest distractor that could affect the SRT distribution should precede the left tail of that distribution by ~125 ms; conversely, for a late distractor to have an influence, it should onset more than 67 ms before the right tail of the baseline distribution falls to zero. Whether or not a distractor’s effect on the SRT distribution will translate into a reliable RDE is less predictable, and may depend on further factors including statistical power and the measure of central tendency chosen. Notably, in our data, the RDE was not reliable with distractor delays of 100 or 150 ms, despite demonstrable dips in the SRT distributions for these conditions.

These considerations imply that the effects of distractors may be characterised more informatively by the shape of SRT distributions, than by any single measure of central tendency. For instance, a late distractor affecting the right tail of the baseline SRT distribution might increase the mean SRT, but leave the median unchanged; on the other hand, the reshaping of SRT distributions by saccadic inhibition may render calculation of the mean SRT inappropriate, as the resulting distributions may be far from normal. An obvious example is the bimodality that will tend to emerge when distractor onset precedes the peak of the baseline SRT distribution by around 100 ms. This was precisely the pattern obtained by Reingold and Stampe (2002) when they timed their flash to precede the median SRT by (on average) 100 ms. In the present study, we observed similar instances of bimodality in individual participants, most commonly in the D50 and D100 conditions, when the distractor happened to precede the peak of their baseline SRT distribution by around 100 ms. With this in mind, it is possible to pinpoint likely examples of the same effect within the prior literature on remote distraction. For instance, Braun and Breitmeyer studied the effects of re-introducing the fixation stimulus in a gap paradigm, and observed a split in the SRT distribution with the separation point roughly 100 ms after the return of fixation (Braun and Breitmeyer 1990, p. 321, Figure 3). A similar split was obtained by Weber and Fischer (1994), 100 ms after the presentation of a simultaneous distractor.

The identification of the RDE with saccadic inhibition is a clear step forward in the eye movement literature. Integration of evidence and theories from these two previously separate sub-litatures should accelerate the quest for a full account of saccadic inhibition, which must necessarily encompass what is known about the RDE. Both effects have been hypothesised to depend upon inhibitory interactions in the intermediate layer of the superior colliculus (see Findlay and Walker 1999; Reingold and Stampe 2002), marking this as the likely locus of their common neurophysiological basis. Distractor onset may stimulate saccade build-up neurons coding for the distractor location, and/or fixation neurons if the distractor is not too eccentric (Findlay and Walker 1999; Reingold and Stampe 2002, 2003). This distractor-related activity would impede target-directed saccades, either directly by lateral inhibition of the build-up neurons coding for the target location (e.g. Reingold and Stampe 2002, 2003), or indirectly by stimulating brainstem omnipause neurons, antagonistic to saccade execution (Findlay and Walker 1999). Our data do not distinguish between these proposed mechanisms, and they need not be mutually exclusive. However, our findings do imply that distractors can affect saccade generation only within a lim-
imated time-window post-onset, here estimated across trials as \( \sim 67–125 \) ms. The lower limit may be imposed by minimum neural delays in the pathway of inhibition, whilst the upper limit presumably reflects the maximum persistence of inhibitory activity. Saccades in preparation within this time-window are vulnerable to distraction.

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References


