Saccadic Inhibition in Voluntary and Reflexive Saccades

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Abstract

■ The present study investigated saccadic inhibition in both voluntary and stimulus-elicited saccades. Two experiments examined saccadic inhibition caused by an irrelevant flash occurring subsequent to target onset. In each trial, participants were required to perform a single saccade following the presentation of a black target on a gray background, 4° to the left or to the right of screen center. In some trials (flash trials), after a variable delay, a 33-msec flash was displayed at the top and bottom third of the monitor (these regions turned white). In all experimental conditions, histograms of flash-to-saccade latencies documented a decrease in saccadic frequency, forming a dip, time-locked to the flash and occurring as early as 60–70 msec following its onset. The fast latency of this effect strongly suggests a low-level, reflex-like, oculomotor effect, which was referred to as saccadic inhibition. A novel procedure was developed to allow comparisons of saccadic inhibition even across conditions, which in the absence of a flash (no-flash trials) produce dissimilar saccadic reaction times (SRTs) distributions. Experiment 1 examined the effects of the fixation stimulus on saccadic inhibition by contrasting three conditions: a gap condition (fixation stimulus disappeared 200 msec prior to target onset), a step condition (offset of the fixation stimulus was simultaneous with target onset), and an overlap condition (the fixation stimulus remained on for the duration of the trial). The overlap condition produced substantially stronger saccadic inhibition, relative to the gap and the step conditions. Experiment 2 contrasted the saccadic inhibition effect obtained for prosaccades (saccades aimed at the target) with the effect obtained for antisaccades (i.e., saccades aimed away from the same target). The onset of saccadic inhibition was earlier, and its magnitude was stronger, for antisaccades, relative to prosaccades. The plausibility that the superior colliculus is the neurophysiological locus of the saccadic inhibition effect was explored. ■

INTRODUCTION

In viewing natural visual scenes, observers produce high-velocity eye movements called saccades, which act to direct the high-acuity foveal region of the visual system (the central two degrees of visual angle) to different areas of the visual field. Natural visual scenes include both structured, unchanging stimuli that are foveated by scanning saccades, as well as changing or sudden onset stimuli that may trigger the ''visual grasp reflex'' (Ingle, 1973; Hess, Burgi, & Bucher, 1946), which is a reflex-like saccadic orienting response (henceforth, stimulus-elicited saccades). In contrast to the reflexive nature of stimulus-elicited saccades, scanning saccades are considered voluntary (e.g., Yarbus, 1967) but may be produced by highly efficient and automated procedures (e.g., in the context of tasks such as reading and visual search). Given the mixture of changing and unchanging stimuli in natural environments (Sommer, 1997), a potentially important interaction between these two types of stimuli concerns the possible effects of irrelevant visual events (such as a flash or flicker) on scanning saccades. Reingold and Stampe (1997, 2000, in press) devised a paradigm that attempted to study this interaction in the context of complex visual tasks. For example, while participants were reading for comprehension, a text screen was replaced for 33 msec by a black screen, at intervals that varied randomly between 300 and 400 msec, resulting in the subjective experience of a flicker. This study documented a decrease in saccadic frequency time-locked to the flicker and occurring as early as 60–70 msec following the onset of the flicker. It was argued that the fast latency of this effect, which approaches the limits imposed by neural delays in the visual and saccadic systems, strongly suggests a low-level, reflex-like, oculomotor effect, which was referred to as saccadic inhibition. Reingold and Stampe (2000) suggested that the neurophysiological locus of the saccadic inhibition they observed may be related to inhibitory processes in the superior colliculus (SC) that were documented by Munoz and colleagues (e.g., Dorris, Paré, & Munoz, 1997; Dorris & Munoz, 1998; Munoz & Wurtz, 1992, 1993a, 1993b, 1995a, 1995b; Munoz & Istvan, 1998; see Munoz, Dorris, Paré, & Everling, 2000 for a review). Based on the available neurophysiological evidence concerning the latencies of neural activity following visual input in each of several saccadic control structures (e.g., Lamme & Roelfsema, 2000; Schmolensky et al., 1998; Schall, 1991; Yin & Mountcastle, 1977; Goldberg & Wurtz, 1972), Reingold

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and Stampe argued that the SC, which receives visual input in as little as 35–47 msec (Rizzolatti, Buchtel, Camarda, & Scandolara, 1980), is the primary candidate for mediating the fast saccadic inhibition onset latencies (60–70 msec).

If saccadic inhibition is a low-level oculomotor effect, then it should be possible to demonstrate this effect across a wide range of saccadic tasks. Accordingly, the major goal of the present study was to investigate saccadic inhibition in both voluntary and stimulus-elicited saccades. For this purpose, a discrete trial version of the paradigm introduced by Reingold and Stampe (2000, in press) was developed. It was hoped that the discrete trial version of the saccadic inhibition paradigm would permit an investigation of this phenomenon under conditions that are more similar to the experimental paradigms typically used in behavioral and neurophysiological studies of the saccadic system. This would enable more direct comparisons to results from such studies and allows a preliminary examination of the hypothesis concerning the involvement the SC in the saccadic inhibition effect. Specifically, we investigated saccadic inhibition of stimulus-elicited saccades in the gap paradigm and saccadic inhibition of voluntary saccades in the antisaccade paradigm. We will first review these experimental paradigms, then briefly summarize relevant neurophysiological findings, and present an overview of the present experiments.

The Gap Paradigm and the Antisaccade Paradigm

To date, the majority of behavioral and neurophysiological studies of the saccadic system have been focused on stimulus-elicited saccades (see Findlay & Walker, 1999 for a review and discussion). One of the most influential experimental manipulations used to study stimulus-elicited saccades is the gap paradigm, which reveals the strong influence of visual events at the fixation location on saccadic reaction times (SRTs). Specifically, introducing a temporal gap between the disappearance of a fixated stimulus and the appearance of a saccadic target has been shown to result in faster SRTs. This finding, commonly referred to as the gap effect, was first reported by Saslow (1967) who manipulated the timing of fixation point offset and the onset of a saccade target. When the fixation stimulus offset occurred 100–200 msec before the target onset (henceforth, the gap condition), SRTs were substantially faster than when fixation point offset and target onset occurred simultaneously (henceforth, the step condition). SRTs in the step condition were in turn faster than when the fixation stimulus remained on after target onset (henceforth, the overlap condition). The gap effect is very robust and was replicated in many subsequent studies (e.g., Forbes & Klein, 1996; Walker, Kentridge, & Findlay, 1995; Kingstone & Klein, 1993a; Ross & Ross, 1980, 1981). It is well established that a component of the gap effect is attributable to a warning signal effect (e.g., Ross & Ross, 1980, 1981). Specifically, the visual offset in the gap condition can be used by participants to predict the impending appearance of the target. In order to equate conditions (i.e., gap, step, overlap) in terms of the participants' ability to predict target appearance, several studies presented a warning tone prior to target onset and demonstrated a smaller magnitude but significant gap effect (e.g., Forbes & Klein, 1996; Reuter-Lorenz, Oonk, Barnes, & Hughes, 1995). Furthermore, the gap effect is due in part to saccades with SRTs of 100 msec or less that are often observed in the gap condition (e.g., Kingstone & Klein, 1993b; Fischer & Ramsperger, 1984). Such saccades were termed express saccades (Fischer & Boch, 1983, Fischer & Ramsperger, 1984) and are produced with a latency that is thought to approach the limits imposed by delays in the visual and saccadic system (Paré & Munoz, 1996; Fischer & Weber, 1993).

Similar to the importance of the gap paradigm for the study of stimulus-elicited saccades, the antisaccade task has proven to be invaluable for the study of voluntary saccades (see Everling & Fischer, 1998 for a review). Hallett (1978) introduced the antisaccade paradigm in order to investigate the ability of participants to suppress a reflexive saccade toward a sudden-onset peripheral stimulus (referred to as a prosaccade), and to voluntarily direct a saccade of equal amplitude to the opposite direction (referred to as an antisaccade). Studies have demonstrated that antisaccades display longer SRTs, more variability in amplitude, and lower peak velocities than prosaccades (e.g., Goldring & Fischer, 1997; Fischer & Weber, 1992, 1997; Smit, van Gisbergen, & Cools, 1987; Hallett, 1978; Hallett & Adams, 1980). Participants erroneously generate prosaccades in a significant number of antisaccade trials, and the frequency of such prosaccade errors is higher when a gap condition is implemented by removing the central fixation target prior to the onset of the peripheral stimulus (e.g., Fischer & Weber, 1992, 1997).

A number of recent studies have compared the magnitude of the gap effect between reflexive saccades and antisaccades. In general, most studies demonstrated a significant gap effect for antisaccades, which was, however, smaller than that found for stimulus-elicited saccades (Craig, Stelmach, & Tam, 1999; Forbes & Klein, 1996; Reuter-Lorenz et al., 1995; Fischer & Weber, 1992; but see Reuter-Lorenz, Hughes, & Fendrich, 1991 for a nonsignificant trend in the same direction). In addition, several studies (Craig et al., 1999; Abrams, Oonk, & Pratt, 1998; Forbes & Klein, 1996) demonstrated gap effects for voluntary saccades made in response to an auditory signal (a tone or a verbal command) rather than a visual signal (i.e., target appearance). Importantly, as was the case for stimulus-elicited saccades, the gap effect for voluntary saccades (i.e., antisaccades or saccades in response to auditory signals) was significant even when a warning tone was presented predicting the impending

appearance of the signal to saccade (Craig et al., 1999; Abrams et al., 1998; Forbes & Klein, 1996; Reuter-Lorenz et al., 1995). This indicates that the warning signal component of the gap effect cannot totally account for the gap effect found in either voluntary saccades or stimulus-elicited saccades.

The Role of the Superior Colliculus in Generating and Inhibiting Saccades

In order to evaluate the hypothesis proposed by Reingold and Stampe (2000, in press) concerning the central involvement of the SC in the saccadic inhibition effect, it is important to briefly review the relevant neurophysiological literature. The SC, located in the midbrain, plays a central role in the saccade control network that generates stimulus-elicited saccades (for reviews see Munoz et al., 2000; Moschovakis, Scudder, & Highstein, 1996; Sparks & Hartwich-Young, 1989; Wurtz & Goldberg, 1989). The SC is uniquely situated to provide fast oculomotor responses to visual inputs as it receives direct retinal input, and collicular output directly activates the saccade generator in the brainstem. The intermediate layer of the SC contains several types of premotor cells (Munoz & Wurtz, 1995a, 1995b): burst neurons, which fire strongly just before and during saccades but are otherwise inactive; buildup neurons, which show increasing activity before saccades and may also fire strongly during saccades; and fixation neurons, which are active during fixations but pause just before and during saccades. These fixation neurons are concentrated in the fixation zone in rostral pole region of the SC (Munoz & Wurtz, 1993a, 1993b). Pharmacological studies (e.g., Munoz & Wurtz, 1992, 1993b; Hikosaka & Wurtz, 1985a, 1985b) and microstimulation studies (Paré, Crommelinck, & Guitton, 1994; Munoz & Wurtz, 1993b; Munoz & Istvan, 1998) have documented a pattern of mutual inhibitory connections that may prevent the fixation neurons and burst neurons from being active at the same time, thus forcing the SC to switch quickly between saccade (burst neurons active) and fixation (fixation neurons active) states. Furthermore, it has been shown that SRT is negatively correlated with the activity of buildup neurons (i.e., higher activity levels lead to shorter latencies), and that SRT is positively correlated with the activation of fixation neurons (i.e., higher activity levels lead to longer latencies) (see Munoz et al., 2000 for a review).

Neurons in the intermediate SC have both a visual receptive field and a movement field. The movement field is the set of saccade directions and amplitudes that a neuron fires to command, which typically moves the eye to a small region of the visual field. The visual receptive field and movement field typically overlap and the combination of these has been called the neuron's response field (Dorris et al., 1997). The foveal area of the visual field is represented at the rostral pole of the SC (i.e., at the fixation zone), and stimulation of the intermediate layer of the SC in this area evokes small saccades (Robinson, 1972) or prevents the production of saccades (Munoz & Wurtz, 1993a, 1993b). The peripheral visual field is represented in the caudal SC, and larger saccades are evoked by stimulation of this region. Saccade (burst and buildup) neurons with distant response fields inhibit each other, but saccade neurons also have an excitatory effect on nearby saccade neurons (Meredith & Ramoa, 1998; Munoz & Istvan, 1998; McIlwain, 1982). Models using this pattern of excitation of nearby neurons, and inhibition of distant neurons, produce a winner-take-all competition to select between distant saccade targets (van Opstal & van Gisbergen, 1989a, b). This connectivity also contributes to the merging of activity that leads to the global effect, in which saccades to two closely spaced visual targets tend to land midway between the targets (Edelman & Keller, 1998; Ottes, van Gisbergen, & Eggermont, 1984; Findlay, 1982).

The intermediate SC also receives projections from several cortical regions that are involved in saccadic control (for a review see Schall, 1997): the frontal eye fields (FEFs) (Segraves & Goldberg, 1987), the supplementary eye fields (SEFs) (Shook, Schlag-Rey, & Schlag, 1990), the lateral intraparietal area (LIP) (Paré& Wurtz, 1997; Lynch, Graybiel, & Lobeck, 1985), and the dorsolateral prefrontal cortex (DPC) (Shook et al., 1990; Stanton, Goldberg, & Bruce, 1988; Fries, 1984; Leichnetz, Spencer, Hardy, & Astruc, 1981). These projections may serve to suppress unwanted saccades and to command saccades by imposing the desired pattern of activation onto the intermediate layers of the SC. The FEF connections are known to achieve this by a pattern of inhibition and excitation of the intermediate SC (Schlag-Rey, Schlag, & Dassonville, 1992). Movement neurons in the FEFs have excitatory connections to neurons in the intermediate SC that produce saccades that are similar in amplitude and direction to those elicited by the FEF neurons that project to them. Other SC neurons that fire during saccades that are incongruent to those that the FEF neuron fires for will be inhibited by the activity of that FEF neuron (Schlag-Rey et al., 1992; Stanton et al., 1988). Another control pathway to the SC is via the basal ganglia. Both the FEF and SEF project to the caudate nucleus, which sends inhibitory projections to the substantia nigra pars reticulata, which in turn sends inhibitory projections to the intermediate SC (for a review see Hikosaka & Wurtz, 1989).

For the present purpose, it is important to focus on the potential role of the SC in mediating performance in the gap paradigm and in the antisaccade paradigm. Recent physiological work has provided strong evidence that the gap effect and the production of express saccades are at least partially mediated by neural activity in the SC. First, lesions of the SC abolish express

saccades in monkeys (Schiller, Sandell, & Maunsell, 1987). Second, several recent studies employed recordings of neural activity from cells in the intermediate SC of monkeys during the generation of saccades in the gap paradigm (e.g., Edelman & Keller, 1996; Paré & Munoz, 1996; Dorris & Munoz, 1995, Dorris et al., 1997). These studies reported that in the gap condition, in which the offset of the central fixation stimulus occurs prior to the onset of the peripheral stimulus, activity of fixation neurons decreases during the gap period (Dorris & Munoz, 1995; Dorris et al., 1997) potentially representing a disengagement of ocular fixation (Dorris & Munoz, 1995; Sommer, 1994; Kingstone & Klein, 1993a; Tam & Stelmach, 1993; Munoz & Wurtz, 1992, 1993b; Reuter-Lorenz et al., 1991). At the same time, presaccadic activity of buildup neurons increases during the gap period preceding target appearance (Dorris et al., 1997; Munoz & Wurtz, 1995a). This increased activity may indicate advanced oculomotor preparation, and possibly reduced inhibition from the fixation neurons whose activity decreases during this period (Dorris et al., 1997; Paré & Munoz, 1996; Kowler, 1990; Becker, 1989). Together, the increased activity of the buildup neurons and the decreased activity of the fixation neurons contribute to the faster SRTs obtained in the gap condition. Furthermore, the increase in presaccadic activity of buildup neurons during the gap period is likely to be responsible for the increased probability of express saccades in the gap condition (Dorris et al., 1997). These saccades are thought to be triggered directly by visual input to the buildup neurons caused by the onset of the saccade target. When presaccadic activity in the buildup neurons is high enough, this additional visual input may cause the total activity to reach the threshold required to produce a saccade immediately (Dorris et al., 1997; Edelman & Keller, 1996).

The role of the SC in mediating performance in the antisaccade paradigm is more complex. There is strong evidence for the involvement of cortical saccade control structures, including the FEF, SEF, and DPC, in the generation of correct antisaccades. Patients with lesions in these cortical regions are very impaired in generating antisaccades, and instead, produce prosaccade errors in the antisaccade condition (e.g., Pierrot-Deseilligny, Rivaud, Gaymard, & Agid, 1991; Fukushima et al., 1988; Lasker, Zee, Hain, & Folstein, 1987; Guitton, Buchtel, & Douglas, 1985; see Everling & Fischer, 1998 for a review). Furthermore, the involvement of the FEF and SEF in mediating correct antisaccades was clearly demonstrated by studies that recorded neural activity in monkeys during the generation of antisaccades (Everling & Munoz, 2000; Schlag-Rey, Amador, Sanchez, & Schlag, 1997; Amador, Schlag-Rey, & Schlag, 1996), by positron emission tomography (PET) studies (Sweeney et al., 1996; O'Driscoll et al., 1995) and by event-related potential (ERP) (Everling, Spantekow, Krappmann, & Flohr, 1998) studies.

Several recent studies have also begun to elucidate the role of the SC in mediating performance in the antisaccade paradigm. Specifically, saccade neurons in the SC, with response fields corresponding to the required saccadic direction and amplitude, show activity in both prosaccade and antisaccade trials, with greater activity in the prosaccade task than in the antisaccade task (Everling, Dorris, Klein, & Munoz, 1999). In addition, both direct and indirect control pathways from the FEF and the SEF to the SC may modulate presaccadic activity (i.e., preparatory set), depending on the task instructions, in order to generate either a prosaccade or an antisaccade (Everling et al., 1999; Everling & Munoz, 2000). Finally, it has been shown that high prestimulus activity in SC saccade neurons with response fields corresponding to the visual stimulus often result in the execution of erroneous prosaccades during the antisaccade trials (i.e., prosaccade errors) (Everling, Dorris & Munoz, 1998).

Overview of the Present Experiments

The findings reviewed above indicate that the SC is instrumental in the preparation and execution of both voluntary and stimulus-elicited saccades. If the SC is also the neurophysiological locus of saccadic inhibition, as proposed by Reingold and Stampe (2000, in press), it should be possible to demonstrate saccadic inhibition for both voluntary and stimulus-elicited saccades. For this purpose, a discrete trial version the paradigm introduced by Reingold and Stampe (2000, in press) was developed. Specifically, in each trial in the present experiments, a target stimulus was abruptly presented 4° to the left or to the right of the center of the screen, and the participants were required to generate a single saccade either toward or away from this stimulus. Target and fixation stimuli were presented in black on a gray background. In some trials (flash trials), following a variable delay from the target onset, a flash was displayed at the top and bottom third of the monitor by changing the color of these regions from gray to white for 33 msec. Performance in flash trials was contrasted with performance in trials without a flash (no-flash trials). Experiment 1 explored the saccadic inhibition in the gap paradigm, while in Experiment 2 this effect was examined in the antisaccade paradigm.

Reingold and Stampe (2000, in press) demonstrated that when reading and visual search were used as the saccade-generating tasks, maximum saccadic inhibition occurs approximately 100 msec following the onset of the visual change (i.e., the flash in the present experiments). If the time course of saccadic inhibition is similar in the present procedure, then SRT histograms should show a dip (i.e., a decrease in saccadic frequency) around SRTs corresponding to saccades made approximately 100 msec after the flash onset. However, the timing of the flash onset relative to the target onset is

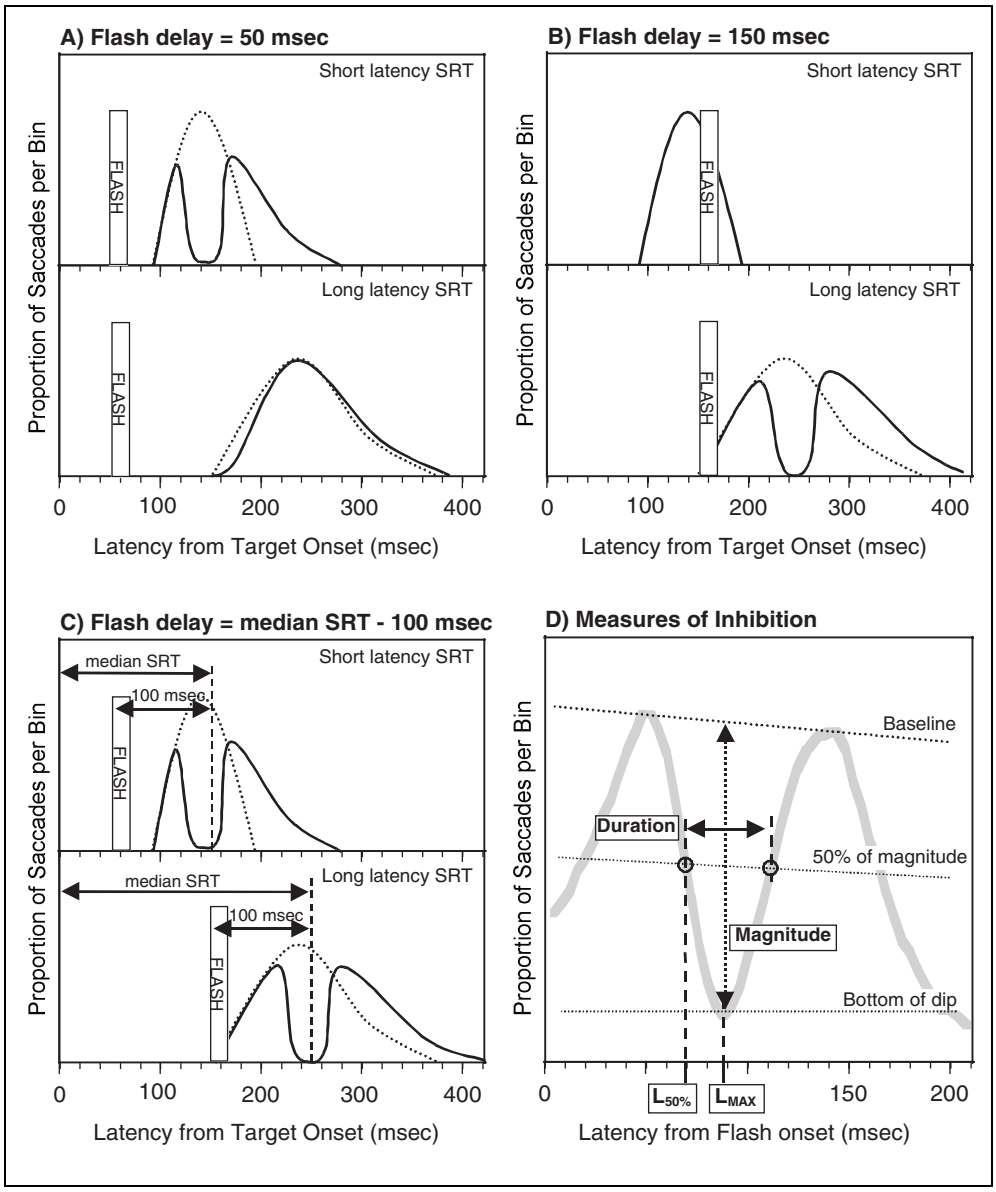

Figure 1. The predicted effect of a flash on histograms of SRT (A and B), a method of determining the optimal timing of the flash (C), and measures of saccadic inhibition (D). A short flash delay of 50 msec (A) will produce saccadic inhibition (i.e., a dip) when median SRT is 150 msec (upper histogram) but will be ineffective when median SRT is 250 msec (lower histogram). In contrast, a long flash delay of 150 msec (B) will produce inhibition when median SRT is 250 msec (lower histogram), but not when median SRT is 150 msec (upper histogram). Thus, optimal flash delay should equal median SRT minus 100 msec (C). The dip in the histogram of flash-to-saccade latencies may then be analyzed to produce four measures of inhibition (D): magnitude, $L_{50\%}$, L_{MAX}, and duration; see Methods section for details. Note, the dotted line histograms in A, B, and C represent SRT histograms in the absence of a flash.

critical if a large number of saccades are to be affected by saccadic inhibition. Panels A and B of Figure 1 illustrate that whether or not dips in SRT histograms should be predicted depends on two important factors: the latency between the onset of the target and the onset of the flash (henceforth, the flash delay), and the characteristics of the SRT histogram obtained in the absence of a flash. Specifically, panel A of Figure 1 illustrates the predicted effects of a flash presented 50 msec after the target onset (i.e., flash delay of 50 msec) on a short-latency SRT distribution (upper histogram, median SRT of 150 msec) and a long-latency SRT distribution (lower histogram, median SRT of 250 msec). As shown in the figure, a 50-msec flash delay is predicted to exert maximum saccadic inhibition with the latency of 100 msec from flash onset (i.e., 150 msec from target onset). This latency is approximately equal to the median SRT of the short-latency SRT distribution, and close to the minimum latency in the long-latency SRT distribution. Consequently, a 50-msec flash delay is predicted to result in a dip in the former, but not in the latter, SRT distribution. In contrast, a flash delay of 150 msec (see panel B of Figure 1) is predicted to produce a dip in the long-latency SRT distribution (where the median SRT equals 250 msec, coinciding with the latency of maximum inhibition), and to have no impact on the short-latency SRT distribution (where the maximum saccadic latency is less than 200 msec). In other words, to maximize the saccadic inhibition produced by the flash, flash delay should equal median SRT minus 100 msec. Panel C of Figure 1 illustrates that this method of controlling flash delay should produce clear dips in both the short-latency SRT distribution (upper histogram), and the long-latency SRT distribution (lower histogram).

This dynamic method for determining the optimal flash delay was implemented for each experimental

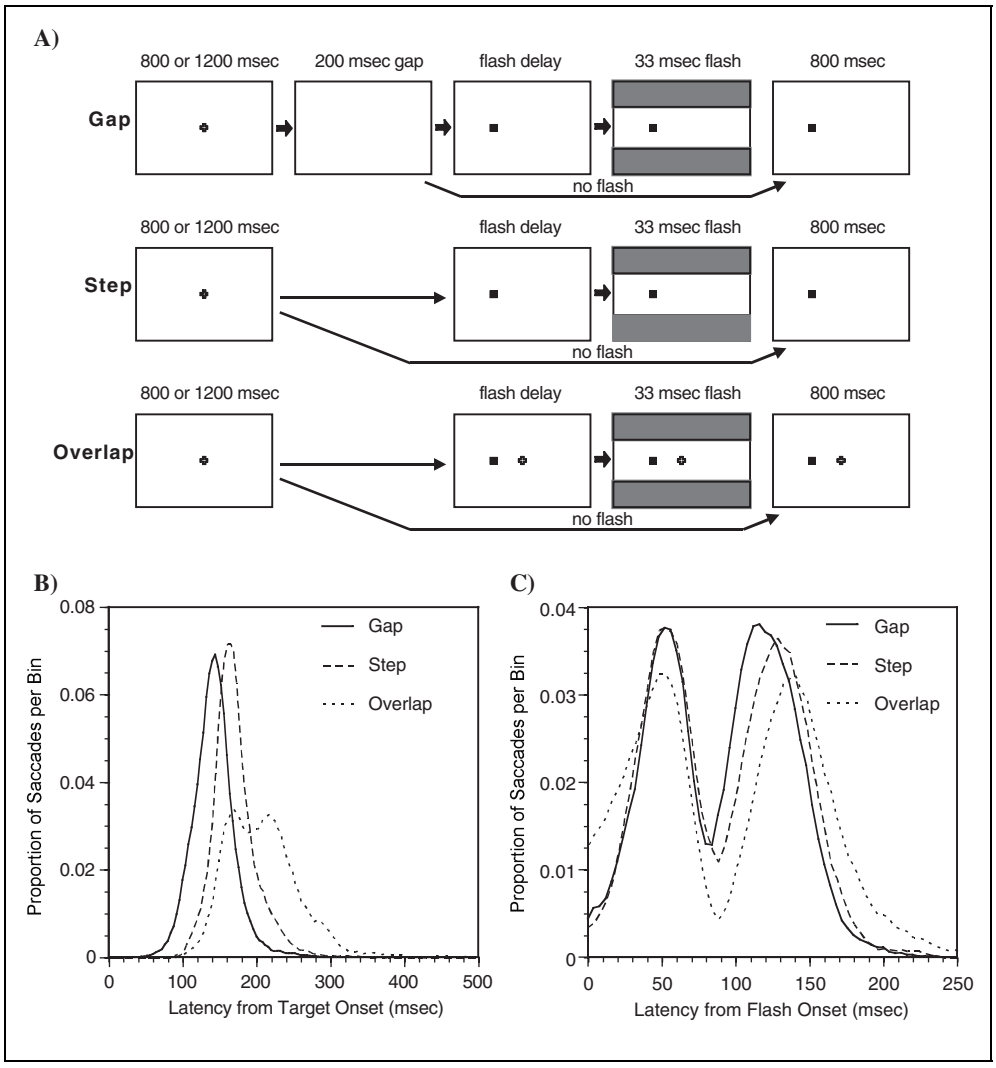

Figure 2. Flash and no-flash trial sequences (A), histograms of SRT in no-flash trials (B), and histograms of flash-to-saccade latencies (C) for the gap, step, and overlap conditions in Experiment 1.

condition in the present experiments using the observed SRT on a trial-by-trial basis. Average flash delay was set to equal the median SRT of the last 50 trials, minus 100 msec. In order to sample a broad range of flash delays and more closely approximate the saccadic inhibition paradigm introduced by Reingold and Stampe (2000, in press), the flash delay for each trial was randomly varied between 30 msec below to 30 msec above this average flash delay. Histograms of saccadic frequency by latency from the flash onset were produced from the experimental data and were analyzed to produce four measures of saccadic inhibition. These measures are illustrated in panel D of Figure 1. The magnitude of saccadic inhibition was defined as the proportion of saccades inhibited when inhibition was at its maximum (i.e., at the center of the dip). Two latency measures were computed: $L_{50\%}$ and L_{MAX}, which represent the latency from flash onset at which inhibition achieved 50% and 100% of its magnitude, respectively. In order to quantify how sustained or transient the inhibition effect was, a duration measure was defined as the temporal interval during which inhibition was greater than or equal to 50% of its magnitude.

EXPERIMENT 1

This experiment examined the effects of the fixation stimulus on saccadic inhibition by contrasting three conditions: a gap condition (in which the fixation stimulus disappeared 200 msec prior to target onset), a step condition (in which the offset of the fixation stimulus was simultaneous with target onset), and an overlap condition (in which the fixation stimulus remained for the duration of the trial). An illustration of trial sequences in these conditions is shown in panel A of Figure 2. One-third of all trials in each condition were presented without a flash.

Results and Discussion

Panel B of Figure 2 plots the SRT histograms in the no-flash trials for the gap, step, and overlap conditions. Panel C of Figure 2 plots the saccadic frequency histo-

grams by time after the flash for each condition. The effects of the flash on saccadic parameters across the experimental conditions are discussed first, followed by an examination of the saccadic inhibition measures.

Saccadic Performance

Table 1 provides the means and standard errors of the percentage of rejected trials, SRT, the saccadic parameters of amplitude, and average and peak velocity for flash and no-flash trials in each of the experimental conditions. For each of these dependent measures, a 2 × 3 within-participants ANOVA was performed, which crossed trial type (flash or no-flash) by condition (gap, step, or overlap). For SRT, a significant main effect of trial type was obtained [$F(1,9) = 69.7$, $MSE = 103$, $p < .001$], indicating that SRTs were slower for flash than for no-flash trials. The main effect of condition (gap, step, or overlap) was also significant [$F(1,9) = 140.5$, $MSE = 154$, $p < .001$]. A gap effect was demonstrated for no-flash trials, consistent with many previous demonstrations (e.g., Forbes & Klein, 1996; Walker et al., 1995; Kingstone & Klein, 1993a; Ross & Ross, 1980, 1981; Saslow, 1967). Specifically, SRT was faster in the gap condition than in the step condition, which in turn was faster than the SRT in the overlap condition (both t's > 7.33, p's < .001). A similar gap effect was also obtained for flash trials (both t's > 8.52, p's < .001). The magnitude of these gap effects did not vary across flash and no-flash trials, resulting in a nonsignificant trial type by condition interaction ($F < 1$). The only other significant effect was a main effect of condition for the peak velocity dependent measure [$F(1,9) = 8.92$, $MSE = 33$, $p < .01$]. Similar to Pratt (1998), peak velocity was slightly, but significantly, higher in the gap condition than in the overlap condition [$t(9) = 3.85$, $p < .01$]. Such a gap effect on saccadic velocity has not been consistently demonstrated (e.g., Bell, Everling, & Munoz, 2000), and consequently further studies of the relationship between the state of fixation (i.e., overlap vs. step vs. gap) and saccadic velocity are required. None of the other main effects or interactions were significant for the saccadic amplitude, or for the average and peak velocity dependent measures (all F's < 1.9, p's > .18).

Saccadic Inhibition Measures

For each experimental condition, Table 1 provides the means and standard errors of $L_{50\%}$, $L_{MAX,}$ duration, and magnitude measures of saccadic inhibition. As can be seen by an inspection of the table and the saccadic inhibition histograms plotted in panel C of Figure 2, the pattern of saccadic inhibition was relatively similar across the gap and step conditions, and both of these conditions differed substantially from the overlap condition. The duration measure is the only aspect of saccadic inhibition that discriminated between all three conditions. Specifically, duration was shorter in the gap condition than in the step condition, which in turn was shorter than the duration in the overlap condition (both t's > 2.62, p's < .05). The magnitude of saccadic inhibition was substantially stronger in the overlap condition, relative to the step and the gap conditions (both t's > 4.87, p's < .001), and did not differ across the latter two conditions ($t < 1$). Maximum inhibition (L_{MAX}) occurred 3 msec earlier in the gap condition than in the step condition [$t(9) = 2.64$, $p < .05$], and 2 msec earlier in the step condition than in the overlap condition [$t(9) = 1.87$, $p = .09$]. The latency to 50% of maximum inhibition ($L_{50\%}$) was not significantly different across conditions (all t's < 1.1, p's > .33), and was in good agreement with the estimate of Reingold and Stampe (2000, in press) that the onset of saccadic inhibition occurred as early as 60–70 msec following the display change (i.e., flicker or flash). This similarity in $L_{50\%}$ and difference in duration of the inhibition across conditions probably explains the difference observed in L_{MAX}, as a longer duration with the same $L_{50\%}$ implies a shift in the center of the dip, resulting in a delayed L_{MAX}.

The clearly visible and well-centered dip caused by saccadic inhibition was present in all conditions (see panel C of Figure 2), despite the significant differences in the corresponding SRT distributions in the no-flash trials (see panel B of Figure 2). This clearly demonstrates the effectiveness of the online procedure for computing flash delay. In particular, even though the SRT distributions in the gap and overlap conditions show very little temporal overlap and have very different widths, the timing of the flash has resulted in flash-to-saccade histograms with well-centered dips. The similarity in the onset of saccadic inhibition as measured by $L_{50\%}$, as well as its short latency, supports the hypothesis that the onset of inhibition is time-locked to the onset of the visual stimulation caused by the flash, and is reflexive in nature. Finally, the stronger and longer lasting saccadic inhibition in the overlap condition is intriguing as this condition has been shown to result in greater activation of SC fixation neurons, which in turn are hypothesized to inhibit SC saccade neurons (Dorris & Munoz, 1995; Dorris et al., 1997). This suggests that saccadic inhibition caused by the flash and the increased activity of the fixation neurons caused by the foveated fixation stimulus may interact to produce a more powerful inhibition of SC saccade neurons. This hypothesis will be elaborated in the General Discussion.

EXPERIMENT 2

Experiment 2 contrasted the saccadic inhibition effect obtained for prosaccades (i.e., saccades aimed at a sudden onset target) with the effect obtained for antisaccades (i.e., saccades aimed away from the same

Table 1. Means and Standard Errors of Saccadic Inhibition Measures and Saccadic Parameters in Experiments 1 and 2

						Rejected Trials (%)		*Average SRT (msec)*		*Amplitude (degrees)*		*Average Velocity (degrees/sec)*		*Peak Velocity (degrees/sec)*	
Experiment	*Condition*	$L_{50\%}$ *(msec)*	L_{MAX} *(msec)*	*Duration (msec)*	*Magnitude proportion*	*No flash*	*Flash*	*No flash*	*Flash*	*No flash*	*Flash*	*No flash*	*Flash*	*No flash*	*Flash*
1	Gap	67.8	86.4	36.8	0.769	5.8	8.0	142.5	166.9	3.47	3.41	124.1	122.8	221.1	216.4
		(1.1)	(2.4)	(3.7)	(0.036)	(0.03)	(0.08)	(4.1)	(5.0)	(0.26)	(0.26)	(5.9)	(5.7)	(16.0)	(14.8)
	Step	69.8	89.4	39.8	0.756	2.2	3.6	170.7	189.4	3.41	3.39	122.6	121.7	212.3	215.6
		(1.2)	(2.0)	(3.5)	(0.035)	(0.01)	(0.06)	(5.0)	(4.9)	(0.23)	(0.27)	(4.8)	(5.7)	(13.8)	(15.2)
	Overlap	69.0	91.3	46.0	0.891	3.9	8.8	208.7	231.3	3.49	3.38	123.3	120.9	211.4	211.1
		(1.3)	(1.8)	(3.4)	(0.017)	(0.03)	(0.09)	(7.9)	(6.9)	(0.30)	(0.28)	(6.0)	(5.7)	(15.8)	(15.2)
2	Prosaccade	70.0	92.6	44.0	0.873	2.3	4.5	172.8	190.6	3.35	3.32	116.9	117.7	199.1	205.4
		(1.7)	(2.6)	(3.7)	(0.041)	(0.01)	(0.01)	(8.0)	(5.5)	(0.22)	(0.23)	(4.5)	(5.0)	(10.8)	(11.9)
	Antisaccade	66.2	86.0	43.8	0.952	15.8	27.4	271.9	291.6	3.23	2.98	109.5	107.0	186.3	184.6
		(1.2)	(1.9)	(4.2)	(0.016)	(0.04)	(0.05)	(10.1)	(13.4)	(0.25)	(0.24)	(3.6)	(5.0)	(12.4)	(13.5)

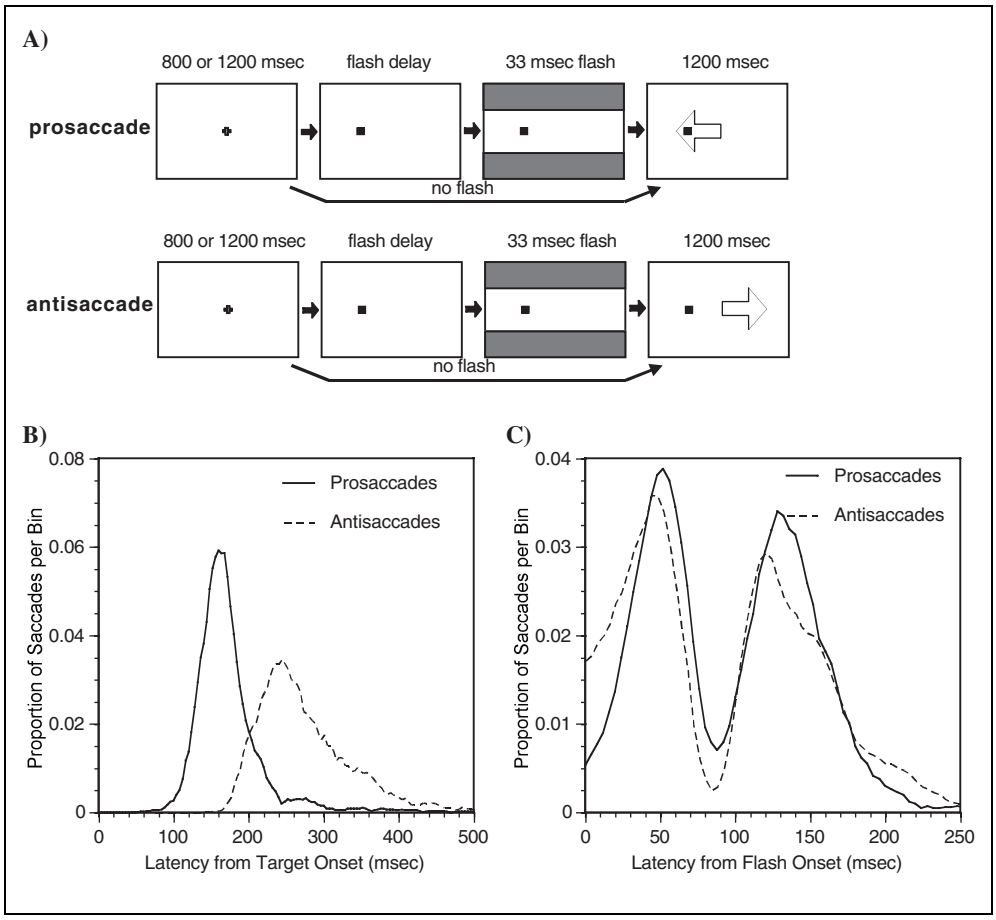

Figure 3. Flash and no-flash trial sequences with an arrow pointing to the direction of the required saccade (A), histograms of SRT in no-flash trials (B), and histograms of flash-to-saccade latencies (C) for the prosaccade and antisaccade conditions in Experiment 2.

sudden onset target). An illustration of trial sequences in these conditions is shown in panel A of Figure 3.

Results and Discussion

Panel B of Figure 3 plots the SRT histograms in the no-flash trials, and panel C of Figure 3 plots the saccadic frequency histograms following flash onset for the prosaccade and antisaccade conditions. The effects of the flash on saccadic parameters across the experimental conditions are discussed first, followed by an examination of the saccadic inhibition measures.

Saccadic Performance

For each experimental condition, Table 1 provides the means and standard errors of the percentage of rejected trials, SRT, and the saccadic parameters of amplitude, and average and peak velocity for both flash and no-flash trials. Prosaccade errors in the antisaccade condition (i.e., trials in which instead of moving in the opposite direction, participants performed a saccade toward the sudden onset stimuli) were excluded prior to the computation of these saccadic parameters and saccadic inhibition measures. The rate of prosaccade errors was 11.2% for no-flash trials, and 15.3% for flash trials [$t(9) = 1.43$, $p = .19$]. For each saccadic parameter, a 2 × 2 within-participants ANOVA, which crossed trial type (flash or no-flash) by condition (prosaccade or antisaccade), was performed. For SRT, a significant main effect of trial type was obtained [$F(1,9) = 17.0$, $MSE = 207$, $p < .01$], indicating that SRTs were slower for flash than for no-flash trials. The main effect of condition (prosaccade vs. antisaccade) was also significant [$F(1,9) = 52.0$, $MSE = 1{,}924$, $p < .001$], replicating previous results showing faster SRTs for prosaccades than for antisaccades (see Everling & Fischer, 1998 for a review). The size of this effect did not vary across flash and no-flash trials, resulting in a nonsignificant trial type by condition interaction ($F < 1$). The only other significant effects were main effects of condition for the average velocity [$F(1,9) = 8.29$, $MSE = 97$, $p < .05$) and the peak-velocity-dependent measures [$F(1,9) = 5.49$, $MSE = 515$, $p < .05$]. These effects reflect the finding that the peak and average velocity were significantly lower in the antisaccade condition than in the prosaccade condition (see also Everling et al., 1999; Amador, Schlag-Rey, & Schlag, 1998; van Gelder, Lebedev, & Tsui, 1997; Smit et al., 1987). None of the other main effects or interactions were significant for the saccadic amplitude, and

average and peak velocity dependent measures (all F's < 1.52, p's $> .24$).

Saccadic Inhibition Measures

For each experimental condition, Table 1 provides the means and standard errors of $L_{50\%}$, $L_{MAX,}$ duration, and magnitude measures of saccadic inhibition. As can be seen by an inspection of the table and the saccadic inhibition histograms plotted in panel C of Figure 3, robust saccadic inhibition was obtained for both the stimulus-elicited saccades in the prosaccade condition, and the voluntary saccades in the antisaccade condition. The magnitude of inhibition was stronger for voluntary saccades than for stimulus-elicited saccades [$t(9) = 4.54$, $p < .001$]. Furthermore, the pattern of saccadic inhibition for antisaccades appears to be shifted earlier in time, relative to the pattern of saccadic inhibition for prosaccades. In support of this observation, both $L_{50\%}$ and L_{MAX} occurred earlier for antisaccades than for prosaccades (both t's > 2.74, p's $< .05$), and there was no difference in the duration of inhibition across conditions ($t < 1$). Thus, the onset of saccadic inhibition was earlier, and its magnitude was stronger, for antisaccades as compared to prosaccades.

DISCUSSION

The most important finding to emerge from the present study is that the transient, task-irrelevant flash produced a robust saccadic inhibition of both voluntary saccades and stimulus-elicited saccades. In addition, this effect was demonstrated across conditions that vary markedly in terms of the SRT distributions they produce. As defined in the present research, across experiments and conditions, the latency to 50% of maximum inhibition averaged 68.6 msec (range 66.2–70.0 msec), the latency to maximum saccadic inhibition averaged 89.1 msec (range 86.4–92.6 msec), the magnitude of the inhibition (i.e., the proportion of saccades inhibited when inhibition was at its maximum) averaged 0.85 (range 0.76–0.95), and the duration of inhibition (i.e., the temporal interval during which inhibition was greater than or equal to 50% of its magnitude) averaged 42.1 msec (range 36.8–46.0 msec). The rapid onset of the saccadic inhibition effect reported here is in good agreement with the demonstration by Reingold and Stampe (2000, in press) that scanning saccades generated in reading and visual search are inhibited as early as 60–70 msec following the onset of a transient, task-irrelevant visual stimulus. The fact that the saccadic inhibition effect was demonstrated across such a wide range of saccadic tasks involving either voluntary saccades or stimulus-elicited saccades strongly suggests that the neurophysiological locus of this effect is a saccadic control pathway shared by most, if not all, saccades. As will be argued below, we believe that the SC is the likely locus of this effect. However, further studies are required in order to test this hypothesis.[1] In the remainder of the Discussion, we explore the potential implications of the present findings for behavioral and neurophysiological investigations of saccadic control.

Saccadic Inhibition and Saccadic Performance

Across experiments and conditions, the flash produced an average SRT 21 msec (range 18–24 msec) slower in flash trials, relative to no-flash trials. Despite this effect on SRTs, the flash did not influence other saccadic parameters, including amplitude and peak and average velocity. This pattern of findings is consistent with the view that separate and parallel processes are involved in the initiation of saccades, and in the computation of the spatial parameters of the saccade (Walker, Deubel, Schneider, & Findlay, 1997; Findlay, 1983; Findlay & Walker, 1999; Becker & Jurgens, 1979).

Based on the present results, it is predicted that a visual change, simultaneous with, or delayed from the onset of the saccade target, may induce saccadic inhibition, resulting in a slowing of SRT. Thus, it is instructive to consider experimental paradigms that may potentially produce such an effect. As a case in point, consider the remote distractor paradigm. When a saccadic target and a distractor stimulus are presented simultaneously at different locations in the visual field, a slowing of SRT occurs. This effect is referred to as the remote distractor effect. In the first study of this type, Levy-Schoen (1969) presented two potential targets simultaneously on opposite sides of the fixation point, resulting in an increase in SRT of 40 msec over that observed for a single target. In this study, participants were allowed to make a saccade to either of the two target stimuli, so it is possible that ambiguity about the identity of the target was responsible for the increase in SRT. However, several subsequent studies replicated the finding of the slowing of SRTs, even when the identity of the targets and distractors were prespecified and discriminable (e.g., Walker et al., 1995, 1997; Weber & Fischer, 1994).

If saccadic inhibition caused by the presentation of the visual change (in these studies, the distractor stimulus) 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 (henceforth, the distractor onset delay) and the characteristics of the SRT histogram obtained when a distractor is not presented. Panels A and B of Figure 1 illustrate that a distractor onset delay (i.e., in this case a flash delay) of 50 msec may produce a saccadic-inhibition effect in the case of a short-latency, but not in the case of a long-latency, SRT distribution, with the reverse pattern predicted for a distractor delay of 150 msec. A comparison of the results of studies by Ross and Ross (1980) and Walker et al. (1995) provides tentative support for such a

prediction. In the first study, Ross and Ross tested the effects of the timing of a warning signal on SRT. In this study, participants made saccades to a target 15° to the left or right. An "O" surrounding the fixation target appeared or disappeared as a warning signal for the target onset (see also Ross & Ross, 1981). This warning signal occurred before, simultaneous with, or after the saccadic target onset. The average SRT without the warning signal was 290 msec. When the onset of the warning signal was delayed by 50, 100, or 150 msec from the onset of the saccade target, SRTs were found to increase significantly. In the second study, Walker et al. presented a saccade target 4.5° or 8.5° to the right of the fixation target, and a distractor to the left of the fixation target. The distractor appeared either simultaneous with, or at several different intervals before or after the target onset. Targets were also presented without distractors to determine the baseline SRT, which was 168 msec in this study. Average SRT was significantly increased for distractor onset delays of 0 (simultaneous), 20, and 40 msec from the target onset. Importantly, SRT was not significantly increased in this study for a distractor onset delay of 100 msec, a delay that caused a large increase in SRT in the study by Ross and Ross. Thus, consistent with the saccadic inhibition prediction, in a short-latency SRT distribution (Walker et al., 1995; mean = 168 msec) slowing of SRTs required shorter distractor onset delays (0–40 msec), relative to the distractor onset delays (50–150 msec) required to demonstrate slowing of SRTs in a long-latency SRT distribution (Ross & Ross, 1980; mean = 290 msec).

The above comparison, although suggestive, should not be taken as conclusive evidence in favor of the saccadic inhibition interpretation of the slowing of SRT caused by the presentation of a nontarget stimulus (i.e., a distractor or a warning signal) observed in these studies. The Ross and Ross (1980) and Walker et al. (1995) studies differed not only in terms of the average SRT obtained but also in terms of many other task dimensions including the visual characteristics and the placement of the target and nontarget stimuli. Most importantly, the slowing of SRT may have resulted from either saccadic inhibition, a low-level reflexive oculomotor effect, or from a higher level disruption associated with the cost of perceptually encoding the distractor and discriminating it from the target. Thus, a crucial aspect of the present demonstration of saccadic inhibition is the fact that the period of decreased frequency observed in histograms of saccades generated in flash trials (i.e., the dip) was time-locked to the flash and that its time course was very consistent across participants.[2]

The Superior Colliculus and the Saccadic Inhibition Effect

In this section of the article we propose an admittedly speculative account of the observed effects in terms of the inhibitory processes in the SC that were reviewed in the Introduction. The basic hypothesis proposed is that saccadic inhibition may be a result of activity in the intermediate SC caused by the transient display change (i.e., the flash in the present experiments). There are two potential mechanisms for neural activity associated with the flash to act through inhibitory connections within the intermediate SC to reduce presaccadic activity in the buildup neurons with response fields corresponding to the required saccadic direction and amplitude. First, these saccade-related buildup neurons may be inhibited by distant buildup neurons with response fields corresponding to the area of the visual field in which the flash was displayed (henceforth, lateral inhibition; see Olivier, Dorris, & Munoz, 1999; Munoz & Istvan, 1998). Second, the visual activity associated with the flash might stimulate fixation neurons, which may inhibit presaccadic activity in buildup neurons throughout the SC (Munoz & Wurtz, 1993a, b). However, in the present experiments, the region of the visual field occupied by the flash was outside the classical fixation zone (Olivier et al., 1999; Munoz & Wurtz, 1993a) that is

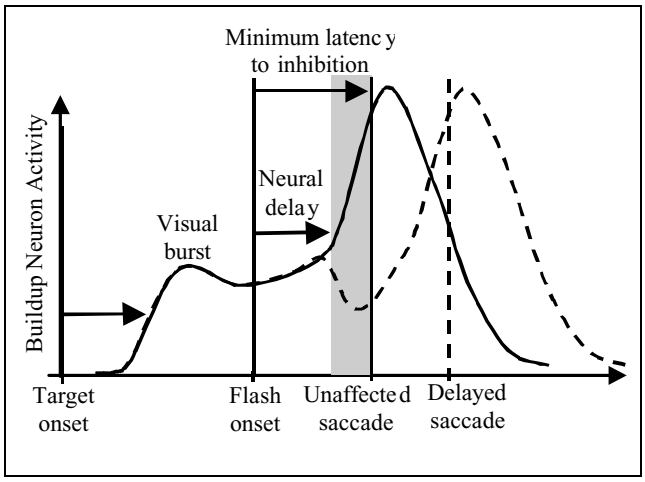

Figure 4. A hypothetical activity pattern of a buildup neuron in the SC during saccadic inhibition. Two patterns of activity are overlaid, with the solid trace indicating a saccade that escaped inhibition, and the dashed trace a saccade that was delayed by the flash. Following target onset both activity patterns overlap showing a visual burst caused by the neural activity in the buildup cell associated with the target onset. As inhibition caused by the flash begins to affect the buildup neuron activity, the patterns corresponding to the unaffected saccade and the delayed saccade begin to diverge. Vertical lines intersecting the motor bursts (i.e., saccade-related activity peaks) indicate when the saccade was detected by the eye tracker. In order to successfully inhibit a saccade, flash-related neural activity must start decreasing the buildup neuron activity prior to the "point of no return" at which the saccade-related motor burst is unstoppable. The bar represents the temporal interval between this point of no return and the beginning of the unaffected saccade. As shown in the figure, the minimum latency to inhibition equals the sum of the neural delays from the onset of the flash to the start of flash-related activity in the intermediate SC and the minimum latency at which flash-related reduction in presaccadic activity can act to delay a saccade (which must exceed the interval marked by the bar).

thought to primarily respond to visual changes near the fovea (but see Findlay & Walker, 1999; Walker et al., 1997; Gandhi & Keller, 1995, 1997 for a proposal of an extended fixation zone as far as 10° from the fovea).

Figure 4 illustrates the hypothetical pattern of activity for a buildup neuron with a response field corresponding to the required saccade. Two activity patterns are shown in the figure: The first corresponds to a saccade unaffected by the flash, and the second corresponds to a saccade that was delayed due to inhibition associated with flash-related neural activity in the intermediate SC. Following target onset, both activity patterns are similar showing a visual burst caused by the neural activity in the buildup cell associated with the target onset (e.g., Everling et al., 1999; Dorris et al., 1997; Munoz & Wurtz,1995a). At some delay from flash onset, determined by neural delays in the visual and saccadic system, the pattern of buildup neuron activity for the unaffected saccade and the delayed saccade begin to diverge as inhibition caused by the flash begins to affect buildup neuron activity. In order to successfully inhibit a saccade, flash-related neural activity must start decreasing the buildup neuron activity prior to the point at which the saccade-related motor burst is unstoppable (henceforth, the point of no return). The bar in Figure 4 represents the temporal interval between this point of no return and the beginning of the unaffected saccade as detected by the eye tracker. Thus, as shown in the figure, the minimum latency to inhibition (i.e., the onset of the saccadic inhibition dip in the histogram of saccadic frequency), equals the sum of the neural delays (from the onset of the flash to the start of flash-related activity in the intermediate SC) and the minimum latency at which flash-related reduction in presaccadic activity can act to delay a saccade (which must exceed the interval marked by the gray bar in Figure 4).

Available neurophysiological evidence can be used to provide rough estimates for each of the components of the minimum latency to saccadic inhibition. Visual latencies in the superficial SC are as low as 35–47 msec for bright flashed stimuli (Rizzolatti et al., 1980) and the delay in transmission between the superficial and intermediate SC is probably about 5–10 msec (Lee, Helms, Augustine, & Hall, 1997) for a predicted minimum latency of 40 msec for neural activity in the intermediate SC. Visual latencies of 60–70 msec were reported for buildup neurons by Munoz and Wurtz (1995a), but these longer latencies may be due to the very dim stimuli used in their study. The latency at which fixation neuron activity can act to delay saccades can be estimated from a study by Munoz and Wurtz (1993b), in which electrical stimulation of fixation neurons could delay saccades only when delivered at least 20 msec before the saccade onset. Munoz and Wurtz (1993b) also demonstrated that when the SC is stimulated during the saccade midflight alterations of saccade trajectory occur within approximately 10 msec. The latency at which lateral inhibition of buildup neurons can act to delay saccades is probably very similar (i.e., 20 msec), as the latency of inhibition of buildup neurons within the SC is similar when either fixation neurons or distant buildup neurons are stimulated (Munoz & Istvan, 1998). Thus, a latency of 20 msec is a reasonable estimate of the time from the point of no return to saccade onset (see gray bar in Figure 4). Accordingly, the estimated minimum latency of saccadic inhibition is equivalent to the visual latency in the intermediate SC (40 msec) plus the latency at which such activity can delay saccades (20 msec), for an estimated latency of 60 msec. Obviously caution must be used in comparing this estimate to the present results given the many differences across studies. Nevertheless, the estimated minimum latency of saccadic inhibition of 60 msec is consistent with the 60- to 70-msec latency to the onset of the saccadic inhibition demonstrated by Reingold and Stampe (2000, in press), and with the latency to 50% of maximum inhibition that averaged 68.6 msec in the present experiments. Taken together, these results provide support for the hypothesis that the SC is the neurophysiological locus of the saccadic inhibition effect observed in the present paradigm.

An important question is whether or not the model presented above can account for the differences in the saccadic inhibition patterns seen across the conditions employed in the present experiments. In general, the conditions in both Experiment 1 and Experiment 2 that produced the strongest saccadic inhibition are also known to involve greater inhibition in the SC (i.e., the overlap condition in Experiment 1, see Dorris & Munoz, 1995, Dorris et al., 1997) or weaker activation of SC saccade neurons (i.e., the antisaccade task in Experiment 2, see Everling et al., 1999). This indicates that inhibition of buildup neurons caused by the flash may interact with other sources of inhibition or activation that act on the same neurons. Specifically, in Experiment 1 the overlap condition produced stronger and longer lasting saccadic inhibition relative to the gap and the step conditions. This may reflect the fact that in the overlap condition, but not in the gap and step conditions, activity of the fixation neurons caused by the foveated fixation stimulus inhibits buildup neurons (Dorris & Munoz, 1995, Dorris et al., 1997) at the time when flash-related activity is hypothesized to inhibit the same neurons. Similarly, in the antisaccade condition in Experiment 2, the flash-related activity acts to inhibit buildup neurons that already display much weaker levels of activation relative to the prosaccade condition (Everling et al., 1999). Furthermore, the earlier onset of saccadic inhibition (as measured by $L_{50\%}$) for antisaccades than for prosaccades may suggest that the point of no return occurs closer to saccade initiation in antisaccades than in prosaccades. This may be related to the weaker motor burst that was observed by Everling et al. (1999) in the antisaccade condition, which is also reflected in the lower peak velocity of the

antisaccades observed both in Experiment 2 and by Everling et al. (1999).

Conclusions

Based on the present findings and the results reported by Reingold and Stampe (2000, in press), it is clear that the saccadic inhibition effect has a very fast and consistent onset latency and is present in a wide range of tasks and saccade types. These findings and the neurophysiological evidence reviewed here suggest that the likely neurophysiological locus of this effect is the SC. The present findings also support the conclusion that saccadic inhibition is a fast reflex of the oculomotor system that acts in response to sudden changes in visual input to inhibit or delay the production of saccades. Saccadic inhibition may serve to give the brain time to process the arrival of abrupt changes in visual input by delaying the execution of saccades.

The present study is an example of a growing effort to integrate behavioral and neurophysiological investigations of the saccadic system (e.g., Trappenberg, Dorris, Munoz, & Klein, 2001; Everling et al., 1999; Findlay, 1987, Findlay & Walker, 1999; Dorris & Munoz, 1995, Dorris et al., 1997; Fischer, 1987, Fischer & Weber, 1992, 1993, 1997; Forbes & Klein, 1996; Munoz & Wurtz, 1995a, 1995b; Reuter-Lorenz et al., 1995). The fine temporal resolution of the saccadic inhibition paradigm introduced in the present article is uniquely suitable for comparisons of the behavioral findings with the available neurophysiological literature. Clearly, combining the present methodology with neurophysiological research techniques is required in order to test the predictions outlined in the above discussion. Finally, employing the present paradigm to study saccadic performance in patients with lesions affecting pathways involved in saccadic control and visual attention may prove informative.

METHODS

Experiment 1

Participants

A group of 10 participants was tested. All participants had normal or corrected-to-normal vision, and were paid US$10.00 per hour.

Apparatus

The SR Research EyeLink eye tracking system used in this research has high spatial resolution (0.005°) and a sampling rate of 250 Hz (4-msec temporal resolution). The three cameras on the EyeLink headband allow simultaneous tracking of both eyes and of head position, computing true gaze position with unrestrained head motion. Only the participant's dominant eye was tracked in these studies. The EyeLink system uses an Ethernet link between the eye tracker and display computers to supply real-time gaze position and saccade event data. The on-line saccade detector of the eye tracker was set to detect saccades with an amplitude of 0.5° or greater, using an acceleration threshold of 9500°/sec^2 and a velocity threshold of 30°/sec. The eye tracker was configured to use only horizontal gaze position to detect saccades in the present study.

Participants viewed a 17-in. ViewSonic 17PS monitor from a distance of 60 cm, which subtended a visual angle of 30° horizontally and 24° vertically. The display was generated using an S3 VGA card and the frame rate was 120 Hz.

Materials and Design

There were three experimental conditions that varied in terms of the timing of the offset of the fixation target (a 0.7° cross) relative to the onset of the saccade target (a 0.7° solid square) (see panel A of Figure 2 for trial sequences of flash and no-flash trials in each condition). Fixation and saccade targets were displayed in black (5.0 cd/m^2) on a gray (20 cd/m^2) background. In some trials, a flash occurred during the trial, generated by changing the color of the top and bottom thirds of the display background from gray to white (60 cd/m^2) for 33 msec. This flash did not alter any part of the display within 4° above or below the center of fixation and saccade targets. Each participant performed four sessions of nine blocks, with 40 trials per block and three blocks per condition. The order of conditions was counterbalanced across sessions and participants. One-third of the blocks in each condition were run with no flash, for a total across sessions of 160 trials without a flash and 320 trials with a flash in each condition. In addition, at the beginning of the experiment, participants performed a block of 30 practice flash trials and 30 practice no-flash trials in each condition.

Procedure

A three-point horizontal-only calibration was performed at the start of the experiment, followed by a three-point calibration accuracy test. Calibration was repeated if the error at any point was more than 1° or if the average error for all points was greater than 0.5°. Throughout each trial, the experimenter was able to view in real time on a separate monitor the target locations overlaid with a cursor corresponding to gaze position. If the experimenter judged that the accuracy of eye tracking was degraded, the experimenter initiated a full calibration before the next screen. This occurred very infrequently.

Panel A of Figure 2 illustrates trial sequences of flash and no-flash trials in each condition. At the start of each trial, a black fixation stimulus was presented at the center of the display. The participant fixated this target

and pressed a button to initiate the trial. After a randomly selected delay of 800 or 1200 msec from the button press, the target was presented 4° to the left or right of the fixation center and remained in view for the duration of the trial. In the gap condition, the fixation stimulus disappeared 200 msec prior to target onset. In the step condition, the offset of the fixation stimulus was simultaneous with target onset. In the overlap condition, the fixation stimulus remained visible for the duration of the trial. In all conditions, the abrupt appearance of the target constituted the signal to make the saccade. In the no-flash trials in each condition, the display remained visible for 800 msec and was followed by a blank screen during the intertrial interval. In the flash trials, following a variable delay from the onset of the saccade target (the flash delay), a 33-msec flash was displayed in the top and bottom thirds of the screen. The timing of this flash was computed on-line using the SRTs from previous trials in the experiment in order to maximize the proportion of saccades that were affected by the flash. Specifically, the median SRT was computed for trials in each experimental condition (to a maximum of the 50 most recent trials). The flash delay for each trial was then computed by subtracting a random number between 130 and 70 msec from this median SRT.

Data Analysis

Saccade data was extracted on-line by the EyeLink tracker and recorded in a data file. During processing, trials with blinks that ended less than 80 msec before the onset of the saccade target were rejected. Other errors that resulted in the rejection of trials were saccades smaller than 1.0°, anticipatory saccades made before the onset of the saccade target or less than 50 msec following this signal, late saccades made more than 500 msec after the onset of the saccade target, and saccades made toward the incorrect direction. In flash trials, the trial was discarded if the saccade was made before the flash.

The remaining trials were then processed to produce measures of average SRT, amplitude, and velocity, as well as histograms of saccade frequency as a function of latency from the target onset. A histogram of saccade latency from the flash was also produced using the data from trials with a flash, which was then analyzed to produce measures of the saccadic inhibition caused by the flash. Separate histograms were compiled for each participant and condition. To maximize the temporal resolution of all histograms, a 4-msec bin width was used (i.e., the maximum temporal resolution of the eye tracker). These narrow bins resulted in noisy individual participant histograms. To reduce this noise, a seven-bin running average filter was applied to individual participant histograms, which replaced each bin with the average of itself, the three previous, and the three following bins.

Four measures of saccadic inhibition were defined, as illustrated in panel D of Figure 1: the magnitude of inhibition, the latency to maximum saccadic inhibition, the latency to 50% of maximum inhibition, and the duration of inhibition. These measures are determined for each participant and condition as described below.

The analysis begins by identifying the higher of the two peaks in the histogram by searching for the bin with the largest value. The algorithm then searches first right and then left of this peak to identify a dip followed by another peak. When multiple candidates are found, the dip and peak pair with the largest difference in bin values is chosen. If two peaks and a dip cannot be identified, no measures can be computed and the algorithm fails. The latency and values of the peaks and dip are not taken directly from the corresponding histogram bins found by the search, as the actual peak or dip may consist of a number of bins of similar value that form a broad peak or dip. Instead, a threshold is used to select an area of the peak or dip. The thresholds are offset from the maxima of the peak or minima of the dip by 10% of the difference between the lowest peak and the dip. The center of gravity of the area over the threshold (for a peak) or under the threshold (for the dip) is computed to calculate the latency, and the average of the selected bins is used to compute the value. The result of this calculation is that the latencies and values of the peak or the dip are less sensitive to single-bin noise.

The ''latency to maximum saccadic inhibition'' (L_{MAX}) is equivalent to the computed latency of the dip. To compute the ''magnitude of inhibition'' (magnitude) a reference value is required. As shown in panel D of Figure 1, this was achieved by connecting the two peaks of the histogram with a straight baseline. The magnitude of inhibition is computed as the baseline value at time L_{MAX} minus the dip value, divided by the baseline value. Next, we can compute the ''latency to 50% of maximum inhibition'' ($L_{50\%}$), which was defined as the latency from the flash onset at which inhibition first reaches 50% of its maximum strength. To minimize the effects of noise, this measure is actually computed by averaging the latency of all bins between the left peak and the center of the dip for which the magnitude of inhibition for the bin is between 33% and 67% of maximum inhibition. We can compute a similar latency for the right side of the dip. Subtracting $L_{50\%}$ from its counterpart latency on the other side of the dip gives the ''duration of inhibition'' (duration), which corresponds to the period during which inhibition remains above 50% of its maximum strength.

Experiment 2

Participants

A group of 10 participants who had not taken part in Experiment 1 was tested. All participants had normal or

corrected-to-normal vision, and were paid US$10.00 per hour for their participation.

Design and Procedure

Two experimental conditions were used (prosaccade and antisaccade), which differed only in the direction of the required saccade. Saccades were required either toward a sudden onset target in the prosaccade condition, or in the opposite direction away from the same sudden onset target in the antisaccade condition. Panel A of Figure 2 illustrates trial sequences of flash and no-flash trials in each condition. Trial sequences were identical across prosaccade and antisaccade trials. In all trials, the offset of the fixation stimulus was simultaneous with target onset (i.e., the same as in the step condition in Experiment 1). In each of the two sessions, participants performed three blocks of prosaccades and five blocks of antisaccades, with the order of conditions counterbalanced across sessions and participants. One-third of the blocks in the prosaccade condition and one-fifth of the blocks in the antisaccade condition were run with no flash. Each block consisted of 48 trials, for a total of 384 antisaccade and 192 prosaccade trials with flash and 96 trials in each condition without flash. Participants were trained on the antisaccade task for 30 trials before each session, with the training repeated until the participants made errors on less than 20% of the trials. The greater number of trials in the antisaccade condition than the prosaccade condition was required due to the increase in errors and the increase in variability of the SRTs produced in the former condition relative to the latter condition. All other details of the method were the same as in Experiment 1.

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Notes

1. Could saccadic inhibition be mediated via the omnipause neurons (OPNs)? Saccades can be prevented or even interrupted by electrical stimulation of the omnipause neurons in monkeys (Keller & Edelman, 1994) and cats (Paré & Guitton, 1994). In cats, the output of the OPNs is modulated by visual, auditory, and tactile sensory input, and very bright flashes of light can stop saccades in progress within 60 msec (Evinger, Kaneko & Fuchs, 1982). The visual responses of cat OPNs appear to arise from the superior colliculus, as the bilateral ablation of the SC removed this visual modulation (King, Precht & Dieringer, 1980). Omnipause neurons in monkeys show much weaker visual modulation: in the cat, a 3.2-cd/m^2 flash produced a 100% increase in the firing rate of the OPNs (Evinger et al., 1982), whereas in the monkey a 2-cd/m^2 LED at 10° eccentricity caused a 7% increase in the firing rate (Everling, Paré, Dorris & Munoz, 1998). Accordingly, the visual response of monkey OPNs is probably too weak to be the sole cause of the robust saccadic inhibition observed in the present paradigm. However, flash-related activation of OPNs might be a contributing factor underlying the saccadic inhibition effect.
2. In addition, to the remote distractor paradigm, further studies are required in order to compare the present paradigm with other behavioral paradigms such as the countermanding task in which participants are instructed to inhibit saccade generation when they receive a stop signal (e.g., Cabel, Armstrong, Reingold & Munoz, 2000; Hanes & Schall, 1995; Hanes & Carpenter, 1999).

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