Are microsaccades responsible for the gap effect?

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Extinguishing a fixation point shortly before, or concurrently with, the onset of a peripheral visual target reduces the latency of saccades to that target. Saslow (1967) hypothesized that this *gap effect* might occur because fixation point offsets reduce the incidence of corrective microsaccades with an associated saccadic refractory period. In the present study, a robust gap effect was obtained. However, using a Purkinje image eyetracker with 1 arcmin of resolution, we found that fixation point offsets had no effect on the occurrence of microsaccades and that the occurrence of microsaccades had no impact on the magnitude of the gap effect. Microsaccades therefore do not appear to play any part in the production of the gap effect.

Saslow (1967) discovered that when a fixation point is offset prior to or coincidently with the onset of a peripheral visual target, the latency of a saccade to the target is shorter than when no offset event occurs. Extrapolating from previous investigations of microsaccades (Cornsweet, 1956; Krauskopf, Cornsweet, & Riggs, 1960; Nachmias, 1959, 1961), Saslow proposed that this *gap effect* might occur because the offset of the fixation point reduces the incidence of fixation microsaccades just prior to the target onset. He reasoned that if such microsaccades have an associated saccadic refractory period, the lower incidence of these refractory periods would serve to shorten the average latency of a subject's saccadic response.

This hypothesis has never been tested, although many subsequent studies have been careful to refer to it (e.g., Fendrich, Hughes, & Reuter-Lorenz, 1991; Kingstone & Klein, 1993a, 1993b; Reuter-Lorenz, Hughes, & Fendrich, 1991; Reuter-Lorenz, Oonk, Barnes, & Hughes, 1995; L. E. Ross & S. M. Ross, 1980; S. M. Ross & L. E. Ross, 1981) and have yielded outcomes commensurate with it (e.g., Fendrich et al., 1991; Kingstone & Klein, 1993a, 1993b; Klein, 1978; L. E. Ross & S. M. Ross, 1980; S. M. Ross & L. E. Ross 1981; Tam & Ono, 1994; Tam & Stelmach, 1993).¹ For example, Ross and Ross discovered that onset events at fixation delay saccadic reac-

tion time (RT) but not manual RT. This finding dovetails with Saslow's theory: If an offset event at fixation reduces the probability of microsaccades, thereby facilitating saccadic RT, it follows that an onset event might increase the incidence of microsaccades, thereby increasing RT. Moreover, if one assumes that any inhibition associated with microsaccades is specific to the oculomotor system, onset events at fixation should delay saccadic responses but not responses in other modalities.

On the other hand, Reuter-Lorenz et al. (1991) have presented evidence that is inconsistent with the microsaccade hypothesis. They found that fixation offsets have no facilitatory effect on saccadic RT when subjects are instructed to direct saccadic responses away from the onset location of a peripheral target (antisaccades) but that there is a gap effect when subjects are instructed to saccade toward a peripheral target (prosaccades). Because Saslow's hypothesis predicts a refractory period that inhibits the saccade system in general, it is difficult to reconcile Saslow's theory with this outcome. This finding has, however, been the subject of some controversy. Fischer and Weber (1992) reported a gap effect for antisaccades and prosaccades alike. However, Reuter-Lorenz et al. (1995) argue that Fischer and Weber's (1992) result reflects the action of a nonspecific warning signal effect occasioned by fixation offset.

The contribution of microsaccades to the gap effect has therefore remained uncertain. Saslow's (1967) hypothesis predicts the following: (1) the refractory period due to microsaccades should lead to longer saccadic latencies on trials with microsaccades than on those without; (2) more microsaccades should occur on overlap than on gap trials; and (3) if only trials without microsaccades

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are considered, the gap effect should be reduced or eliminated. To address these interrelated predictions, we used a Purkinje image eyetracker to document the incidence of microsaccades in a gap paradigm.

METHOD

Subjects

Four naive subjects, 3 male and 1 female, with a mean age of 25 years (range, 22–30 years), were paid \$6.00/h for their participation. All subjects had normal or corrected-to-normal vision.

Equipment

Visual stimuli were presented at eye level on a Hewlett-Packard 1310A large-screen display with a fast (P15) phosphor CRT. A Purkinje image eyetracker with resolution of 1 arcmin was used to monitor subjects' eye motions. The subjects' right eye position was sampled, and the display screen refreshed, at 500 Hz. A warning tone was generated by a Wavetek Model 116 function generator. The display oscilloscope, function generator, Purkinje eyetracker, and finger key were interfaced via Data Translation analog to digital (AD) and digital to analog (DA) converters with an IBM PC AT computer which controlled stimulus presentations and response tabulation.

Stimuli and Procedure

Subjects sat alone at a table in a dark testing room with their heads positioned by a bite board, 1 m from the display screen. They were dark adapted for approximately 5 min before the beginning of each testing session. Stimuli had a luminous intensity of 10 cd/m², and were shown against a nominally black (<1 cd/m²) background. The eyetracker was calibrated at the start of each testing session, and this calibration was checked at the start of each trial block. Calibration entailed recording a subject's eye position at each of five stimulus locations: center, and 4° above, below, left, and right of center. Eyetracker gains were set so that 1 AD bit equaled 1 arcmin of eye displacement.

The appearance of an X (subtending .5° × .5°) in the center of the CRT screen signaled that a trial was about to begin. Subjects fixated this X and initiated each trial with a keypress. Following this keypress, a random time delay of 900–1,200 msec preceded target onset. On each trial the eye was sampled for a total of 1,500 msec, beginning 800 msec prior to target onset and continuing for 700 msec after target onset.

The target, a plus sign subtending .5° × .5°, was presented randomly 4° to the left or right of center. On all trials, a 1280-Hz warning tone was sounded 200 msec before target onset. The tone was turned off when the target appeared. Subjects were instructed to maintain central fixation until the appearance of a target. They were then to make a saccadic eye movement response to the target as quickly and as accurately as possible. The target remained present for 1,000 msec. The display was then extinguished, and after an intertrial interval of 500 msec an X appeared in the center of the screen signaling that the next trial was ready to begin.

There were three types of trials. In gap trials, the fixation stimulus was extinguished 200 msec before target onset. In no-gap trials, fixation was extinguished coincidentally with target onset. In overlap trials, fixation was not extinguished. Each trial type occurred equally often and at random.

Each testing session lasted about 1 h and comprised four blocks of 72 trials, for a total of 288 trials. Rest periods separated each block. All subjects received about 15 practice trials before testing.

Data Analysis

Eye records were analyzed off line. No filtering of the records was deemed necessary. Microsaccades had a distinctive profile,

consisting of a sudden shift in the horizontal and/or vertical position trace with a small but conspicuous overshoot and return to the new baseline position within 4–8 msec (see Figure 1). Because of their small size, we were not confident that microsaccades would be found reliably by an automatic detection algorithm, and we therefore chose to identify these saccades by visual inspection. Microsaccades of 2 arcmin or greater were easily detected. Microsaccade size was taken to be the difference between the presaccadic eye position and the postsaccadic position at the end of the overshoot. Overshoots associated with saccades are characteristic of Purkinje image eyetracker records and may be a tracker artifact. In any case, in the present investigation, they were useful insofar as they helped to make the microsaccades in our records distinctive. The latency of target directed saccades was assessed automatically using a velocity criterion (50°/sec). However, in all cases, the correct identification of these responses was confirmed by visual inspection of the eye record.

The latency of microsaccadic and saccadic responses was defined in relation to target onset (e.g., a saccade that occurred before target onset was recorded as having a negative RT, and a saccade that occurred after target onset was recorded as having a positive RT). Because we did not wish to exclude any potential eye movements from our analysis, we defined those smaller than 1° as microsaccades and those larger than 1° as saccades. The microsaccades that we observed (see Table 1) were characteristic of those found in previous studies (cf. Ditchburn & Foley-Fischer, 1967). If subjects executed more than one saccade in a trial, the first saccade was used during the data sort. Both the horizontal and the vertical magnitudes of microsaccades were recorded.

The data analysis was conducted as follows. Trials were excluded if there was an anticipation or direction error, or if a blink preceded a saccadic response. A saccade initiated before, or less than 100 msec after, target onset was classified as an anticipation. A saccade executed 100 msec or more after target onset and toward the target visual field was considered correct. A saccade was classified as a directional error when executed after target onset but to the nontarget visual field. Failure to execute a saccade within 700 msec was considered a miss.

Figure 1. An illustrative horizontal eye movement record for a gap trial. One second of data is shown. The left vertical line marks the time at which the fixation point was turned off. The right vertical line marks the time at which the target appeared, 200 msec later. The small discontinuities in the eye motion trace are microsaccades. The microsaccade that occurs within the gap interval (i.e., between the vertical lines) measures 4 arcmin. The leftmost microsaccade measures 30 arcmin. The large eye movement at the end of the record is the saccade to the target, which occurs 168 msec after target onset.

Table 1
Incidence and Topology of Microsaccades

Subject	% of Trials on Which Microsaccades Occurred			Mean Amplitude		Time Before Target Onset
	Overlap	No Gap	Gap	Horizontal	Vertical	
J.F.	0.0	2.1	1.1	11	13	145
G.M.	14.1	10.4	10.1	15	6	156
J.E.	0.0	0.0	0.0			
M.E.	3.2	4.4	1.1	19	6	48
Overall	4.3	4.2	3.1	15	8	116

Note—Amplitude is given in arcmin; time before target onset, in milliseconds.

RESULTS

Previous investigators have found that as the temporal gap between fixation offset and target onset lengthens, saccadic RT declines, reaching asymptote at approximately 200 msec (Saslow, 1967; L. E. Ross & S. M. Ross, 1980; S. M. Ross & L. E. Ross, 1981). Therefore, to test the hypothesis that microsaccades contribute to the gap effect, we looked for microsaccades on every trial within a critical interval that began 200 msec before target onset and ended with the occurrence of a saccadic response. An example of a gap trial with a microsaccade occurring within the critical interval is shown in Figure 1.

The incidence and topology of the recorded microsaccades are summarized in Table 1. The combined data from 4 subjects (1,090 trials) yielded just 40 microsaccades within the critical interval. In individual subjects, the incidence of microsaccades varied from 0 to 29 (0 to 12% of trials). The horizontal magnitude of the microsaccades ranged from 2 to 44 arcmin, and the vertical magnitude ranged from 0 to 27 arcmin (the vertical size of 0 indicating that some microsaccades were exclusively horizontal). The average onset time of these microsaccades was 116 msec prior to target presentation. An analysis of variance (ANOVA) revealed that the incidence of microsaccades varied significantly between subjects [$F(3,8) = 37.26, p < .0001$] but did not vary significantly between the gap conditions [$F(2,6) < 1$]. The latter finding should not be interpreted as a failure to replicate previous microsaccade investigations (e.g., Cornsweet, 1956; Nachmias, 1959, 1961). Unlike in previous studies, in which the incidence of eye movements was measured over relatively long time intervals (e.g., Cornsweet, 1956, took measurements over 45-sec intervals; Nachmias, 1959, 1961, took measurements over 30-sec intervals), we confined our analysis to a brief interval critical to the gap effect beginning 200 msec before target onset and continuing until the start of a saccadic response. Any effect of the fixation point on the frequency of microsaccades may simply not have had time to manifest itself.

To evaluate the effect of microsaccades on saccadic latencies in the overlap, no-gap, and gap conditions, we eliminated all trials with microsaccades in the interval beginning 200 msec before target onset. The data from this subset of trials were compared with the original data

set (see Figure 2). A two-way ANOVA indicated that there was a significant effect of gap condition [overlap RT > no-gap RT > gap RT; $F(2,6) = 28.36, p < .001$] but no effect of microsaccades [$F(1,3) = 2.46, p > .20$] and no interaction [$F(2,6) < 1$].

When this analysis was also conducted for the individual subjects, it yielded the same pattern of results. For Subject G.M., who had the greatest incidence of microsaccades, the overlap versus no-gap versus gap pattern was 226 > 209 > 190 msec with microsaccades included, and 224 > 209 > 188 msec with microsaccades removed, yielding a significant effect of gap condition but no effect of microsaccades. For this subject we also measured the condition mean saccadic latency of the specific trials with microsaccades (233, 209, 206) and compared these with the condition mean values of trials without microsaccades (224, 209, 188). In no case do these means differ statistically ($p > .20$).

Data on saccadic accuracy and response latency are given in Table 2. An analysis of the accuracy data shows that neither error rates [$F(2,6) = 3.24, p > .10$] nor saccadic amplitudes [$F(2,6) = 2.92, p > .10$] varied significantly between conditions. Anticipations (RT < 100 msec) accounted for 21 of the 24 response errors executed by subjects (1.89% of the total trials). The average amplitude of correct saccades was 4.2°, which is close to the optimal 4° response.

Figure 2. Saccadic latencies for overlap, no gap, and gap conditions, with and without trials containing microsaccades.

Table 2
Saccadic Accuracy and Response Latency

Subject	% Correct			Amplitude			Latency					
							Overlap		No Gap		Gap	
	Overlap	No Gap	Gap	Overlap	No Gap	Gap	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
J.F.	100.0	99.0	97.9	4.6	4.2	4.2	247	24.1	229	17.5	199	29.6
G.M.	97.7	98.9	95.2	4.5	4.1	4.2	226	35.2	209	26.6	190	38.1
J.E.	99.0	100.0	93.7	4.3	4.2	4.3	200	42.2	179	23.6	157	25.9
M.E.	96.9	97.8	97.9	4.0	4.1	3.8	227	50.3	211	52.2	207	49.4
Overall	98.4	98.9	96.2	4.4	4.2	4.1	225	38.0	207	30.0	188	35.8

Note—Amplitude is given in degrees of arc; latency, in milliseconds.

DISCUSSION

The pattern of saccadic latencies observed in the present study replicates the original work of Saslow (1967): RTs are fastest when fixation offset precedes target onset by 200 msec (gap condition), longer when fixation offset and target onset coincide (no-gap condition), and slowest when there is no offset event (overlap condition). Saslow hypothesized that fixation offset facilitates RT by reducing the incidence of microsaccades that can increase RT.

We found that saccadic latencies and the gap effect were unaffected by whether subjects produced many (Subject G.M.), few (Subjects J.F. and M.E.), or no microsaccades (Subject J.E.) measured from 200 msec before the target onset. In addition, whether and when the fixation point was offset had no effect on the incidence of microsaccades. Finally, eliminating all trials that contained microsaccades had no effect on saccadic latency for any subject in any condition. We take these findings as strong disconfirmation of the microsaccadic explanation of the gap effect.

When Saslow (1967) proposed the microsaccade hypothesis, it was known that small corrective microsaccades can occur more frequently in the presence than in the absence of a fixation stimulus (see, e.g., Cornsweet, 1956; Nachmias, 1959). There was also evidence of a refractory period following saccadic eye movements, with the latency of a second saccade increasing if the target for this saccade occurs closely in time to the initial saccade (e.g., Bartlett, Eason, & White, 1961). Our work shows, however, that for the time interval relevant to the gap effect (e.g., 200 msec prior to target onset) the occurrence of microsaccades is unaffected by the presence versus absence of a fixation stimulus. Moreover, we found that the magnitude of the gap effect was unaffected when only trials without microsaccades were evaluated. Finally, we found that for the subject with many microsaccades, the condition mean for trials with microsaccades did not differ from the condition mean for trials without microsaccades. This suggests that any refractory period associated with microsaccades must be very brief. Illustrating this point, Figure 3 shows a trial with a 7-arcmin microsaccade occurring 104 msec after target onset. The subject's saccade to the target, which has a latency of 196 msec, occurs only 80 msec after the completion of

this microsaccade. This outcome is commensurate with the possibility that microsaccades are generated at a low level in the oculomotor system (i.e., within the subcortical output stages), so that they do not interfere with the programming of goal-directed saccades.

These data appear to unequivocally disconfirm Saslow's (1967) microsaccade explanation of the gap effect. What then produces the gap effect? One hypothesis is that engaged attention inhibits the saccadic system, and that the offset of an attended stimulus (e.g., the fixation point) removes this inhibition by disengaging attention (see, e.g., Braun & Breitmeyer, 1988, 1990; Fischer & Breitmeyer, 1987; Mayfrank, Mobashery, Kimmig, & Fischer, 1986). Reuter-Lorenz et al. (1991) question this hypothesis, noting that, unlike attentional disengagement, the gap effect appears to have a premotor rather than sensory-perceptual locus. Kingstone and Klein (1993a) tested the attentional predisengagement theory (APT) directly and found that, contrary to the predictions of APT, RTs were equally facilitated by attended and unattended stimulus offsets (see also Klein, Taylor, & Kingstone, 1995; Tam & Stelmach, 1993).

Kingstone and Klein (1993a) suggest that the gap effect might be driven by two independent processes (see also Kingstone, Klein, & Taylor, 1994). One component

Figure 3. A horizontal eye movement (Subject M.E.) is illustrated. The left vertical line marks the time of fixation offset. The right vertical line marks the time of target onset, 200 msec later. A 7-arcmin microsaccade occurs 104 msec after target onset. A goal-directed saccade, with a latency of 196 msec, occurs only 80 msec after completion of the microsaccade.

is a general motor preparation (warning) effect produced by the onset of the gap. The other component is a fixation release effect that is specific to the oculomotor system. It is the first component, for example, that appears to produce the robust gap effect for antisaccades as reported by Fischer and Weber (1992). Reuter-Lorenz et al. (1995) found that the gap effect for antisaccades is reduced from 32 to 14 msec if the warning signal effects of the fixation point offset are controlled for by sounding an acoustic warning tone 200 msec before target onset in both the gap and the overlap conditions. In contrast, prosaccades show a 43-msec gap effect even with warning effects controlled.

Fendrich et al. (1991) and Reuter-Lorenz et al. (1991) have suggested that the second component, the fixation release effect, involves a facilitation of saccadic premotor processes mediated by the superior colliculus. This suggestion has recently received corroborative support from Munoz and Wurtz (1992, 1993a, 1993b; see also Hikosaka & Wurtz, 1983). They demonstrated that chemical deactivation of the rostral pole of the superior colliculus serves to reduce saccadic latencies while electrical stimulation of this region serves to prevent either saccadic initiation or continuation. These data have led investigators to hypothesize that engaging the oculomotor system at fixation is a function of the rostral pole of the superior colliculus, and that disengaging the oculomotor system from fixation is one stage in the normal sequence of saccadic programming. Removal of a fixation stimulus may disengage this fixation system, allowing for the more efficient generation of saccades (e.g., Fendrich et al., 1991; Kingstone & Klein, 1993a, 1993b; Reuter-Lorenz et al., 1991; Tam & Ono, 1994; Tam & Stelmach, 1993). To the extent that antisaccades depend on the frontal eye fields rather than a collicular-based reflex pathway (see, e.g., Guitton, Buchtel, & Douglas, 1985), the effects of fixation release on these saccades should be minimal (Reuter-Lorenz et al., 1991; Reuter-Lorenz et al., in press).

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NOTE

1. It is interesting to note that Carpenter (1977) reasoned that if the variability of target-directed saccadic latencies was due to the presence

versus absence of a microsaccade refractory effect, the distribution of saccadic latencies should be bimodal. Some investigators have reported that bimodal distributions are frequently observed (see, e.g., Fischer & Weber, 1993, for a recent review) but many others have found that bimodality is not robust (e.g., Inhoff, Topolski, Vitu, & O'Regan, 1993; Kingstone and Klein, 1993b; Reuter-Lorenz et al., 1991; Reuter-Lorenz et al., in press; Tam & Ono, 1994; Tam & Stelmach, 1993; Wenban-Smith & Findlay, 1991).

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