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Fixation-point offsets reduce the latency of saccades to acoustic targets

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If an observer's fixation point is extinguished just prior to the onset of a peripheral target, the latency to saccade to that target is reduced. We show that this "gap effect" is not specific to visual targets. Observers made saccades to a light flash or to a white-noise burst. A warning tone was presented on every trial to control for the possible warning effect of the fixation-point offset. For both target modalities, saccade latencies were significantly reduced when the fixation point was extinguished 200 msec prior to the target onset. Implications of this outcome for interpretations of the gap effect are considered. It is argued that the presence of a gap effect for tones, in conjunction with previous findings, is consistent with the hypothesis that the gap effect is produced by a facilitation of premotor processes in the superior colliculus.

When human or monkey observers saccade to the onset of a visual target, their saccadic latency is reduced by the prior offset of the fixation point (e.g., Fischer, 1987; Fischer & Boch, 1983; Fischer & Ramsperger, 1984, 1986; Saslow, 1967). In human subjects, the magnitude of this reduction is about 50 msec. The reduction of saccadic latency—frequently referred to as the "gap effect"—is maximal if the fixation point is offset 200-300 msec before the target's appearance. The gap effect occurs even if a warning signal is employed to control for the alerting effect produced by the fixation-point offset (Reuter-Lorenz, Hughes, & Fendrich, 1991). Fischer and his co-workers (Fischer, 1987; Fischer & Boch, 1983; Fischer & Ramsperger, 1984) have suggested that this reduction of saccadic latency is the consequence of the appearance of a distinct subpopulation of "express saccades" with modal latencies of 120 msec in humans. However, fixation-point offsets decrease saccadic latencies even when the bimodal latency distribution that is characteristic of express saccades has not been found (Kingstone & Klein, 1990; Reuter-Lorenz et al., 1991).

Several studies have indicated that the superior colliculus plays an important role in generating the short-latency saccades that are produced in the gap paradigm. In monkeys, the gap effect is eliminated following ablation of the superior colliculus (Sandell, Schiller, & Maunsell, 1984; Schiller, Sandell, & Maunsell, 1987) . In addition, Rohrer & Sparks (1986) report that the interval between the visual and presaccadic activity bursts of cells within the deeper layers of the superior colliculus is reduced prior to such short-latency saccades. One probable role of the colliculus is to initiate rapid reflexive saccades toward peripheral visual events (Sparks & Mays, 1980; Wurtz & Albano, 1980). These reflexive saccades may be inhibited during active fixation (Munoz & Guitton, 1989). Sparks and Mays (1983) have found that during fixation the threshold to elicit saccades by electrical stimulation of the colliculus is increased. The offset of a fixation point could enable short-latency saccades by reducing this inhibition (Reuter-Lorenz, et al., 1991).

Saccades can be directed toward nonvisual targets. There are cells in the deep layers of the superior colliculus that receive acoustic inputs and increase their rate of discharge prior to saccades to acoustic targets (Jay & Sparks, 1987, 1990). Thus, a disinhibition of collicular orienting mechanisms might well enable short-latency saccades to acoustic stimuli. This suggests that if the gap effect is caused by a disinhibition of collicular reflexes, a gap effect ought to be observable with auditory targets.

Other explanations of the gap effect also predict the effect with acoustic targets. Saslow (1967) suggested that the elimination of the fixation point may serve to reduce the probability of corrective microsaccades just prior to the onset of the target, thereby reducing the refractory periods microsaccades produce. Kalesnykas and Hallett (1987) have suggested that the offset of the fixation point may increase the likelihood of anticipatory saccades, with express saccades forming a population of direction-appropriate anticipations prepared before, but executed after, the target onset. Fischer and his colleagues (Fischer, 1987; Fischer & Breitmeyer, 1987; Mayfrank, Mobashery, Kimmig, & Fischer, 1986) have proposed that the offset of the fixation point serves to release a subject's attention, so that attention is more quickly engaged by the target. According to all of these views, the gap effect should be present irrespective of the modality of the saccadic target. On the other hand, one explanation of the gap effect does not predict saccades to acoustic targets, at least in its present

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form. Reulen (1984a, 1984b) has proposed that the offset of the fixation point may serve to facilitate the visual processing of the target.

The occurrence of a gap effect to nonvisual targets is therefore relevant to a number of explanations of this effect. However, the existence of a gap effect for saccades to such targets has never actually been demonstrated. Here, we show that fixation-point offsets do, in fact, reduce the latency of saccades to auditory targets. In addition, we compare the magnitudes of the gap effect obtained with auditory and visual targets.

METHOD

The subjects were seated 114 cm from a stimulus panel aligned on an arc with a radius of 114 cm. A central, green light-emitting diode (LED) served as the fixation stimulus. Two red LEDs mounted on the panel 10° to the left and to the right of the fixation point provided the visual targets, and two small (4 cm) speakers mounted directly below the red LEDs provided the auditory targets. The visual targets consisted of 300-msec, 0.7-cd/m² LED flashes; the auditory targets were 300-msec, 90-dB white-noise bursts. These intensity levels were chosen on the basis of preliminary testing, which indicated that they would produce similar saccadic response times. Warning-tone bursts (at 2.8 kHz) were provided by a small oscillator module mounted just above the fixation point. To minimize echoes, the apparatus was housed in a large enclosure (1.54 × 1.54 × .9 m), which was lined with sound-absorbing foam (Sonex).

The subjects sat just within the open front end of this enclosure, their heads positioned by a bite plate. The subjects were run in a dark room after at least 5 min of dark adaptation and could not see either the extinguished LEDs or the speakers.

At the start of each trial, the subjects fixated the green LED. An experimenter initiated the trial when an oscilloscope display of the subject's eye position indicated proper fixation. On each trial, the warning tone sounded for 100 msec. Two hundred msec after the offset of this tone, either the visual or the auditory target was presented. The subjects were instructed to saccade to the target as rapidly as possible.

Testing was carried out in blocks of 72 trials. Within each block, there were 32 trials with visual targets and 32 with auditory targets. Half of the targets were on the left, and half were on the right. To discourage anticipatory responses, 8 trials in each block were catch trials in which there was no target. Half of the trials for each modality and half of the catch trials were "gap" trials. In gap trials, the fixation LED was turned off at the offset of the warning tone. The 200-msec interval between the fixation-point offset and the target presentation constituted the gap. The remaining trials were "overlap" trials, in which the fixation point remained on from the start of the trial until 1 sec after the target onset. The order of the various types of trials was completely randomized within each block.

An IBM PC XT microcomputer controlled the display presentations via a 16-bit parallel-output port and a custom-built interface unit. Eye motions were monitored with a scleral infrared-reflection device (Narco Biosystems Model 200) sampled via a 12-bit A/D converter at 200 Hz. The Eye-trac 200 was calibrated at the start of each block of trials. On each trial, the subject's horizontal eye position was sampled for 1,000 msec, starting at the onset of the warning tone. The eye records were stored on disk for subsequent analysis.

Saccades were detected by a computer program that used a velocity criterion (50°/sec.). In addition, accurate saccade detection was verified by visual inspection of a CRT display of each eye record. Trials in which computer detection errors occurred were corrected.

Six naive observers served as the subjects. Each observer received at least one block of practice trials prior to formal data collection. Formal data were collected over seven blocks of trials, yielding a total of 112 observations per subject in each of the experimental conditions (gap-visual target, overlap-visual target, gap-auditory target, overlap-auditory target).

RESULTS

Any trial with an initial saccade in the wrong direction was discarded. In addition, for each subject in each condition, trials with saccadic latencies more than 2.5 standard deviations (*SD*) from the mean latency of that condition were excluded from the final analyses. Finally, saccades with latencies of less than 100 msec were taken to be anticipations and removed (Kalesnykas & Hallett, 1987). Altogether, these procedures eliminated 4.6% of the data points, 4.1% of the no-gap trials, and 5% of the gap trials.

Means were computed for each subject in the four experimental conditions. With visual targets, the mean saccadic latency across the 6 subjects was 287.8 msec (*SD* = 53) in the overlap condition and 244.8 msec (*SD* = 46) in the gap condition. With acoustic targets, the mean latency was 264.2 msec (*SD* = 52) in the overlap condition and 233.8 msec (*SD* = 37) in the gap condition. Thus, mean gap effects of 43 and 30.4 msec were obtained with visual and auditory targets, respectively. These data are graphed in Figure 1.

An ANOVA was run on the subject means in the four conditions. Although acoustic targets produced faster responses and a smaller gap effect than did visual targets, only the main effect of the gap was significant [$F(1,5) = 10.76, p < .03$]. Paired comparisons using the Newman-Keuls procedure indicated that for both visual and acous-

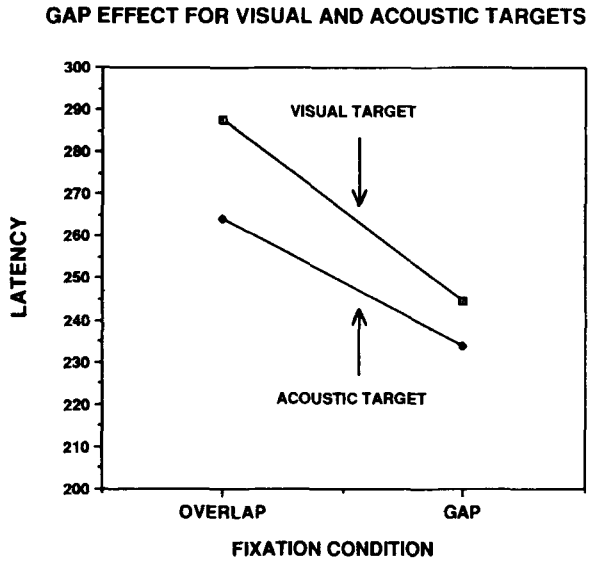

Figure 1. Mean saccadic latencies for visual and acoustic targets in the gap and overlap conditions.

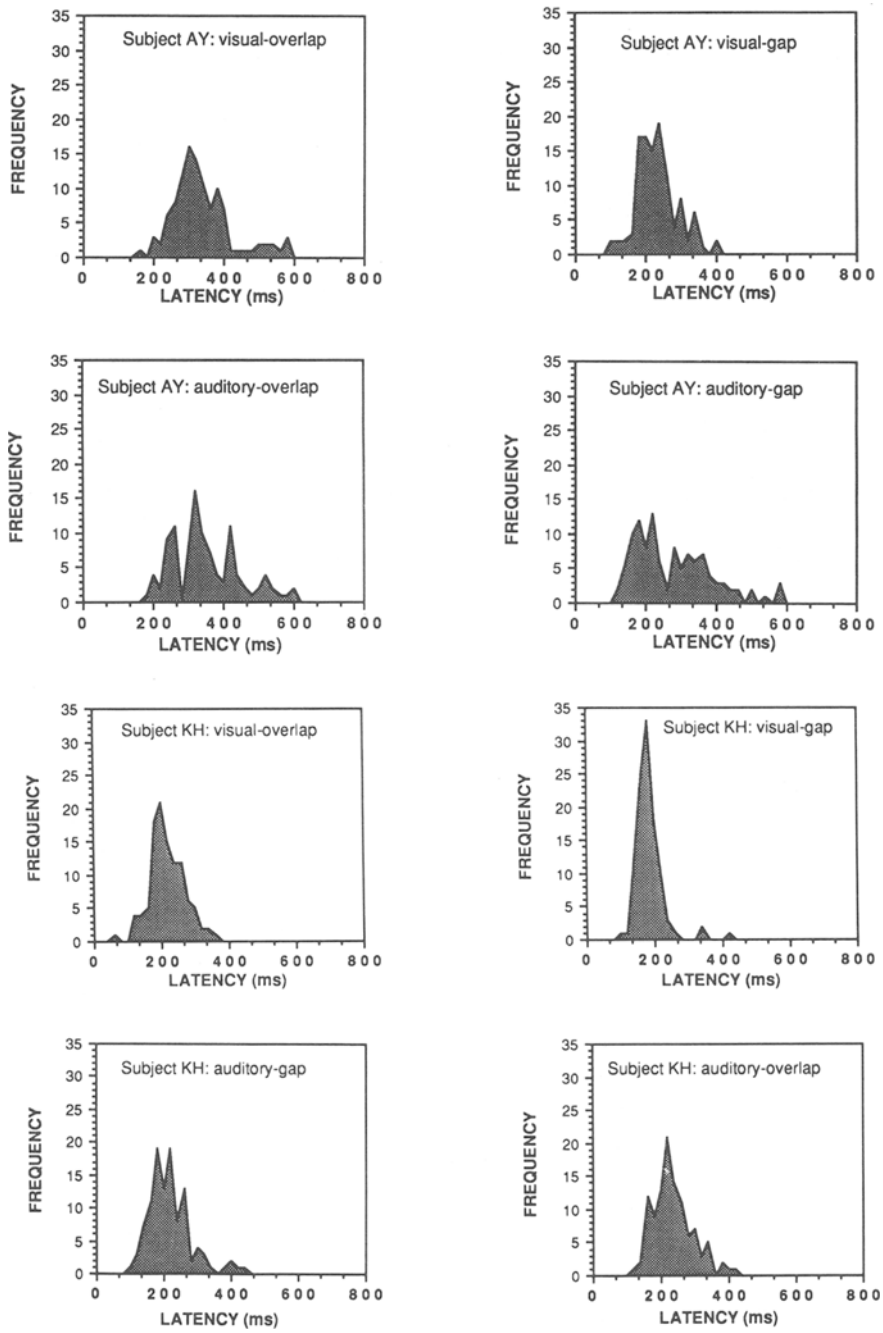

Figure 2. Distribution of saccadic latencies for 2 representative subjects in the four experimental conditions.

tic targets, latencies were significantly faster in the gap than in the overlap condition ($p < .05$). It should be noted, however, that the gap-modality interaction came quite close to significance [$F(1,5) = 5.4, p < .07$], which suggests that the tendency for the gap effect to be smaller with acoustic than with visual targets may be genuine.

Latency histograms for two representative observers are illustrated in Figure 2. Similar to the findings of Reuter-Lorenz et al. (1991), most of the obtained latency distributions failed to show evidence of bimodality. In the distributions illustrated, a suggestion of bimodality is observable only in the auditory target data for Subject A.Y. Generally, a gap effect was found because fixation offsets tended to shift or compress entire distributions toward shorter latencies.

The false alarm rate was .08 (4.5 per 56 catch trials), with half the subjects showing 2 or fewer catch-trial saccades. Most (19 of 24) of the saccades occurred in gap

catch trials. The average latency of the catch-trial saccades, measured from the time the target would have onset had one been presented, was 182 msec (SD = 66.5). There was no apparent relationship between the number of saccades a subject made during the catch trials and the magnitude of a subject's gap effect; averaged across modality, virtually identical gap effects of 28 and 27 msec were obtained from the subjects with the highest (9) and lowest (0) number of catch-trial saccades.

DISCUSSION

The results indicate that the prior offset of a fixation point facilitates saccades to acoustic, as well as to visual targets. This finding suggests that the gap effect cannot be attributed simply to enhanced visual processing (Reulen, 1984a, 1984b). The fact that the gap effect is not modality-specific is consistent with an interpretation of this effect that is based on a facilitation of motor or premotor processes. The deep layers of the superior colliculus appear a likely candidate for the locus of this premotor facilitation, since this structure receives convergent visual and auditory inputs (e.g., Jay & Sparks, 1990; Meredith & Stein, 1986) and is involved in initiating saccades (see Sparks & Hartwich-Young, 1989, for a review).

There is an indication that the magnitude of the gap effect may be stronger for visual than for auditory targets. Assuming this interaction is real, we can only offer suggestions as to how such a difference might arise. The magnitude of the gap effect varies with gap duration (Saslow, 1967). The gap duration we employed is optimal for visual targets (Saslow, 1967) but might not be optimal for auditory targets. In addition, although visual and auditory inputs converge in the colliculus, the characteristics of saccades to auditory targets differ from visually triggered saccades. For example, auditory targets have a lower peak velocity and are more likely to be double saccades (Jay & Sparks, 1990). These discrepancies imply a difference in saccadic programming for targets of varying modalities, which could also affect the magnitude of the gap effect.

As noted in the introduction, several alternative accounts of the gap effect are also consistent with the fact that it occurs with auditory targets. Saslow's (1967) attribution of this effect to an increased incidence of microsaccades during the gap would predict this outcome. However, Saslow's hypothesis fails to account for the absence of a gap effect with antisaccades (Reuter-Lorenz et al., 1991).

The attribution of the gap effect to an increased incidence of anticipatory saccades (Kalesnykas & Hallett, 1987) is also consistent with the present finding. As this hypothesis would predict, more saccades occurred in gap than in overlap catch trials. Furthermore, the mean latency of these saccades (with respect to the time a target would have appeared in a test trial) was short. However, the gap effect was present in the subjects who made few or no saccades in catch trials; across subjects, the number of catch-trial saccades was not related to the magnitude of the gap effect. We therefore acknowledge that anticipations may sometimes contribute to the gap effect, but we believe that they are unlikely to be the primary source of this effect. (A further discussion of the role of anticipations in the gap effect can be found in Reuter-Lorenz et al., 1991.)

An attribution of the gap effect to the release of attention (Braun & Breitmeyer, 1988; Fischer, 1987; Fischer & Breitmeyer, 1987) agrees with the current outcome if one assumes that, once released, attention will be engaged by acoustic, as well as by visual stimuli. Precuing experiments have failed, however, to demonstrate any effect of spatial attention on responses to auditory targets (Buchtel & Butter, 1988; M. I. Posner, 1978). Admittedly, this observation needs to be regarded with caution, since it has not been specifically demonstrated that spatial precues do not influence saccades to auditory targets. In addition, spatial precues interact with target luminance (Hawkins, Shafto, & Richardson, 1988), whereas the gap effect is additive with luminance (Reuter-Lorenz et al., 1991), and the absence of a gap effect with either manual responses or antisaccades (Reuter-Lorenz et al., 1991) does not seem to agree with the attentional hypothesis. On the other hand, it has been proposed that the superior colliculus serves to control movements of attention (Posner & Petersen, 1990) and that movements of attention are tied to oculomotor programming (Rizzolatti, Riggio, Dascola, & Umilta, 1987). To the extent that these proposals are correct, an interpretation of the gap effect based on movements of attention might prove compatible with an account based on collicular premotor processes.

An explanation of the gap effect based on the release of collicular orienting reflexes does account for its absence with antisaccades and manual responses, since the colliculus does not control manual responses and antisaccades are not directed toward a sensory target. The present finding of a gap effect with auditory targets strengthens the case for such an explanation.

REFERENCES

Braun, D., & Breitmeyer, B. G. (1988). Relationship between directed visual attention and saccadic reaction times. *Experimental Brain Research*, **73**, 546-552.

Buchtel, H. A., & Butter, C. M. (1988). Spatial attention shifts: Implications for the role of polysensory mechanisms. *Neuropsychologia*, **26**, 499-510.

Fischer, B. (1987). The preparation of visually guided saccades. *Review of Physiological Biochemistry Pharmacology*, **106**, 1-35.

Fischer, B., & Boch, R. (1983). Saccadic eye movements after extremely short reaction times in the monkey. *Experimental Brain Research*, **260**, 21-26.

Fischer, B., & Breitmeyer, B. (1987). Mechanisms of visual attention revealed by saccadic eye motions. *Neuropsychologia*, **25**, 73-83.

Fischer, B., & Ramsperger, E. (1984). Human express saccades: Extremely short reaction times of goal directed eye movements. *Experimental Brain Research*, **57**, 191-195.

Fischer, B., & Ramsperger, E. (1986). Human express-saccades: Effects of randomization and daily practice. *Experimental Brain Research*, **64**, 569-578.

Hawkins, H. L., Shafto, M. G., & Richardson, K. (1988). Effects

of target luminance and cue validity on the latency of visual detection. *Perception & Psychophysics*, **44**, 484-492.

JAY, M. F., & SPARKS, D. L. (1987). Sensorimotor integration in the primate superior colliculus: I. Motor convergence. *Journal of Neurophysiology*, **57**, 22-34.

JAY, M. F., & SPARKS, D. L. (1990). Localization of auditory and visual targets for the initiation of saccadic eye movements. In M. A. Berkley & W. Stebbins (Eds.), *Comparative perception: Vol. 1. Basic mechanisms* (pp. 351-374). New York: Wiley.

KALESNYKAS, R., & HALLETT, P. E. (1987). The differentiation of visually guided and anticipatory saccades in gap and overlap paradigms. *Experimental Brain Research*, **68**, 115-121.

KINGSTONE, A., & KLEIN, R. (1990, November). *Attention and express saccades*. Paper presented at the 31st Annual Meeting of the Psychonomic Society, New Orleans.

MAYFRANK, L., MOBASHERY, M., KIMMIG, H., & FISCHER, B. (1986). The role of fixation and visual attention on the occurrence of express saccades in man. *European Archives of Psychiatry & Neurological Science*, **235**, 269-275.

MEREDITH, M. A., & STEIN, B. E. (1986). Visual, auditory, and somatosensory convergence on cells in superior colliculus results in multisensory integration. *Journal of Neurophysiology*, **56**, 640-662.

MUNOZ, D. P., & GUITTON, D. (1989). Fixation and orientation control by the tecto-reticulo-spinal system in the cat whose head is unrestrained. *Revue Neurologique*, **145**, 567-579.

POSNER, M. I. (1978). *Chronometric explorations of mind*. Hillsdale, NJ: Erlbaum.

POSNER, M. I., & PETERSEN, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience*, **13**, 25-42.

REULEN, J. P. H. (1984a). Latency of visually evoked saccadic eye movements: I. Saccadic latency and the facilitation model. *Biological Cybernetics*, **50**, 251-263.

REULEN, J. P. H. (1984b). Latency of visually evoked saccadic eye movements: II. Temporal properties of the facilitation mechanism. *Biological Cybernetics*, **50**, 263-271.

REUTER-LORENZ, P. A., HUGHES, H. C., & FENDRICH, R. (1991). The reduction of saccadic latency by prior offset of the fixation point: An analysis of the gap effect. *Perception & Psychophysics*, **49**, 167-175.

RIZZOLATTI, G., RIGGIO, L., DASCOLA, I., & UMILTA, C. (1987). Reorienting attention across the horizontal and vertical meridians: Evidence in favor of a premotor theory of attention. *Neuropsychologia*, **25**, 31-40.

ROHRER, W. H., & SPARKS, D. L. (1986). Role of the superior colliculus in the initiation of express saccades. *Investigative Ophthalmology Visual Science*, **27**, 156. (Abstract)

SANDELL, J. H., SCHILLER, P. H., & MAUNSELL, J. H. R. (1984). The effect of superior colliculus and frontal eye field lesions on saccade latency in the monkey. *Perception*, **13**, 66.

SASLOW, M. G. (1967). Effects of components of displacement-step stimuli upon latency of saccadic eye movement. *Journal of the Optical Society of America*, **57**, 1024-1029.

SCHILLER, P. H., SANDELL, J. H., & MAUNSELL, J. H. R. (1987). The effect of frontal eye field and superior colliculus lesions on saccadic latencies in the rhesus monkey. *Journal of Neurophysiology*, **57**, 1033-1049.

SPARKS, D. L., & HARTWICH-YOUNG, R. (1989). The deep layers of the superior colliculus. In R. H. Wurtz & M. E. Goldberg (Eds.), *The neurobiology of saccadic eye motions* (pp. 213-255). New York: Elsevier Science Publishers.

SPARKS, D. L., & MAYS, L. E. (1980). Movement fields of saccade-related burst neurons in the monkey superior colliculus. *Brain Research*, **190**, 39-50.

SPARKS, D. L., & MAYS, L. E. (1983). Spatial localization of saccade targets: I. Compensation for stimulus-induced perturbations in eye position. *Journal of Neurophysiology*, **49**, 45-63.

WURTZ, R. H., & ALBANO, J. E. (1980). Visual-motor function of the primate superior colliculus. *Annual Review of Neuroscience*, **31**, 189-220.

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