Saccadic Performance as a Function of the Presence and Disappearance of Auditory and Visual Fixation Stimuli

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Abstract

■ Relative to when a fixated stimulus remains visible, saccadic latencies are facilitated when a fixated stimulus is extinguished simultaneously with or prior to the appearance of an eccentric auditory, visual, or combined visual-auditory target. In a study of nine human subjects, we determined whether such facilitation (the "gap effect") occurs equivalently for the disappearance of fixated auditory stimuli and fixated visual stimuli. In the present study, a fixated auditory (noise) stimulus remained present (overlap) or else was extinguished simultaneously with (step) or 200 msec prior to (gap) the appearance of a visual, auditory (tone), or combined visual-auditory target 10° to the left or right of fixation. The results demonstrated equivalent facilitatory effects due to the disappearance of fixated auditory and visual stimuli and are consistent with the presumed role of the superior colliculus in the gap effect. ■

INTRODUCTION

Saccadic latencies are facilitated when a fixated visual stimulus is extinguished simultaneously with (step) or prior to (gap) the appearance of a peripheral visual target (e.g., Fischer & Ramsperger, 1984; Saslow, 1967). Known as the gap effect, this facilitatory effect of fixation disappearance is specific to oculomotor responses (e.g., Reuter-Lorenz, Hughes, & Fendrich, 1991; Ross & Ross, 1980; Tam & Stelmach, 1993; Taylor, Klein, & Kingstone, 1993) and does not appear to (1) be due entirely to a warning effect engendered by the disappearance of a stimulus at fixation (e.g., Ross & Ross, 1980, 1981), (2) be related to fixation-maintaining microsaccades (cf. Saslow, 1967; see Kingstone, Fendrich, Wessinger, & Reuter-Lorenz, 1994), (3) result from improved efficiency of visual processing (cf. Reulen, 1984a, 1984b; see Kingstone & Klein, 1993; Reuter-Lorenz et al., 1991), or (4) be the result of anticipatory execution of a preprogrammed saccadic response (cf. Jüttner & Wolf, 1992 and Kalesnykas & Hallett, 1987; see Kingstone & Klein, 1993; Reuter-Lorenz et al., 1991). Instead, evidence converges on oculomotor mechanisms of the superior colliculus (SC) as the basis for the gap effect.

The SC and the frontal eye fields (FEF) are two structures critical for producing saccadic eye movements (Schiller, 1977; Schiller, True, & Conway, 1980). The SC is responsible for reflexive orienting to visual targets (e.g., Dorris, Pare, & Munoz, 1997; Mohler & Wurtz, 1976; Schiller, Sandell, & Maunsell, 1987; Sparks, 1978). In contrast, the FEF is responsible primarily for goal-directed saccades (e.g., Schiller et al., 1987) and is one of a number of cortical projections that exert higher-level control over the reflexive machinery of the SC (e.g., Guitton, Buchtel, & Douglas, 1985) through both direct connections to the SC (e.g., Segraves & Goldberg, 1987) and indirect connections via the caudate nucleus to the substantia nigra pars reticulata to the SC (see Hikosaka & Wurtz, 1989 for a review).

In addition to this control of the SC by fronto-collicular projections, SC activity is also influenced by intracollicular inhibition (Munoz & Istvan, 1998). This inhibition derives from the activity of fixation cells in the rostral pole of the SC that code for the central 1 to 2° of the visual field (Munoz & Wurtz, 1993a, 1993b). During active fixation, these cells inhibit saccade-related activity in the intermediate layer. That this inhibition affects saccadic latency is suggested by a positive correlation wherein the maximum decrease in fixation cell discharge rates corresponds with the fastest saccades (Dorris & Munoz, 1995; Dorris et al., 1997). As such, it appears that release from the inhibitory consequences of fixation cells is likely to be responsible for the decrease in saccadic latencies that occurs when a fixated visual stimulus is extinguished prior to target appearance.

Approximately 18% of SC cells in the monkey are

responsive to auditory stimuli and another 11% are maximally responsive to combined visual-auditory stimuli (Wallace & Stein, 1996). To the extent that the gap effect reflects the release of inhibition on saccade-generating mechanisms of the SC, this suggests that saccades made to auditory and combined visual-auditory targets should also benefit from the prior or coincident release of visual fixation. Indeed, this is the case. A gap effect has been observed for auditory targets (Fendrich, Hughes, & Reuter-Lorenz, 1991) and for visual-auditory compounds that are spatially and temporally congruent (Munoz & Corneil, 1995; Nozawa, Reuter-Lorenz, & Hughes, 1994).

The occurrence of a gap effect for visual, auditory, and combined visual-auditory targets demonstrates that the release of visual fixation facilitates saccades to target stimuli that are known to be represented at the level of the SC. The goal of the present research is to determine whether the release of *auditory fixation* is likewise capable of facilitating saccades to visual, auditory, and combined visual-auditory targets. If the spatial registration of an auditory stimulus at fixation generates saccadic inhibition, the disappearance of such a stimulus should result in an auditory gap effect akin to that observed for the disappearance of a fixated visual stimulus.

ASSESSING THE EQUIVALENCE OF VISUAL AND AUDITORY FIXATED STIMULI

Three speakers were mounted on a horizontal perimeter arc, with 10° visual angle between adjacent pairs. A red light-emitting diode (LED) was centered in front of each speaker. On auditory fixation trials, subjects fixated white noise generated by the central speaker; on visual fixation trials, subjects fixated the (illuminated) central LED. Auditory and visual fixation trials were mixed within blocks. Three gap conditions were employed: In the overlap condition, the fixated stimulus remained present throughout the trial in the step condition, the fixated stimulus was extinguished simultaneously with the appearance of a peripheral target; and in the gap condition, the fixated stimulus was extinguished 200 msec prior to the appearance of a peripheral target. Whether subjects fixated a central visual or auditory stimulus, the saccade target was an illuminated peripheral LED (visual), a tone from a peripheral speaker (auditory), or an illuminated peripheral LED that was presented along with a tone from the corresponding speaker (visual-auditory). Note that the visual, auditory, and visual-auditory saccade targets were identical under conditions of visual and auditory fixation.[1] As such, gross differences in the speed to make responses following the disappearance of the fixated stimulus would suggest that visual and auditory stimuli do not exert similar inhibitory control over the saccadic eye movement system. Conversely, similarities between the latencies to make saccades under the two fixation stimulus conditions would point to equivalence in the release of visual and auditory fixation.

In this analysis, note that it is the comparison of the step condition to the overlap condition that is most telling. This is because the 0 msec foreperiod duration (i.e., the time between fixation disappearance and target appearance) in the step condition is insufficient for the operation of warning effects. As such, any facilitation in the step relative to overlap conditions is believed to be due only to the release of ocular fixation (cf. Taylor et al., 1993) and is therefore the purest measure of whether the disappearance of auditory and visual fixation stimuli may result in equivalent oculomotor disengagement. Nevertheless, a 200-msec gap condition was retained to examine any differences that might arise between auditory and visual fixation conditions as a function of warning interval and also because the effects of ocular disengagement may not be fully blown until 200 msec after the fixation stimulus is extinguished (cf. Dorris & Munoz, 1995).

Results

The overall error rate was 8%. Error trials were excluded from the following analyses.

Figure 1 shows the mean correct saccadic reaction times (RTs) for the visual fixation (a) and auditory fixation (b) conditions. The overall pattern of results appears similar between (a) and (b) of Figure 1. This observation was confirmed by a three-way repeated measures analysis of variance (ANOVA), with fixation modality (visual, auditory), target modality (visual, auditory, visual-auditory), and gap condition (overlap, step, gap) as factors. Fixation modality did not interact with any other factor—not with target modality ($F(2, 16) = 2.43$, *ns*), gap condition ($F < 1$), or their combination ($F < 1$). This indicates that saccadic RTs were affected equivalently by the disappearance of auditory and visual fixated stimuli. There was also no overall difference in the mean saccadic RT on auditory versus visual fixation trials ($F(1, 8) = 3.30$, *ns*).

The disappearance of a fixated stimulus resulted in an overall facilitation of saccadic RTs with increasing gap duration ($F(2, 8) = 19.42, p < 0.01$). As seen in Figure 1, however, this gap effect interacted with target modality ($F(4, 16) = 8.28$, $p < 0.01$). Whereas a saccadic RT reduction was observed in the step condition when targets were visual ($F(1, 16) = 99.53, p < 0.01$) or visual-auditory ($F(1, 16) = 5.37, p < 0.03$), when targets were auditory, there was no facilitation from overlap to step ($F < 1$). For auditory targets, an RT reduction was not obtained until the 200-msec gap condition ($F(1,16) = 40.44, p < 0.01$).

The differential pattern of gap effects that occurred as a function of target modality cannot be explained by differences in the overall saccadic RTs. The significant main effect for target modality ($F(2, 8) = 15.13, p < 0.01$)

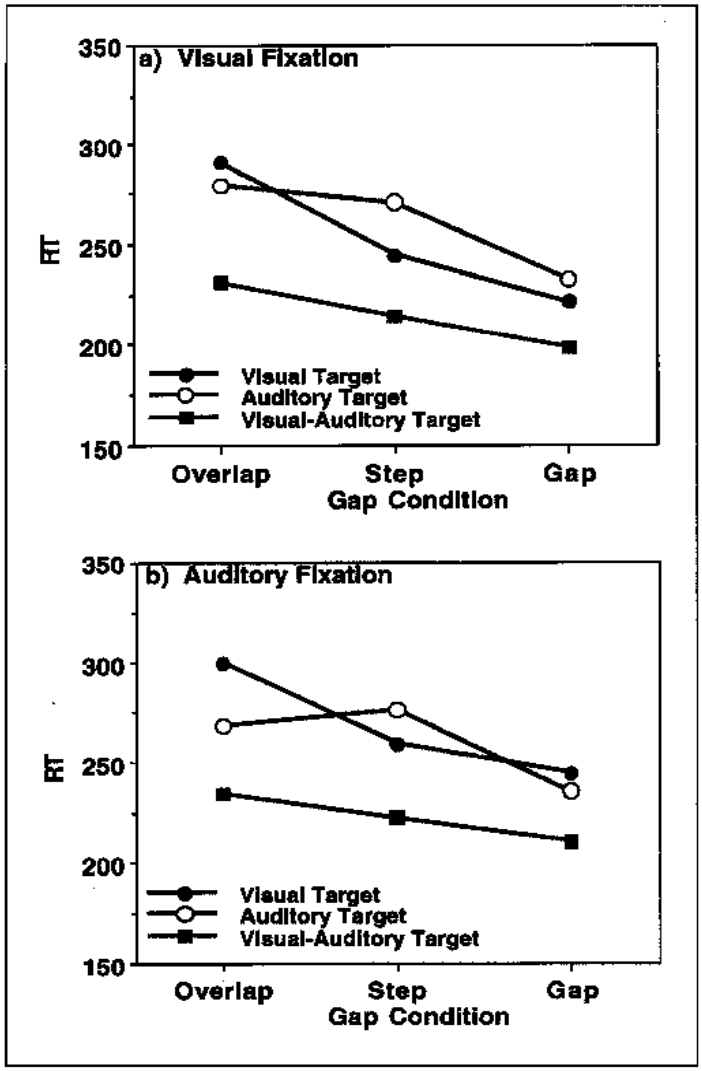

Figure 1. Saccadic RTs (msec) to visual, auditory, and visual-auditory targets in visual (a) and auditory (b) overlap, step, and gap conditions.

was due to the overall fastest saccadic RTs to visual-auditory targets; despite showing a different pattern of gap effects, there was no difference in the overall saccadic RTs to visual and auditory targets ($F < 1$). Indeed, the main effect of target modality confirms other research (e.g., Corneil & Munoz, 1996; Hughes, Reuter-Lorenz, Nozawa, & Fendrich, 1994; Lueck, Crawford, Savage, & Kennard, 1990; Nozawa et al., 1994) in demonstrating faster responding to visual-auditory targets than to visual-only ($F(1, 8) = 22.49, p < 0.01$) or auditory-only targets ($F(1, 8) = 22.91, p < 0.01$).

DISCUSSION

The disappearance of a fixated visual stimulus prior to or coincident with the appearance of a visual, auditory, or combined visual-auditory target results in a facilitation of saccadic RTs that is believed to be due to the release of inhibition on saccade-related cells in the intermediate SC by rostral pole fixation cells (Dorris & Munoz, 1995). Given that the SC receives and generates saccades based on auditory input (Jay & Sparks, 1984, 1987a, 1987b; VanOpstal & Frens, 1996) and that saccadic RTs to targets with an auditory component therefore enjoy the facilitatory effects of ocular disengagement, the present investigation explored whether auditory stimuli might similarly control ocular fixation. The present experiment compared saccadic RTs that followed the disappearance of a fixated visual versus a fixated auditory stimulus prior to or simultaneous with the appearance of a peripheral visual, auditory, or visual-auditory target.

For targets with a visual component, we replicated the typical finding of saccadic RT facilitation in step and gap conditions relative to overlap. For targets that were purely auditory, however, there was no apparent facilitation in the step relative to overlap condition. The step condition is thought to represent the effects of oculomotor disengagement, uncontaminated by warning effects (Taylor et al., 1993). As such, if saccades to auditory targets were influenced by the release of oculomotor inhibition, they should have revealed a gap effect in the step condition of our experiment. Although it is unclear why a gap effect was not observed for auditory targets in the step condition, we can rule out several alternatives and suggest several more plausible ones.

It is tempting to suggest that the failure to observe a

Table 1. Saccadic RTs (msec) for visual, auditory, and visual-auditory targets in visual and auditory overlap, step, and gap conditions. Standard errors are given in parentheses.

		Gap Condition		
Fixation	*Target Modality*	*Overlap*	*Step*	*Gap*
Visual	Visual	291 (10)	245 (7)	222 (13)
	Auditory	279 (11)	271 (16)	233 (18)
	Visual-auditory	231 (9)	215 (8)	199 (9)
Auditory	Visual	300 (19)	260 (13)	245 (11)
	Auditory	269 (18)	277 (12)	236 (15)
	Visual-auditory	235 (10)	222 (11)	210 (11)

gap effect for our auditory targets may have been due to interference from our auditory fixation stimulus (see Note 1); however, this suggestion is belied by the fact that a gap effect was also not obtained for auditory targets in the visual fixation step condition. Given that the overall latency of saccades to auditory-only targets did not differ from those made to visual-only targets, it is also not the case that the delay between target appearance and response was insufficient to benefit from ocular disengagement. Moreover, the RT facilitation for the step versus overlap conditions that was observed with visual and visual-auditory targets was roughly the same magnitude whether the fixation stimulus was visual or auditory. This suggests that the disappearance of fixated visual and auditory stimuli effectively disengaged the oculomotor system. And, although we did not equate the intensity of the visual and auditory stimuli, the gap effect has been shown not to interact with target intensity (Kingstone & Klein, 1993; Reuter-Lorenz et al., 1991). This argues against intensity differences as the source of the discrepancy between the effect of gap condition on saccadic RTs to auditory versus visual and visual-auditory targets.

The SC codes motor error—the difference between current and desired eye position—in an oculocentric frame of reference. In the case of targets that have a visual component, oculocentric information about desired eye position is available directly from the sensory stimulation; in the case of auditory targets, however, craniotopic coding of stimulus location must be transformed to an oculocentric coding of desired eye position. Possibly because of this required transformation from craniotopic to oculocentric coordinates (based on the analysis of spectral content and relative timing at the two ears), auditory-evoked saccades tend to be less accurate than visually evoked saccades; the accuracy of auditory-evoked saccades increases with increasing amplitude. As such, it could be suggested that the 10° target eccentricity that we used in our study did not provide localization information that was sufficient to establish a collicular motor program in the same manner as for a visual stimulus.[2] However, the data do not support this hypothesis.

Consider that if our auditory targets were particularly difficult to localize, this would be reflected in the mean saccade amplitude and/or in the amount of within-subject variability around the mean. Collapsed over right and left target locations, the mean amplitude was identical, at 10°, for all three target modalities. An analysis of the standard deviations around each subject's mean amplitude likewise revealed no significant differences ($F(2, 16) = 1.20, p < 0.33$): The mean standard deviation was 2.10° for visual targets, 2.18° for auditory targets, and 2.07° for visual-auditory targets.

What else could account for the failure to observe a gap effect for auditory targets in the step condition? One possibility is based on the fact that the activity of saccade-related burst neurons (SRBNs) in the SC is modulated according to target modality (VanOpstal & Frens, 1996). Compared to visually evoked saccades, the SRBN activity associated with auditory-evoked saccades is reduced by almost 60%. Relative to unimodal activity, some visuomotor responses to visual-auditory compounds are enhanced, and others are suppressed. Thus, to the extent that the disappearance of a fixated stimulus may release inhibition on movement-related cells, this would suggest that the occurrence and/or magnitude of the gap effect may be determined by the baseline activity of the SRBNs: the more active the SRBNs, the greater the impact of fixation stimulus removal. This view would predict the largest gap effect for visual targets and the smallest gap effect for auditory targets, with visual-auditory targets showing an intermediate effect based on the overall degree of enhancement versus suppression. Indeed, this is the pattern of results that was obtained. Collapsed across fixation condition, for visual, auditory, and visual-auditory targets, respectively, the difference between overlap and step was 43, −1, and 15 msec.

It could also be the case that auditory control of fixation is modulated by the nature and/or spatial relations of the fixated versus target stimuli. Shafiq, Stuart, Sandbach, Maruff, and Currie (1998) performed a study in which subjects fixated a central tone and made saccades to white noise bursts that occurred at 15° to the left or right of fixation.[3] The fixated auditory stimulus remained present throughout the trial (overlap), was extinguished simultaneously with target onset (step), or was extinguished 200 msec prior to target onset (gap). Saccade latencies were 178 msec in the overlap condition, 166 msec in the step condition, and 153 msec in the gap condition. On our view that the comparison of overlap versus step is uncontaminated by warning effects, the estimate of the gap effect in Shafiq et al.'s (1998) study was 12 msec and was significant (compare this to our nonsignificant 8-msec effect). Consider that whereas (1) our fixated auditory stimulus was noise, theirs was a tone; (2) our auditory targets were tones, theirs were noise; and, (3) our stimuli were at 10° eccentricity, theirs were at 15°. Any one or a combination of these factors may have been responsible for the lack of a significant gap effect in our auditory fixation step condition.

Another possibility is that—with our stimuli—saccades made to auditory targets are affected by, but have a different timecourse for, the influence of oculomotor disengagement. In a gap paradigm that used visual fixated stimuli and visual targets, Taylor, Kingstone, and Klein (1998) eliminated the influence of warning effects across gap intervals and demonstrated that oculomotor disengagement occurs with a 0-msec gap interval (i.e., in the step condition) and that the effects of oculomotor disengagement increase only slightly with increasing gap duration. Whereas targets with a visual component benefit from the disengagement of the oculomotor sys-

tem immediately following the disappearance of the fixated stimulus, auditory targets may be affected only when there is a longer delay between the disappearance of the fixated stimulus and the appearance of the target. According to this view, the gap effect we observed for auditory targets at a 200-msec gap interval represents the combined influence of warning effects and oculomotor disengagement such that, if warning effects were eliminated, a facilitatory effect would remain.

Regardless of the lack of saccadic RT facilitation for auditory targets in the step condition of our study, there clearly was facilitation of saccadic RTs to visual and visual-auditory targets following fixation disappearance. Critically, for present purposes, this facilitation was the same whether the extinguished fixated stimulus was visual or auditory.[4] This is strong evidence that visual and auditory stimuli do exert similar effects on the saccadic system and suggests that the presumed SC-based inhibitory relation between saccades and the presence of a fixated visual stimulus also exists between saccades and the presence of a fixated auditory stimulus.

To the extent that the SC is responsible for the release of saccadic inhibition following fixation removal, the present findings suggest that there is auditory input to the rostral pole fixation cells and that the inhibitory influence of these cells on the saccade-related cells of the intermediate layer can be activated by visual and auditory stimuli alike (cf. Frens & VanOpstal, 1995; Peck & Baro, 1997). Single-cell recording from rostral pole fixation cells in monkeys trained to fixate auditory stimuli would therefore seem critical to developing primate models of the relation between stimulus control over fixation and saccades.

METHODS

Subjects

Nine Dalhousie undergraduates volunteered in exchange for credit toward their introductory psychology grade. Subjects reported normal or corrected-to-normal vision and were naive to the experimental purpose.

Apparatus and Stimuli

Stimulus presentation and data collection were controlled by a Macintosh IIx, running custom software. Three LEDs were mounted in front of three Radio Shack speakers and attached to a horizontal, semicircular rod positioned at eye level. The fixation stimuli were generated by illuminating the central LED or presenting broad band white noise (Scientific Prototype 381G) through the central speaker. The visual target stimuli were generated by illuminating one of the peripheral LEDs; the auditory target stimuli were generated by presenting a tone through one of the peripheral speakers. Subjects initiated each trial by depressing a hand-held thumb-switch.

Subjects were tested individually in a completely darkened, sound-attenuating room in which they sat with the head stabilized in a chin rest mounted 163 cm from the center stimulus. At this distance, the target LEDs/speakers were 10° visual angle to the left and right of fixation. Horizontal eye movements were sampled every 1 msec using an Eyetrac Model 210 scleral reflection monitor mounted on eyeglass frames and supported by headstraps.

Design and Procedure

The stimuli and trial events are depicted in Figure 2. The $2 \times 3 \times 3$ within-subjects design was comprised of the factorial combination of fixation modality (auditory, visual), target modality (visual, auditory, visual-auditory), and gap condition (overlap, step, gap). Each of the resulting 18 conditions were repeated twice in a practice block (36 trials) and were repeated 20 times in an experimental block (360 trials). Targets occurred to the left of fixation on a random half of the trials and to the right of fixation on the other half of the trials.

The fixation stimulus (the central noise or LED) was presented during the intertrial interval. Once fixated, the subject initiated the trial events by depressing the thumb-switch; the target followed 1200 msec after trial initiation. Subjects were instructed to maintain fixation on whatever stimulus was presented at the start of the trial and to maintain fixation until the presentation of a peripheral target. Whether the fixated stimulus was auditory or visual, in the step condition, it was extinguished simultaneously with target presentation; in the gap condition, it was extinguished 200 msec prior to target presentation. Visual targets consisted of the illumination of a peripheral LED; auditory targets consisted of tones emitted from a peripheral speaker; and, visual-auditory targets consisted of the illumination of a peripheral LED and simultaneous emission of a tone from the spatially congruent peripheral speaker (i.e., the speaker mounted behind the illuminated LED). Targets remained on until there was a response or else until 1200 msec had elapsed. There was a 2- sec interval during which no LED or noise stimulus was presented. This interval was followed by the intertrial interval during which the fixation stimulus for the next trial was presented and subjects were free to initiate the trial at their own discretion.

Eye movements were monitored throughout the trial duration and saccades were detected using a velocity criterion of 50°/sec. Failures to maintain fixation between trial initiation and target presentation were not coded as errors, and the trials were recycled later in the block. Errors included a failure to attain sufficient amplitude following the onset of a saccade and an eye movement made in the nontarget direction. Subjects were

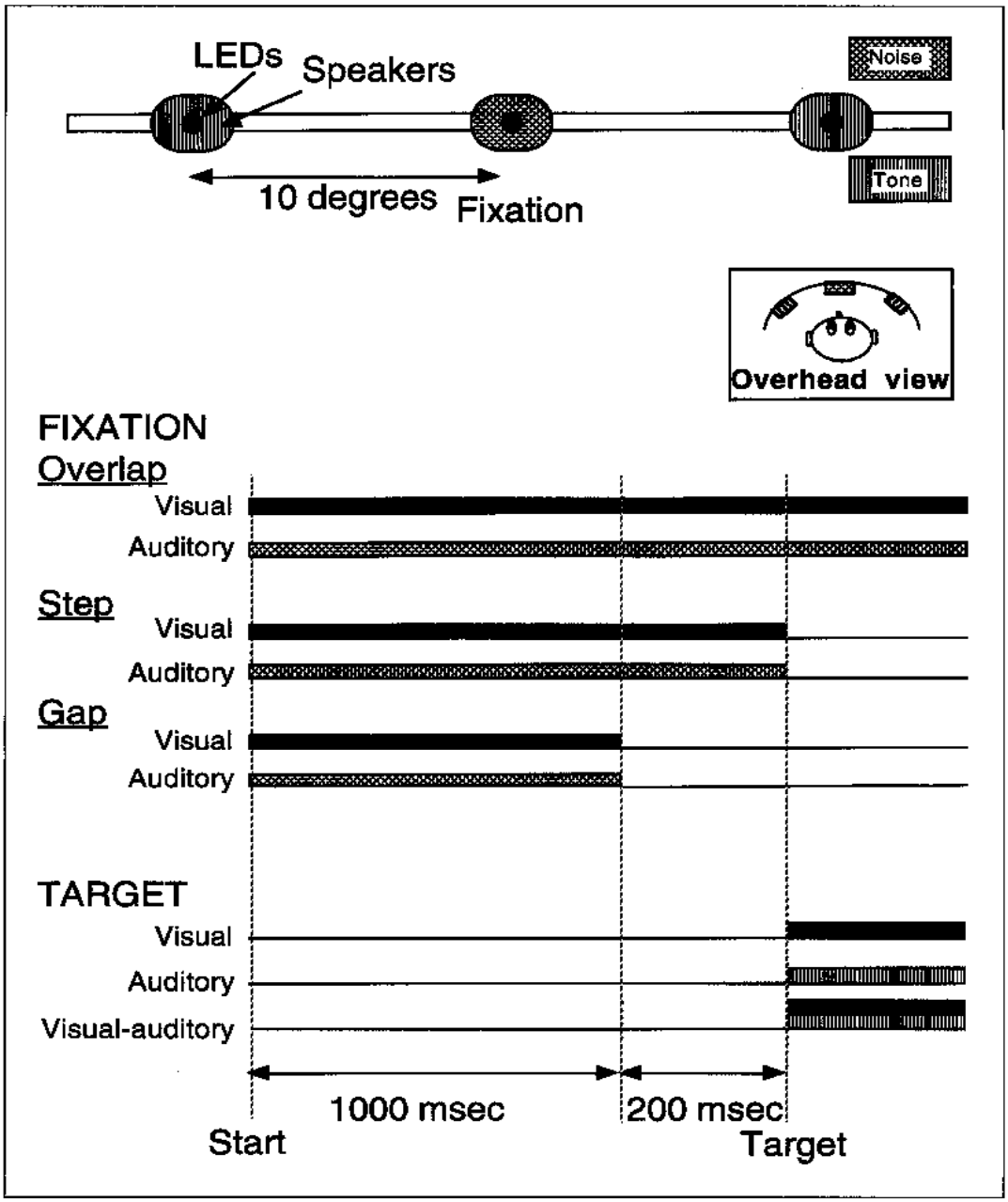

Figure 2. Stimuli and trial events. At the top of the figure is a representation of the arrangement of visual and auditory stimuli and an overhead view of the apparatus. At the bottom of the figure is a representation of the fixation and target events. For visual and auditory fixation conditions, the fixated stimulus remained visible throughout target presentation (overlap), disappeared at the same time as the target appeared (step), or disappeared 200 msec (gap) before the target appeared. The target was auditory, visual, or visual-auditory. See text for further details.

alerted to errors via the illumination of all three LEDs and all three speakers. Error trials were not recycled.

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Notes

1. In a pilot investigation, we examined auditory and visual step and gap conditions for auditory, visual, and auditory-visual targets. However, the auditory fixated stimulus and the auditory and visual-auditory targets were the same (noise). The results of this investigation suggested the equivalence of visual and auditory control of fixation when targets were visual. However, when the targets had an auditory component, there appeared to be interference in the step condition for auditory fixation.

Unfortunately, because of this postulated interference between the simultaneous disappearance of a fixated noise stimulus and the appearance of a peripheral noise target, it was impossible to determine whether fixated visual and auditory stimuli do, in fact, control fixation equivalently. Furthermore, because we did not include an overlap condition in this pilot investigation, facilitation of saccadic RTs due to fixation disappearance could not be evaluated.

To disambiguate auditory fixation from peripheral auditory targets—and thereby obviate the possible interfering effects of using identical stimuli for both—the reported experiment presented noise at the central speaker and tones at the peripheral speakers. Although humans are relatively poorer at localizing peripheral tones than broad-band noise stimuli, responses tend to suffer minimally along the horizontal meridian (e.g., Frens and VanOpstal, 1995; Frens, VanOpstal, & VanderWilligen, 1995). As such, the desire to use the same auditory fixated stimulus in the reported experiment as in our pilot investigation motivated the use of a central noise stimulus and peripheral tones for fixation and target stimuli, respectively. The reported experiment also included an overlap condition in which the fixated stimulus remained present throughout the trial. Whereas our pilot investigation allowed the evaluation of the relative effects of extinguishing visual versus auditory fixation stimuli, the introduction of an overlap condition in the reported experiment allowed for the measurement of facilitation due to ocular disengagement.

2. We would like to thank an anonymous reviewer for suggesting this possibility.

3. We would like to thank an anonymous reviewer for bringing this recently published study to our attention.

4. Note that Shafiq et al. (1998) have claimed that auditory fixation and visual fixation removal do *not* produce equivalent gap effects. In addition to the auditory experiment described in the text, Shafiq et al. included an experiment that extinguished a visual fixated stimulus under step and gap conditions. Their estimates of the magnitude of the gap effect were based on the difference in saccadic latency between step and gap conditions. Using this measure, they reported greater saccadic RT facilitation following visual fixation disappearance than following auditory fixation disappearance and, on these grounds, argued that visual and auditory control of fixation are not equivalent. However, Shafiq et al.'s conclusion is unwarranted. First, the gap effect is defined as saccadic facilitation due to the removal of a fixated stimulus. By taking the difference between step and gap conditions—both of which involve the removal of fixation—Shafiq et al. have failed to isolate the effects of fixation removal per se. Even more critically, however, under auditory fixation they presented subjects with auditory targets, whereas under visual fixation, they presented subjects with visual targets. In other words, their comparison of auditory versus visual control of fixation was confounded with target modality. Our design eliminates this confound and finds no differences between auditory versus visual control of fixation.

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