



Afferent delays and the mislocalization of perisaccadic stimuli

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Abstract

Determining the precise moment a visual stimulus appears is difficult because visual response latencies vary. This temporal uncertainty could cause localization errors to brief visual targets presented before and during eye movements if the oculomotor system cannot determine the position of the eye at the time the stimulus appeared. We investigated the effect of varying neural processing time on localization accuracy for perisaccadic visual targets that differed in luminance. Although systematic errors in localization were observed, the effect of luminance was surprisingly small. We explore several hypotheses that may explain why processing delays are not more disruptive to localization performance. © 2001 Elsevier Science Ltd. All rights reserved.

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1. Introduction

Neural information processing is not instantaneous. It takes time for sensory information to be detected by the central nervous system, and even more time to use that information to generate an appropriate motor response. In the visual system, response latencies can be quite long. For example, the response latency of V1 neurons ranges from 30 to over 70 ms (e.g. Maunsell & Gibson, 1992; Schmolesky et al., 1998). Saccadic eye movements are fast enough to move the eyes as much as 20° during this period of time. Thus, if a brief visual stimulus occurs immediately prior to a saccade, the eyes can move a substantial distance before information about the target arrives at any given point within the central visual pathways. Indeed, stimuli flashed briefly just before and during eye movements are mislocalized, at least under conditions of complete darkness (e.g. Matin, Matin, & Pola, 1970; Matin, 1972; Honda, 1989; Dassonville, Schlag, & Schlag-Rey, 1992).

Accurate visual localization relies on the integrity of two sources of information: the retinal location of the stimulus and the position of the eyes with respect to the world. Retinal stimulus location can be readily deci-

phered from the distribution of neural activity in the retinotopic maps characteristic of so many of the structures that comprise the early visual pathways. Neural signals representing eye position may be derived from an efference copy of oculomotor commands, from reafferent proprioceptive feedback, or both. Imprecise information about either the retinal location of the stimulus or position of the eyes *at the moment the stimulus appeared* will lead to misperceptions of the target's location.

The magnitude of the visual processing delay should therefore adversely affect the localization of perisaccadic stimuli, by varying degrees of temporal mismatch between retinal and eye position information (e.g. Matin & Pearce, 1965; Dassonville et al., 1992; Schlag & Schlag-Rey, 1995). Manipulating the processing time of the targets should cause systematic changes in the magnitude and timing of the errors. Longer processing delays should lead to larger localization errors because the eye would move through a greater distance during longer, as compared to shorter, latencies. For the same reason, longer visual latencies should also increase the period of time over which errors occur.

We investigated the role of visual processing delays in localization by varying the luminance of brief visual targets presented around the time of a saccade. The magnitude and time course of the localization errors for the target whose luminance varied were the principal

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measures of interest. Since visual latencies are inversely proportional to luminance (Lennie, 1981), we predicted larger localization errors to dim targets than bright targets because the eyes can travel farther during the longer visual processing delay associated with the dimmer stimuli.

Surprisingly, we found only a very small effect of luminance on the magnitude of localization errors. Although it was statistically reliable, the difference in errors was not as large as we predicted based on the estimated average latencies of the bright and dim targets. This minimal effect of luminance coupled with the prolonged time course of the errors cannot be explained by a simple model involving the combination of retinal and eye position information that are mismatched in time, but are otherwise accurate. When we attribute the errors to the sluggishness of the neural representation of eye position as others have suggested (e.g. Matin et al., 1970; Matin, 1972; Dassonville et al., 1992), the fit is somewhat improved between the model and the data. However, the degree of dampening needed to improve the fit is unrealistic, and further, we find no evidence of a sluggish eye position signal when subjects localize perisaccadic sounds. In sum, our results suggest that the errors in localizing visual stimuli presented in close temporal proximity to saccades cannot be fully accounted for by any of the existing hypotheses.

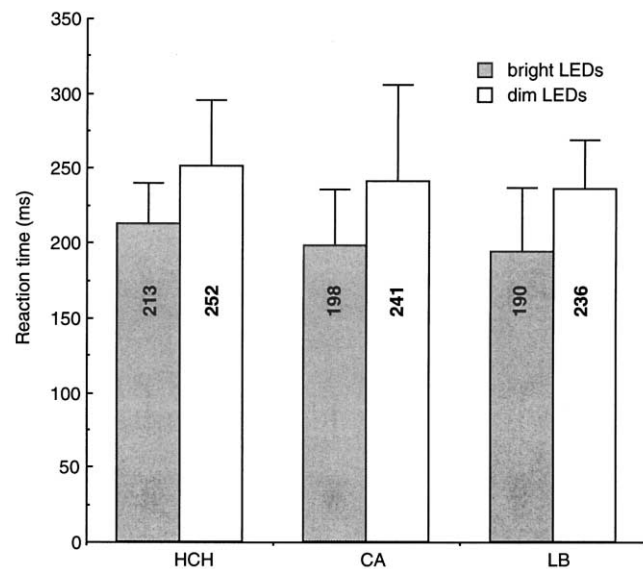


Fig. 1. Saccade reaction time of each subject for bright and dim targets in a simple saccade task. The bars indicate 1 S.D. For all three subjects, the difference in RT was approximately 40 ms.

2. Methods

2.1. Subjects

Four human subjects participated in these experiments (CA, HCH, LB, and LS). Each subject had normal or corrected to normal visual acuity and hearing. Each was informed about the nature of the recording procedures, the general goals of the experiment, and signed an informed consent document. The Dartmouth College Committee for the Protection of Human Subjects approved the experimental protocol followed in this report.

2.2. Experimental procedures

2.2.1. Estimating processing times

A saccadic reaction time task was used to obtain an estimate of the difference in visual latency for high and low luminance targets. A fixation LED (5.1×10^2 cd m^{-2}) was presented directly in front of the subject at ($0^\circ, 0^\circ$) and remained illuminated for a variable duration (300–500 ms). Subjects were instructed to steadily direct their gaze on the fixation LED until it was extinguished and a target LED was illuminated. Subjects then had to redirect their gaze as quickly and accurately as possible to the target LED. Targets were located at an elevation of 8.5° and an eccentricity of 8.5° in either the left or right visual field (randomly interleaved). Flash duration was 1 ms, and the luminance was either low (30 cd m^{-2}) or high (4.2×10^4 cd m^{-2}).

We defined the saccadic reaction time as the time that elapsed between target onset and saccade onset. We computed the arithmetic mean reaction time to the bright and dim targets for each subject using a sample of at least 65 saccades in each condition. The difference between the mean reaction times to the bright and dim targets was taken as the estimate of the difference in the average processing times for these two target luminances (Fig. 1), and the variance of the reaction time distributions was taken as the estimate of the variability in visual latency. For all subjects, the difference in mean reaction time was approximately 40 ms.

2.2.2. Evaluating localization accuracy

The experimental tasks consisted of two versions of the double-step paradigm (Hallett & Lightstone, 1976a,b; Becker & Jürgens, 1979) (Fig. 2). In Experiment I (a in Fig. 2), two visual targets were presented in rapid succession. In Experiment II (b in Fig. 2), the second target was either visual or auditory. In both experiments, subjects were instructed to direct their gaze to the location of each successive stimulus as accurately as possible. Trials began with the presenta-

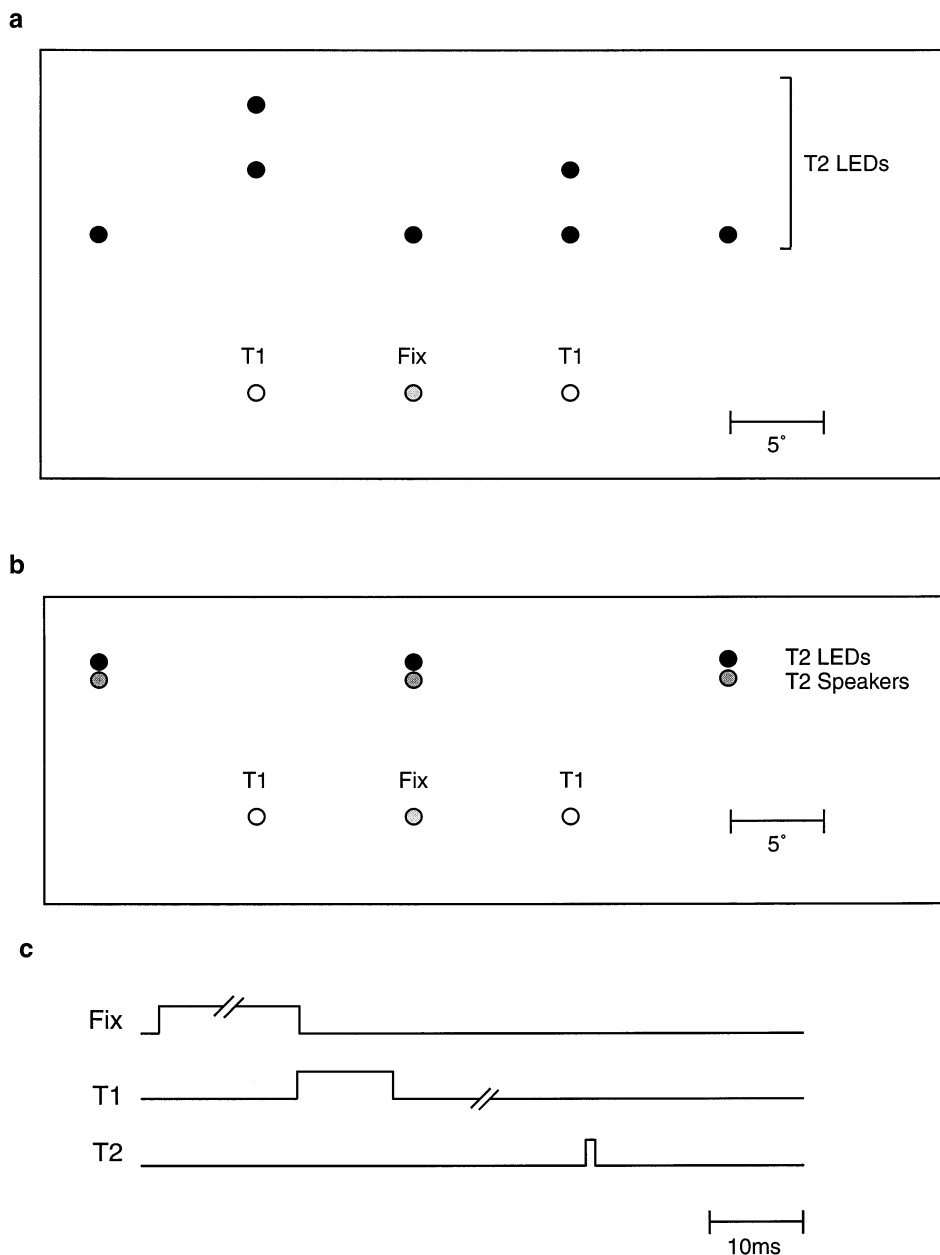


Fig. 2. A schematic representation of the events in the double step task. Panel A shows the spatial layout of the targets for Experiment I, while panel B shows the spatial configuration of targets for Experiment II. FIX refers to the fixation target used on every trial; T1 and T2 refer to the first and second target locations, respectively. Panel C shows the events of the task in time. The duration of the fixation stimulus was 1200–1700 ms, and the interval between T1–T2 was 100–550 (Experiment I) or 50–550 ms (Experiment II, see text for details). Subjects were instructed to make a saccade to each target as accurately as possible.

tion of a fixation LED (1200–1700 ms duration). In Experiment I, the fixation light remained on until the first target (T1) appeared 8.5° to the left or right (10 ms duration). The second target (T2; 1 ms duration) was presented 100–550 ms after T1 was extinguished. The luminance of the fixation point and T1 were both $5.1 \times 10^2 \text{ cd m}^{-2}$. The luminance of T2 varied on a trial-by-trial basis and was either 30 cd m^{-2} (low luminance condition) or $4.2 \times 10^4 \text{ cd m}^{-2}$ (high luminance condition).

In Experiment II, the second target (T2) could be either visual or auditory, and was presented 50–550 ms after T1 was extinguished. On visual-auditory trials, T2 was a click (1 ms duration). As a control for these visual-auditory trials, blocks of visual-visual trials were presented in the same target locations with the same timing parameters. The luminance of the T2 LED was $2.8 \times 10^3 \text{ cd m}^{-2}$ and the intensity of the T2 click was 72 dB. The experiments were run in complete darkness leaving no visual landmarks to serve as localization cues.

2.2.3. The analysis of localization accuracy

The variable of primary interest is the accuracy of the saccade to T2 as a function of the temporal relationship between T2 and the first saccade (to T1). We refer to the interval between T2 onset and the saccade to T1 as the *delay time*. Negative delays denote cases in which T2 was presented before the saccade to T1, and positive delay times denote cases in which T2 was presented after the onset of the saccade to T1.

2.2.4. Recording procedures

Horizontal and vertical eye position was monitored using the scleral search coil technique (Robinson, 1963). Head position was stabilized using a chin rest. We also monitored head position using a second search coil placed on the forehead. The latter recordings confirmed that the subjects kept their heads stationary, and were not analyzed further. The resolution of the recording system is 10 minutes of arc. Eye and head position was digitized (16-bit resolution, 250 Hz) and stored on disk. A calibration procedure required fixation of each possible target position; calibration data were obtained at the beginning of each experimental session. These data were used to transform the raw voltage records to measures of angular displacement. Saccades were detected automatically using a velocity criterion ($37.5^\circ \text{ s}^{-1}$). As suggested by the manufacturer of the scleral coils (Skalar Medical), experimental sessions were confined to 30 minutes and subjects were tested on alternate days. All data analyses were performed offline.

2.3. Data analysis

During an initial perusal of the data we discovered that the accuracy of each subjects' saccades to T2 varied slightly with the position of T1, even when T2 was presented well after the eyes had arrived at the T1 location. These biases due to the initial eye position were not affected by target luminance, and involved primarily saccade overshoot in two subjects (HCH and LB) and saccade undershoot in the third (CA). We controlled for these biases using the following procedure. Prior to pooling the data across stimulus location, we calculated a baseline eye position error for each combination of T1 and T2 location, independent of the luminance of T2. To avoid including perisaccadic errors into this correction, the correction procedure was performed using data from those trials in which T2 came on at least 150 ms after the beginning of the saccade to T1 (i.e., a delay time of at least +150 ms). Thus, the data using long positive delay times represents the baseline accuracy of the second saccade when little interference is expected from the initial saccade. The average baseline errors for each condition were then subtracted from all the data for that condition. This

correction only served to normalize the baseline errors at long delay times to zero. It did not change the overall pattern of perisaccadic errors.

3. Results

3.1. A simple target localization model

We first generated predictions for how the magnitude and timing of saccadic localization errors should vary as a function of stimulus luminance in the double-step paradigm. In order to derive these predictions, we began by assuming that the saccadic control system has access to a veridical representation of eye position as it changes over time. We also assumed that in order to compute the location of T2, a signal specifying the location of T2 on the retina must be combined with this veridical eye position signal. Finally, we assumed that there is no compensation for any of the delays in the neural response to T2. Thus, this model predicts the pattern of errors that would occur if all errors were due to delays in visual processing.

Before we could actually implement the model, we had to estimate the afferent delays in the neural response to T2. We assumed that it takes a minimum of 34 ms for the retinal location signal to arrive at a point in the nervous system where it can be combined with eye position information. This 'base time' corresponds to the shortest latency of cells in V1 (c.f. Maunsell & Gibson, 1992; Schmolesky et al., 1998). The choice of this latency is somewhat arbitrary, but plausible as recent research has shown that eye position signals modulate responses in V1 (Trotter & Celebrini, 1999). Thus, retinal inputs appear to coexist with eye position information in this area. Based on the saccadic reaction times to the bright and dim targets for each subject (Fig. 1, also see Section 2), we assumed an additional luminance-dependent delay of 39 (HCH), 40 (LB) or 43 (CA) ms (i.e. a total latency of 34 ms for bright targets, 73–77 ms for dim targets).

These parameter values were used to generate the predicted pattern of errors illustrated in Fig. 3. Because all targets are processed with some non-zero afferent delay, localization errors occur over a range of pre-saccadic intervals. The specific range is determined by the visual latency for that target and the duration of the subsequent saccade. Since the response latency for dim targets is longer than the latency for bright ones, the errors to dim targets begin to occur before the errors to bright targets (measured relative to the onset of the saccade). The relative temporal offset in the timing of these errors is determined by the difference in their visual latencies, which we estimated to be about 40 ms for this subject. The errors to both types of stimuli end at the end of the saccade. This is because afferent

delays have no impact on localization accuracy once the eye is stationary. Finally, because the eyes can travel farther during a longer latent period, the magnitude of the simulated errors is greater for dim stimuli than for bright stimuli. The results of these simulations provided us with a context for considering our empirical results, which are described in Section 3.2.

3.2. The effects of target luminance on saccadic localization errors

All of our subjects mislocalized perisaccadic stimuli, but none conformed to the model predictions shown in Fig. 3. The pattern of errors differed from the model prediction in two important respects (Fig. 4). First, the effect of target luminance was not nearly as great as predicted. Luminance did have a small effect however, and it was in the direction expected: errors to dim stimuli tended to be slightly larger than errors to bright stimuli over a large range of negative delay times. We tested the reliability of this difference using a Wilcoxon signed ranks test for delay times ranging from -60 ms

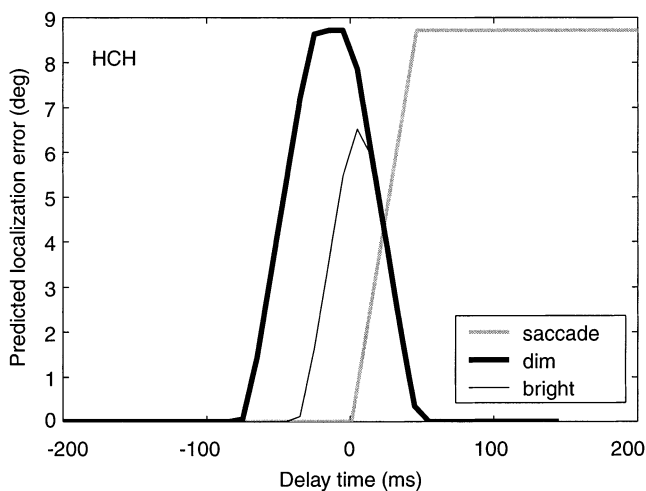


Fig. 3. The errors predicted by a simple model of oculomotor localization that assumes a veridical eye position signal (EPS) is combined with a signal specifying the retinal location of the target. In this model, the retinal signal is delayed by an amount that depends on the luminance of the target. The estimated latencies in this case were those obtained in a saccadic reaction time task for subject HCH. In this simulation, we assumed that a minimum visual latency of 34 ms, and a luminance-dependent latency difference of 39 ms (see text for additional details). The saccade (gray line) begins at time 0. The duration and the amplitude of the saccade conformed to the average values for this subject's average saccade to T1. The heavy solid line shows the pattern of errors predicted using dim targets, and the light solid line shows the pattern of errors using bright targets (averaged in 10 ms bins). Negative and positive delay times indicate the target was presented before or after the eyes actually began to move. Errors with positive values indicate the error is in the same direction as the saccade. Errors with negative values would indicate errors in the direction opposite the saccade (but note that the model does not predict errors in the opposing direction).

to 0 ms, the interval over which our model predicts a difference. For all subjects, the dim targets produced a higher mean error than the bright targets ($P = 0.028$ for CA; $P = 0.018$ for HCH; $P = 0.028$ for LB). A paired t-test in which bright and dim trials were matched by delay time confirmed this finding ($P = 0.018$ for CA; $P = 0.030$ for HCH; $P < 0.001$ for LB). Although the effect is statistically reliable, it must be acknowledged that it is also quite small, especially when compared to the model predictions.

The second major discrepancy between the model and the observed pattern of errors was in their overall time course. Errors began more than 150 ms prior to the saccade, whereas the model predicts accurate performance for targets appearing as late as 70 ms prior to the saccade. The slope of the errors as a function of the time prior to the saccade was also much shallower than what is predicted by our simulations. The temporal pattern of errors we observed is consistent with previous experiments (e.g. Dassonville et al., 1992). In succeeding sections, we will attempt to account for as much of this pattern as possible. Our strategy will be to embellish the model with additional features in an attempt to assess how closely this general class of model can match the empirical data.

3.3. Can variability in afferent delays and saccadic motor output account for the pattern of errors?

Clearly, a model based entirely on average values of afferent delays cannot account for localization errors for targets presented as much as 200 ms prior to a saccade. The slow rate of growth in error magnitudes as a function of time is also not readily predicted by average afferent delays. Beyond that, a model based on averages is unrealistic because it is well established that processing latencies are not fixed but vary from trial to trial. This variability in the afferent delays could contribute to the localization errors.

In addition to variability in the afferent latencies, variability in the metrics of the motor output might also contribute to the errors. Specifically, the saccades to T1 vary in their amplitude and velocity on a trial-by-trial basis. It is possible that the variability in saccade metrics might not be represented in the saccadic motor command, but might arise in the execution of the saccade by the motor pathway. If the neural representation of eye position (the eye position signal or EPS) is derived from this invariant motor command, then it would not contain information about the variation in the eye movement itself, and this would in turn affect the pattern of errors.

We incorporated estimates of the variability in the afferent delays and saccade metrics into the next version of the model. We took the variance in the saccade reaction time distributions obtained from our prelimi-

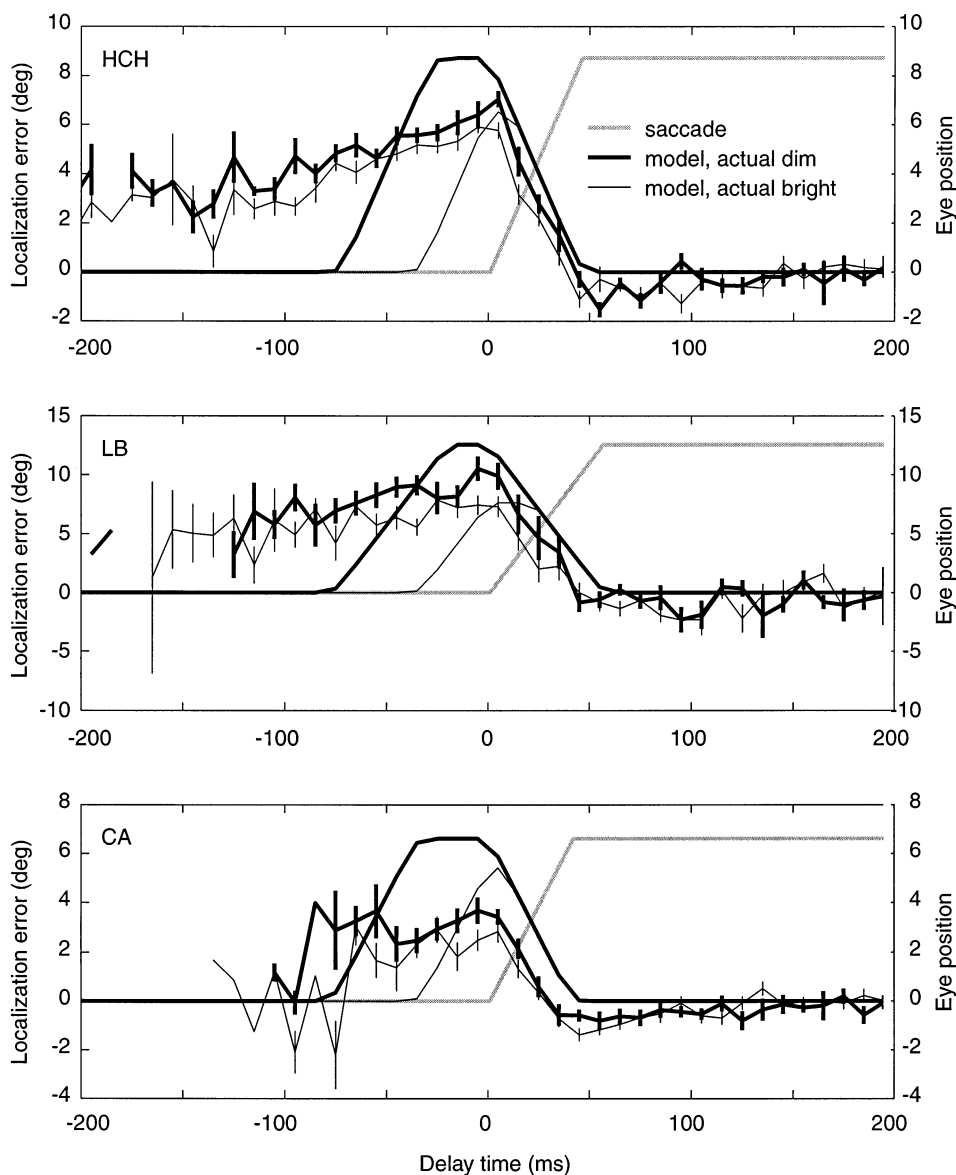


Fig. 4. The actual localization errors as a function of delay time for each subject. The abscissa indicates the relative timing between the onset of T2 and the saccade to T1. The ordinate represents the magnitude of the localization error. Positive errors are in the direction of the saccade to T1 whereas negative errors represent errors in the opposite direction. The lines with vertical marks represent each subject's averaged errors (10 ms time bins) and the standard errors associated with those means. Also included are the simple model predictions for dim and bright targets, and the average saccade to T1 (gray line). The parameters of the model were adjusted to match each subject's average reaction time data and the average magnitude and duration of their T1 saccades.

nary saccade reaction time task as an estimate of the variability in visual processing time for each subject. Since temporal variability in both the sensory and motor pathways contribute to the variance in the saccade reaction time distribution, this estimate probably overestimates the afferent variability, but it is at least a reasonable start. We also measured the variability in the amplitude and velocity of saccades to the T1 and incorporated these sources of variability into the model.

Each trial of the simulation of the modified model randomly selected values for both the visual latency and saccade variance from a normal distribution whose

mean and standard deviation were set to the experimentally obtained estimates. We first constructed an eye trace consisting of an initial fixation period, an 'actual' saccade with a randomly chosen velocity and amplitude, and a period of steady fixation after the saccade. The retinal location (R) of a stimulus at a specified position in the world (S) was then computed by subtracting the position of the eyes (E) at the time of stimulus onset (t) from stimulus location (Eq. (1)). In this and subsequent equations, parameters that incorporate variability are underlined:

$$R(t) = S - E(t, \text{velocity}, \text{amplitude}) \quad (1)$$

The ‘perceived’ location of the stimulus (P) was calculated by assuming that this retinal location was combined with an internal eye position signal (I) whose metrics did not change from trial to trial. That is, the eye position signal did *not* take into account the variations in metrics of the saccade that actually were produced, but was instead based on the average saccade velocity and amplitude. Note, though, that both fixed and variable components of visual processing delays were included at this stage of the simulation by combining the retinal information with a suitably delayed time point in the internal eye position signal. Thus, the perceived location of a target presented at time t is given by the following expression:

$$P(t) = R(t) + I(t + \text{latency, average velocity, average amplitude}) \quad (2)$$

In this simulation, the internal eye position signal does not vary from trial to trial (the ‘average’ saccade metrics are used), but the *actual* saccade produced does (in the form of $E(t, \text{velocity, amplitude})$ from Eq. (1)). To summarize, errors predicted from this model can be attributed not only to visual processing delays but also to the *variability* in both the visual response latency and the metrics of the saccadic response to T1. Eq. (3) combines Eqs. (1) and (2), (again with the parameters that incorporate variability underlined):

$$P(t) = S - E(t, \text{velocity, amplitude}) + I(t + \text{latency, average velocity, average amplitude}) \quad (3)$$

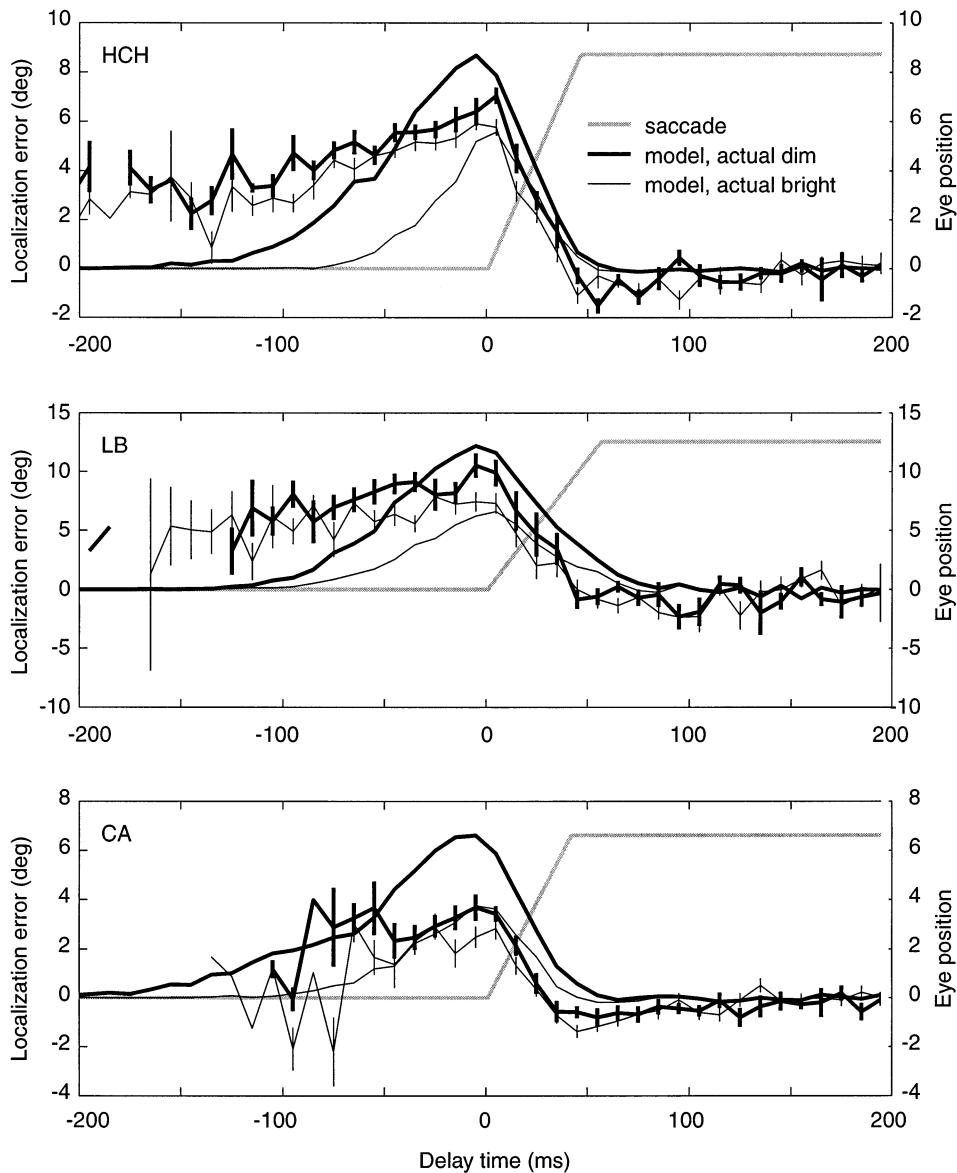


Fig. 5. Predictions from a model modified to incorporate the trial by trial variability in visual latency (for T2) and trial by trial variability in the metrical properties of the saccade to T1. The experimental data are re-plotted from Fig. 4. See text for details. The plotting conventions are the same as in Figs. 3 and 4.

Table 1
Quality of fit of different versions of the model

Subject	Luminance	Simple model (Fig. 4)	Variable model (Fig. 5)	iEPS model (Fig. 6)	Constrained Model (Fig. 7)
<i>(A) Mean squared error</i>					
HCH	High	11.06	8.97	1.23	3.05
	Low	11.34	7.2	1.28	4.3
LB	High	26.91	19.07	3.13	6.43
	Low	22.21	15.46	5.46	8.19
CA	High	3.6	2.58	4.36	11.08
	Low	6.6	4.33	3.28	9.63
<i>(B) Percent reduction in mean squared error (comparison to simple model)</i>					
HCH	High	0	19	89	72
	Low	0	37	89	62
LB	High	0	29	88	76
	Low	0	30	75	63
CA	High	0	28	–21	–208
	Low	0	34	50	–46
Mean		0	30	62	3

(A) The mean squared error of the data with respect to various models was calculated. Because all but the simplest model incorporated variability, no smooth function described the predicted results for those models. Thus, the mean squared errors are estimates derived from one particular run of the simulation for each model, and are taken as the average of the squares of the separation between the observed and predicted localization errors in 10 ms time bins for delay times ranging from –200 ms to 0 ms. The simulation runs involved 30 replications for every delay time at 1 ms resolution, so that each 10 ms time bin included 300 simulation trials. (B) Percent reduction in mean squared error of later models in comparison to the simple model that involved only a temporal mismatch between accurate visual and eye position information. The percent reduction in error was calculated as:

$$\text{percent reduction} = \left(1 - \frac{MSE_{\text{model2}}}{MSE_{\text{model1}}}\right) \times 100 \quad (4)$$

where model 2 is the model in question and model 1 is the simple model. Note that this statistic is equivalent to the R^2 statistic for linear regression.

Including these inherent sources of variance improves the fit of the model to the data (Fig. 5) because adding temporal variability in the afferent delays effectively shuffles the individual points along the time axis. This effectively blurs the transition between steady fixation and the onset of the saccade with the consequence that the lead-time of the errors increases and the slope relating error magnitude with time prior to the saccade decreases. There is a 19–37% improvement in the fit of this variable model over the previous model (Table 1). However, the variable model still predicts a more substantial difference in the errors to bright and dim stimuli than we observed experimentally. Thus, the time course of pre-saccadic errors and the relative absence of an effect of luminance on those errors remain unexplained.

3.4. Does misinformation about eye position contribute to the errors?

The timing of the errors observed in localizing perisaccadic targets lead previous researchers to suggest that the localization errors might result from a severe dampening of the neural representation of eye position. A damped EPS is a distorted representation of the eye position in that it has a slower velocity and a longer

duration than the actual saccade. Given this, the EPS could begin to change well in advance of the actual saccade, and could end after the actual saccade has ended. According to this hypothesis localization errors result in large part from a mismatch between the EPS and the actual eye movement.

We attempted to model the data by incorporating a damped EPS into the simulation. There are, however, inherent problems with this approach. In all of the previous simulations we were able to estimate the relevant parameters (latency, latency variability, saccade amplitude variability, and saccade velocity variability) from measurements that were independent of the data we were hoping to approximate (the time course of the localization errors). Because it is completely internal to the observer, there is no way to measure the possible degree of dampening of the EPS independent of the data we hope to model.

Following previous research (i.e. Dassonville et al., 1992), we estimated the degree of dampening in the EPS that was needed to best fit the data by first calculating an *inferred eye position signal* (iEPS). Normally the inferred eye position signal value for a given trial would be calculated by adding the localization error to the actual eye position at the time the target was presented on that particular trial. This method

includes trial-by-trial variability in saccade metrics. Since we had already included this source of variability, albeit in a different fashion, we used a modified method of estimating the inferred eye position signal. We instead added the measured error to what the eye position would have been on that trial assuming that the saccade had the average velocity and amplitude (the expected value of a fixed EPS). We then fit a regression line to the iEPS using data from dim targets using delay times over the interval from 100 ms before the saccade to the onset of the saccade (-100 ms to 0 ms delay time), a period of time in which the data are roughly linear. The slope and intercept of this regression line was then used to derive the onset time and average

velocity of the ‘saccade’ represented by the iEPS for each subject. This damped iEPS then replaced the accurate internal representation of eye position (I in Eq. (3)) in the previous version of the simulation. The variability described in the previous simulation remained present in the model.

Fig. 6 shows the fit when the EPS is damped so as to optimize the fit to the data. Note that this fit is the best we have achieved (see Table 1). However, a good fit here is not at all surprising since the parameters of the iEPS were obtained by fitting the model to the data we are attempting to simulate. It is therefore difficult to attribute a great deal of significance to the improved fit to the data as such an improvement was inevitable. Yet

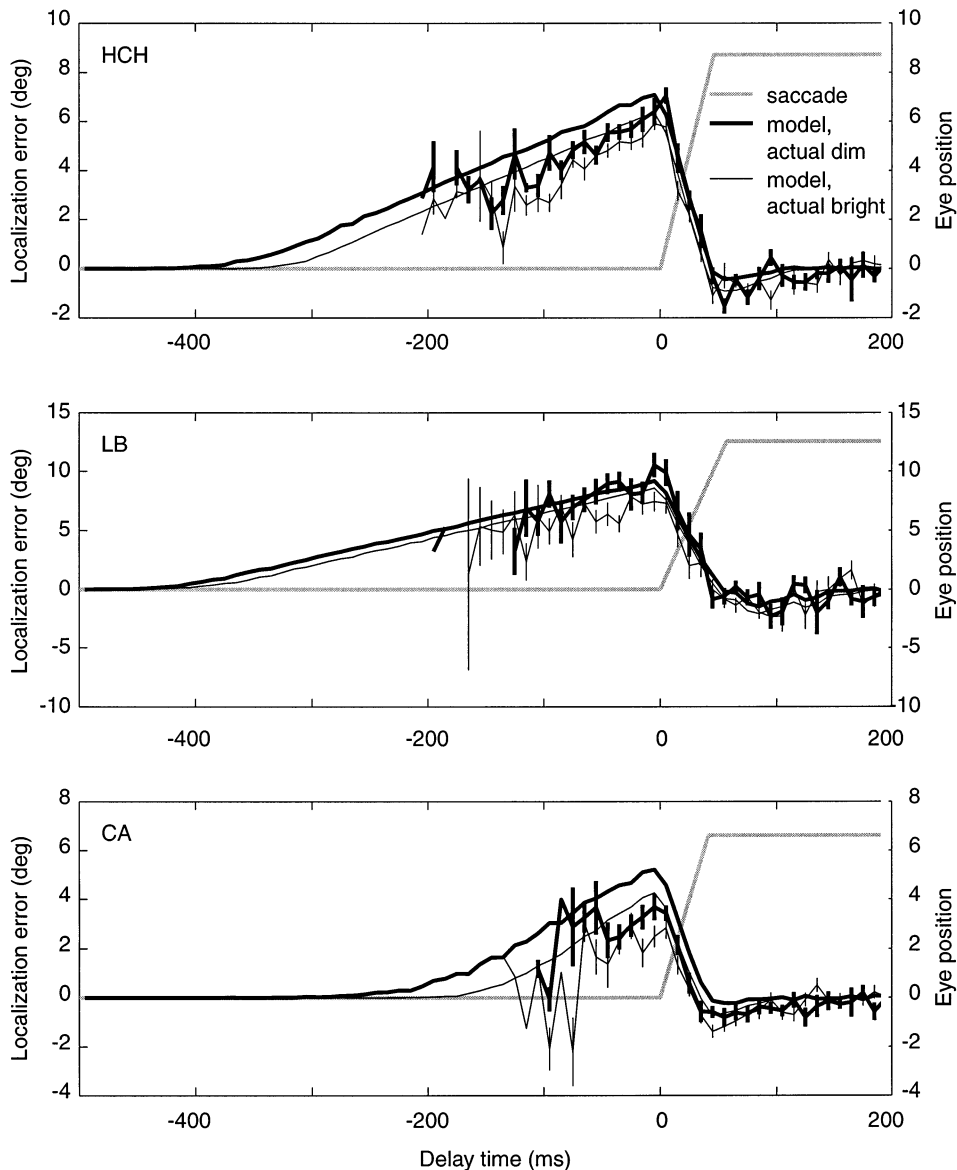


Fig. 6. Predictions from a model which incorporates an inferred eye position signal calculated from the data. The experimental data are re-plotted from Fig. 4. See text for details. The plotting conventions are the same as in Figs. 3–5. For these simulations, the inferred eye position signal contains a saccade that is 9.4 (HCH), 9.9 (LB), and 5.4 (CA) times slower than the actual saccade. The onset of the saccade in the inferred eye position signal begins -279.2 (HCH), -340.2 (LB) and -100.2 (CA) ms before the onset of the real saccade.

in the absence of an independent way to approximate the EPS, there is no alternative method of evaluating the effect of a damped EPS.

Despite the above caveat, there are several noteworthy aspects of this simulation. The first noteworthy feature relates to the major new finding from Experiment 1: the curious lack of a luminance effect on localization performance. The simulation shows that a severely damped EPS actually *predicts* a very modest luminance effect. This is because if eye position (or the eye position signal) changes slowly, it cannot travel much farther during the long latency for dim targets than it does during the shorter latency for bright targets. This minimizes the predicted luminance difference in the error pattern to a point that is at least qualitatively similar to the actual pattern of errors that we observed. Thus, adding a damped EPS to the model produces predictions that are at least qualitatively similar to the actual pattern of errors that we observed.

The second noteworthy feature is the severity of dampening needed to approximate the data. This poses some practical problems for the hypothesis that the errors are due to a damped EPS. The estimated velocity of the iEPS is 5–10 times slower than the velocity of a typical saccade of this magnitude. This difference in velocity between the actual saccade and the iEPS has implications for how far in advance of the saccade the iEPS must begin to change. In two of our subjects (HCH and LB) the internal representation of the saccade begins to change approximately 300 ms before the actual saccade. That is, 300 ms *before* the eye moves toward the target, the neural representation of the eye position (the EPS) begins to change. Consider, however, that the interval between successive saccades is frequently less than 300 ms, and can be as short as 100 ms (e.g. Fischer & Boch, 1983; Reuter-Lorenz, Oonk, Barnes, & Hughes, 1995). Thus, according to these estimates of the EPS, the internal representation of saccade onset could occur before the execution of the previous saccade has been completed. This is highly improbable, as the EPS would contain the summed effects of successive saccades, producing enormous errors in the internal representation of eye position. Under these circumstances, it becomes hard to see how the double step paradigm could be performed with any accuracy at all.

Not only is the time course of the damped EPS inconsistent with typical intersaccadic intervals, but it is also inconsistent with the saccade reaction times we observed in our study, which could also be much shorter than 300 ms. The dampening estimated from our data produce the untenable inference that the EPS could begin to change before the eliciting target was presented. Since this cannot be so, the interval between the onset of T1 and the onset of the impending saccade provides an upper bound on the lead-time of the sac-

cade as represented in the EPS. We repeated the simulation using a damped EPS constrained by the average reaction time of the first saccade. We assumed that the EPS began to reflect the upcoming saccade 34 ms (the latency of visual responses in V1) after the presentation of T1, and completed the ‘saccade’ at the end of the actual saccade. The latency and saccade variability of the previous two versions was also included. As expected, the model does not fit the data quite as well as previously (Table 1, Fig. 7). Thus, even if we accept a damped EPS but simply constrain it to conform to physiological plausibility, there is still considerable discrepancy between the model and the data.

At this point it seemed prudent to approach the problem from a different perspective. We decided to look for confirmatory evidence for the existence of a damped EPS. If the EPS were so drastically damped, it would virtually never be accurate in ordinary circumstances in which saccades occur at rates up to 2–3 per second. Presumably, the effects of such a distorted EPS should be apparent in other contexts. We therefore turned to an investigation of whether perisaccadic localization errors also occur in the double step task when T2 is an auditory stimulus.

3.5. Oculomotor localization accuracy for perisaccadic acoustic targets

Localizing sounds is a fundamentally different process than localizing visual stimuli. Sound localization relies on comparisons between the signals arriving at each ear, and the auditory system is assumed to initially compute the location of the sound with respect to the head. However, neurophysiological studies have shown that eye position influences the response of auditory neurons in classic oculomotor areas such as the superior colliculus (e.g. Jay & Sparks, 1987) and in early auditory processing areas such as the inferior colliculus (Groh, Trause, Underhill, Clark, & Inati, 2001) and auditory cortex (Trause, Werner-Reiss, Underhill, & Groh, 2000). This influence of eye position on auditory responses is thought to reflect a process in which the locations of auditory targets are transformed from head-centered to eye-centered coordinates. Because the direction and amplitude of saccades appears to be specified in eye-centered coordinates in oculomotor areas, this transformation is needed in order to produce a saccade to a sound source. Accordingly, we hypothesized that saccades to sounds should also depend on the internal representation of eye position, and if so, may be mislocalized in the double-step paradigm in a manner analogous to that observed using visual targets.

We used the double step paradigm once again, but this time the second target was either visual or auditory. As Fig. 8 shows, neither of our subjects showed evidence of a systematic pattern of localization errors

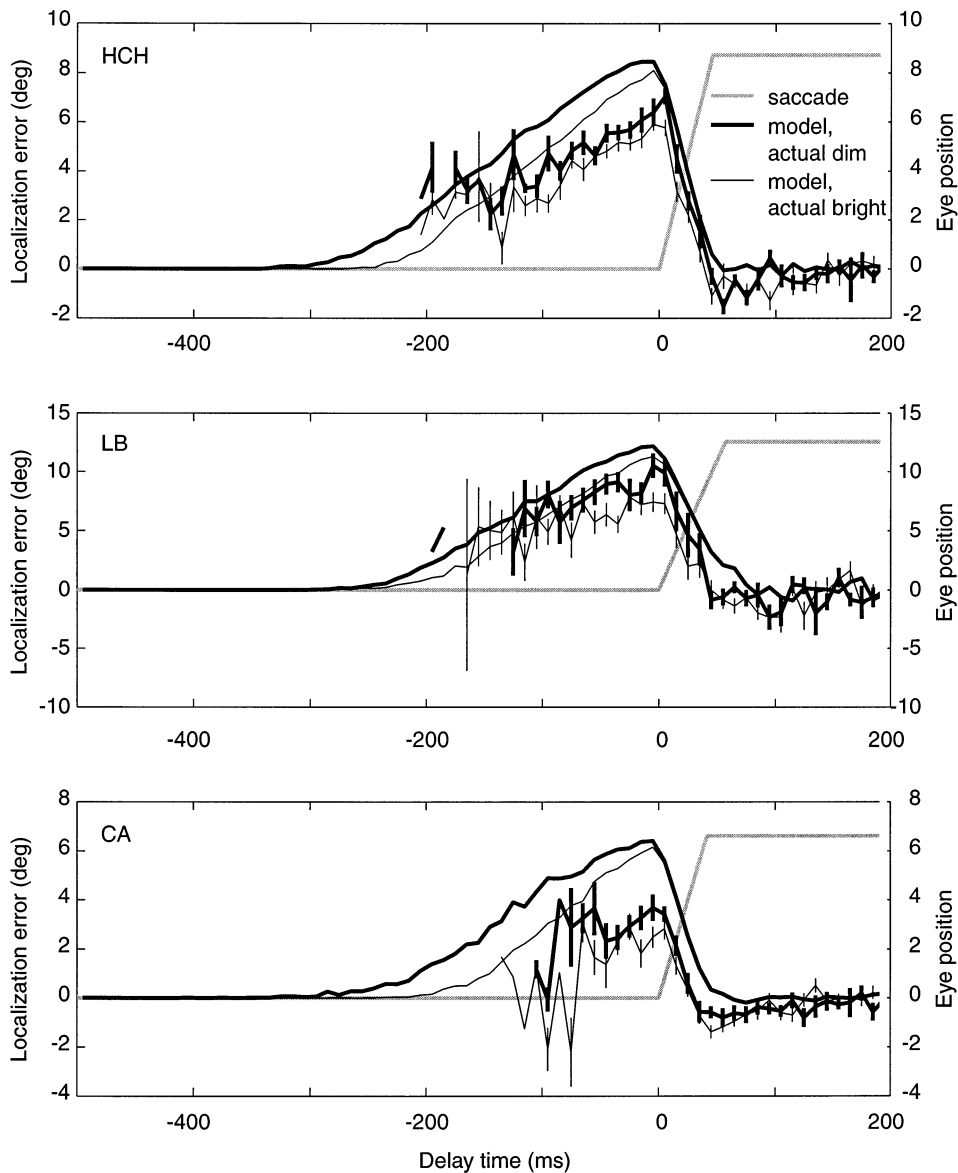


Fig. 7. Predictions from a model which incorporates an inferred eye position signal constrained by the latency of the saccade to the first target. The experimental data are re-plotted from Fig. 4. See text for details. The plotting conventions are the same as in Figs. 3–6. For these simulations, the inferred eye position signal contains a saccade that is 5.0 (HCH), 3.7 (LB), and 4.0 (CA) times slower than the actual saccade. The onset of the saccade in the inferred eye position signal begins -184.3 (HCH), -153.1 (LB) and -122.6 (CA) ms before the onset of the real saccade.

for brief sounds that occurred immediately before a saccade. In other words, there is no indication that the system is utilizing a damped EPS when guiding saccades to auditory stimuli. This finding leaves three possibilities open. One, the EPS is not damped, and something else must account for the mislocalization of perisaccadic visual stimuli. Two, more than one EPS exists: a damped representation for visual localization, and an accurate representation for sound localization. Why this should be so is unclear. Three, there is only one EPS, and it is damped, but when localizing acoustic stimuli, the oculomotor system ‘waits’ until information about eye position becomes accurate (i.e. after the eye has stopped moving) *before* planning a saccade to the

target. This last possibility is discussed further in Section 4.

3.6. Controls

In view of the evidence that saccades may be programmed in parallel (e.g., Becker & Jürgens, 1979), we wondered whether programming of the second saccade might interfere with either the programming or execution of the first saccade. It seemed logically possible that, when two saccades occur in close succession, there might be interactions between them (such as saccade vector averaging) that might contribute to the pattern of errors that we and others have observed. If this were

the case, then the metrics of the saccade to T1 should be affected by the production of a later saccade to T2. A thorough examination of the saccades to T1 found no evidence for a retroactive effect of the saccade to T2 on the saccade to T1. Specifically, the amplitude and velocity of the first saccade were independent of both the interval between the T1 and T2 onsets and the interval between the T2 onset and the first saccade

(delay time). Moreover, variations in the amplitude of the first saccade had no systematic effect on the errors in localizing T2. The two saccades appear to have been generated independently of one another. Perhaps this is not so surprising in view of the fact that our subjects were instructed to maximize the accuracy of their saccades; since there was no premium placed on speed, the intervals between the saccades were relatively long.

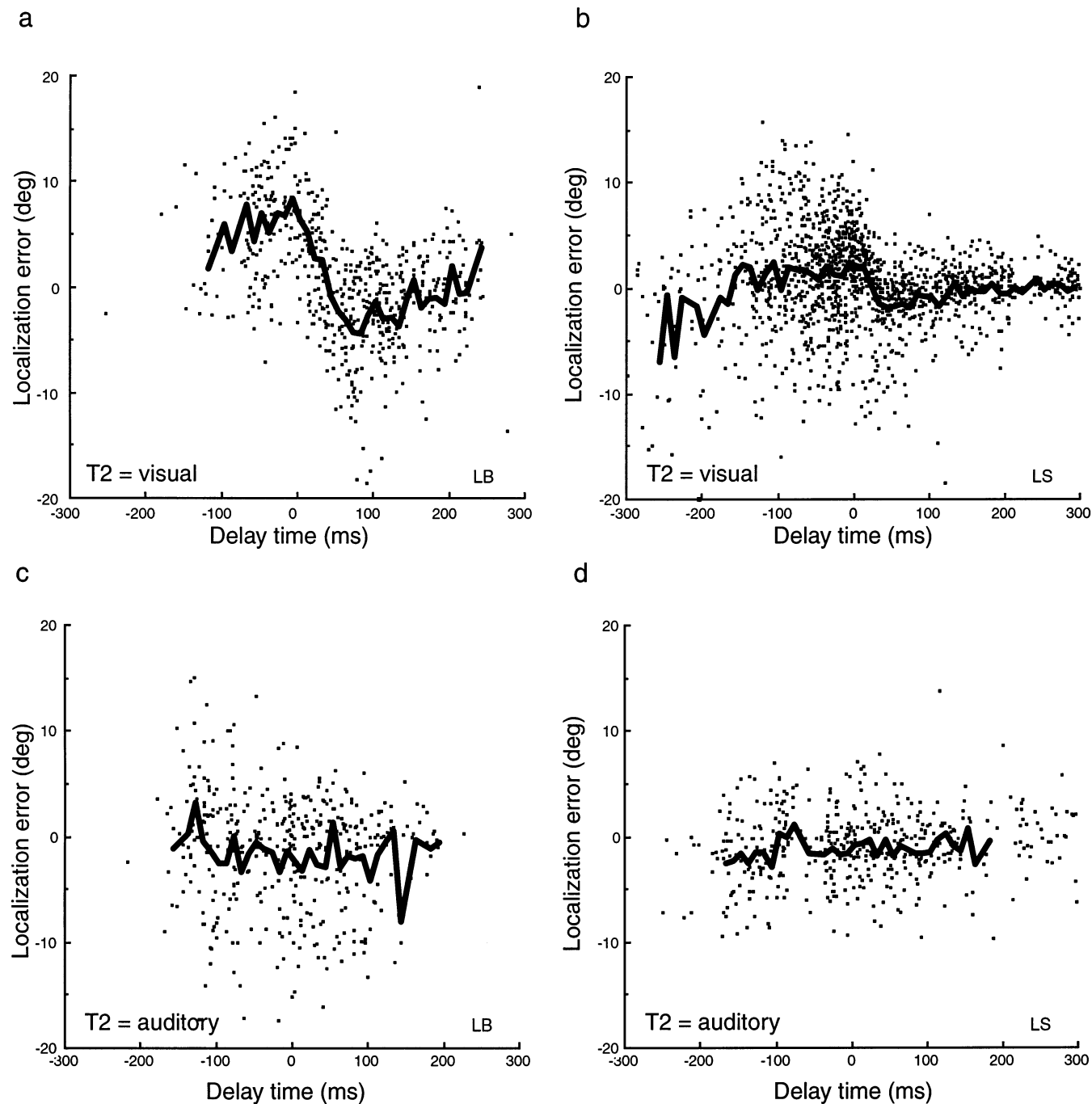


Fig. 8. Perisaccadic localization errors for visual (panels A and B) and auditory targets (panels C and D) in two subjects. Individual points correspond to the error on individual trials and the solid line illustrates the average error in 10 ms time bins. The target configuration shown in Fig. 2 B was used for all data shown here. Plotting conventions are the same as in Figs. 3–6.

4. Discussion

In view of the difficulty in specifying the position of a rapidly moving eye at the precise moment a visual stimulus occurred, it would be surprising if visual stimuli presented around the time of a saccade were *not* mislocalized to some degree. Accurate localization of such perisaccadic stimuli requires two things: a veridical representation of the position of the eye and an ability to ‘back-date’ the visual signal from the moment the target was detected by an amount of time equivalent to the visual response latency. The inherent difficulty of this scenario has long been recognized (e.g. Matin & Pearce, 1965; Dassonville et al., 1992; Schlag & Schlag-Rey, 1995).

Our results show that the localization accuracy of perisaccadic visual targets is not influenced by visual processing delays nearly as much as one might have expected. We initially predicted that the neural delays inherent in processing visual information would result in the mislocalization of a perisaccadic visual stimulus by an amount equivalent to the distance the eyes had traveled during relevant afferent delays. In our experiment we estimated the processing difference between the bright and dim lights to be about 40 ms. At saccadic velocities, the eyes are capable of moving up to 20° during that interval of time. Yet we find a difference in the localization curves of about 1–2°!

One possible explanation for this curious lack of a large luminance effect is that the neural systems involved in visual localization are capable of compensating for delays resulting from changes in stimulus luminance. The brain might indeed ‘back-date’ the detection time of a visual signal by an amount equivalent to the visual latency. How might this happen, since the visual system presumably has no way to measure its own response latency directly? One possibility is that the latency could be estimated using some other parameter of the neural response that is highly correlated with latency. Overall discharge rate of retinal ganglion cells is correlated with flash luminance (Lennie, 1981), and thus might be a candidate. Latency compensation has also been suggested on the basis of several other findings (e.g. Nijhawan, 1994), although this point is understandably controversial (e.g. Purushothaman, Patel, Bedell, & Ogmen, 1998).

Since our data suggest that the system may be able to compensate for *differences* in visual processing delays based on luminance, it is surprising that the system *does not* appear to compensate for the overall latency. In fact, the errors are more severe overall than they should be without any compensation for delay at all. The observed errors were considerably greater than those predicted from a simple model that attributes the errors entirely to temporal misalignments between the two critical sources of information: the retinal locus of the

target and the position of the eyes (Fig. 4). Measured relative to the onset of a previous saccade, localization errors began well before the model predicted they should and the magnitudes of the errors were generally larger than expected. This was the case even when the model incorporated variability in visual latency and saccade metrics (Fig. 5).

The fit between the model and the data can be substantially improved by assuming that the neural representation of eye position is damped, which has the interesting side effect of reducing the predicted effects due to afferent delays (Figs. 6 and 7). The degree of dampening required for this improvement is quite severe however, and we regard it as implausible based on several considerations. First, given the rate at which saccades naturally occur, the EPS would be in a virtually continuous state of flux. Second, if the EPS is so dramatically different from the actual eye position, and if it is indeed in a constant state of flux, why does it not produce a similar pattern of errors for sounds presented around the time of a saccade (Fig. 8)?

Admittedly, the situation for saccadic localization of sounds is qualitatively different than for visual stimuli, since there is no intrinsic need to know the precise position of the eyes at the moment the sound occurred. There is, however, ample evidence that the brain must eventually calculate the position of a sound with respect to the eyes before a saccade to that sound can be made (Jay & Sparks, 1984, 1987, 1990; Groh & Sparks, 1992; Russo & Bruce, 1994; Stricanne, Anderson, & Mazzone, 1996). In fact, this process appears to begin within the auditory pathway proper: eye position modulates auditory activity as early as the inferior colliculus (Groh et al., 2001). Thus, an accurate EPS is eventually needed to localize sounds as well. Although it is possible that the system may wait to sample the EPS until it is accurate before making a saccade to an acoustic stimulus, this requires the assumption that the saccadic control system ‘knows’ when the EPS has become accurate. Further, given the above considerations of the severity of the dampening and its consequences on the time over which the EPS is inaccurate, the periods of accuracy would indeed be few and far between.

Thus, the severity of perisaccadic visual mislocalization remains a conundrum. Our data show that differential neural processing delays which result from varying stimulus luminance do not make a large contribution to the pattern of errors. We have attempted to model the data with as many parameters as possible including variability in visual response latencies, variability in the saccadic response, and a possible mismatch between the actual eye position and the neural representation of that eye position. None of these explanations seem to capture the data fully. Therefore, we are left with the conclusion that there are no extant theories capable of providing a completely convincing

explanation for the accuracy of localization in the presence of eye movements.

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References

- Becker, W., & Jürgens, R. (1979). An analysis of the saccadic system by means of double step stimuli. *Vision Research*, *19*, 967–983.
- Dassonville, P. R., Schlag, J., & Schlag-Rey, M. (1992). Oculomotor localization relies on a damped representation of saccadic eye displacement in human and non-human primates. *Visual Neuroscience*, *9*, 261–269.
- Fischer, B., & Boch, R. (1983). Saccadic eye movements after extremely short reaction times in the monkey. *Brain Research*, *260*, 21–26.
- Groh, J. M., & Sparks, D. L. (1992). Two models for transforming auditory signals from head-centered to eye-centered coordinates. *Biological Cybernetics*, *67*, 291–302.
- Groh, J. M., Trause, A. S., Underhill, A. M., Clark, K. R., & Inati, S. (2001). Eye position influences auditory responses in primate inferior colliculus. *Neuron*, *29*, 509–518.
- Hallett, P. E., & Lightstone, A. D. (1976a). Saccadic eye movements due to stimuli triggered during prior saccades. *Vision Research*, *16*, 99–106.
- Hallett, P. E., & Lightstone, A. D. (1976b). Saccadic eye movements to flashed targets. *Vision Research*, *16*, 107–114.
- Honda, H. (1989). Perceptual localization of visual stimuli flashed during saccades. *Perception and Psychophysics*, *45*, 162–174.
- Jay, M. F., & Sparks, D. L. (1984). Auditory receptive fields in primate superior colliculus shift with changes in eye position. *Nature*, *309*, 345–347.
- Jay, M. F., & Sparks, D. L. (1987). Sensorimotor integration in the primate superior colliculus. II. Coordinates of auditory signals. *Journal of Neurophysiology*, *57*, 35–55.
- 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. C. Stebbins, *Comparative perception: basic mechanisms*, vol. 1 (pp. 351–374). New York: Wiley.
- Lennie, P. (1981). The physiological basis of variations in visual latency. *Vision Research*, *21*, 815–824.
- Matin, L. (1972). Eye movements and perceived visual direction. In D. Jameson, & L. M. Hurvich, *Handbook of sensory physiology: visual psychophysics*, vol. 7(4). New York: Springer.
- Matin, L., & Pearce, D. (1965). Visual perception of direction for stimuli flashed during voluntary saccadic eye movements. *Science*, *148*, 1485–1488.
- Matin, L., Matin, E., & Pola, J. (1970). Visual perception of direction when voluntary saccades occur: II. Relation of visual direction of a fixation target extinguished before a saccade to a subsequent test flash presented before the saccade. *Perception and Psychophysics*, *8*(1), 9–14.
- Maunsell, J. H. R., & Gibson, J. R. (1992). Visual response latencies in striate cortex of the macaque monkey. *Journal of Neurophysiology*, *68*(4), 1332–1344.
- Nijhawan, R. (1994). Motion extrapolation in catching. *Nature*, *370*, 256–257.
- Purushothaman, G., Patel, S. S., Bedell, H. E., & Ogmen, H. (1998). Moving ahead through differential visual latency. *Nature*, *396*, 424.
- Robinson, D. A. (1963). A method of measuring eye movement using a sclera search coil in a magnetic field. *IEEE Transactions on Biomedical Engineering*, *10*, 137–145.
- Reuter-Lorenz, P. A., Oonk, H. M., Barnes, L. L., & Hughes, H. C. (1995). Effects of warning signals and fixation point offsets on the latencies of pro- versus antisaccades: implications for an interpretation of the gap effect. *Experimental Brain Research*, *103*(2), 287–293.
- Russo, G. S., & Bruce, C. J. (1994). Frontal eye field activity preceding aurally guided saccades. *Journal of Neurophysiology*, *71*(3), 1250–1253.
- Schlag, J., & Schlag-Rey, M. (1995). Illusory localization of stimuli flashed in the dark before saccades. *Vision Research*, *35*(16), 2347–2357.
- Schmolesky, M. T., Wang, Y., Hanes, D. P., Thompson, K. G., Leutgeb, S., Schall, J. D., & Leventhal, A. G. (1998). Signal timing across the macaque monkey visual system. *Journal of Neurophysiology*, *79*, 3272–3278.
- Stricanne, B., Andersen, R. A., & Mazzoni, P. (1996). Eye-centered, head-centered, and intermediate coding of remembered sound locations in area LIP. *Journal of Neurophysiology*, *76*, 2071–2076.
- Trause, A. S., Werner-Reiss, U., Underhill, A. M., & Groh, J. M. (2000). Effects of eye position on auditory signals in primate auditory cortex. *Society for Neuroscience Abstracts*, *26*, 1977.
- Trotter, Y., & Celebrini, S. (1999). Gaze direction controls response gain in primary visual-cortex neurons. *Nature*, *398*, 239–242.