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Short-Latency Fixational Saccades Induced by Luminance Increments

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Horwitz, Gregory D. and Thomas D. Albright. Short-latency fixational saccades induced by luminance increments. *J Neurophysiol* 90: 1333–1339, 2003; 10.1152/jn.00146.2003. We investigated the effect of peripheral visual stimulation on small-amplitude saccades that occur naturally during fixation. Two macaque monkeys were rewarded for fixating while a colorful stimulus flickered randomly in the periphery. Reverse correlation revealed a lawful relationship between the stimulus sequence and saccade occurrences: on average, a transient increase in stimulus intensity evoked saccades at a latency of ~ 70 ms. The spectral tuning of this increase was roughly, but not exactly, consistent with a pure luminance increase. We conclude that peripheral luminance increases can evoke fixational saccades.

INTRODUCTION

Peripheral visual stimuli can capture attention, but whether such stimuli obligatorily engage the oculomotor system is a matter of active debate. Allocation of visual attention is closely yoked to the generation of saccadic eye movements (Corbetta et al. 1998; Kustov and Robinson 1996; Theeuwes et al. 1999). On the other hand, a subject may attend covertly to a peripheral visual stimulus while maintaining visual fixation, indicating disengagement of the attentional and saccadic systems (e.g., Posner et al. 1980). The extent of this disengagement is unclear; for example, it is unknown whether peripheral visual stimuli influence the occurrence of small-amplitude saccades that occur naturally during fixation. The present experiments address this issue.

Theeuwes et al. (1998, 1999) demonstrated that abrupt onsets can attract visual attention and induce involuntary saccades. Subjects in their task were instructed to make saccades to color-defined targets among distractors. The sudden appearance of a distractor during the subject's reaction time frequently attracted a short-latency saccade of which the subject later claimed to be unaware. Theeuwes et al. hypothesized that these short-latency saccades derive from a fast, reflexive pathway that initiates saccades to novel stimuli even when such saccades are inappropriate.

Tse et al. (2002) asked whether sudden onsets elicit reflexive eye movements in subjects instructed explicitly to maintain fixation. In contrast to Theeuwes' result, they found that subjects' eye positions were not related clearly to the occurrence of sudden onsets. Their analysis, however, was restricted to mean eye position and may therefore have masked important relationships between onsets and saccade occurrences. We used a saccade-triggered analysis technique to assess directly the impact of a visual stimulus on saccade initiations.

We studied small-amplitude saccades made by monkeys during fixation while a visual stimulus flickered randomly in the periphery. Monkeys were rewarded for maintaining their eye position within a small electronically defined window: large-amplitude saccades arrested trials and resulted in brief time-out periods. Cross-correlation of the stimulus sequence with the occurrence of small saccades within the fixation window revealed a systematic relationship between the two: on average, a fixational saccade followed a transient increase in luminance at a latency of ~ 70 ms. Saccade directions were distributed broadly with a slight bias away from the stimulus. Our results demonstrate that peripheral luminance increments can evoke saccades even when the task demands do not require saccades at all.

METHODS

Subjects

Two monkeys (*Macaca mulatta*) served as subjects in these experiments. In an initial surgical procedure, performed with sterile technique and under general anesthesia, each monkey was implanted with a stainless steel head post and monocular scleral search coil (Judge et al. 1980). During experimental sessions, the head post was secured to a mating piece on the monkeys' chair to eliminate head movements. Eye position was digitized at 250 Hz for off-line analysis. Experimental protocols were approved by the Salk Institute Animal Care and Use Committee, and conform to U. S. Department of Agriculture regulations and to the National Institutes of Health guidelines for the humane care and use of laboratory animals.

Behavioral paradigm

Juice rewards were provided on a random schedule while the monkeys maintained fixation on a $0.2 \times 0.2^\circ$ black square presented against a gray background (CIE coordinates: $x = 0.33$, $y = 0.33$, 65 cd/m^2) on a computer monitor (Sony F500). On stimulated trials, a 3° diam stimulus disk was presented at an eccentric location 500 ms after the monkeys acquired the fixation point. Importantly, the stimulus was irrelevant to the monkeys' behavior; rewards were given for fixation only, and the monkeys had never been trained to saccade to this stimulus or use it otherwise in any behavioral task. In some sessions, stimulated trials were randomly interleaved with nonstimulated trials, which proceeded identically except that the stimulus never appeared. If the eye position left a $1 \times 1^\circ$ electronically defined window surrounding the fixation point, the stimulus and fixation point disappeared and data collection ceased. After 1 s, the fixation point reappeared and the next trial began. Trials concluded also if the animal maintained fixation for 10 s whereupon a reward was given.

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Visual stimulation

The color of the stimulus disk, which was determined by the intensities of the red, green, and blue monitor phosphors, changed randomly and independently on every screen refresh (every 10 ms), as illustrated in Fig. 1A. Phosphor intensities were drawn from independent quantized and truncated Gaussian distributions, shown in Fig. 1B. The mean intensity of each phosphor in the patch was identical to that phosphor's contribution to the background. In most experiments, the SD of each distribution was 9% of the range physically achievable. This range corresponds to luminance contrasts of 3.9, 11.5, and 2.5% for the red, green, and blue phosphors, respectively.

One monkey (*monkey C*) viewed the stimulus through a tunnel lined with aluminum foil to increase retinal illuminance and stabilize adaptation state (Chichilnisky and Wandell 1999). For this animal, the average position of the stimulus was 7° left and 1° below the fixation point. During most of these sessions, electrodes were inserted transdurally to record the electrical activity of visual cortical neurons. For *monkey T*, the stimulus was presented consistently 7° to the right and 1° below the fixation point and electrodes were not inserted. Figure 1C illustrates the distribution of stimulus positions. The foil-lined tunnel was omitted for *monkey T* to control for the possibility that results obtained in *monkey C* were related to global luminance changes caused by stimulus reflection.

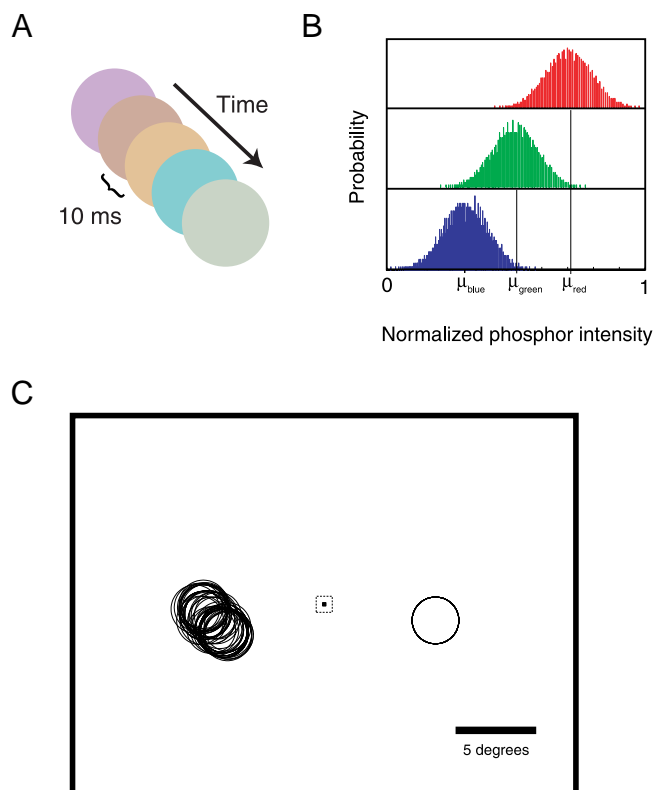


FIG. 1. Dynamic color stimulus. *A*: the stimulus was a 3° diam disk that changed color every 10 ms. *B*: color changes were achieved by adjusting, on every screen refresh, the intensity of the red, green, and blue monitor phosphors according to independent approximately Gaussian probability distributions. Intensity is expressed as the proportion of the maximum intensity achievable for each phosphor. Red and green probability densities have been shifted vertically. *C*: stimuli (\circ) were placed in several locations to the left of the fixation point (\blacksquare) for *monkey C*, and in 1 location to the right of the fixation point for *monkey T*. The screen subtended $31 \times 23^\circ$ (outer border). Monkeys were required to maintain fixation within a $1 \times 1^\circ$ electronically defined window around the fixation point (---).

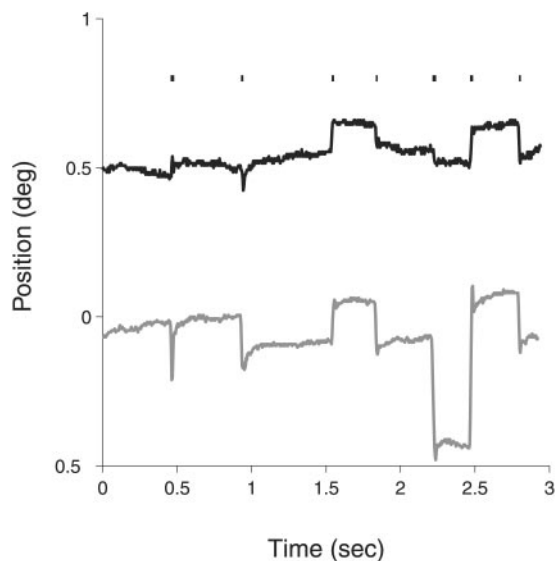


FIG. 2. Sample eye position record. Horizontal (black) and vertical (gray) eye position as a function of time. Nominal fixation is composed of periods of relatively stable gaze, smooth drifts, and small-amplitude saccades. Tick marks at top illustrate saccade occurrences and their (brief) durations. The horizontal trace has been shifted upward by 0.5° .

Data analysis

SACCADE DETECTION. Eye velocity was determined by convolving eye position records with a Gaussian filter kernel (SD of 4 ms = cutoff frequency of 49 Hz) and then a differencing operator. Fixational saccades were identified as deflections during which eye speed exceeded $6^\circ/s$ for at least eight consecutive milliseconds. Pairs of deflections satisfying this criterion but separated by <30 ms were considered a single movement. This algorithm reliably detected small-amplitude saccades, which have correspondingly low velocities, but may have detected some nonsaccadic eye movements as well.

As described in RESULTS, most of the detected movements appeared to be saccades by standard criteria, but an appreciable fraction had looping trajectories for which the start and end points were nearly coincident. Such trajectories are consistent with blinks (Bair and O'Keefe 1998; Collewyn et al. 1985; Goossens and Van Opstal 2000). We did not measure the position of the eyelids and therefore do not know how much they moved during the eye movements we studied. Several lines of evidence, however, suggest that few if any of the movements we analyzed were blinks. First, during a blink, the eye is typically displaced by $>1^\circ$ with a peak speed $>50^\circ/s$ (Bair and O'Keefe 1998; Collewyn et al. 1985; Goossens and Van Opstal 2000). Such movements would have resulted in fixation breaks in our task, and none of the movements we studied fell within this range. Second, fixating monkeys performing other tasks rarely make eye movements consistent with blinks (Bair and O'Keefe 1998), and we failed to observe, via video monitoring, frequent blinking during performance of our task. Nonetheless, *monkey C* made approximately one small amplitude looping eye movement per second when the stimulus was present and also when it was absent. Third, we analyzed separately movements with straight and looping trajectories but were unable to find convincing differences between them with respect to their stimulus dependence. We therefore include all detected movements in the analyses that follow and refer to them as "saccades" for ease of exposition, but note that some of them may have been high-velocity, nonsaccadic movements. A typical eye position record, with saccade detections indicated, appears in Fig. 2.

To estimate the noise in our eye position measurements, we calculated the SD of eye position during the 50 ms after movement offset, during which the saccadic system may be refractory. Changes in eye position during this period reflect physiological noise including slow

drifts and tremors as well as electrical noise inherent to our measurement system. Averaged across movements, horizontal and vertical channels, and monkeys, the SD was 0.015° , indicating relatively stable eye position. Assuming Gaussian distributed noise that is independent across samples, the probability of the eye speed exceeding our $6^\circ/\text{s}$ threshold by chance is 3×10^{-5} . This implies a false alarm probability of $<1\%$ during each second of recording.

SACCADE-TRIGGERED AVERAGE. Previous studies have employed the reverse-correlation method to study the relationship between sensory stimuli and neuronal or psychophysical responses (e.g., Ahumada 1996; Marmarelis and Marmarelis 1978). We used it to relate our stimulus to the occurrence of fixational saccades. For each saccade, the sequence of 20 stimulus frames (200 ms) preceding saccade initiation were extracted and represented numerically as the intensities of the three monitor phosphors. The saccade-triggered average stimulus (STA) was calculated as the intensity of each phosphor at each of the 20 frames leading up to the saccade, averaged across saccades. Saccade initiations were assigned to the beginning of each frame: initiations occurring from 0 to 9 ms after a screen refresh were considered to be coincident with the refresh for the purpose of computing the STA.

The stochastic nature of our stimulus simplified hypothesis testing on the STA. By construction, the intensity of each phosphor in a given stimulus frame is an approximately Gaussian random variable with mean μ_{phosp} and variance σ^2 . The average of n randomly selected intensities, for a given phosphor, thus has a Gaussian distribution with variance σ^2/n . Under the assumption that the stimulus sequence does not influence the probability of saccade initiation, the STA is simply the average of many randomly selected stimulus sequences. In this case, every point in the STA has a Gaussian distribution with mean 0 and variance σ^2/n (after having subtracted off the mean intensity values, μ_{phosp}). Departures from this distribution indicate a relationship between the stimulus sequence and saccade initiations. We express STAs in units of SEs to correct for the fact that its variance depends on n .

RESULTS

General properties: saccade frequencies, directions, and amplitudes

Saccades that caused the eye to leave the fixation window were excluded from all analyses. Saccades obtained during nonstimulated trials were likewise excluded except where stated otherwise. *Monkey C* made 118,704 saccades during 46,976 s of accumulated stimulated trials, resulting in an average frequency of 2.5 saccades/s. *Monkey T* made 201,428 saccades in 89,429 s, or 2.3 saccades/s.

Distributions of saccade directions differed between monkeys, as shown in Fig. 3, *A* and *B*. *Monkey C*'s saccades were distributed broadly but biased downward. *Monkey T*'s saccades were usually directed downward, less frequently upward, and rarely horizontally. Saccade directions were not obviously related to the stimulus location: the stimulus was presented to the left for *monkey C* and to the right for *monkey T*, as shown Fig. 3 *A* and *B*, \rightarrow . Saccades in these directions were not particularly common.

Distributions of saccade amplitudes also differed between monkeys as shown in Fig. 1, *C* and *D*. The largest saccade allowed, theoretically, was $\sqrt{2}^\circ$ (corner to opposite corner of the fixation window). Empirically, however, amplitudes tended to be well below this upper bound: $0.14 \pm 0.08^\circ$ and $0.27 \pm 0.13^\circ$ (means \pm SD) for *monkeys C* and *T*, respectively.

Peak eye speed covaried with movement amplitude for both

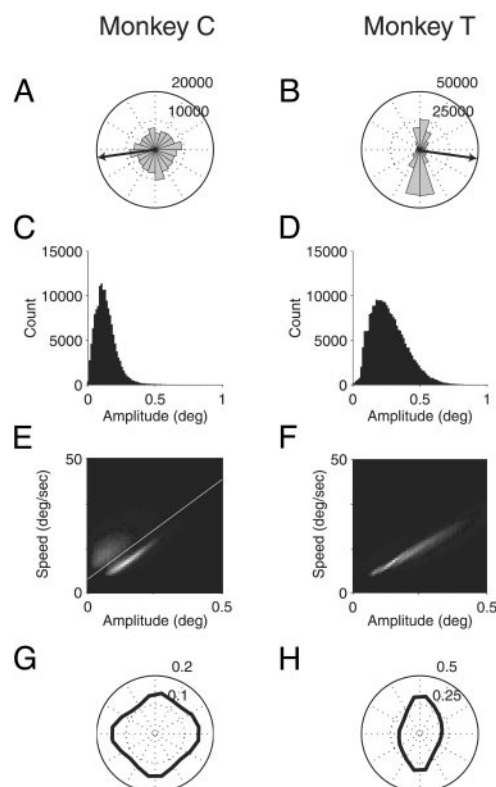


FIG. 3. Distributions of saccade parameters. *Left*: data from *monkey C*; *right*: data from *monkey T*. *A* and *B*: circular histograms of saccade directions. \rightarrow the angular position of the stimulus relative to the fovea. *C* and *D*: histograms of saccade amplitudes. *E* and *F*: density map of saccade peak velocity as a function of amplitude. The cloud above the diagonal line in *E* corresponds to high-velocity movements with looping trajectories. *G* and *H*: mean amplitude as a function of saccade direction. SEs are smaller than the line width.

monkeys as shown in Fig. 3, *E* and *F*. Most movements we studied belonged to the “main sequence” of saccades: their amplitudes and speeds lay on a line passing through the origin. Interestingly however, *monkey C* made many small amplitude movements with looping trajectories and velocities higher than expected from the main sequence: 53% of *monkey C*'s movements lay above the diagonal line in Fig. 3*E* (only 5% of *monkey T*'s movements were in this range). The first eye-position deflection in Fig. 2 is a small movement with a looping trajectory. Saccade amplitude covaried with direction for both monkeys. Figure 3, *E* and *F*, shows mean saccade amplitude as a function of saccade direction. Linear-circular correlation coefficients (Batschelet 1981) for *monkey C* (0.048) and *monkey T* (0.031) were highly significant ($P < 0.0001$). Interestingly, directions of high saccade frequency tended to be directions of high saccade amplitude (compare Fig. 3, *A* and *B* with *G* and *H*).

Luminance transients elicit saccades

Irwin et al. (2000) showed that luminance transients can elicit reflexive saccades in human subjects, suggesting that some of the saccades that we recorded may have been related to luminance changes in the stimulus. To test this hypothesis, we extracted the sequence of stimulus frames preceding each saccade and, by averaging phosphor intensities, derived the

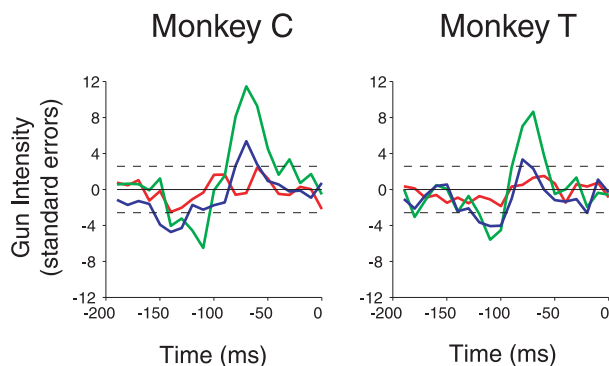


FIG. 4. Saccade-triggered average stimuli. Average intensities of the red, green, and blue phosphors preceding a saccade (at *time 0*) are plotted as a function of time. The y axis is expressed in units of SEs of the mean (each point has been divided by σ/\sqrt{n} , see METHODS). ---, the 99% confidence interval. On average, a saccade is preceded by a decrease, followed by an increase, in phosphor intensity. The green phosphor undergoes the clearest fluctuation, which peaks 70 ms before the saccade.

average sequence of frames preceding a saccade. As shown in Fig. 4, these saccade-triggered averages (STAs) were surprisingly consistent between monkeys: on average, the stimulus that preceded a saccade was a small decrease, followed by a larger increase, in phosphor intensity.

The three monitor phosphors differed in their ability to elicit a saccade. On average, increments in the green phosphor were most effective, increments in the blue phosphor were less effective, and the red phosphor did not appear to drive saccades at all. This is roughly, but not exactly, the pattern expected if the saccades were driven by the luminance of the stimulus. We return to this issue in the DISCUSSION.

Stimulus-evoked saccades had remarkably short latencies. STAs peaked ~ 70 ms before saccade initiation for both monkeys. This indicates that the entire chain of events, from visual transduction to contraction of the extraocular muscles, is completed within 70–80 ms. In fact, points in the STA lie outside the 99% confidence interval even closer in time to saccade initiation, suggesting that the minimum latency may be slightly shorter.

Directions of stimulus-evoked saccades

Our monkeys made saccades of many directions (Fig. 3, *A* and *B*). We considered the possibility that luminance transients in our stimulus attracted saccades and that saccades in other directions were spontaneous. We tested this hypothesis in two ways, neither of which supported the hypothesis.

In the first analysis, we binned saccades by direction, calculated STAs for saccades in each bin, and quantified intensity transients in each STA with an STA index. Only green and blue phosphors were considered in this analysis because the red phosphor was ineffective at driving saccades (see Fig. 4). The STA index is defined as the average difference between the phosphor intensities from 60 to 90 ms preceding each saccade and from 100 to 120 ms preceding each saccade. This index will be positive for STAs exhibiting a trough near 110 ms and a peak near 70 ms (like those shown in Fig. 4), will be negative for STAs with trough and peak reversed, and will be close to zero for STAs that are flat or random. Under the null hypothesis, this index has a Gaussian distribution with a mean of 0 and a variance of $\sigma^2/(14n)$. We express STA indices in units of

standard errors to correct for the fact that its variance depends on n . Importantly, positive and negative indices are equally likely under the null hypothesis.

Figure 5, *A* and *B*, shows the STA index as a function of saccade direction. Surprisingly, the index was uniformly positive, indicating that saccades in all directions were preceded, on average, by a decrease and then an increase in stimulus luminance. Saccades toward the stimulus (to the left for *monkey C* and to the right for *monkey T*) did not lead to particularly large indices. Luminance transients therefore evoke saccades of many directions not just toward the stimulus.

Additionally, we investigated the stimulus-dependence of saccade directions by measuring fixational eye movements made during interleaved stimulated and nonstimulated trials. Polar frequency histograms of saccade directions appear in Fig. 5, *C* and *D*, for saccades made in the presence (black curve) and the absence (gray curve) of the stimulus. Although the histograms are quite similar visually, the distribution of saccade directions differed significantly between stimulus conditions for both monkeys (Mardia-Watson-Wheeler tests, $P < 0.001$). Counterintuitively, saccade directions were biased weakly away from the stimulus for both monkeys, by 1.3° for *monkey C* and by 6.9° for *monkey T*. This result suggests that the stimulus did not attract fixational saccades but rather repelled them slightly.

Amplitudes of stimulus-evoked saccades

We considered the possibility that luminance transients evoked saccades of only a particular range of amplitudes. To

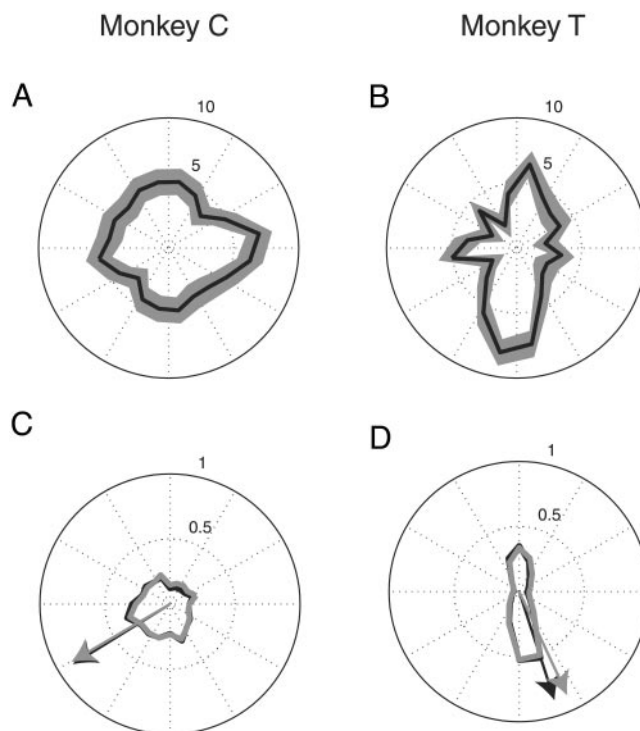


FIG. 5. Analysis of saccade directions. *A* and *B*: saccade-triggered average stimulus (STA) index as a function of saccade direction. Indices are expressed in units of SE under the null hypothesis. The gray band indicates ± 1 SE computed from the sample variance. *C* and *D*: frequency histogram of saccade directions in interleaved stimulated (black) and nonstimulated (gray) trials. Arrows indicate mean directions.

test this hypothesis, we binned saccades by amplitude and calculated an STA index for saccades in each bin. The results of this analysis appear in Fig. 6, *A* and *B*. STA indices for both monkeys were positive and of similar magnitude across a broad range of amplitudes, indicating that luminance transients drove saccades of many amplitudes similarly.

We also compared saccade amplitudes in the presence and absence of the stimulus. Frequency histograms for stimulated (black) and nonstimulated (gray) conditions appear in Fig. 6, *C* and *D*. For *monkey C*, the stimulus decreased the mean amplitude by 0.007° . This effect was reversed, however, for *monkey T* for whom the stimulus increased the mean amplitude by 0.018° . Both changes were significant (*t*-test, $P < 0.0001$). We thus conclude that peripheral visual stimulation affects the amplitudes of fixational saccades significantly but inconsistently.

DISCUSSION

The main finding of this study is that peripheral visual stimulation can elicit small amplitude saccades during nominal fixation with a latency of ~ 70 ms. Saccades varied idiosyncratically between monkeys in amplitudes and direction. The average stimulus preceding a saccade, however, was remarkably consistent: saccades followed a decrease, then an increase, in phosphor intensity. Saccades were biased weakly away from the stimulus.

Spectral sensitivity

Saccade initiations were related most closely to intensity changes in the green phosphor. This is what one would expect if saccades were triggered by changes in stimulus luminance: although each phosphor in our stimulus varied over a similar radiometric range, they varied over different luminance ranges due to their different emission spectra. The green phosphor

underwent the greatest luminance changes, and it drove saccades effectively; the red and the blue phosphors underwent smaller luminance changes and drove saccades much less effectively. If saccades were driven purely by luminance, however, the red phosphor would have been more effective than the blue. This was not the case, so we infer that the mechanism that drove saccades in our experiment is relatively more sensitive to short wavelength light (i.e., the light emitted by the blue phosphor).

Enhanced sensitivity to short wavelength light, relative to luminance, could arise from a number of sources. First, the stimulus was presented in the peripheral visual field, where lack of macular pigment increases psychophysical sensitivity to short wavelength light (Wyszecki and Stiles 1982). Second, macaque monkeys may be more sensitive to short wavelength light than humans are (DeValois et al. 1974). Consistent with this idea, we find that many macaque V1 neurons exhibit similarly enhanced sensitivity at short wavelengths relative to luminance (G. D. Horwitz, E. J. Chichilnisky, and T. D. Albright, unpublished observations).

The saccades we observed may have been driven by multiple mechanisms with varied spectral sensitivities. In this case, the STA would reflect the average sensitivity of the underlying mechanisms but not necessarily the sensitivity of any single one. Likewise, saccades may have been driven by different mechanisms at different latencies. For instance, our data are consistent with a single mechanism that drove saccades in response to the sequential presentation of a luminance decrement followed by an increment or alternatively, two independent mechanisms: one that drove short-latency saccades in response to luminance increments and another that drove saccades at a longer latency in response to luminance decrements. Our analysis method does not allow us to determine how many spectrally or temporally distinct mechanisms drove saccades in our experiment.

Saccades in our experiment were driven primarily by luminance changes on average, but express saccades, which have a similar latency, can be made to both luminant and isoluminant targets (McPeck and Schiller 1994; Weber et al. 1991). Despite this apparent discrepancy, a common mechanism may underlie the saccades we studied and express saccades. First, if multiple mechanisms contributed to the saccades we studied, those sensitive to pure color modulations may have been hidden in the STA. This would be expected if saccades were driven equally well by opponent colors (which would cancel in the STA) or if our stimulus activated luminance mechanisms more effectively than chromatic mechanisms. Second, we found that fixational saccades were more readily evoked by short wavelength light than expected from luminance. Isoluminant modulations, which by definition do not activate a luminance mechanism, might therefore still activate the mechanism responsible for driving fixational saccades, if weakly.

Comparison with previous work

Tse et al. (2002) looked for, and failed to reveal, a relationship between fixational eye movements and peripheral luminance transients. A variety of methodological differences between our study and that of Tse et al. could account for the difference in result. First, Tse et al. tested human subjects, whereas we tested monkeys. Monkeys may be less able to

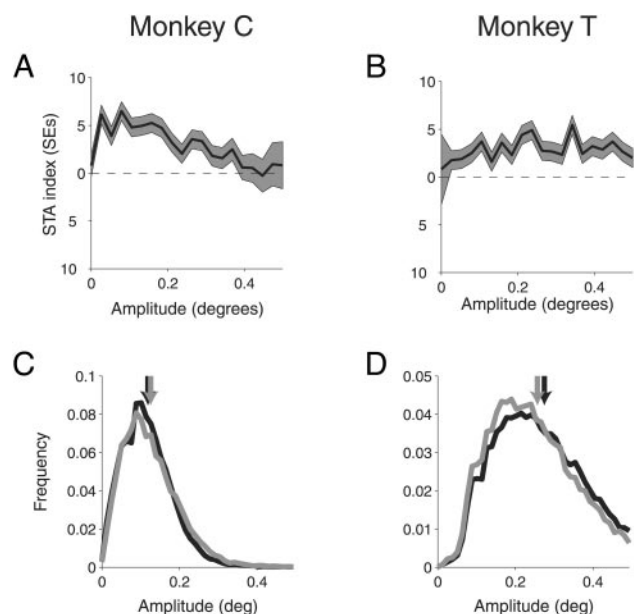


FIG. 6. Analysis of saccade amplitudes. *A* and *B*: STA index as a function of saccade amplitude. Conventions are as in Fig. 5. *C* and *D*: frequency histogram of saccade amplitudes in interleaved stimulated (black) and nonstimulated (gray) trials. Arrows indicate mean amplitudes.

suppress reflexive saccades than humans (but see Amador et al. 1998; Bell et al. 2000; Steinman et al. 1973). Second, subjects in the Tse et al. study performed a demanding novelty detection task during data collection and were instructed explicitly to ignore the peripheral flash. Our monkeys fixated without performing any other task; they were therefore free to attend to the stimulus and may have done so. Third, we analyzed saccades specifically, whereas Tse et al. considered only average eye position during periods of nominal fixation. We found that saccades induced by luminance transients were broadly distributed in direction and amplitude. If saccades in the study of Tse et al. were similarly distributed, it would be therefore unsurprising that stimulus position was not related clearly to mean eye position.

Saccade frequencies in our study were somewhat higher than in previous studies of fixating monkeys (Bair and O'Keefe 1998; Leopold and Logothetis 1998). This discrepancy is presumably due in part to differences in saccade detection algorithms: we set a fairly low velocity threshold and included eye movements with strongly curved trajectories, whereas previous studies have used stricter criteria. Details of our behavioral task including the display luminance and the presence of a salient peripheral stimulus may also have increased saccade frequencies.

Directions of stimulus-evoked fixational saccades

The amplitudes of saccades evoked by visual onsets or by electrical stimulation are diminished during active fixation (Sparks and Mays 1983; Weber et al. 1993). This raises the possibility that the saccades we observed were directed toward the stimulus but were hypometric as a result of fixation and therefore failed to leave the fixation window. This was not the case. Luminance transients preceded saccades in many directions, including away from the stimulus. Furthermore, presentation of the stimulus actually biased saccades weakly toward the opposite hemifield. We conclude that, under the conditions of our experiment, peripheral luminance transients increase the probability of a saccade and that these saccades are not directed toward the stimulus.

A priori, one might expect peripheral luminance transients to attract saccades. Such a reflex is of obvious utility, it has been documented in previous studies (e.g., Theeuwes et al. 1999), and candidate neural mechanisms are becoming understood (see Isa 2002 for a review). Details of our behavioral task may have prevented the saccades we studied from being directed toward the stimulus. Our fixation window, for example, was sufficiently small that even a few small co-directional saccades, without intervening compensatory eye movements, would have resulted in a fixation break and thus a penalty for the monkey. Our monkeys may therefore have adopted a strategy to compensate for the reflex to acquire the stimulus. Preparing small amplitude saccades away from the stimulus, for instance, might have prolonged fixation durations. Enlarging the fixation window might reveal a relationship between the stimulus location and saccade direction.

Stimulus-evoked saccades may have been directed toward the fixation point. Two lines of reasoning lend credibility to this notion: first, peripheral luminance transients may have enhanced the contrast of the black fixation point by raising the mean luminance of the background. The fixation point may

have then provided a particularly salient saccade target. Second, stimulus-evoked saccades may have been directed toward the weighted average position of the fixation point and the stimulus with very little weight assigned to the stimulus. In this case, stimulus-evoked saccades would be expected to land near the fixation point. Unfortunately, our experiment does not permit us to test these hypotheses rigorously: slow drifts in the eye-tracking system, combined with uncertainty in mean fixation position relative to the fixation point, prevent us from classifying decisively saccades as centripetal or centrifugal.

Neural mechanisms

The superior colliculus may participate in generating the saccades we studied. First, saccade latencies in our experiment were extremely short, indicating a relatively direct pathway from retina to extraocular musculature. By comparison, voluntary visually guided saccades typically have latencies of ≥ 120 ms. The colliculus receives input from the retina and visual cortex, and it projects to oculomotor nuclei in the brain stem, appropriate for driving short-latency saccades. Second, superficial layer SC neurons, like the saccades we observed, are tuned roughly for luminance (Marrocco and Li 1977). We do not know whether the deviation from luminance tuning we observed is reflected in the spectral sensitivity of collicular neurons. Third, collicular ablation eliminates express saccades, which have latencies similar to those we measured and may therefore derive from a common neural circuit (Schiller et al. 1980). A testable extension of this idea is that the short latencies we observed are related to the highly predictable location of our stimulus: express saccades occur more frequently when target position is predictable than when it is not (Pare and Munoz 1996).

In apparent contradiction to our findings, Reingold and Stampe (2002) showed that a peripheral luminance increment can inhibit a visually guided saccade with a latency of ~ 70 ms. In their study, human subjects were instructed to execute 4° horizontal saccades while luminance flashes were presented either above or below the fixation point. Their study differed from ours in a number of respects including subject species, task demands, and stimulus parameters. Any of these methodological differences could contribute to the difference in result.

On the other hand, a common mechanism may underlie both results. Reingold and Stampe proposed that collicular neurons in whose response field a flash occurs may inhibit other collicular neurons that mediate the intended behavior. Saccades in their task required the activity of saccade-related burst neurons at the 4° site in the collicular map (Robinson 1972). The peripheral flash might have inhibited these neurons. Monkeys in our experiment were required only to fixate, which presumably requires the balanced activity of "fixation neurons" at the rostral pole of the colliculus (Munoz and Wurtz 1993). If fixation neurons are inhibited by a peripheral flash, one might expect fixation to be impaired, as we observed. Support for this idea comes from the existence of lateral inhibition within the colliculus and the apparent continuum between fixation neurons and those involved in saccade preparation (Munoz and Istvan 1998; Munoz and Wurtz 1995). Luminance flashes may therefore disrupt saccades and fixation nonspecifically.

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DISCLOSURES

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REFERENCES

- Ahumada A.** Perceptual classification images from Vernier acuity masked by noise. *Perception*, 26: 18, 1996.
- Amador N, Schlag-Rey M, and Schlag J.** Primate antisaccades. I. Behavioral characteristics. *J Neurophysiol* 80: 1775–1786, 1998.
- Bair W and O'Keefe LP.** The influence of fixational eye movements on the response of neurons in area MT of the macaque. *Vis Neurosci* 15: 779–786, 1998.
- Batschelet E.** *Circular Statistics in Biology*. New York: Academic, 1981.
- Bell AH, Everling S, and Munoz DP.** Influence of stimulus eccentricity and direction on characteristics of pro- and antisaccades in non-human primates. *J Neurophysiol* 84: 2595–2604, 2000.
- Chichilnisky EJ and Wandell BA.** Trichromatic opponent color classification. *Vision Res* 39: 3444–3458, 1999.
- Collewijn H, van der Steen J, and Steinman RM.** Human eye movements associated with blinks and prolonged eyelid closure. *J Neurophysiol* 54: 11–27, 1985.
- Corbetta M, Akbudak E, Conturo TE, Snyder AZ, Ollinger JM, Drury HA, Linenweber MR, Petersen SE, Raichle ME, Van Essen DC, and Shulman GL.** A common network of functional areas for attention and eye movements. *Neuron* 21: 761–773, 1998.
- DeValois R, Morgan H, Polson M, Mead W, and Hull E.** Psychophysical studies of monkey vision. I. Macaque luminosity and color vision tests. *Vision Res* 14: 53–67, 1974.
- Goossens HH and Van Opstal AJ.** Blink-perturbed saccades in monkey. I. Behavioral analysis. *J Neurophysiol* 83: 3411–3429, 2000.
- Irwin D, Colcombe A, Kramer A, and Hahn S.** Attentional and oculomotor capture by onset, luminance and color singletons. *Vision Res* 40: 1443–1458, 2000.
- Isa T.** Intrinsic processing in the mammalian superior colliculus. *Curr Opin Neurobiol* 12: 668–677, 2002.
- Judge SJ, Richmond BJ, and Chu FC.** Implantation of magnetic search coils for measurement of eye position: an improved method. *Vision Res* 20: 535–538, 1980.
- Kustov AA and Robinson DL.** Shared neural control of attentional shifts and eye movements. *Nature* 384: 74–77, 1996.
- Leopold DA and Logothetis NK.** Microsaccades differentially modulate neural activity in the striate and extrastriate visual cortex. *Exp Brain Res* 123: 341–345, 1998.
- Marmarelis P and Marmarelis V.** *Analysis of Physiological Systems: The White Noise Approach*. New York: Plenum, 1978.
- Marrocco RT and Li RH.** Monkey superior colliculus: properties of single cells and their afferent inputs. *J Neurophysiol* 40: 844–860, 1977.
- McPeck RM and Schiller PH.** The effects of visual scene composition on the latency of saccadic eye movements of the rhesus monkey. *Vision Res* 34: 2293–2305, 1994.
- Munoz DP and Istvan PJ.** Lateral inhibitory interactions in the intermediate layers of the monkey superior colliculus. *J Neurophysiol* 79: 1193–1209, 1998.
- Munoz DP and Wurtz RH.** Fixation cells in the monkey superior colliculus. I. Characteristics of cell discharge. *J Neurophysiol* 70: 559–575, 1993.
- Munoz DP and Wurtz RH.** Saccade-related activity in monkey superior colliculus. I. Characteristics of burst and buildup cells. *J Neurophysiol* 73: 2313–2333, 1995.
- Pare M and Munoz DP.** Saccadic reaction time in the monkey: advanced preparation of oculomotor programs is primarily responsible for express saccade occurrence. *J Neurophysiol* 76: 3666–3681, 1996.
- Posner MI, Snyder CRR, and Davidson BJ.** Attention and the detection of signals. *J Exp Psychol Gen* 109: 160–174, 1980.
- Reingold EM and Stampe DM.** Saccadic inhibition in voluntary and reflexive saccades. *J Cogn Neurosci* 14: 371–388, 2002.
- Robinson DA.** Eye movements evoked by collicular stimulation in the alert monkey. *Vision Res* 12: 1795–1807, 1972.
- Schiller P, True S, and Conway J.** Deficits in eye movements following frontal eye-field and superior colliculus ablations. *J Neurophysiol* 44: 1175–1189, 1980.
- Sparks DL and Mays LE.** Spatial localization of saccade targets. I. Compensation for stimulation-induced perturbations in eye position. *J Neurophysiol* 49: 45–63, 1983.
- Steinman RM, Haddad GM, Skavenski AA, and Wyman D.** Miniature eye movement. *Science* 181: 810–819, 1973.
- Theeuwes J, Kramer A, Hahn S, and Irwin D.** Our eyes do not always go where we want them to go: capture of the eyes by new objects. *Psychol Sci* 9: 379–385, 1998.
- Theeuwes J, Kramer AF, Hahn S, Irwin DE, and Zelinsky GJ.** Influence of attentional capture on oculomotor control. *J Exp Psychol Hum Percept Perform* 25: 1595–1608, 1999.
- Tse PU, Sheinberg DL, and Logothetis NK.** Fixational eye movements are not affected by abrupt onsets that capture attention. *Vision Res* 42: 1663–1669, 2002.
- Weber H, Fischer B, Bach M, and Aiple F.** Occurrence of express saccades under isoluminance and low contrast luminance conditions. *Vis Neurosci* 7: 505–510, 1991.
- Weber H, Latanov A, and Fischer B.** Context dependent amplitude modulations of express and regular saccades in man and monkey. *Exp Brain Res* 93: 335–344, 1993.
- Wyszecki G and Stiles W.** *Color Science: Concepts and Methods, Quantitative Data and Formulae*. New York: Wiley, 1982.