Effects of Saccades on the Activity of Neurons in the Cat Lateral Geniculate Nucleus

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Lee, Daeyeol and Joseph G. Malpeli. Effects of saccades on the activity of neurons in the cat lateral geniculate nucleus. *J. Neurophysiol.* 79: 922–936, 1998. Effects of saccades on individual neurons in the cat lateral geniculate nucleus (LGN) were examined under two conditions: during spontaneous saccades in the dark and during stimulation by large, uniform flashes delivered at various times during and after rewarded saccades made to small visual targets. In the dark condition, a suppression of activity began 200–300 ms before saccade start, peaked ~100 ms before saccade start, and smoothly reversed to a facilitation of activity by saccade end. The facilitation peaked 70–130 ms after saccade end and decayed during the next several hundred milliseconds. The latency of the facilitation was related inversely to saccade velocity, reaching a minimum for saccades with peak velocity >70–80°/s. Effects of saccades on visually evoked activity were remarkably similar: a facilitation began at saccade end and peaked 50–100 ms later. When matched for saccade velocity, the time courses and magnitudes of postsaccadic facilitation for activity in the dark and during visual stimulation were identical. The presaccadic suppression observed in the dark condition was similar for X and Y cells, whereas the postsaccadic facilitation was substantially stronger for X cells, both in the dark and for visually evoked responses. This saccade-related regulation of geniculate transmission appears to be independent of the conditions under which the saccade is evoked or the state of retinal input to the LGN. The change in activity from presaccadic suppression to postsaccadic facilitation amounted to an increase in gain of geniculate transmission of ~30%. This may promote rapid central registration of visual inputs by increasing the temporal contrast between activity evoked by an image near the end of a fixation and that evoked by the image immediately after a saccade.

INTRODUCTION

The lateral geniculate nucleus (LGN) receives a multiplicity of nonretinal inputs (for a recent review, see Sherman and Guillery 1996). For this reason and because of its early position in the visual pathway, the LGN has long been considered a site where visual- and saccade-related signals might converge. Indeed, there is considerable evidence that saccades modulate LGN transmission of visually evoked activity in both the cat (Fischer et al. 1996; see also Noda 1975) and monkey (Bartlett et al. 1976; Büttner and Fuchs 1973; Chukoskie et al. 1995).

Thoughts about the function of saccadic modulation often have centered on suppressing visual perception during saccades (Erdmann and Dodge 1898). Perceptual saccadic suppression appears to begin before saccade start, peak during the saccade, and dissipate by saccade end (Latour 1962; Volkmann et al. 1968; Zuber and Stark 1966). Such suppression, however, need not depend entirely on oculomotor signals. Three categories of mechanisms have been proposed to account for it: centrally originating suppression of visual transmission (Holt 1903), visual masking (Dodge 1905; MacKay 1970), and mechanical shearing forces in the retina (Richards 1969). In this context, centrally mediated suppression usually is assumed to involve oculomotor signals (either corollary discharge or proprioceptive feedback), and visual masking refers to any destructive interference with perception resulting from the spatiotemporal pattern of the image (whether or not retinal image motion is caused by an eye movement). There is psychophysical evidence for both masking (Campbell and Wurtz 1978; MacKay 1970; Matin et al. 1972; for a review, see Volkmann 1986) and central suppression (Burr et al. 1994; Latour 1962; Riggs and Manning 1982; Volkmann 1962; Zuber and Stark 1966).

Another function that has been linked to saccadic modulation of visual transmission is the rapid establishment of new images after saccades. Jung (1972), proposed that ''cancellation'' of previous images promotes ''undisturbed transfer of the following retinal image,'' and centrally mediated suppression might underlie such a resetting mechanism. However, postsaccadic facilitation has been invoked more commonly as an aid in registration of new scenes (Jüttner and Röhler 1993; Lal and Friedlander 1990; Wolf et al. 1978, 1980). It also has been suggested that postsaccadic facilitation plays a role in evaluating saccade accuracy and programming corrective saccades (Becker 1976; Deubel et al. 1982).

None of the functions proposed for centrally mediated, saccade-related modulation of visual inputs suggest a need to block such modulation in the dark. However, effects on LGN activity of saccades made in darkness have proven relatively difficult to detect. For the cat, they have been reported absent (Fischer et al. 1996; Noda 1975) and for the monkey, present in <5% of cells (Büttner and Fuchs 1973) or of much smaller magnitude than in the light (Bartlett et al. 1976). This is odd, given that proprioceptive signals from the extraocular muscles modulate LGN activity in anesthetized cats (Donaldson and Dixon 1980; Lal and Friedlander 1990; see also Molotchnikoff and Casanova 1985 for similar results in the rabbit) and that cells in the pretectum projecting to the LGN carry strong saccade-related signals in the dark (Schmidt 1996). Furthermore, cat LGN activity is modulated during the saccade-like rapid eye movements of sleep (Bizzi 1966) and during vestibular nystagmus in encéphale isolé preparations (Jeannerod and Putkonen 1971). If oculomotor inputs to the LGN are actively gated, the circumstances under which saccadic modulation of geniculate transmission is present or blocked might provide important clues as to the role of such modulation in percep-

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tion. On the other hand, it might be that these inputs are undiminished in the dark but that their effects on LGN gain are obscured by low, variable rates of activity.

The purpose of the present study was to obtain a more complete understanding of the nature and purpose of saccade-related modulation of LGN activity. One goal was to reveal the sign, magnitude, and time course of effects of saccades on LGN activity with sufficient resolution to see if they are consistent with saccadic suppression and/or postsaccadic facilitation. Another goal was to determine whether there is active cancellation of saccade-related signals in the dark or if the effects of these signals are independent of the conditions under which saccades take place.

In choosing a strategy for investigating the effects of eye movements on the LGN, a major consideration is the technical difficulty of stabilizing images on the retina during natural saccades. Each method of getting around this problem has its limitations. For paradigms in which the eye moves across patterned visual stimuli, there is a fixed temporal relationship between the visual stimulus and the eye movement, requiring indirect methods to extract the effects of the saccade per se (Bartlett et al. 1976; Fischer et al. 1996; Noda 1975). Examining saccades in the dark potentially sidesteps these problems by eliminating any contribution of masking and allowing oculomotor influences to be directly detected. However, as pointed out above, relatively low rates of activity in the dark may obscure changes in excitability. Experiments with anesthetized animals (Donaldson and Dixon 1980; Lal and Friedlander 1990; Molotchnikoff and Casanova 1985) allow precise control of stimulus parameters but at the expense of dealing with an abnormal brain and limiting eye-movement signals to proprioceptive input. In the present study, we decided to reexamine the effects of saccades in the dark on LGN activity with extensive averaging of neural responses to reveal saccade-related signals. For saccades in the light, we gave up the possibility of using optimal, patterned stimuli in return for the ability to decouple the timing of visual stimuli from saccade dynamics. Head-fixed cats made rewarded saccades to small targets, and at various times during and after saccades, visual stimuli were flashed onto the receptive fields of LGN cells. These stimuli were very large, uniform flashes that always extended well beyond the borders of the receptive fields of recorded LGN cells. We assumed that responses evoked several hundred milliseconds after saccade end could be taken as baseline and took differences between these and responses evoked during and immediately after saccades as measures of saccade-related changes. Our results suggest that oculomotor influences on LGN activity are substantial and have the same time course and are of similar magnitude, whether in the dark or at the peak of visually evoked activity. Activity undergoes a modest drop before and during saccades, followed by a larger postsaccadic facilitation beginning at saccade end. These changes appear to be related only to saccade dynamics and are independent of the timing or even the presence of visual stimuli.

METHODS

Animal preparation

Data were collected from seven LGN in four cats. All surgeries were done aseptically under barbiturate anesthesia (sodium thiopental, intravenous, supplemented by buprenorphine, 0.0075 mg/kg im). Two or three doses of buprenorphine were given every 12 h after surgery for postoperative analgesia. Care and use of animals were in accordance with the guidelines of the American Physiological Society, the Society for Neuroscience, and the University of Illinois Laboratory Animal Care Advisory Committee.

The method of mounting and protecting skull implants was that of Malpeli et al. (1992). An aluminum ''crown'' was bonded with antibiotic-doped bone-cement (Palacos R. Smith and Nephew Richards) to stainless-steel studs tapped into the skull. Scleral search coils (Robinson 1963) were implanted on both eyes (Judge et al. 1980a). During all training and experimental sessions, the head was immobilized by a fixture attached to the crown. A fiberglass cap mounted on the crown protected skull implants between experimental sessions.

When the cats had recovered completely from the initial surgery, they were trained to fixate a small spot of light (0.1°) for food rewards (a/d Prescription Diet, Hill's Pet Nutrition) and to follow it when it jumped from one location to another. Food was delivered by pressure in small boluses through a tube positioned near the mouth. The animals usually were allowed to work to satiation. They were weighed weekly and, when necessary, given supplemental food to keep them >80% of their preoperative weights. Visual stimuli used to assess excitability of LGN neurons (described in following text) were always irrelevant to the task, and their presentation soon had no effect on the animals' fixation of the laser target.

Once training was complete (typically several weeks), microdrive bases were implanted in a second surgery. A small hole was drilled through the bone-cement and skull, one or two bases with microdrives attached were positioned over the LGN, and recording electrodes were advanced into the nucleus. The animals were paralyzed briefly (vecuronium bromide, 1 ml/h) to allow final adjustments of the microdrive bases by physiological criteria before the bases were cemented in place with dental cement. The duration of paralysis was usually 30–60 min, and during this time, anesthetic was delivered continually at the same rate that had proven adequate during the 2–3 h of surgery before paralysis. The electrocardiogram was monitored during paralysis, and heart-rate variability used to assess the adequacy of anesthesia.

On full recovery from surgery, recording sessions began. The microdrive system allowed microelectrodes to be left in place from session to session and afforded easy and precise changes of electrode position (Malpeli et al. 1992). During recording sessions, the electrode was advanced by a stepping motor via a telescoping shaft connected to the microelectrode drive by universal joints.

Visual stimuli and data collection

All aspects of stimulus delivery and data collection (including behavioral tasks used in the visual-stimulation condition described in following text) were under computer control. Images were presented on a 58 × 58 cm rear-project screen located 47 cm from the eyes (subtending 63 × 63°). The fixation target was a spot produced by a laser beam the location of which was controlled with mirror galvanometers. Its intensity was adjusted with neutral density filters so that it was brighter and much more salient than other images. Sinusoidal gratings (used only for cell classification) or rectangles of uniform brightness (used to test effects of saccades on visually evoked responses) were produced by an image generator (Picasso CRT Image Synthesizer, Innisfree), displayed on a Tektronix 608 monitor, and projected onto the screen by a lens. The general background level of the screen was 1.7×10^{-4} cd/m^2, which, for fully dilated pupils, is ~3 log units below the mesopic range (Lee et al. 1992). The projection screen was the only source of light in the recording chamber. To dark-adapted human observers, the screen was clearly visible at background level but it appeared uniform and featureless.

For examining spontaneous activity in the dark, several steps were taken to ensure that all sources of light were eliminated. The recording chamber, a 1.5×5 m room with a light-tight door, was constructed specifically with this requirement in mind. A remotely manipulated, double-interlocking shutter blocked the persistent afterglow from the oscilloscope monitor used to produce visual stimuli. As an additional precaution, an infrared light source used for monitoring the animal via a CCD camera was turned off for the dark condition. To check for light leaks, four observers who first were familiarized thoroughly with possible source of light (the display screen, electronic equipment, cable light traps, door frame, etc.) sat in the chamber until fully dark-adapted (45 min). They were instructed to search continuously for stray light during the entire adaptation period. Concurrently, flood lights were played on the outside of the chamber to maximize the detectability of light leaks. The adequacy of light exclusion was rechecked several times during the course of these experiments.

Single cells were recorded extracellularly with glass-insulated tungsten microelectrodes, and arrival times of action potentials stored at a resolution of 0.2 ms. Eye-position signals generated with the scleral search-coil circuit of Remmel (1988) were sampled at 250 Hz by 12-bit A/D converters. Motorized flaps controlled the exposure of the eyes independently. Once a cell was isolated, small rectangles, either brighter or darker than the background, were used to locate its receptive field and determine center polarity (ON or OFF).

Sinusoidal gratings of 0.26 cd/m^2 mean intensity (low mesopic) and 80% contrast [(max − min)/(max + min)] were used to classify X and Y cells (Enroth-Cugell and Robson 1966). After the cat acquired the fixation target, a large ($\geq 20 \times 20°$), fixed-position, contrast-reversing (2 Hz), sinusoidal grating was projected over the receptive field; elsewhere, the screen was illuminated at the general background level of 1.7×10^{-4} cd/m^2. Temporally, the contrast reversals were abrupt not sinusoidal. A cell was classified X if excited by only one phase of contrast reversal and if no responses were obtained for a particular position of the eye on the grating (nulling of the response; Fig. 1, *top*). It was classified Y if excited by contrast reversals of both phases for all eye positions (frequency doubling) (Hochstein and Shapley 1976) and not nulled for any position of the eye relative to the grating (Fig. 1, *bottom*). With paralyzed, anesthetized animals, one normally accomplishes this by shifting the zero crossings of contrast-reversing gratings relative to the stationary eye. However, in the present study, the counterphased grating was stationary on the screen, and its position on the retina was varied by the cat's natural fixation behavior. Small eye movements typically broke up a trial into several stable fixation periods, each of which was sufficiently long to evaluate the effects of several cycles of contrast reversal. Multiple trials provided the opportunity to assess many combinations of grating and eye position. Small variations in fixation position were further encouraged by pseudorandomly drawing from several (3–5) fixation targets, horizontally separated by 1–2°. Failure to null the response was associated always with obvious frequency doubling, whereas null positions (of the eye relative to the grating) were observed readily in those cells that responded only to one phase of contrast reversal. We previously have used systematically shifted, contrast-reversing gratings to classify LGN cells in paralyzed, anesthetized cats (Lee et al. 1992). In comparison, relying on the cat's natural eye movement behavior to shift the grating is faster, and the classifications are at least as clear-cut. We have no reason to believe that these classifications differed from those obtained in anesthetized animals, including those previously reported by this laboratory (Lee et al. 1992). As will be documented in RESULTS, effects of eye movements on LGN activity differed substantially for X and Y cells, an outcome that we take as evidence for the adequacy of our classification method.

Experimental paradigms

To assess effects of oculomotor behavior on LGN activity in the absence of visual stimuli, data were collected for 10–15 min in the dark for each cell. The animals had no task to perform while in the dark, and during this time free rewards occasionally were given. They usually looked about spontaneously, but when their eyes were stationary for more than a few seconds, we tapped on the walls of the experimental chamber to encourage saccades. Cats have the reputation of being difficult to keep awake, but we did not experience this problem with animals trained to work for food even during the periods in the dark.

To determine the effects of saccades on visually evoked responses, large ($20 \times 20°$), uniform, square stimuli, roughly centered on the receptive field, were presented for 500 ms at different times relative to saccades made to the laser targets. Flashes were presented only to the dominant eye, the other eye being covered by a motorized flap. For ON-center cells, the luminance of the flash was 8.6×10^{-3} cd/m^2, against a background of 1.7×10^{-4} cd/m^2. For OFF-center cells, the luminance of the stimulus and background were reversed. To keep flashes adequately centered on the receptive field, eye-position signals were used to update flash location every 4 ms. The monitor producing the flashes was refreshed at 200 Hz, but because of interactions with the computer sampling cycle, updating the display could take as long as 10 ms. The peak velocity of saccades under these conditions rarely exceeded 300°/s (see Fig. 3), so the center of the receptive field was always within ~3° of the center of the $20 \times 20°$ stimulus. However, stabilization was usually much better than this because most flashes were delivered not at peak saccade velocity but at late phases of saccades or after the eye was stationary.

Two different paradigms were used in the visual-stimulation condition. In the "centrifugal saccade" paradigm, a central target was presented at the beginning of a trial, and the animal had to fixate it for 0.8–1.5 s (Fig. 2). Then the central target was extinguished, and a peripheral target was presented 8° away, which the cat had to fixate within 1.5 s to obtain a reward. Although we allowed a generous time for acquiring targets, saccades usually occurred with much shorter latency [319.5 ± 185.5 (SD) ms]. Only the first saccade launched from the central target contributed to the data. For some cells, two peripheral target positions were employed (left and right); for others, two additional targets, displaced vertically (above and below the central target), also were used. Because the location of flashes on the screen differed according to saccade target, any apparent variations in response as a function of saccade direction might have resulted from nonuniformities in the display or from gaze angle rather than the saccade itself. To evaluate these possibilities, a "centripetal saccade" paradigm also was used, reversing the temporal order of target presentation by requiring the cat to make saccades from a peripheral target toward the central fixation target (Fig. 2). This paradigm had the advantage that flashes delivered near and after saccade end always were presented on the same screen location (and gaze angle). For the centripetal saccade paradigm, the position of the saccade target was totally predictable, whereas for the centrifugal saccade paradigm, the animal did not know which target would appear next.

For each saccade direction in either paradigm, two methods of stimulus presentation were used for determining the timing between flash and saccade (Fig. 2). In the velocity-triggered condition, flash onset occurred either when eye velocity exceeded 25°/s or 50 ms later (velocity-triggered stimulus onset delay, SOD_V = 0 or 50 ms). In the position-triggered condition, the flash was presented after variable delays from the time when the eye entered a $6 \times 6°$ window centered on the target (position-triggered stimulus onset delay, SOD_P = 0, 50, 100, or 500 ms). Not all conditions were used for every cell. The 0- and 50-ms SOD_V conditions were added midway through the study, but the latter was abandoned after 14 cells when it became apparent that the timing of the flash

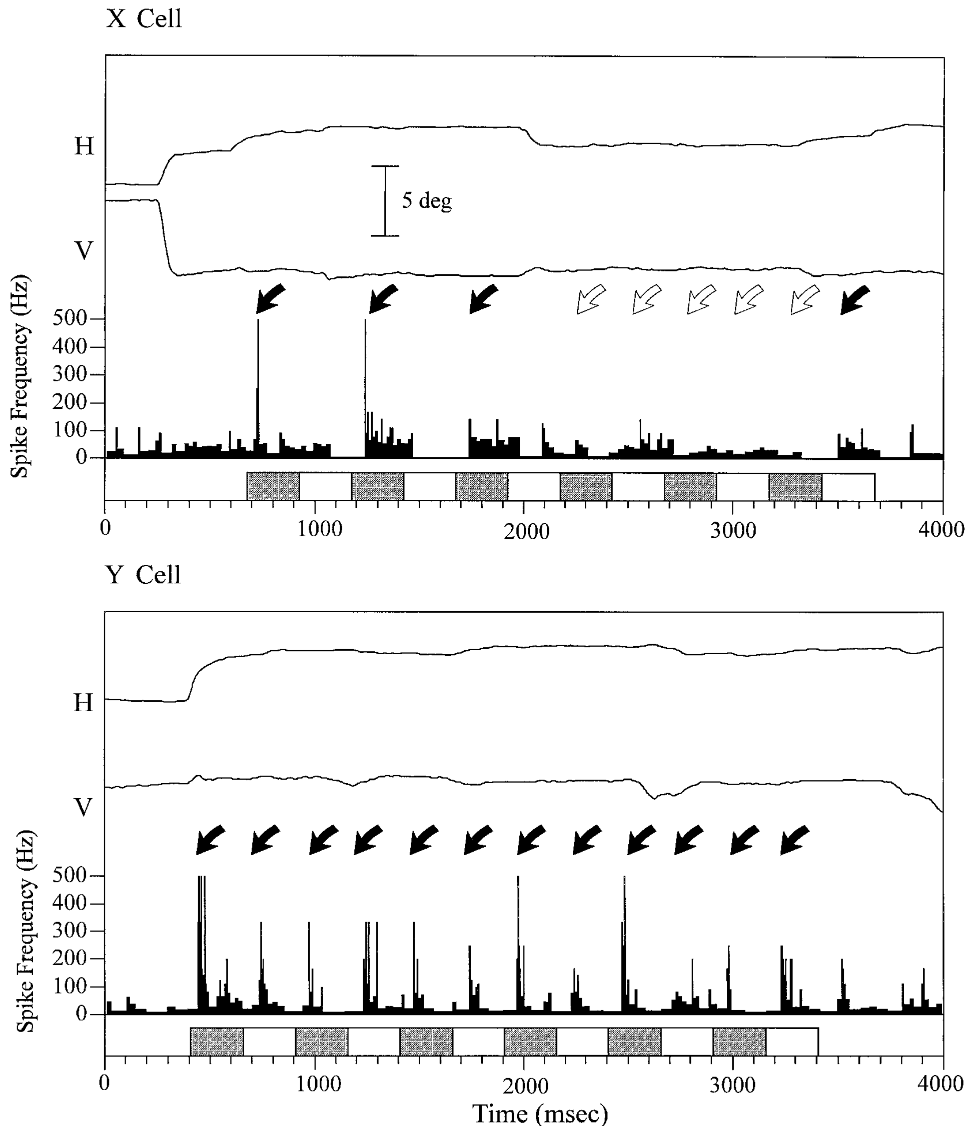

FIG. 1. Method of categorizing X and Y cells. Response histograms (■) show the inverse of the interspike interval during presentation of a counterphased (2 Hz), sinusoidal grating during a single behavioral trial. Horizontal (*H*) and vertical (*V*) eye position are given as separate traces. Horizontal bars *(bottom)* indicate (by ▨ and □) the points in time when the grating was counterphased abruptly. Tick marks on the abscissa indicate 100 ms. *Top*: example of a X cell recorded in layer A1. At the beginning of the trial, it responded to only 1 phase of reversal (1st 3 filled arrows). After a small spontaneous shift of gaze midway through the trial, there were no clear responses to either phase (nulling of the response; open arrows). Near the end of the trial, another change in eye position resulted in a response to the opposite phase of the grating (last filled arrow). Spatial frequency of the grating was 0.54 cycles/deg. *Bottom*: example of a Y cell recorded in layer A. This cell responded to both phases of reversal (filled arrows), and no nulling of the response was observed for this or several other trials. Response associated with the 10th arrow was smeared out because the eye was in motion during the phase reversal of the grating (spatial frequency = 1.0 cycle/deg).

relative to the saccade was about the same as for the 0-ms SOD_P condition (all of these data were nevertheless included in the analyses that follow). Each combination of SOD and target direction was repeated at least 10 times with presentation order randomized across trials. Responses to flashes in the 500-ms SOD_P condition were used as a baseline with which responses in other conditions were compared. Alternatively, we could have taken responses to flashes delivered before the second target as baseline. We chose the postsaccadic period because (in experiments not dealt with here) we have observed effects of maintained eye position on LGN excitability. For the position-triggered SODs, these changes would be factored out by our choice of baseline. Consider also that a saccade was required for fixating the central target at the beginning of each trial, so for either strategy, the baseline flash would be delivered shortly after the completion of a saccade.

Occasionally, the retinotopic location of a cell was such that the receptive field was swept across the laser target during saccades, directly evoking a response. Cells with receptive fields in these locations were avoided, and any recorded were excluded from analysis.

Data analysis

For both dark and visual-stimulation conditions, saccade start was taken as the point when eye velocity exceeded 25°/s, saccade end as the point where velocity dropped below 10°/s, and saccade duration as the interval between these two points. Usually, an eye movement was considered a saccade only if its velocity exceeded 40°/s. However, for examining the relationship between saccade amplitude and peak velocity, any eye movement with velocity exceeding 25°/s was counted as a saccade. Trials were discarded if the velocity of secondary eye movements exceeded 25°/s within 1 s of saccade end.

For most individual cells, activity in the dark was too low for effects of saccades to be revealed with high time-resolution, so we compared the total numbers of spikes occurring during the 500-ms periods before (N_{PRE}) and after (N_{POST}) saccade end. Saccade end was taken as the natural dividing point for this comparison because, as shown later in RESULTS, it separated periods characterized by suppression and facilitation. For any given neuron, these spike counts were summed over all saccades (regardless of direction), and the ratio of the sums minus one, $(\Sigma N_{POST}/\Sigma N_{PRE}) - 1$, taken as the "dark postsaccadic facilitation index." This index is positive for greater activity after saccade end and negative for lesser activity. To examine effects of saccade direction, the Michelson contrast, $(N_{POST} - N_{PRE})/(N_{POST} + N_{PRE})$, was calculated for each saccade, and a directional tuning curve computed with a binwidth of 45°, smoothed with 90° wide moving boxcar. The difference

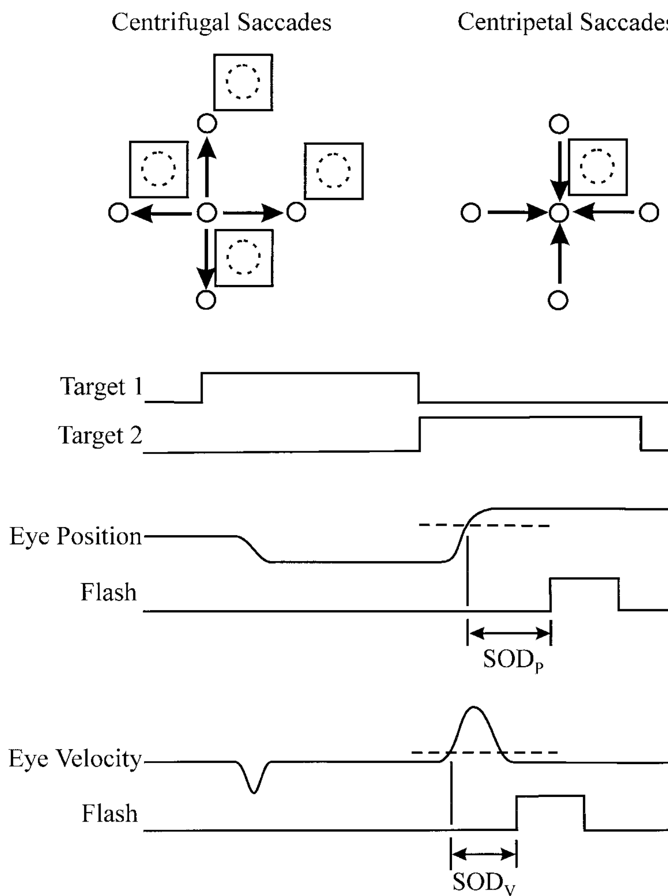

FIG. 2. Spatial and temporal arrangement of visual stimuli used to study the effects of saccades in centrifugal and centripetal saccade paradigms. →, saccades, which were directed either from the central fixation target to 1 of 4 peripheral targets (centrifugal saccade paradigm) or from peripheral targets to the central target (centripetal saccade paradigm). Squares (not to scale) represent large (∼20 × 20°), uniformly bright, 500-ms flashes that were initiated at various phases of the saccade. Eye-position signals were used to keep the flashes roughly centered on the receptive field during saccades. Traces labeled *Target 1* and *Target 2* give the temporal relationship between the initial fixation target and the target to which the saccade was directed. In the position-triggered condition, the flash was triggered after a variable delay (position-triggered stimulus onset delay, SOD_P) from when the eye entered a 6 × 6° window (dashed line) centered on the saccade target. In the velocity-triggered condition, the flash was triggered after a variable delay (velocity-triggered stimulus onset delay, SOD_V) from the point when eye velocity exceeded 25°/s (dashed line).

between the largest and smallest values of this curve was used as a measure of the influence of saccadic direction on postsaccadic facilitation.

For numerical estimates of effects of saccades on individual cells in the visual-stimulation condition, the spike data were collapsed over all perisaccade flashes (i.e., all with SOD <500 ms). Fractional changes in activity associated with saccades were determined by dividing mean spike counts for five successive 100-ms intervals by the corresponding counts for the baseline flash (SOD_P = 500 ms) and subtracting 1 from these quotients. Positive values indicate a facilitation and negative values a suppression of the response. Effects of saccade direction were assessed separately for each 100-ms interval by obtaining the fractional change in activity separately for each of the four saccade directions and taking the difference between maximum and minimum values.

To analyze effects of saccades on LGN activity with sufficient resolution to reveal their time course, it was necessary to collapse the data across cells. For saccades in the dark, the spike train bracketing each saccade was smoothed using a fixed Gaussian filter (SD = 20 ms). The smoothed data were averaged across cells, aligned on either saccade start or saccade end, and expressed as continuous functions, either as absolute rate of activity or as fractional changes in activity relative to activity in the 500-ms interval beginning 500 ms after saccade end. For the visual-stimulation condition, mean fractional changes in activity relative to this same postsaccadic baseline period were calculated for comparison with the dark condition. However, because the distribution of flashes relative to the saccade was neither continuous nor uniform, this was done for discrete intervals rather than as continuous functions. The number of spikes was counted separately for each trial in five successive 100-ms intervals, starting at flash onset. To determine the fractional change in activity for any given interval, five sequential 100-ms intervals of activity spanning the most delayed flash (500–1,000 ms after saccade end) were taken as baseline for the five corresponding 100-ms intervals of flashes with shorter SODs. These fractional changes were averaged separately for successive 50-ms bins, beginning at saccade start or end. Because of variability in saccade duration and velocity profile and because the eye was in motion when it entered the positional window around the second target, in most cases, flashes were not delivered in a constant temporal relationship relative to saccade start or end. Therefore, a given 50-ms bin (relative to saccade start or end) included data from flashes of several different SODs. To reduce the effects of uncontrolled variability, for most analyses, a particular 50-ms bin was used only if ≥20 cells each contributed five or more saccades to that bin. When the sample was subdivided for comparisons of different subgroups of cells (Fig. 12), these criteria were relaxed to five or more cells each contributing five or more saccades.

Response latency for each cell was taken as the time to reach half the peak amplitude of the initial response relative to mean activity in the 500-ms period preceding flash onset. Spike density functions used to determine this were smoothed with adaptive kernel estimation (Richmond et al. 1990; Silverman 1986). This optimizes temporal resolution by continually adjusting the width of the smoothing window to keep it inversely proportional to the square root of spike frequency (σ_p = 10 ms was used as the initial estimate of the spike density function) (see Richmond et al. 1990). Latencies were adjusted for delays related to the refresh of the display. There was an uncertainty of ±2.5 ms in this adjustment, but errors were distributed randomly and did not vary systematically across conditions. Because the data were averaged over many trials, any residual errors would not have significantly affected computations of mean effects of saccades on response latency.

High-frequency bursts, attributed to activation of a low-threshold Ca^{2+} conductance as cells switch from a hyperpolarized to a relatively depolarized state, have been observed in the LGN (Guido et al. 1992, Lu et al. 1992; Steriade and Llinás 1988). To see if such bursts contribute significantly to saccadic modulation of LGN activity, we identified them, using the criteria employed by Lu et al. (1992): two or more spikes with interspike intervals ≤4 ms preceded by a silent period ≥100 ms. For the dark condition, bursts were counted in intervals that averaged 1,964 ms, consisting of a 800 ms-period before saccade start, the saccade itself and a 1,000-ms after saccade end. Because of the required 100-ms silent period, any burst occurring within the first 100 ms of a counting interval could not be identified as such, so 100 ms was subtracted from each counting interval for calculating burst frequency. For the visual-stimulation condition, each flash was divided into five 100-ms intervals, and burst were counted separately for each interval.

Unless otherwise noted, statistical significance was determined by permutation tests (also known as randomization tests), which have the advantage of being independent of distribution assumptions. For example, for the dark postsaccadic facilitation index, we

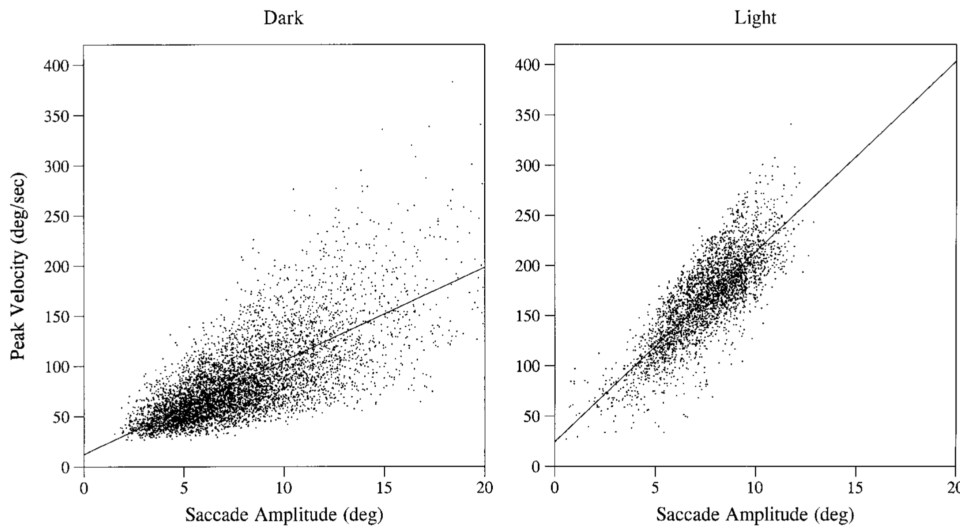

FIG. 3. Saccade amplitude vs. peak saccade velocity for saccades in the dark (dark: n = 6,605, slope = 9.29°/s per deg, intercept = 12.1°/s) and saccades to visual targets (light: n = 3,494, slope = 19.1°/s per deg, intercept = 23.2°/s). These relationships are distorted somewhat by the exclusion of saccades with peak velocities <25°/s from the analysis: it is likely that the true intercepts are closer to 0°/s.

repeatedly shuffled spike counts during the 500-ms periods before and after saccade end for every trial, calculated the index for each shuffle, and took as a measure of statistical significance the fraction of shuffles that produced an index larger than the experimental value. For all permutation tests, we performed at least 2,000 shuffles of the original data and considered differences significant when the experimental effect was exceeded in <5% of the shuffles. When multiple comparisons were made, Bonferroni's inequality was used to adjust the P values accordingly.

RESULTS

Comparison of saccade metrics in the dark and in the light

As will be pointed out later, the latency of saccade-related facilitation of LGN activity varied with saccade velocity. There was a fairly linear relationship between peak velocity and saccade amplitude, similar to that reported by Evinger and Fuchs (1978), but the slope of the function for saccades in the dark was about half that for saccades to visual targets: 9.29 versus 19.1°/s per deg (Fig. 3). Mean saccade amplitude was similar in both cases (8.0° in the dark, 7.5° to visual targets), whereas mean peak velocity was much higher for saccades to the laser target (165.7 vs. 86.1°/s). Saccade duration averaged 164.7 ± 69.2 (SD) ms in the dark and 89.9 ± 26.6 ms for saccades to visual targets. Although duration was quite variable in the light, it did not vary significantly with amplitude (the slope of the duration vs. amplitude function was 0.49 ms/deg; r = 0.038). Because the slope of the velocity versus amplitude function was lower for saccades in the dark than for saccades to visual targets, one would expect duration to increase with amplitude in the dark, and this was the case (the slope of the duration versus amplitude function was 11.0 ms/deg, r = 0.559).

Activity during saccadic eye movements in the dark

Effects of saccades on "spontaneous" activity in the dark were examined for 71 cells (an analysis by cell type and LGN layer is provided later—see Table 1). The mean number of saccades analyzed for each cell was 55.8 ± 29.9. The mean rates of activity in the 500-ms periods before and after saccade end were 29.8 and 33.0 spikes/s, respectively. These rates differed significantly for 47 cells: 46 (65%) had relatively higher activity in the postsaccadic interval, and one cell had lower activity in this interval (Fig. 4, *right*). Figure 4, *left,* shows the pre- and postsaccadic activity for each cell, plotted on a log-log scale. The absolute level of activity varied among cells by a factor of 6. The data fall on a line parallel to the main diagonal, which, on a log-log plot, indicates a constant fractional increase in activity after the saccade. This suggests that over a wide range of firing rates the effect of saccades is multiplicative, rather than additive, i.e., a change in gain. Saccade direction was not a significant factor in the postsaccadic facilitation: the magnitude of the facilitation varied with direction for only five cells (7%).

Even for cells showing comparatively large increases in postsaccadic activity, firing rates in the dark were too low and variable for effects of saccades to be obvious on a trial-by-trial basis (Fig. 5). However, the time course and magnitude of these effects were revealed clearly when activity was averaged across all cells (Fig. 6). There was a modest decrease in activity, beginning 200–300 ms before saccade start and extending through most of the saccade itself. Near saccade end, an increase in activity began that peaked ~70–150 ms later and gradually declined during the next several hundred milliseconds. The increase was more rapid and peaked sooner and higher when the data were aligned on

TABLE 1. *Distribution of sample with regard to experimental condition, LGN layer, cell type, and receptive-field center polarity*

	LGN Layer			Cell Type		Polarity		
Condition	A	A1	C	X	Y	ON	OFF	Total
Dark	38	23	10	42	26	36	33	71
Light	41	19	9	45	24	43	26	69

Dark refers to cells for which data were obtained for saccades in the dark; light refers to cells for which data were obtained for saccades to visual targets. A few cells were not categorized by type or polarity. The dark and light samples consist mostly of the same cells: 49 were tested in both conditions.

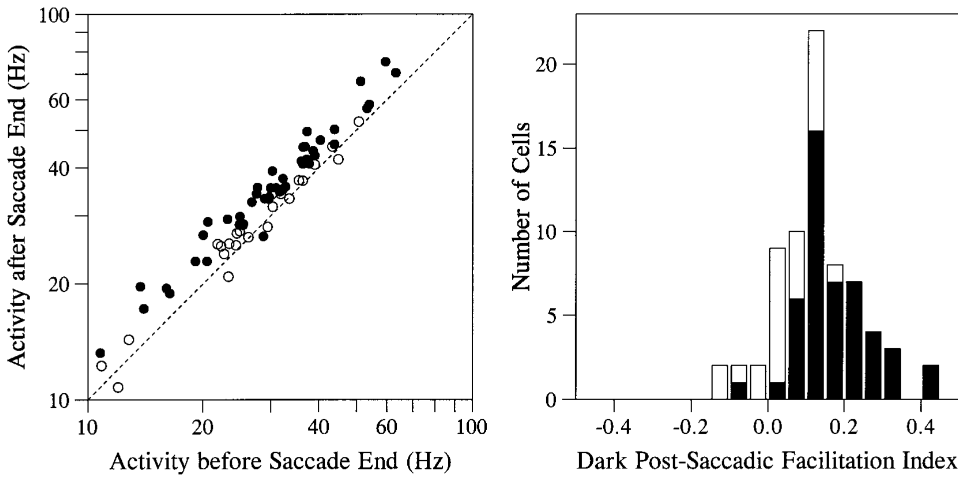

FIG. 4. Effects of saccades in the dark on the activity of 71 LGN cells. Filled symbols or bars indicate statistical significance ($P < 0.05$). *Left*: comparison of activity in 500-ms periods before and after saccade end, plotted on a log-log scale. For the majority of cells, activity increased after saccade end. *Right*: distribution of dark post-saccadic facilitation index (see METHODS). Mean value of this index was 0.13 for all cells and 0.18 for those cells showing a statistically significant facilitation.

saccade end than when they were aligned on saccade start (Fig. 6). Both the presaccadic decrease and postsaccadic increase were highly significant. Ten thousand Monte-Carlo simulations of the underlying data produced not a single dip or peak, relative to the mean activity during the entire analysis epoch, equal in size to those actually observed (for each simulation, the temporal relationship between the original spike train and saccade was varied randomly for every sac-

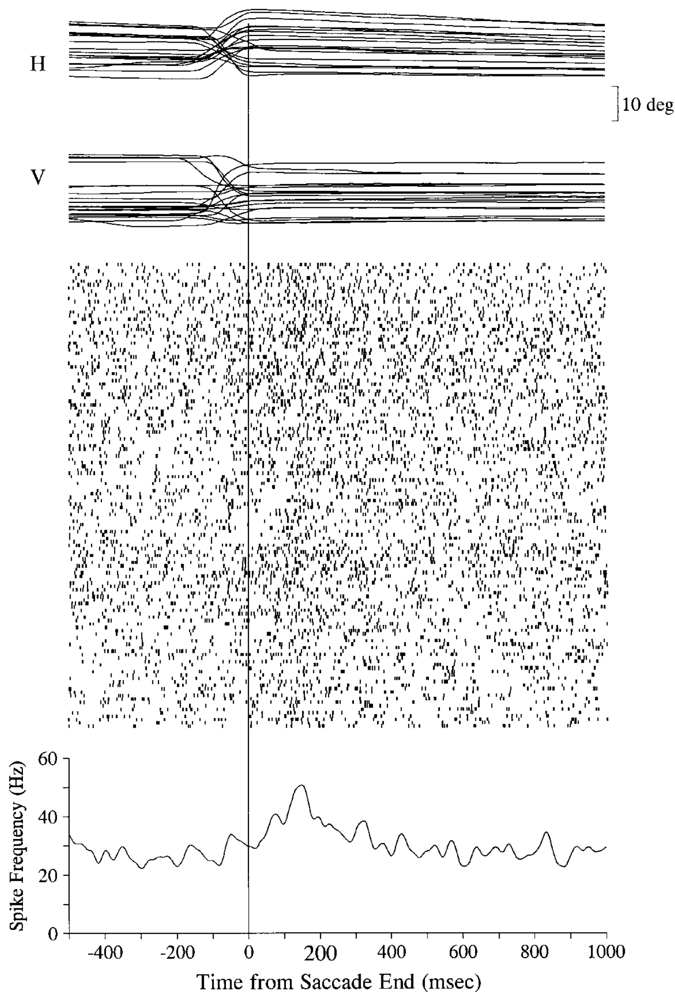

FIG. 5. Effects of spontaneous saccades in the dark on the activity of an ON-center X cell located in layer A, aligned on saccade end. *Top*: horizontal (H) and vertical (V) eye movement traces. *Middle*: raster showing each action potential for all trials meeting the criteria for analysis. *Bottom*: spike density function computed by convolving the spike train with a Gaussian filter with a standard deviation of 10 ms. Postsaccadic facilitation index for this cell was 0.28 ($P < 0.001$), which is among the highest observed.

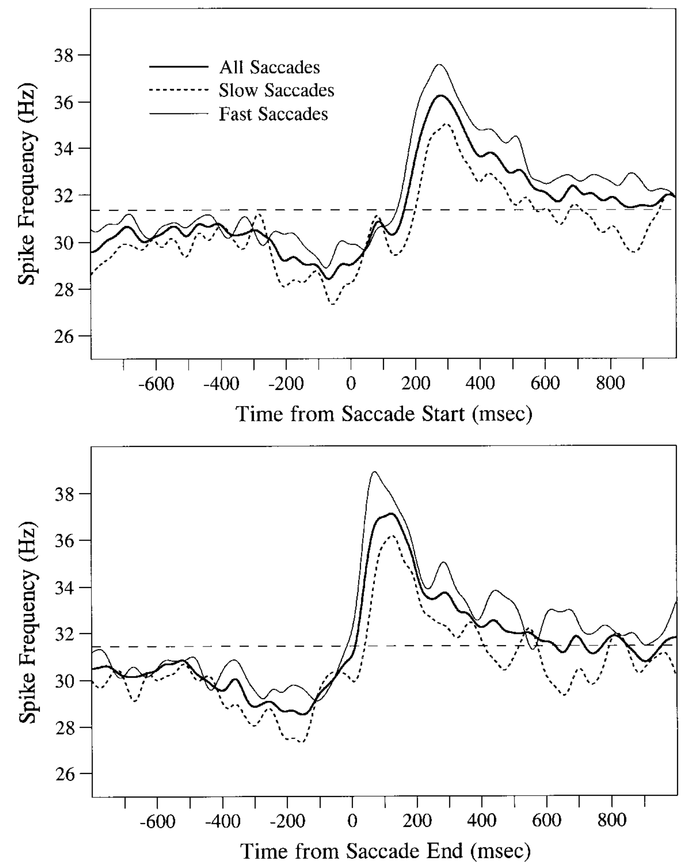

FIG. 6. Activity during saccades in the dark, averaged for all 71 cells, aligned on saccade start (*top*) and end (*bottom*). Bold curves are for all saccades (median peak velocity = 68.1°/s); thin curves are for saccades with peak velocities above the median (mean = 104.5°/s); dashed curves, saccades with peak velocities below the median (mean = 52.8°/s).

cade of the data set). We will refer in the following text to the decrease and increase as a suppression and facilitation, but this should not be taken to imply any particular synaptic mechanism.

The time between saccade end and the peak of the facilitation was related inversely to saccade velocity (Fig. 6). Note that the mean rate of firing of LGN cells was slightly lower for slower saccades than for faster saccades throughout the entire analysis period, suggesting that reduced saccade velocity and "spontaneous" rates of firing are both characteristics of lower arousal states. These curves eventually must wrap around because the postsaccadic level of activity for one saccade is the presaccadic level for the next saccade. Because the intersaccadic interval was quite variable and because pre- and postsaccadic intervals of the same length were analyzed for each cell, the right and left boundaries of Fig. 6 do not represent the same point in the saccade cycle. In fact, activity at these boundaries still differed by 3.2%. However, logic demands that the far right and left portions of the underlying functions must somewhere merge.

Interactions of saccadic eye movements with visually evoked responses

Sixty-nine cells were examined in the visual-stimulation condition for left and right saccade directions, and 36 of these also were examined for up and down directions. Twenty-eight cells were tested in the centrifugal saccade paradigm and 41 in the centripetal saccade paradigm. The mean receptive-field eccentricity was 8.0 ± 2.73° (range 3.2–15.1°). The relationships between saccade and flash timing in different conditions (SOD_V = 0, SOD_P = 0, 50, and 100 ms) are shown in Fig. 7. The velocity-triggered condition essentially probed the beginning phase of saccades, and longer SOD_Ps probed periods of stable eye position immediately after saccades. The eye was generally still moving during the 0-ms SOD_P condition so that condition probed the late phase of saccades.

For each cell, the fractional change in activity (see METHODS) was calculated for each of five 100-ms periods of the response beginning at flash onset, using the corresponding 100-ms epoch of the 500-ms SOD_P condition as baseline. Thirty-six cells (48%) showed a statistically significant facilitation in at least one of the 100-ms intervals examined (collapsed across all saccade directions and corrected for multiple comparisons), and 9 (13%) showed suppression. For nine cells (13%) there was a significant effect of saccade direction, but overall there was no bias toward any particular direction (either for these 9 cells or for pair-wise comparisons of the overall population response). Also, there were no significant differences in the frequency of facilitation or suppression for centrifugal and centripetal saccade paradigms, either for overall effects or for direction-specific effects. We conclude that neither display homogeneity nor the location of the saccade endpoint influenced any of the parameters investigated. In all subsequent analyses the data from centrifugal and centripetal saccades are pooled.

Most cells were excited vigorously by the flash for all SODs employed. A typical response consisted of a primary excitation, a strong suppression, and a rebound followed by a relatively sustained discharge (Fig. 8). All three components

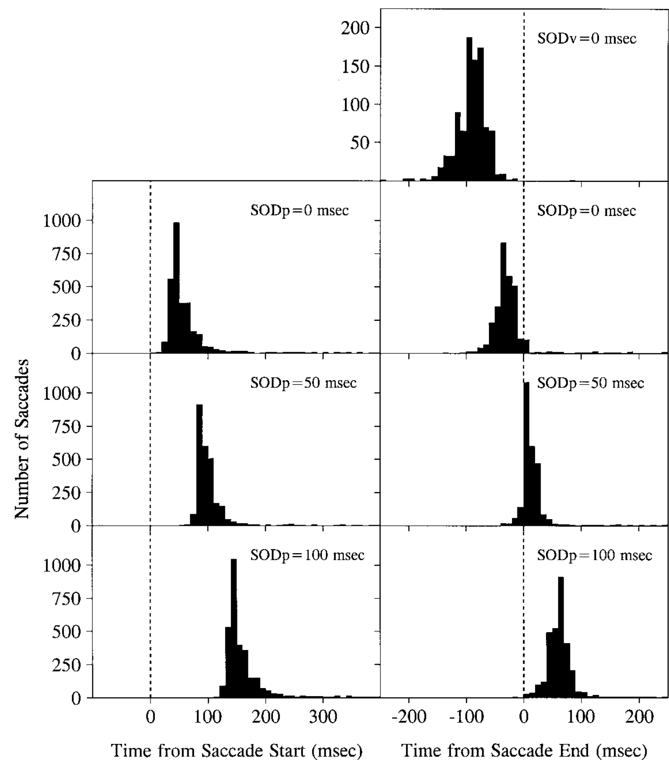

FIG. 7. Timing of flash onset with respect to saccade start (*left*) and saccade end (*right*) for several stimulus conditions. Individual panels show data for a velocity-triggered stimulus (SOD_V = 0 ms) and 3 position-triggered stimulus onset delays (SOD_P = 0, 50, and 100 ms). There is no panel for the 0-ms SOD_V condition for saccade start because this point in time defined saccade start. Data for the 50-ms SOD_V condition are not shown because this condition was used for only a few cells. Data for the 500-ms SOD_P condition are not shown because doing so would require a much compressed scale and for such long delays, small variations in timing are inconsequential.

clearly were present in most cells, although there was considerable variation in magnitude and time course. In addition, among 56 cells the activity of which was reliable enough to judge fine details of the response profile, 20 (36%) showed a small secondary peak at the falling phase of the primary response (Fig. 8). An inflection in the falling phase of the primary response of an additional 16 cells (29%) presumably had the same origin as this secondary peak but was obscured partially by the primary response. Averaged across all cells, the mean firing rate in the 500-ms preceding the flash was 37.6 spikes/s, and after flash onset this increased to a mean peak firing rate of 202.1 spikes/s (measured after smoothing with adaptive kernel estimation; see METHODS).

Saccades had only minor effects on the latency of the flash-evoked response (Fig. 9). We used the response latency for the 500-ms SOD_P flash as baseline, assuming that dynamic effects of saccades on response latency would be gone 500 ms after saccade end. For flash onset immediately after the saccade, there was a small shortening of response latency for X cells (2.05 ms, $P < 0.001$); an even smaller shortening of latency for Y cells (0.71 ms) was not statistically significant. Saccades had no effect on response latency for flashes delivered during the saccade for either X or Y cells.

Although visual responses evoked by flashes were com-

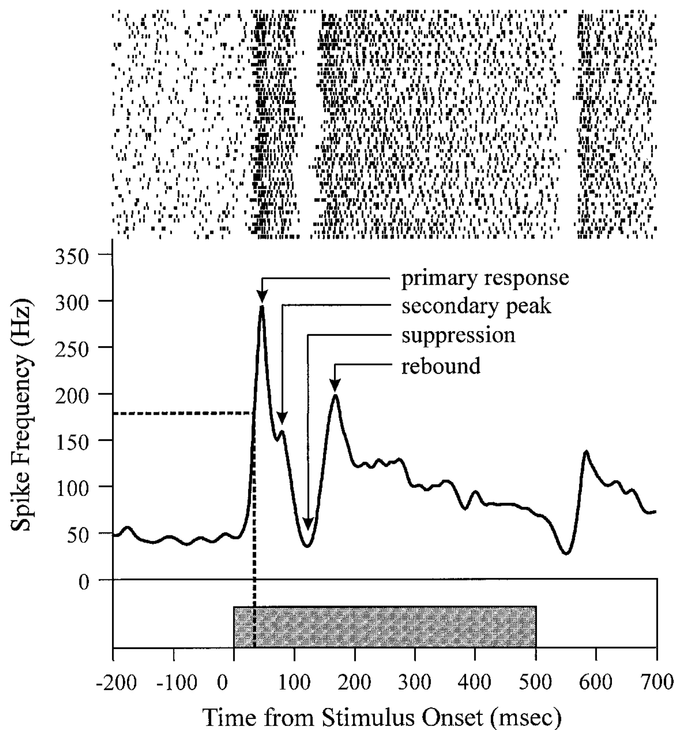

FIG. 8. Typical response of a lateral geniculate nucleus (LGN) neuron (an ON-center Y cell in layer A1) averaged across all flash SODs employed. Flash timing is indicated (▨). There is a large primary response with a secondary peak on its falling phase, followed by a suppression and a rebound. Response is smoothed using adaptive kernel estimation, as described in METHODS. Dashed line illustrates how response latency was determined: time from flash onset for the primary response to reach half-maximum amplitude, relative to the preflash level of activity.

plex and included peak firing rates many times larger than in the dark, the effects of saccades were still similar in magnitude and time course to those observed in the dark. Fractional changes in activity for the dark and visual-stimulation conditions are compared in Fig. 10, with saccades in the dark again separated into fast and slow groups (as in Fig. 6). The main feature was a facilitation of the response beginning at saccade end (there are no data in the visual-stimulation condition for times preceding saccade start). When the data are aligned on saccade start (Fig. 10, *top*), the facilitation in the visual-stimulation condition leads that of the dark condition, whereas the time courses of the facilitations are more similar for alignment on saccade end (Fig. 10, *bottom*). This difference is largely, but not entirely, attributable to longer saccade durations in the dark. As previously pointed out, the latency of the facilitation was related to peak saccade velocity, and saccades in the dark were substantially slower than those in the visual-stimulation condition. Consequently, even when saccade duration is factored out by aligning the data on saccade end (Fig. 10, *bottom*), the facilitation associated with slower saccades in the dark still lags that of the visual-stimulation condition. A good temporal match between facilitations in the two conditions is obtained only when saccade velocities are reasonably similar: for the faster group of saccades in the dark (mean peak velocity = 104.5°/s), the facilitation was essentially identical to that in the visual-stimulation condition (mean peak velocity = 165.7°/s). It should be stressed that this striking similarity occurs in the face of great differences in absolute activity, both between the dark and visual-stimulation conditions and among the various phases of flash-evoked responses in the visual-stimulation condition. This further supports the notion that saccades result in an invariant change in the gain of geniculate transmission that is relatively independent of factors other than saccade parameters themselves.

To evaluate interactions between saccade parameters and the postsaccadic facilitation more systematically, we compared the facilitation in the dark and visual-stimulation conditions, sorting saccades by amplitude, peak velocity, and duration (Fig. 11). In each case, saccades in both conditions were sorted into thirds containing the lowest, middle, and highest values of the parameter examined (there were not enough data for finer subdivision to be meaningful). When neural responses are aligned on saccade end (Fig. 11, *bottom*), the onset of the facilitation is sharper and there is a better match between gain changes in the dark (curves) and during visual stimulation (individual symbols) than when responses are aligned on saccade start (Fig. 11, *top*). This is consistent with the idea that the critical events triggering the facilitation occur closer to saccade end than saccade start, and so we will confine the rest of this description to the data

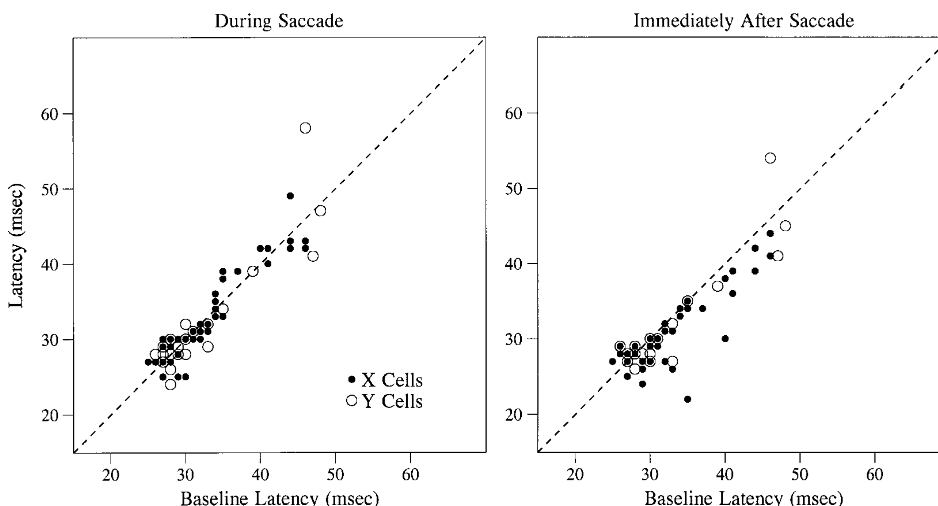

FIG. 9. Effects of saccades on latency of visually evoked response for 68 cells (1 cell was not included because the onset of its response was too sluggish and irregular for a meaningful response latency to be assigned). *Left*: latencies for stimuli delivered during saccades; *right*: latencies for stimuli delivered after saccade end. These are the saccades contributing data to the *left* and *right* of the vertical dashed line in the right panel of Fig. 7, respectively. Abscissa gives the baseline latency for stationary eye position (SOD_P = 500 ms). ●, X cells; ○, Y cells. Mean number of trials contributing to baseline latencies, latencies during saccades, and latencies immediately after saccades were 35.8 ± 17.7, 58.0 ± 17.9, and 60.0 ± 34.8 (SD) ms, respectively.

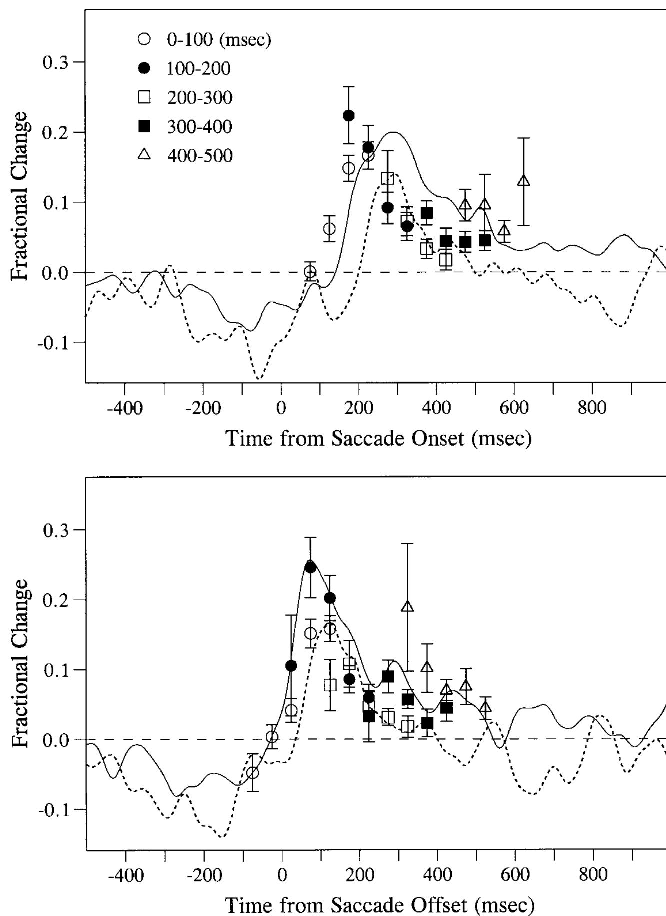

FIG. 10. Time course of fractional changes in activity, aligned on saccade start (*top*) and end (*bottom*), relative to postsaccadic baseline activity. Curves are for activity during saccades in the dark: continuous (broken) curves are for saccades with peak velocity above (below) the median. These are similar, but not identical to curves in Fig. 6; those curves show averaged absolute activity. For the visual-stimulation condition, symbols indicate fractional changes in activity during five consecutive 100-ms intervals starting from flash onset, averaged across all 69 cells, and grouped into 50-ms bins relative to saccade start or end. Fractional changes associated with flashes of different SOD are not explicitly distinguished. Criteria for minimum data in a particular 50-ms bin (see METHODS) resulted in 20 bins (*top*) and 25 (*bottom*), each based on 963–2,456 saccades. Error brackets indicate standard error of the mean. None of the fractional changes for any 100-ms flash interval (individual symbols) differed significantly from the mean fractional change for all 100-ms flash intervals falling in the same 50-ms bin.

aligned on saccade end (Fig. 11, *bottom*). For saccades in the dark, there was a progressive shortening of latency as peak velocity increased (Fig. 11, *bottom middle*; see also Fig. 6). Most of this shortening occurred between the slow- and medium-velocity groups of saccades, suggesting a saturation at 70–80°/s. Mean peak saccade velocity was >80°/s for all three velocity groups in the visual-stimulation condition; this could account for the absence of an observable difference in latency among any of these groups. Because saccade velocity and amplitude are correlated (Fig. 3), it is not clear to what extent the main saccade parameter affecting facilitation latency is velocity per se. The comparisons of the facilitation for the data sorted by saccade amplitude and peak velocity are inconclusive on this point (Fig. 11, *bottom left* and *bottom middle*). However, saccade duration is unlikely to be directly related to the latency of the facilitation because latencies were identical for the dark and visual-stimulation conditions even though the ranges of saccade durations were quite different (Fig. 11, *bottom right*).

Comparisons across cell categories

The distribution of our sample with regard to cell type (X or Y), LGN layer, and polarity of receptive-field center is given in Table 1. Saccade-related, fractional changes in activity for both the dark and visual-stimulation conditions are compared across different categories of cells in Fig. 12. The main difference is that the postsaccadic facilitation was substantially larger (by roughly a factor of 2) for X cells relative to Y cells both in the dark and during visual activation. The data revealed no significant differences in effects of saccades for the other comparisons. Not shown in Fig. 12 are similar comparisons for centripetal versus centrifugal saccade paradigms and for ipsiversive versus contraversive saccades, neither of which yielded significant differences.

Burst responses

For saccades in the dark, the overwhelming number of low-threshold Ca^{2+} bursts consisted of only two spikes (on average, 2.1 spikes/burst). Mean burst frequency was 0.094/s, which is identical to the rate reported by Guido and Weyand (1995). On average, a burst occurred only once every 5.7 saccades (including the entire perisaccade epoch). There were no statistically significant variations in burst rate with respect to saccade timing. During flashes delivered in the visual-stimulation condition, the mean number of spikes per burst was higher than in the dark, averaging 3.3 spikes, but bursts were even rarer, occurring exclusively during the first 100 ms after flash onset. There were 0.021 bursts/flash for the baseline flash (SOD_P = 500 ms), 0.031 bursts/flash for flashes delivered during saccades, and 0.021 bursts/flash for flashes presented within 250 ms after saccade end. The increase during saccades was statistically significant, but even for these flashes there was only a single burst for every 32.3 trials. There were no significant differences between X and Y cells for any of the analyses of burst frequency in either the dark or visual-stimulation conditions.

DISCUSSION

Presaccadic suppression

The data for saccades in the dark revealed a modest suppression of activity that began 200–300 ms before saccade start, extended through most of the saccade, and terminated at saccade end (Fig. 6). By the nature of our paradigms, we could not assess changes in LGN activity preceding the saccade for the visual-stimulation condition, but there is no reason to believe that the underlying effect is absent in the light. Indeed, a hint of a suppression for the earliest flash intervals can be seen in Fig. 10, *bottom.*

The presaccadic suppression was detected only when responses were averaged across many cells. Previous investigators who found no effects of eye movements in the dark for the cat LGN focused on aspects of activity other than response magnitude and did not average responses of large

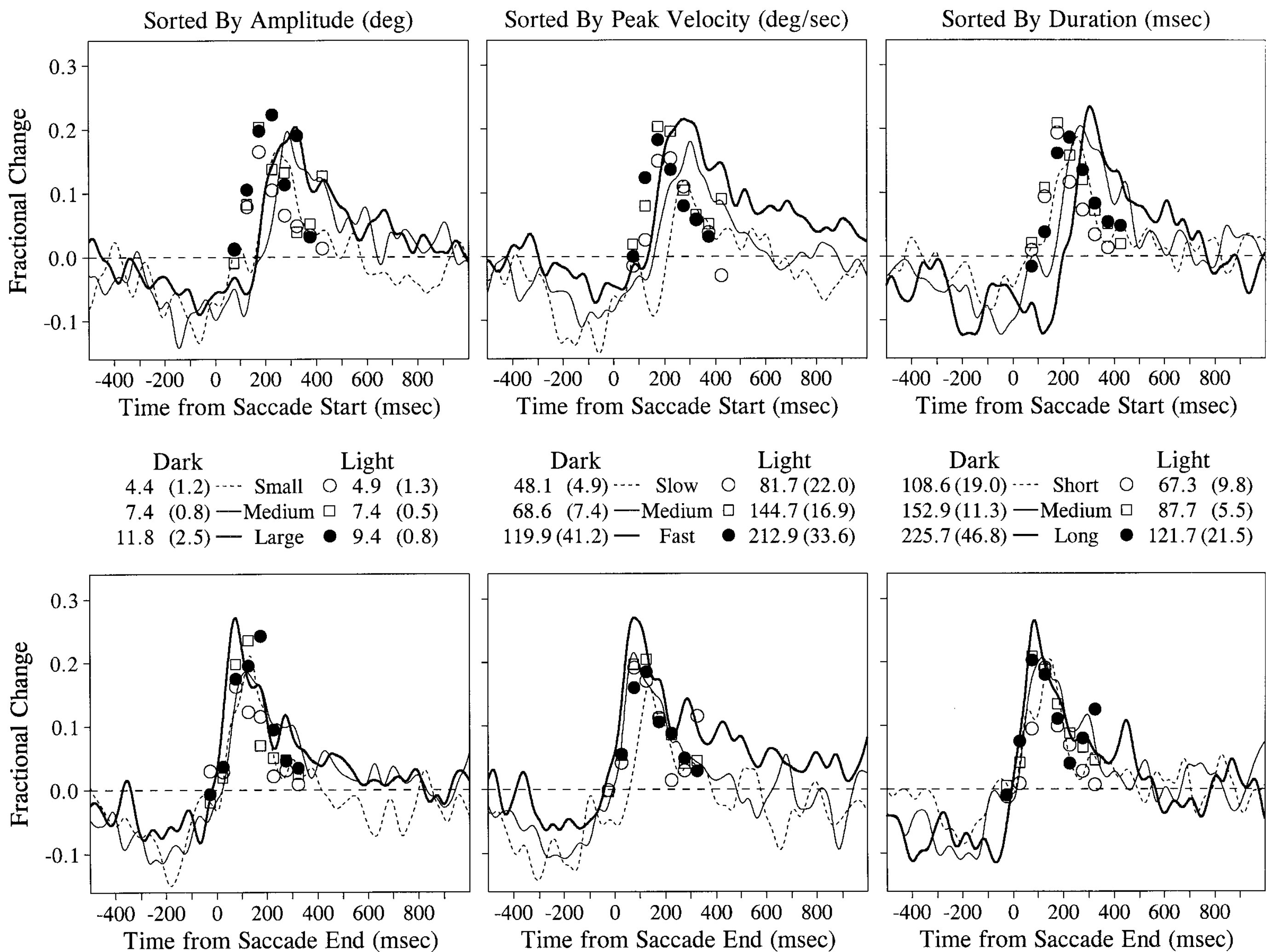

FIG. 11. Time courses of fractional changes in activity, aligned on saccade start (*top*) and end (*bottom*) for each of 3 ranges of saccade amplitude (*left*), peak velocity (*middle*), and duration (*right*). Curves are for activity during saccades in the dark; symbols are for activity during saccades to visual targets. For each panel, saccades are equally apportioned into 3 groups: the thirds with the lowest, middle, and highest amplitudes, peak velocities, and durations. Keys between *top* and *bottom panels* give the mean amplitude, peak velocity, and duration of each group (numbers in parentheses are standard deviations). Conventions are the same as in Fig. 10, except that the number of symbols used is reduced to a minimum by no longer distinguishing among the 5 100-ms intervals into which each flash is subdivided.

numbers of cells (Fischer et al. 1996; Noda 1975). The relatively weak influences of saccades in the dark on the monkey LGN (Bartlett et al. 1976; Büttner and Fuchs 1973) may reflect true species differences, but it is also possible that extensive averaging would reveal saccade-related modulations of activity similar to those in the cat.

Postsaccadic facilitation

The largest effect of saccades on the activity of LGN cells was a facilitation that began at saccade end and peaked 70–130 ms later. The facilitation was independent of the motivation for the saccade in that it was similar for spontaneous saccades in the dark and for rewarded saccades made to discrete visual targets (Fig. 10). It was also independent of the location from which the saccade was launched and the predictability of the saccade-target location because it did not differ for centrifugal and centripetal saccade paradigms. Finally, it was largely independent of the state of the retina: the time course and magnitude of the postsaccadic facilitation were quite similar in the dark and visual-stimulation conditions, even though the firing rate of the LGN cells (and presumably the retinal ganglion cells driving them) differed radically. The only factor definitively influencing the facilitation was saccade velocity with higher velocities producing a larger facilitation that peaked more rapidly up to a critical peak velocity of 70–80°/s (Fig. 11). We conclude that this facilitation is a generalized, saccade-related increase in the gain of LGN transmission, resulting from the interaction of oculomotor and/or proprioceptive signals with retinal signals in a simple multiplicative fashion. Although we cannot exclude the possibility that patterned stimuli might have resulted in a different degree of facilitation, we think it unlikely because of the apparent automatic nature of this process.

The facilitation of the present study was similar to that

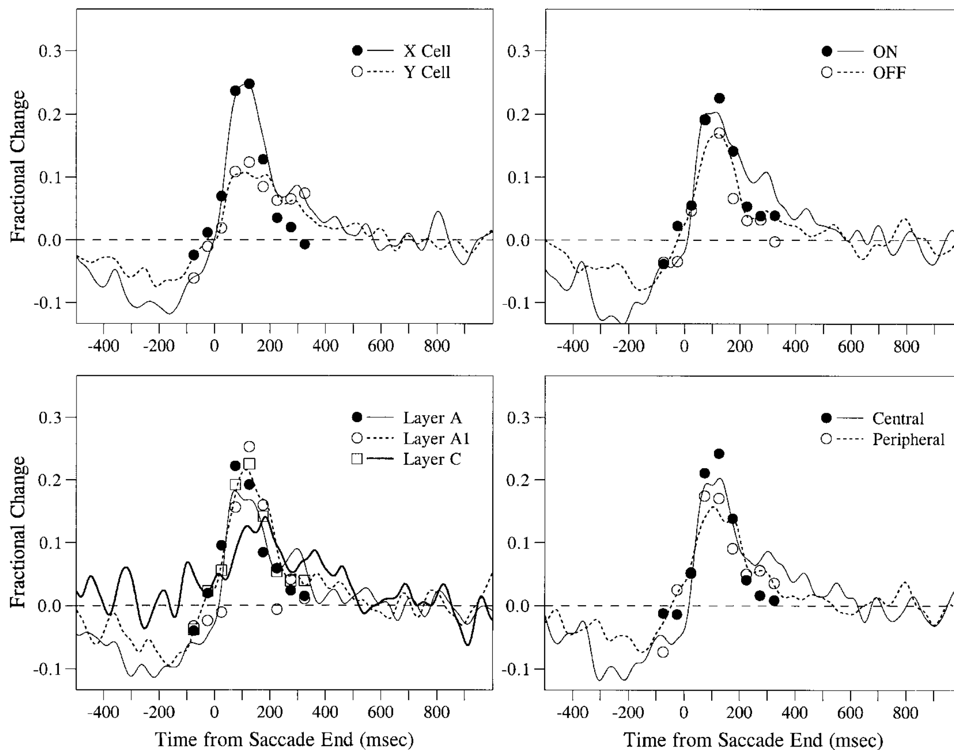

FIG. 12. Time course of fractional changes in activity aligned on saccade end, comparing different categories of cells for saccades in the dark and during visual stimulation: X and Y cells (*top left*), ON- and OFF-center cells (*top right*), different LGN layers (*bottom left*), and cells with receptive fields central to and peripheral to the median receptive field eccentricity of 8.0° (*bottom right*). Conventions are the same as in Fig. 11. Only significant difference for saccades in the dark is in the postsaccadic facilitations of X and Y cells ($P = 0.03$ for the interval from 0 to 250 ms after saccade end). For the 4th and 5th intervals during visually evoked activity (the peak of the facilitation), there was a significant difference between groups only for the X-Y comparison ($P = 0.03$).

observed by Lal and Friedlander (1990) for rapid, passive rotations of the eye in anesthetized cats. Their facilitation was abolished by retrobulbar blocks, implying a proprioceptive source. For the range of visual-field eccentricities corresponding to our sample, it peaked 70–170 ms after the onset of ramp-like, 15° movements lasting 75 ms (they reported suppressive effects only for receptive fields with eccentricity >15°). This is somewhat earlier than the peak we observed, but this is to be expected because they employed a 200°/s velocity ramp. With a ramp, high velocity is reached immediately, whereas with natural eye movements, peak velocity is not reached until well into a saccade.

Low-threshold Ca^{2+} bursts (Guido et al. 1992; Steriade and Llinás 1988) were too infrequent to account for a significant portion of the postsaccadic facilitation. In the dark, they contributed to <1% of the spikes of LGN cells. In the visual-stimulation condition, LGN cells fired, on average, 10.2 spikes in the first 100 ms of visual stimulation, and bursts contributed only ~0.1 spike to this activity. The conditions required for such bursts are the arrival of a depolarizing input after a relatively long period of hyperpolarization (Guido et al. 1992; Steriade and Llinás 1988), and we have no reason to believe that the conditions of the current experiment were conducive to the burst mode.

Noda (1975) reported that the excitability of cat LGN cells was depressed after saccades. However, his results and ours are in no way contradictory: the two experiments address different phenomena, and by virtue of the design of each, the phenomenon revealed by one is largely factored out in the other. Noda (1975) found that when cat LGN cells were driven to high rates of firing by saccades made across patterned stimuli, there was a substantial reduction in the frequency of postsynaptic responses evoked by single electrical shocks of the optic tract. Because moving the pattern at saccade velocities with the eye stationary produced comparable effects, he concluded that most of the reduction was not due to saccades per se but to high rates of firing induced by rapid stimulus motion on the retina however achieved. In the current experiment, the retinal input to the LGN was decoupled from the metrics of the saccade by comparing the postsynaptic responses of LGN cells receiving (presumably) identical retinal input at different times relative to saccade start or end. Noda's phenomenon, which is essentially a gain compression at high firing rates (which he interpreted in the context of visual masking), should have reduced the postsaccadic facilitation to the extent that firing rates were high enough to lead to significant gain compression. This may have been a factor at the highest firing rates evoked in the visual-stimulation condition. Although the difference is not statistically significant, the fractional change in activity at the peak of the facilitation was smaller for the first 100-ms epoch of the flash, which spanned the peak of the visually evoked response, than for subsequent epochs, for which firing rates were substantially less (Fig. 10). Obviously, in the extreme case where LGN cells were driven to the point of response saturation, the postsaccadic facilitation would disappear entirely.

One advantage of using flashes for visual stimuli is that they simplify the measurement of response latencies. The efficiency of retinogeniculate transmission is generally less than unity (Noda 1975) so the activation of facilitative signals to the LGN might lead to a slight shortening of response latency through temporal summation, and this was what we found. The shortening was more detectable for X cells than for Y cells (Fig. 9); this is consistent with the greater postsaccadic facilitation observed for X cells (Fig. 12). Fischer et al. (1996) examined effects of saccades on response latency by comparing latencies during natural saccades across

large, patterned, visual stimuli with those obtained during movement of the same stimuli at saccade velocity while the eye was stationary (they did not quantitatively examine effects of saccades on response magnitude). They also found that saccades shortened latency, but in other ways, their results differed substantially from ours: the latency shifts were several times larger (on the order of 10 ms and >30 ms for some individual cells), they were larger for Y cells than for X cells (on average, ~7 and 15 ms for X and Y cells, respectively), and they occurred during the saccade (where we observed response suppression and no change in response latency). However, the paradigms and method of analysis differ so greatly that these results cannot be directly compared. For example, Fischer et al. (1996) used the first burst of activity to measure latency, and there is no particular reason why this should be related directly to the time for a visually evoked response to reach half-maximum amplitude.

Possible sources of oculomotor signals

Presently, none of the many potential sources of oculomotor or proprioceptive signals to the LGN can be eliminated as contributors to gain changes associated with saccades. This is so even for structures for which such signals have not been previously observed: effects of saccades in the LGN were rarely obvious for single cells, and signal averaging across cells comparable with that of the current study has seldom been employed for other structures. Nevertheless, there are some obvious candidates. The striate cortex appears to receive proprioceptive signals (Ashton et al. 1984; Buisseret and Maffei 1977; Enomoto et al. 1983) and a corollary discharge linked to saccades (Toyama et al. 1984), either or both of which could provide the oculomotor component of an integrated response. To the extent that these signals play a role in the phenomena of the current study, it presumably would be in the postsaccadic facilitation of LGN activity: proprioceptive signals are available only after the movement begins, and the corollary discharge observed by Toyama et al. (1984) peaked 10–70 ms after saccade end (their Figs. 5*B*, 9*A*, and 10*A*). The superior colliculus contains cells the activity of which is strongly time-locked to saccades (Peck et al. 1980). Their discharges (depending on cell category) span the period from 200 ms before to 300 ms after saccade onset (Peck et al. 1980), so collicular activity might play a role in either presaccadic or postsaccadic effects on LGN activity (see Lo 1988; Lo and Xie 1987 for a possible role of the rabbit superior colliculus in saccadic suppression). Other potential sources of oculomotor inputs include the midbrain reticular formation (Singer and Bedworth 1974), the frontal eye fields (Tsumoto and Suzuki 1976), and the pretecto-geniculate pathway (Schmidt and Hoffmann 1992).

Recently, Schmidt (1996) reported that cat pretectal cells projecting to the LGN are excited during saccades, both for saccades made across visual patterns and for saccades in the dark. He proposed that this projection facilitates LGN responses through inhibition of inhibitory interneurons via an intrageniculate circuit identified anatomically by Cucchiaro et al. (1993). If pretectal activity is the source of LGN postsaccadic facilitation, the two should have similar time courses, with the latter lagging the former by an interval equal to the conduction time from the pretectum, plus a period sufficient for two synaptic delays (a couple of milliseconds) (see Fig. 2*C* in Schmidt 1996) and associated integration times of unknown duration. Data on individual pretectal cells seem reasonably consistent with a pretectal source for the LGN postsaccadic facilitation. For example, in Fig. 5 of Schmidt (1996), the peak response of a pretectal cell occurred ~50 ms after saccade end for saccades in the dark. One impediment to considering the pretectum a likely candidate for the postsaccadic facilitation is the report by Funke and Eysel (1995) in which they concluded that pretectal activity suppresses the excitability of LGN relay cells and therefore might contribute to saccadic suppression. However, they examined the effects of visually evoked pretectal signals on LGN activity in anesthetized animals, and it is not clear how this relates to saccade-induced pretectal activity.

Function of saccade-related gain changes

The effect of saccades on the LGN can be summarized as a biphasic change in the gain of LGN transmission: a presaccadic decrease in gain that smoothly changes to a postsaccadic increase at saccade end. One could interpret these two phases as partial solutions to distinctly different problems: the suppression raising perceptual thresholds to avoid confusion resulting from rapid image motion and the facilitation aiding analysis of new images after each saccade. However, although there is both perceptual and physiological evidence for centrally originating saccadic suppression, there is at present no need to assume that the function of this suppression is to avoid perceptual confusion. As pointed out by Judge et al. (1980b), the threshold elevation associated with saccadic suppression is insufficient to prevent perception of much of the image for natural scenes. To illustrate this point, consider the demonstration of centrally mediated saccadic suppression of Burr et al. (1994). They had subjects make saccades parallel to sinusoidal gratings, a simple and elegant way of eliminating the confounding of image motion across the retina during saccades. They found an increase in threshold for the perception of gratings that was specific to isoluminant gratings of low spatial frequency. This increase, which they attributed to magnocellular LGN, raised the contrast threshold from ~1 to 10% for the maximally affected spatial frequency. A threshold elevation of a factor of 10 is certainly a large effect, but nevertheless, the contrast of natural images is commonly well above 10%. The fact is that LGN cells respond vigorously as images are swept across the retina during natural saccades (Bartlett et al. 1996; Büttner and Fuchs 1973; Noda 1975). Were suppression at the LGN level required to prevent perceptual confusion during saccades, we continually would experience such confusion, but because we do not, other functions for centrally mediated saccadic suppression should be considered. We remind the reader that our study was designed to isolate oculomotor signals to the LGN, and as such, it purposely excluded visual masking effects that might more effectively suppress perception during saccades. Thus we do not argue against the importance of perceptual saccadic suppression. However, we see no reason to invoke it as an explanation for the saccade-related gain changes observed in the current experiment.

As an alternative to separate roles for saccadic suppression and facilitation at the level of the LGN, we suggest that both phenomena can be interpreted as facilitating the registration and analysis of new images after each saccade (Jung 1972; Singer 1977). Perhaps one should consider the presaccadic suppression and the postsaccadic facilitation to be a coordinated change in gain directed toward a single goal: maximizing temporal contrast between the pattern of activity near the end of one fixation and that which will need to be evaluated when the eye settles on the next location. The underlying assumption is that the impact on perception of a new pattern of cortical activation will be determined as much by the change and/or rate of change of activity as by the absolute level of activity. Looked at in this way, the overall gain change is substantial: an increase from the presaccadic to the postsaccadic period of ∼30%. Larger facilitations might result in too many cells being driven into saturation. This proposal is consistent with the time course of LGN gain changes. If the primary function of the suppression is to prevent perceptual confusion, one would expect it to be in full force through most of the saccade. However, it begins well before the saccade, is maximum near the beginning of the saccade, begins to dissipate at saccade start, and is gone by saccade end (Fig. 10).

The postsaccadic facilitation was considerably larger for X cells than for Y cells (Fig. 12). Perhaps it takes longer to analyze the details of a new image than to register the general outlines, and the facilitation of the X-cell channel is larger because it is the high-resolution channel. Another reason could be related to different response dynamics of X and Y cells. Insofar as the response of Y cells is more transient under natural viewing conditions, the contrast in activity before and after an eye movement would be greater for Y cells than for X cells (because the response of a Y cell would have a greater tendency to decay during the period of stationary gaze just before a saccade). Thus the postsaccadic facilitation may be less of a benefit for Y-cell input.

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