Exp Brain Res (2006) 168: 62–75
DOI 10.1007/s00221-005-0093-z

RESEARCH ARTICLE

D. W. Royal · Gy. Sáry · J. D. Schall
V. A. Casagrande

Correlates of motor planning and postsaccadic fixation in the macaque monkey lateral geniculate nucleus

Received: 8 January 2005 / Accepted: 5 May 2005 / Published online: 7 September 2005

Abstract There is significant controversy regarding the ability of the primate visual system to construct stable percepts from a never-ending stream of brief fixations and rapid saccadic eye movements. In this study, we examined the timing and occurrence of perisaccadic modulation of LGN single-unit activity in awake-behaving macaque monkeys while they made spontaneous saccades in the dark and made visually guided saccades to discrete stimuli located *outside* the receptive field. Our hypothesis was that the activity of LGN cells is modulated by efference copies of motor plans to produce saccadic eye movements and that this modulation depends neither on the presence of feedforward visual information nor on a corollary discharge of signals directing saccadic eye movements. On average, 25% of LGN cells demonstrated significant perisaccadic modulation. This modulation consisted of a moderate suppression of activity that began more than 100 ms prior to the initiation of a saccadic eye movement and continued beyond the termination of the saccadic eye movement. This suppression was followed by a large enhancement of activity after the eyes arrived at the next fixation. Although members of all three LGN relay cell classes (magnocellular, parvocellular, and koniocellular) demonstrated significant saccade-related suppression and enhancement of activity, more cells demonstrated postsaccadic enhancement (25%) than perisaccadic suppression (17%). In no case did the timing of the modulation coincide directly with saccade duration. The degree of modulation observed did not vary with LGN cell class, LGN receptive field center location, center sign (ON-center or OFF-center), or saccade latency or velocity. The time course of modulation did, however, vary with saccade size such that suppression was longer for longer saccades. The fact that activity from a percentage of LGN cells from all cell classes was modulated in relationship to saccadic eye movements in the absence of direct visual stimulation suggests that this modulation is a general phenomenon not tied to specific types of visual stimuli. Similarly, because the onset of the modulation preceded eye movements by more than 100 ms, it is likely that this modulation reflects higher order motor-planning rather than a corollary of mechanisms in direct control of eye movements themselves. Finally, the fact that the largest modulation is a postsaccadic enhancement of activity may suggest that perisaccadic modulations are designed more for the facilitation of visual information processing once the eyes land at a new location than for filtering unwanted visual stimuli.

Keywords Primates · Vision · Thalamus · Eye movement · Saccade

D. W. Royal · V. A. Casagrande
Center for Molecular Neuroscience, Vanderbilt University,
Nashville, TN 37232-2175, USA

Gy. Sáry · V. A. Casagrande
Department of Cell and Developmental Biology,
Vanderbilt University Medical School,
Medical Center North U3218,
Nashville, TN 37232-8240, USA

J. D. Schall · V. A. Casagrande
Center for Integrative and Cognitive Neuroscience,
Vanderbilt University,
Nashville, TN 37232-2175, USA

J. D. Schall · V. A. Casagrande
Department of Psychology, Vanderbilt University,
Nashville, TN 37232-2175, USA

J. D. Schall · V. A. Casagrande
Vanderbilt Vision Research Center,
Vanderbilt University, Nashville, TN 37232-2175, USA

V. A. Casagrande (✉)
Department of Ophthalmology and Visual Sciences,
Vanderbilt University,
Nashville, TN 37232-2175, USA
E-mail: vivien.casagrande@vanderbilt.edu
Tel.: +1-615-343-4538
Fax: +1-615-936-5673

Gy. Sáry
Department of Physiology,
University of Szeged, Szeged, Hungary

Introduction

The fact that the visual world appears stable in spite of multiple saccadic eye movements is remarkable. Mammals with well-developed central vision are the most likely to make saccadic eye movements even though such movements present special challenges for their visual systems. Rapid ballistic eye movements (saccades) can: (1) blur the image by sweeping the visual scene and (2) require that separate "snap shots" of the visual scene from different fixations be integrated with each other in order to maintain visual stability. Many psychophysical studies have shown that some, but not all, visual information is suppressed during saccadic eye movements (see Volkmann 1986 for review). For example, it is well known that we cannot see our eyes move when looking in a mirror although some spatial details can be detected when the eyes are in flight. Controversy still exists over where saccadic suppression occurs in the brain and what information is actually suppressed (Castet and Masson 2000; Thilo et al. 2004). Controversy also exists about how visual information acquired during each fixation is linked across fixations (see Khayatt et al. 2004). At the level of the lateral geniculate nucleus (LGN), there is some evidence both in cats and primates that signals can be modulated during saccadic eye movements (Jeannerod and Putkonen 1971; Bartlett et al. 1976; Noda 1975; Lee and Malpeli 1998; Ramcharan et al. 2001; Reppas et al. 2002). Results, however, have been quite variable with some investigators finding little modulation of LGN cells by saccadic eye movements (Büttner and Fuchs 1972), some investigators finding modulation limited to one cell class (magnocellular cells in primates) (Ramcharan et al. 2001), and some investigators finding different types of modulation in different LGN cell classes (Lee and Malpeli 1998; Reppas et al. 2002). The variability in effects reported could be explained, in part, by species differences (e.g., cats vs. monkeys), the experimental design, or hypotheses that motivated the design.

In light of these conflicting results within the literature, we examined saccade-related changes recorded in single LGN neurons in macaque monkeys making saccades under two conditions, target-directed visually guided saccades made to stimuli presented outside the LGN receptive field (RF) and spontaneous saccades made in total darkness. Our hypothesis was that activity in the LGN is modulated by efference copies of motor plans and is not dependent directly on the presence of visual signals nor a corollary discharge of signals that directly control saccadic eye movements. Instead, the system is optimized to enhance information when the eyes land at each new location following a movement by suppressing signals before the eyes begin to move and boosting signals once a stable new position is achieved regardless of the visual conditions. Comparing LGN activity during saccades made in both the presence and absence of visual cues but without stimulating the LGN RF directly is informative for two reasons. First, changes in activity within the LGN caused by direct stimulation of the RF are difficult to dissociate from changes caused by eye movements given the very small RFs of LGN cells and the complex responses of LGN cells to direct stimulation. Second, comparing visually guided saccades and spontaneous saccades helps address the question of whether saccadic suppression is a general mechanism linked to *all* saccadic eye movements or is predicated upon feedforward retinal stimulation. Finally, using a more precise method to define the time course of modulation (the Poisson analysis), in addition to more traditional methods, can measure more accurately the likely extraretinal sources of LGN cell modulation by saccadic eye movements.

Materials and methods

Subjects

Two macaque monkeys (*Maccaca radiata*, 6 kg, male) served as subjects. Monkeys were cared for in accordance with the National Institutes of Health Guide for the Care and Use of Laboratory Animals and the guidelines of the Vanderbilt University Animal Care and Use Committee under an approved protocol.

LGN localization and surgeries

LGNs were localized via images created with either a GE Signa 1.5-Tesla MRI or a GE Signa 3-Tesla MRI scanner. Under general anesthesia [10 mg/kg ketamine (IM) with 0.4 mg/kg xylazine (IM) supplemented as needed], monkeys were secured in a titanium stereotaxic apparatus fitted with hollow ear bars filled with distilled water. The tips of the ear bars were aligned with the bases of the orbits to serve as landmarks to define the horizontal plane. After locating both ear bars in the same coronal section perpendicular to this horizontal plane, a series of 1 mm thick coronal images were taken anterior to the ear bars in overlapping 0.5 mm increments. LGN coordinates were calculated by G.E. 3.9 software and compared to a standard stereotaxic atlas for the macaque monkey (Paxinos et al. 2000).

Under general anesthesia [10 mg/kg ketamine (IM)] and 1.5–3.0% isofluorane gas (tracheal tube) and using aseptic procedures, a stainless steel head post (courtesy Dr. Ralph Siegel, Rutgers) was secured to the rear of each monkey's skull using titanium maxillofacial screws (2.7 mm, Synthes, West Chester, PA, USA) and Methyl Methacrylate cement (BIOMET, Warsaw, IN). A recording chamber (stainless steel, 20 mm diameter, Crist, Hagerstown, MD) was centered over the left LGN using coordinates calculated from the MRI and secured to the skull in the same manner described above. Finally, a search coil was implanted underneath the conjunctiva

of the monkeys' right eye using either sutures (Ethicon 6.0 or 7.0, Johnson and Johnson, Piscataway, NJ, USA) or tissue glue (Nexaband S/C, Closure Medical Corporation, Raleigh, NC, USA) placed at cardinal locations around the eye (Judge et al. 1980). The bone enclosed within the recording chamber was removed in a subsequent aseptic surgery.

Training and tasks

The monkeys were trained to enter and exit a primate chair on command and permit head fixation for extended periods. Then they were placed in a completely darkened room and conditioned to fixate a fixation spot (single white pixel, 1/17°) centered on a computer monitor 57 cm in front of the monkey. The room was examined for light leaks by having one investigator remain in the room for at least 45 min in darkness to ensure full dark adaptation. This investigator paid close attention to potential sources of light during this period. Training was performed using a controlled water access paradigm and monkeys were reinforced with water or juice for maintaining gaze within a 1°×1° invisible window centered over the fixation spot.

Visually guided saccades

The monkeys were trained to make a visually guided saccade to a target following a visual cue (Fig. 1a). After 350–650 ms of constant fixation, the fixation spot changed color from white to green, cueing the monkey to prepare to make a saccade to an impending target. The cue was presented for 350–650 ms. Simultaneous with the removal of the cue, the target stimulus (see below) was placed in the hemifield opposite to the plotted LGN RF at the same relative eccentricity and elevation as the RF (mean eccentricity = 8.8°). Target placement and timing of cue removal prevented direct LGN RF stimulation during the trial (Fig. 1b). They were rewarded only when they shifted gaze to the target within 250 ms of target onset and remained within the invisible target window (~2°) for ~350–650 ms. The duration of the fixation, cue and target periods varied randomly so as to eliminate the monkeys' ability to predict trial events (see *Conditional Failure Density Function*, Johnson and Balakrishnan 1994). Incorrect trials were aborted and removed from the analysis. Trials were blocked and at least 20 correct trials were recorded for each cell.

Spontaneous saccades in the dark

LGN single units were recorded while the monkeys made saccades freely in the dark for at least 200 s (Fig. 2). Then they were rewarded with juice at the conclusion of each recording period only after data acquisition had terminated. Reward was not contingent upon eye movements or any other behavior.

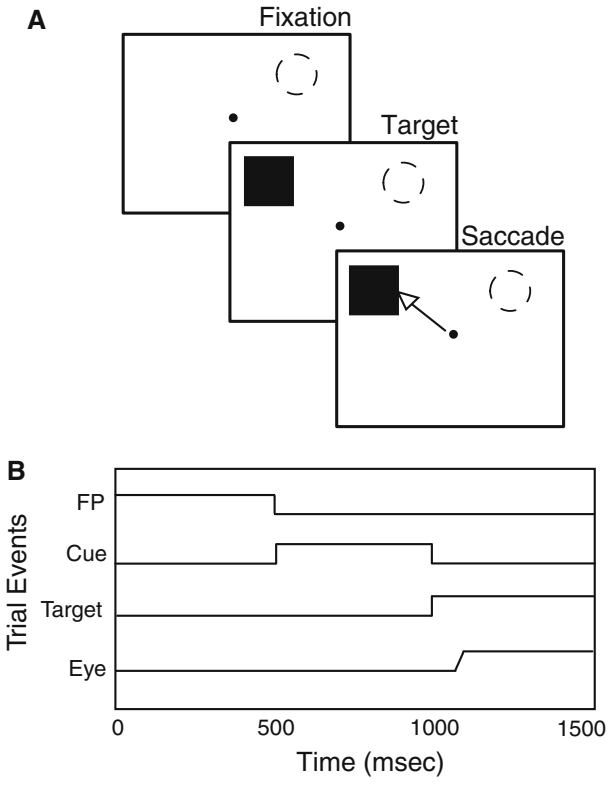

Fig. 1 Diagrams demonstrating the behavioral requirements **(a)** and chronology **(b)** of the visually guided saccade task. **a** The three frames indicate sequential events in the visually guided saccade task. Presented this way, moving down and right marches you forward in time. The small central circle represents the fixation spot. The dashed circle represents the mapped receptive field (RF). The black square represents a stimulus presented outside the RF. The arrow outlines the path of the monkey's eyes as he shifts gaze from the fixation spot to the target stimulus. Trials begin with the monkey fixating the fixation spot (Fixation). The target appears (Target) outside the cell's RF. Finally, the monkey makes a visually guided saccade to the target (Saccade) for reward. **b** Chronology of the visually guided saccade task. Trials begin with the monkey fixating the fixation point (FP). The FP is then replaced with a green cue (Cue) of the same size and luminance in order to instruct the monkey to make a saccade to the impending target. Continued fixation of the cue is followed by the presentation of a target stimulus (Target) outside the LGN cell's RF. After a short latency, the monkey responds by making a saccade to the target and fixating the target for reward. The exact timing of trial events was presented randomly (±30%) in order to prevent the monkey from predicting trial events

Recording and stimulus presentation

Extracellular single-unit activity from the LGN was recorded with Parylene-coated tungsten microelectrodes 1–3 MΩ (FHC, Bowdoinham, ME, USA). Cellular responses were amplified, bandpass filtered and fed into a window discriminator (BAK Instruments, Mount Airy, MD, USA), an audio monitor, and an oscilloscope for

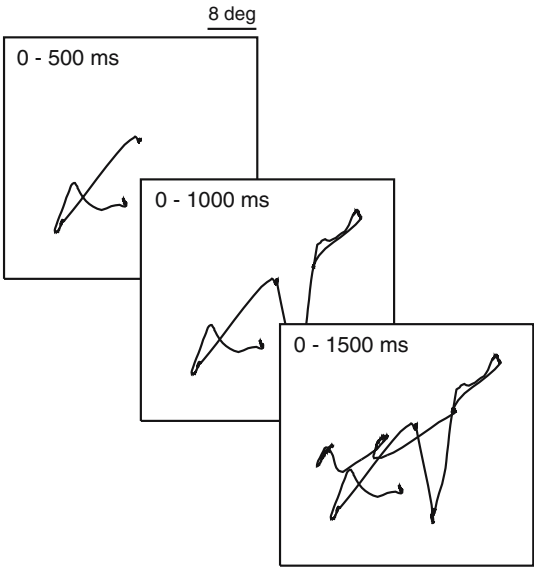

Fig. 2 Example record of the monkey's shift in gaze while moving his eyes freely in a completely darkened room (spontaneous saccade condition). Each frame illustrates the cumulative path of the monkey's gaze. As before, moving down and right marches you forward in time

monitoring. A PC-based real-time data acquisition system (Visual TEMPO, Reflective Computing, St. Louis, MO, USA) controlled trials, recorded spike times (1 kHz), recorded eye position (250 Hz) and presented stimuli. Eye position data were then interpolated using a method of cubic splines and resampled at 1 kHz. Analysis of the data was performed using MATLAB (Math Works Inc., Natick, MA, USA) and SPSS statistical software.

A Sony Multiscan GPS 500 computer monitor (70 Hz refresh rate, 640 × 480 pixel resolution, 36°× 29° visible area, stimulus luminance 1.8 cd/m^2, contrast ~100%, San Jose, CA, USA) presented visual stimuli in a completely darkened room. Calibrated look-up tables corrected for luminance non-linearity and colors were evaluated periodically by color photometry (Minolta CA 100, Mahwah, NJ, USA) to ensure isoluminance. To help reduce light scatter and shield from electrical noise, our stimulus monitor is fitted with a diffuser screen. To test whether any light scatter was being reflected from the monitor, two researchers sat in the darkened room for ~30 min to become dark adapted before scanning the monitor with and without stimuli present. Outside the stimulus itself, the rest of the monitor appeared invisible.

Data analysis

Saccade detection

Analyzed saccades met the following criteria: at least 8 ms of monotonic change in eye position and a peak velocity of at least 40°/s. The beginning of every saccade was defined as the first millisecond of the 8 ms of monotonic change in eye position and the ending of every saccade was defined as the millisecond the eye velocity had slowed to 5°/s. These criteria alone were sufficient to isolate individual saccades during the visually guided saccade task because there was but one saccade during each trial and each saccade was flanked by at least 350 ms of fixation (as per task requirements). Such a design ensured that pre- and postsaccadic modulations from one saccade did not "blend" with those of an adjacent saccade. Saccades produced during the spontaneous saccade task however, were made following variable, if any, fixation intervals. Therefore, additional constraints, based on observations from the visually guided saccade task, were placed on spontaneous saccades selected for analysis. Specifically, pre- and postsaccadic modulations observed during the visually guided saccade task never exceeded a combined total of 500 ms. Thus, requiring that at least 1,000 ms of constant fixation flank each spontaneous saccade ensured that modulations from adjacent saccades did not "blend" with one another. Although these selection criteria are designed to optimize detection of modulations related to saccades, our criteria by necessity excluded from the analysis a subset of saccades. For this study, 12% of spontaneous saccades, on average, did not meet these criteria and therefore were not included in the analysis.

Poisson analysis

Significant modulations of activity and visual response latencies were examined using a Poisson spike train analysis described originally by Legendy and Salcman (1985) and applied by Hanes et al. (1995). By comparing recorded spike trains to a Poisson distribution based on the mean firing rate over the entire trial, we identified nonrandom patterns of spiking. After saccades were detected (see *Saccade detection* above), the corresponding segments of the spike train were aligned on either saccade beginning or ending before running the Poisson analysis to identify the beginning of the significant modulation in spike density relative to saccade endpoints. In order to identify the beginning and ending of a period of suppression of activity, trials were aligned on saccade start. From the set of trials that demonstrated significant saccade-related suppression, both the modal start point and end point were calculated according to the following formula

$$p[(t_i + t_{i+J})/2] = J/N(t_{i+J} - t_i)$$

where N is the total number of significant deviations detected and J was set to a value that provided the most accurate estimates of the mode (usually $N/4$, but not less than 3). The time of $(t_i + t_{i+J})/2$ that generates the largest value of p was the estimated mode, which corresponded to the time when the spike rate deviated significantly from what would have been predicted by a Poisson distribution based upon the mean firing rate over the duration of the trial. To examine for post-

saccadic modulation, trials were aligned on saccade end and the modal start points and end points of these changes were again calculated from trials where significant modulation was detected.

Magnitude of modulation

The relative magnitude of modulation was calculated in two ways. First, we divided trials into epochs based upon the Poisson analysis described above using the modal analysis to establish the beginning and ending of each epoch on a trial-by-trial basis. The baseline was arbitrarily defined as the first 100 ms of fixation beginning 500 ms prior to a saccade. This avoided sampling during any period that might be defined as a presaccadic suppression epoch. Spike density functions were convolved using a growth constant (1.0 ms) and decay constant (20 ms) based upon values reported in the literature (Sayer et al. 1990; Mason et al. 1991; Kim and Connors 1993; Sato and Schall 2001). These values were selected in order to approximate the time-course of the postsynaptic potential, and thereby create spike density functions that conform to electrophysiology. The number of spikes per second generated during this baseline epoch was then compared to the spikes per second during the presaccadic modulation epoch, the saccade epoch and the postsaccadic modulation epoch using an ANOVA and post-hoc Scheffé test. Second, in order to compare more closely to analyses performed by others, we operationally defined epochs with the baseline and saccade epochs defined as above, the presaccadic epoch defined as the 50 ms block prior to saccade onset and the postsaccadic epoch as the 100 ms block after saccade end. Again these epochs were compared using an ANOVA and post-hoc Scheffé test. Alpha levels were adjusted using a modified Bonferroni technique to control for multiple *t*-tests (Hochberg 1988). The Poisson analysis differed from the more traditional epoch analysis in that fewer cells were found to be modulated significantly by saccadic eye movements in both the visually guided and spontaneous saccade conditions. This was particularly true for cells with very low baseline rates of firing where the Poisson was incapable of detecting significant suppression, suggesting that floor effects may be a limiting factor with the Poisson analysis. Although the Poisson analysis had difficulty detecting significant decreases in activation when baseline rates of firing were low, more than 50% of our population had sufficient baseline activity for the Poisson analysis to determine the point in time where significant suppression began, thus providing us with a reliable measure of the time course of this suppressive signal.

LGN cell classification

Receptive fields were mapped using a bar that swept the length and breadth of the single unit's RF while the monkey fixated the fixation point. The bar's height, width, orientation, direction of motion, and color were defined by user input and controlled by TEMPO software (Reflective Computing, St. Louis, MO, USA) in order to define the borders of the cell's RF. LGN cell RF center polarity (e.g., ON or OFF response to target stimulus) was determined by using colored stimuli optimized for the cell's RF center response. Isoluminant stimuli of several colors [neutral gray (CIE, $x = 345$, $y = 467$), red (CIE, $x = 630$, $y = 338$), green (CIE, $x = 290$, $y = 606$), and blue (CIE, $x = 143$, $y = 058$)] were used. The borders of target stimuli extended beyond the edges of the single unit's plotted RF center to compensate for the effect of fixational microsaccades. LGN cells which increased their rate of firing significantly to RF center stimulation were classified as being ON-center, while cells that decreased their rate of firing significantly to RF center stimulation were classified as being OFF-center.

Our monkeys are still participating in experiments, thus histological confirmation of recording locations was not possible. Therefore, we classified LGN cells into K, M, and P cells using other established criteria. Not every LGN cell within our sample population could be classified using all of the following criteria; therefore, we classified only those cells that could be sorted according to at least three of the following seven criteria: (1) shifts in ocular dominance, (2) depth within the LGN, and (3) RF position. Shifts in ocular dominance between layers are only useful as a criterion if the location of the RFs along a vertical recording pass remain at or below the horizontal meridian. This RF position allows one to pass consecutively from the P layers at the top of the nucleus to the M layers at the bottom of the nucleus without the danger of re-penetrating the P layers (Lee and Malpeli 1998). If the penetration is appropriate, the RFs located at the bottom of the contralaterally innervated M layer will lie closest to the horizontal meridian at the most eccentric location. (4) RF size. In general, it has been found that K cell RFs are larger than M cell RFs, which, in turn, are larger than P cell RFs (see Martin et al. 1997; Xu et al. 2001). (5) Color selectivity. P and K LGN cells have been shown to be sensitive to wavelength and M cells have been found not to be (Wiesel and Hubel 1966; Martin et al. 1997). Cells clearly selective for red/green were considered P cells, those selective for blue were considered K cells and those that were unselective were considered M cells assuming this classification was supported by our other criteria. (6) Latency to stimulus onset or offset. Although there is considerable overlap, K LGN cells show the longest latencies and M cells the shortest latencies to stimulus onset (Irvin et al. 1993; Schmolesky et al. 1998; Ichida et al. 2003). Latency has been measured in various ways. We used a Poisson spike train analysis described above to calculate visual response latencies to an optimized target. Empirical response latency values were compared with our own prior study and previously reported literature values to ensure our cell classifications conformed

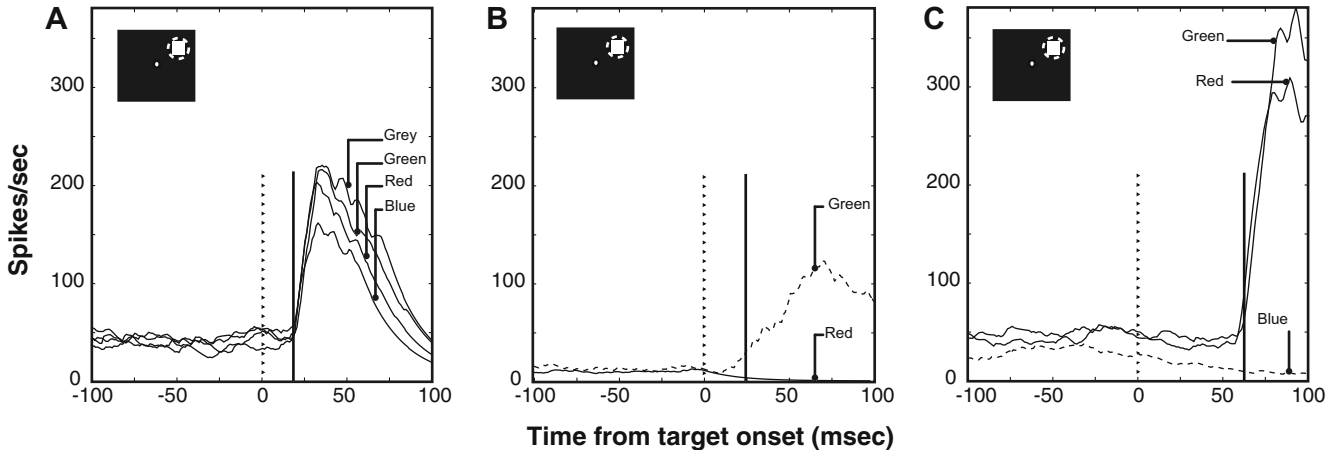

Fig. 3 Peristimulus time histograms (PSTH) demonstrating the mean responses of three LGN cells to presentations of isoluminant red, green, blue, and gray stimuli inside the cell's RF. Trials have been aligned to target onset (0 ms). Traces are labeled to indicate the cell's average response over 20 trials to a preferred stimulus inside the cell's RF. The vertical black line indicates the cell's response latency to target onset as determined by the Poisson analysis (see text for details). **a** PSTH of an LGN M cell which was color non-selective, showing increased activation to all colored stimuli. Response latency to target onset for this LGN cell was determined to be 18 ms. **b** PSTH of an LGN P cell that was characterized by a red/green color opponent RF center. Response latency to target onset for this LGN cell was determined to be 24 ms. **c** PSTH of an LGN K cell that was characterized by a blue-OFF/red/green-ON color opponent RF center. Response latency to target onset was determined to be 66 ms

to expectations (Irvin et al. 1993; Schmolesky et al. 1998; Royal et al. 2004; see Fig. 3). (7) Response transience. M cells generally give a more transient response compared to P cells (see Casagrande and Norton 1991). The latter criterion cannot be used for K cells since K cells can exhibit either property.

According to these criteria our population of cells recorded in the visually guided saccade task included 41 ON-center cells, 34 OFF-center cells, 10 M, 51 P, and 14 K cells. In the spontaneous saccade task there were 30 ON-center cells, 36 OFF-center cells, 9 M, 55 P, and 2 K cells. Twenty-three cells could not be classified sufficiently by the above criteria and were not analyzed further.

Results

Spontaneous saccades: Poisson analysis

As can be seen from the record shown in Fig. 2, saccades made in the dark were made at irregular intervals with both short and very long pauses in between. During these pauses the monkey's eyes would often drift slowly rather than remain at one location—presumably because the room was in fact completely dark and lacked any visual stimuli on which the monkey could fix his gaze. Rhythmic vertical drifts were excluded as these are general indicators of sleep; however, we included sufficiently long, slow lateral drifts (at least 1000 ms at $< 3°$/s) as baseline fixation. We recorded from 66 LGN neurons while the monkeys made spontaneous saccades ($N = 125 \pm 33$ saccades) in the dark. Given the restriction that we could not analyze saccades occurring too close together (see "Methods"), our analysis was based upon 55 ± 6 saccades per cell. Of these 66 cells, 20% (13/66; 9P and 4M cells) showed significant suppression of activity prior to saccade start, based upon the trial-by-trial Poisson analysis (see Fig. 4a, b). The average magnitude of presaccadic suppression relative to baseline across all cells and trials was 53%. The modal time at which suppression started prior to the initiation of a saccade was 121 ms but the range was broad (from 109 to 267 ms prior to saccade start). The suppression extended beyond saccade end by an average of 53 ms. It is also noteworthy that the Poisson analysis had difficulty identifying significant suppression on trials where the firing rate was extremely low. For example, in the cell demonstrating the most robust modulations in firing rate relative to saccade endpoints, the Poisson analysis determined that only 65% (38/58) of the trials demonstrated a significant suppression in firing rate. A comparison of the baseline rate of activity between modulated trials and non-modulated trials revealed that on average, the baseline activity during trials where suppression was detected (27 ± 4 spikes/s) was more than twice that of trials where suppression was not detected (12 ± 3 spikes/s), pointing to a potential floor effect in cells failing to demonstrate significant suppression. However, when we used a *t*-test ($p < 0.01$) to compare the mean baseline activity for cells that did and did not demonstrate presaccadic suppression (27 ± 4 and 15 ± 9 spikes/s, respectively), although a trend was observed, the difference was not significant, suggesting that the modulation cannot be explained fully by a floor effect.

We also examined for modulation during each saccade and found that the same 13 cells that exhibited presaccadic suppression also exhibited suppression dur-

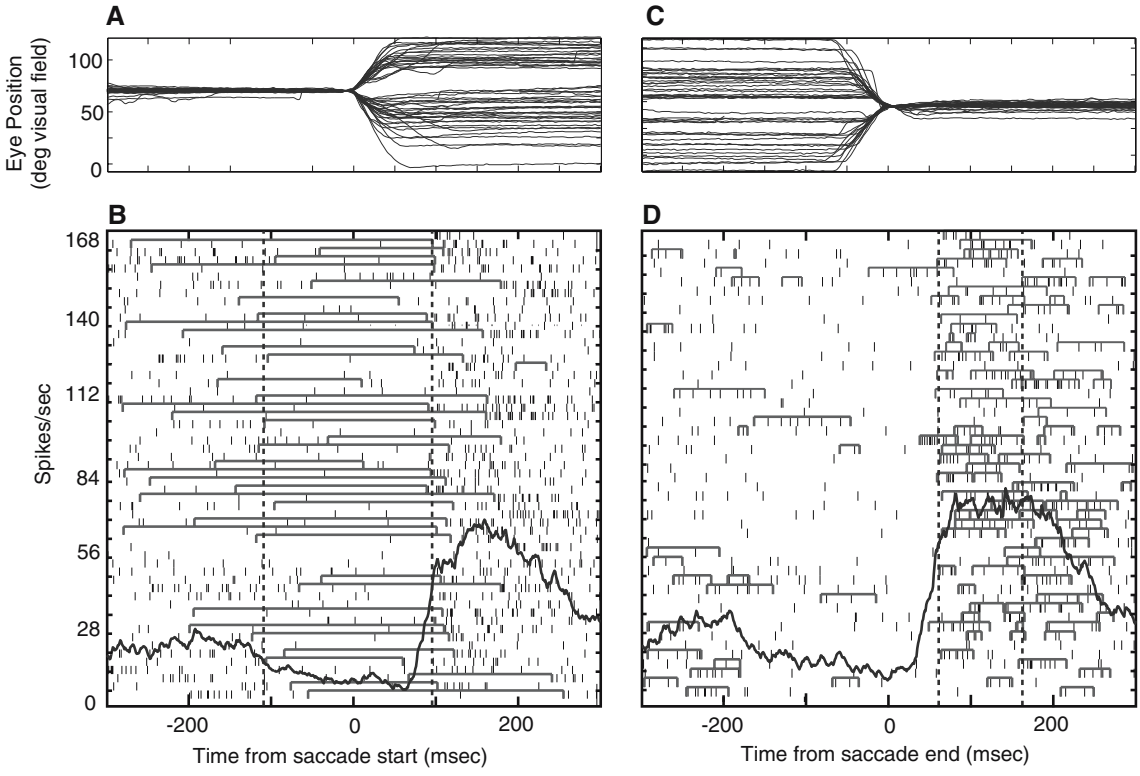

Fig. 4 Effect of spontaneous saccades on LGN P cell activity. **a** Change in eye position as a function of time while the monkey made 58 spontaneous saccades in a completely darkened room. Saccades are aligned on saccade start. **b** PSTH for an LGN cell recorded while the monkey produced the spontaneous saccades displayed in **(a)**. The small tick marks represent spikes. The horizontal brackets represent significant increases in interspike intervals as determined by the Poisson analysis. The vertical dashed lines represent the modal onset (−109 ms) and offset (99 ms) of the periods of significant modulation relative to saccade start. **c** Saccades shown in **(a)** aligned to saccade end. **d** The horizontal brackets represent significant decreases in interspike interval as determined by the Poisson analysis. The modal onset and offset of significant modulation was 65 and 169 ms, respectively

ing the saccade. This suppression was basically an extension of the presaccadic suppression as can be seen in Fig. 4a, b, and lasted ~53 ms beyond the end of the saccade (range 37–68 ms). On average, 36% of trials showed saccadic suppression, and the percentage change relative to baseline during the saccade was similar to that seen during the presaccade epoch, 58%. No cell demonstrated a consistent facilitation of activity during the saccade relative to baseline.

Finally, we examined for postsaccadic modulation (see Fig. 4c, d). This postsaccadic enhancement of activity occurred in a greater percentage 27% (18/66) of cells than the suppression of activity that occurred before and during the saccade but involved the same subset of cells with the exception that five cells showed only postsaccadic enhancement of activity according to the Poisson analysis. Figure 5 is a perisaccadic time histogram of an LGN P cell that demonstrated only postsaccadic facilitation. This enhancement of activity averaged 208% of baseline and started an average of 95 ms after saccade end and ended ~70 ms later. The postsaccadic enhancement was also evident in a larger subset of trials (72%) than was the case for suppression events.

Spontaneous saccades: epoch analysis

A larger percentage of cells demonstrated significant saccade-related modulation when epochs were compared by ANOVA and post-hoc *t* tests against baseline. Thirty-eight percent (25/66; $F = 24.50$, $p < 0.01$; 5M, 19P,

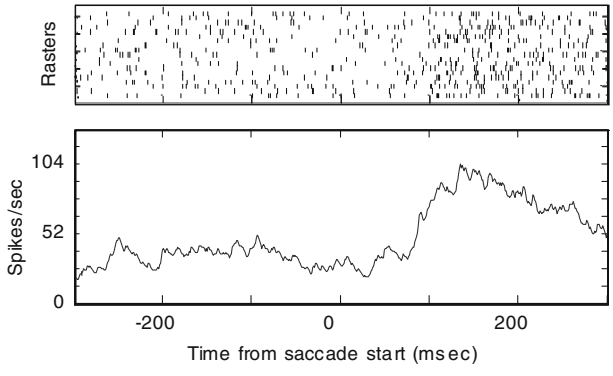

Fig. 5 Activity of an LGN P cell that demonstrated postsaccadic facilitation in the absence of saccadic suppression. Top panel: raster plot of the LGN P cell's perisaccadic activity. Trials have been aligned to saccade start (0 ms). Bottom panel: convolved spike density function of the rasters displayed in the top panel

and 1K cell) showed saccade-related modulation. The same 13 cells revealed by the Poisson analysis to be modulated significantly by saccades were also identified by the epoch analysis. The epoch analysis also detected two cells that demonstrated only a postsaccadic enhancement of activity. It is likely that some cells revealed saccade-related modulation only in the epoch analysis due to an advantage of spike averaging done in the epoch analysis (see also Lee and Malpeli 1998). When all trials from all the cells were aligned to saccade onset or saccade offset (see Fig. 4), as shown for spontaneous saccades in the dark by Lee and Malpeli (1998), presaccadic suppression cannot be detected, however, postsaccadic enhancement of activity is obvious. Using operationally defined epochs, the magnitudes of suppression and enhancement, however, were lower than when epochs were defined by the Poisson analysis: 44% suppression during saccade and 144% enhancement postsaccade. The reason the magnitudes may have been higher using the Poisson analysis is that no epochs were included that did not show an effect by definition.

Visually guided saccades: Poisson analysis

We recorded from 75 LGN relay neurons while the monkeys made saccades to a target located outside of the RF. At least 20 saccades were included for each of these cells. Of these 75 cells, 15% (11/75, 9 P, 1 M, and 1 K, 2 ON-center, 9 OFF-center) showed significant suppression of activity prior to saccade start (see Fig. 6a, b) based upon both the trial-by-trial Poisson analysis and the epoch analysis that compared average firing rate. The same 11 cells were selected based upon both methods of analysis. The average magnitude of this presaccadic suppression relative to baseline across all cells and trials was 78%. The modal time at which suppression started prior to the initiation of a saccade was −87 ms but the range was broad (from −75 to −98 ms). The suppression often extended beyond saccade end by 80 ms. As in the spontaneous saccade condition, cells that showed a significant presaccadic suppression of activity did not show this suppression on every trial. On average, 88% of trials per cell showed a significant change in activity as measured by the Poisson analysis. We also compared trials on which suppression was detected with those in which no suppression was detected to determine if baseline activity was lower during the latter. On average, the baseline activity during trials, where suppression was detected, was significantly higher (34 ± 11 spikes/s compared to 15 ± 9 spikes/s, $p < 0.01$) than when no modulation was detected, suggesting that the difference could be explained as a floor effect. However, a comparison of the mean baseline activity of cells that demonstrated presaccadic suppression (31 ± 14 spikes/s) with those that did not (26 ± 15 spikes/s) found no statistical difference, suggesting that the differences between cells that are or are not modulated cannot be explained by a floor effect.

We also examined for saccadic modulation and found that the same 11 cells that exhibited presaccadic suppression also exhibited suppression during the saccade. As seen in the spontaneous saccade condition, this suppression was basically an extension of the presaccadic suppression (see Fig. 6a, b) and lasted ~25 ms beyond the end of the saccade (range 24–80 ms). The average

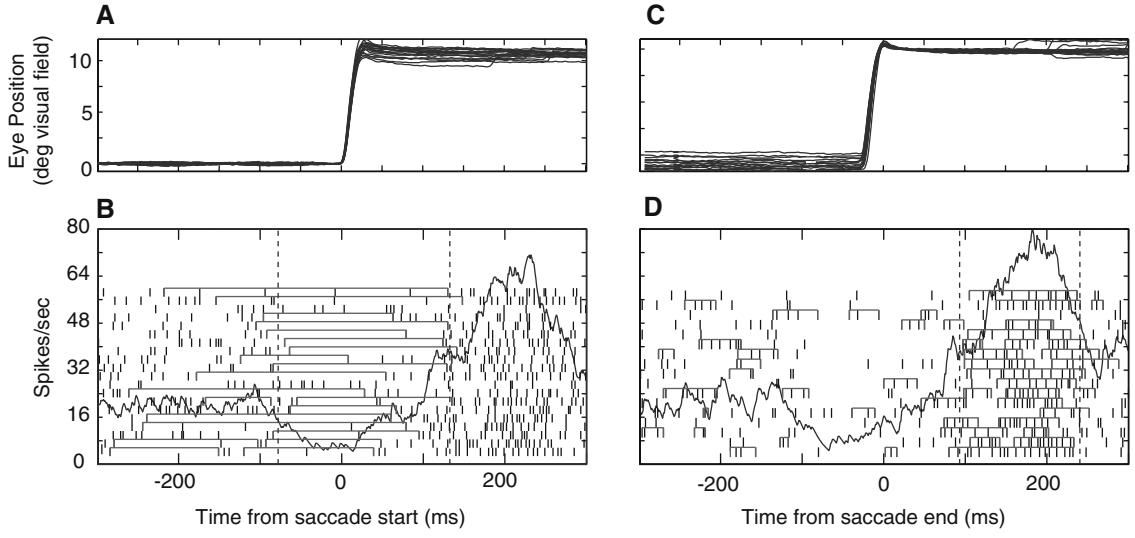

Fig. 6 Effect of visually guided saccades on LGN P cell activity. **a** Change in eye position as a function of time while the monkey made 20 visually guided saccades from a fixation spot to a target stimulus presented outside the LGN cell's RF. **b** Corresponding activity of LGN cell recorded while the monkey produced saccades illustrated in **(a)**. The horizontal brackets represent significant increases in interspike interval as determined by the Poisson analysis. The vertical dashed lines represent the modal onset (−81 ms) and offset (131 ms) of the periods of significant modulation relative to saccade start. **c** Saccades shown in **(a)** aligned to saccade offset. **d** Corresponding activity of cell recorded while monkey produced saccades illustrated in **(c)**. The horizontal brackets represent significant decreases in interspike interval as determined by the Poisson analysis. The modal onset and offset of significant modulation was 98 and 242 ms, respectively

percentage of trials that showed saccadic suppression (88%) was the same as for the presaccadic modulation but the magnitude of the suppression was less (62%).

Finally, we examined for postsaccadic modulation (see Fig. 6c, d). As in the spontaneous saccade condition, this postsaccadic enhancement of activity occurred in a greater percentage (23%, 17/75) of cells than the suppression of activity that occurred before and during the saccade but involved the same subset of cells with the exception that 6 cells showed only postsaccadic enhancement of activity according to the Poisson analysis. This enhancement of activity was substantial and averaged 225% of baseline and started an average of 99 ms after saccade end and ended ~178 ms later. The postsaccadic enhancement was also evident in a larger subset of trials (99%) than was the case for suppression events.

Visually guided saccades: epoch analysis

More cells [20% (15/75 cells; $F = 33.55$, $p < 0.01$; 3 M cells, 10 P cells, and 2 K cells)] exhibited significant suppression of activity during saccades using the epoch analysis compared to the Poisson analysis. This was likely the result of the Poisson's inability to detect significant reductions in firing rate in cells with already low baseline rates of firing. Additionally, significant increases in activity during saccades were detected in one LGN P cell.

As in the case of the Poisson analysis, the most prominent modulation coincident with saccades in the visually guided saccade task was a significant postsaccadic enhancement of activity, although a much larger percentage was identified using the epoch analysis than using the Poisson analysis [40% (30/75 cells; $F = 43.23$, $p < 0.01$; 26 P cells, 2 M cells, and 2 K cells)]. As found using the Poisson analysis, evidence of saccadic suppression was not necessary for a cell to demonstrate postsaccadic enhancement; 29% of all cells (22/75 cells; 18 P cells and 4 M cells) showed postsaccadic enhancement but not suppression.

The spontaneous activity of these 22 cells was significantly ($p = 0.05$) lower (mean = 8.7 ± 4 spikes/s) than 15 cells that showed both suppression and enhancement (mean = 24.6 ± 11 spikes/s) suggesting that absence of suppression was due to a floor effect, namely the spontaneous rate was too low and too variable to detect suppression. These 22 cells are nearly identical along other dimensions (cell classes, types, RF center polarities, etc.) to those cells that demonstrated both saccadic suppression and postsaccadic facilitation reinforcing the conclusion that they did not belong to a separate population.

Modulation by cell class and RF center polarity

Although members of every LGN cell class demonstrated significant perisaccadic modulations in activity, a higher percentage of LGN M than P or K cells demonstrated significant saccadic modulation. Figure 7 shows examples of perisaccadic time histograms of six different cells, two from each cell class, which demonstrated or failed to demonstrate significant saccadic suppression. Whereas 42% of the M cells demonstrated significant saccade-related modulation, only 28% and 25% of P and K LGN cells, respectively, demonstrated these effects. It should be noted, however, that the number of sampled M and K cells is much smaller than for P cells. Furthermore, because baseline rates of firing were low overall and the Poisson analysis had difficulty determining significant suppression in situations with a low firing rate, the numbers reported in this section are based on an analysis of epochs only (see "Methods"). For cells that demonstrated significant saccadic suppression, no significant differences were found in baseline rates of activity between cells that demonstrated ($N = 40$; mean = 27 ± 8) and failed to demonstrate ($N = 101$; mean = 27 ± 10) significant modulations of activity during saccades, arguing against a floor effect.

Forty-two percent (15/36) of OFF-center cells and 33% (10/30) of ON-center cells were suppressed significantly during spontaneous saccades. As with the analysis of modulation by cell class above, using an analysis of epochs revealed that reductions in firing rate during the initial 100 ms of suppression relative to the initial 100 ms of fixation ranged from 15% to 46%. On average, cells demonstrating significant saccadic suppression during spontaneous saccades showed a 28% decrease in activity. Figure 8 contains perisaccadic time histograms aligned to saccade start and end showing the average response of all ON-center and OFF-center cells that demonstrated significant saccadic suppression during spontaneous saccades.

Seven percent (3/41) of ON-center cells and 35% (12/34) of OFF-center cells were suppressed significantly during visually guided saccades. Reductions in firing rate during the initial 100 ms of suppression relative to the initial 100 ms of fixation ranged from 51% to 100%. On average, cells demonstrating significant saccadic suppression during visually guided saccades demonstrated an 84% decrease in activity during saccades. Figure 9 contains summary perisaccadic time histograms aligned to saccade start and end for all ON-center and OFF-center cells demonstrating significant saccadic suppression during visually guided saccades.

Modulation in relationship to other parameters

Saccade metrics were also examined for a relationship to the modulations reported here. The degree of saccadic suppression or postsaccadic facilitation (the percent change from baseline) was evaluated for cells recorded during the visually guided saccade condition as this condition provided the tightest control over the mon-

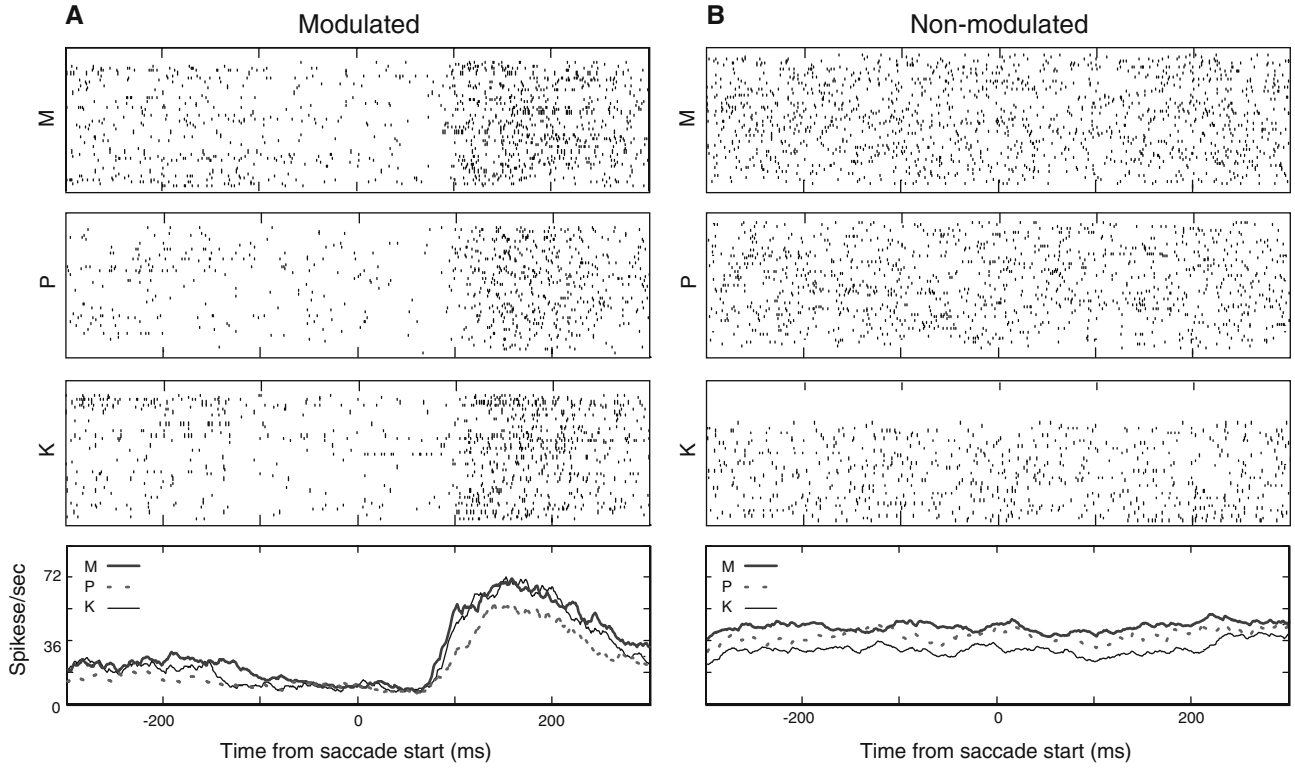

Fig. 7 Activity of LGN cells that demonstrate or fail to demonstrate perisaccadic modulation during spontaneous saccades. **a** Top panels: Raster plots of an LGN M (top raster), P (middle raster), and K (bottom raster) cell's perisaccadic activity demonstrating significant saccadic suppression and postsaccadic facilitation. Trials have been aligned to saccade start (0 ms). Bottom panel: Convolved spike density functions for the same M (bold trace), P (dotted trace), and K (thin trace) cells. Trials are aligned on saccade start (0 ms). **b** Top panel: raster plots of an LGN M (top raster), P (middle raster), and K (bottom raster) cell's perisaccadic activity demonstrating no significant saccadic suppression or postsaccadic facilitation. Trials have been aligned to saccade start (0 ms). Bottom panel: convolved spike density functions for these same M, P, and K cells. Trials are aligned on saccade start (0 ms)

keys' eye movements. Perisaccadic modulations were found to be weakly correlated to saccade amplitude (r = .16) and saccade latency (r = .13). No significant correlation was found to exist between the degree of perisaccadic modulation and saccade direction (r = .08) (Fig. 10). However, both saccade amplitude and saccade velocity impacted the time course of saccadic modulation. This is revealed by an inspection of the summary perisaccadic time histograms for the ON-center and OFF-center cells recorded during both tasks (Figs. 8 and 9). In the visually guided saccade condition, the monkeys made small amplitude saccades (mean = 8.8°). In the spontaneous saccade condition, however, the monkeys' eye movements were not constrained to the target area of the stimulus monitor so the monkeys tended to make much larger amplitude saccades (mean = 32.6°), and correspondingly, higher velocity saccades. The result is that longer saccades produced suppression that lasted longer.

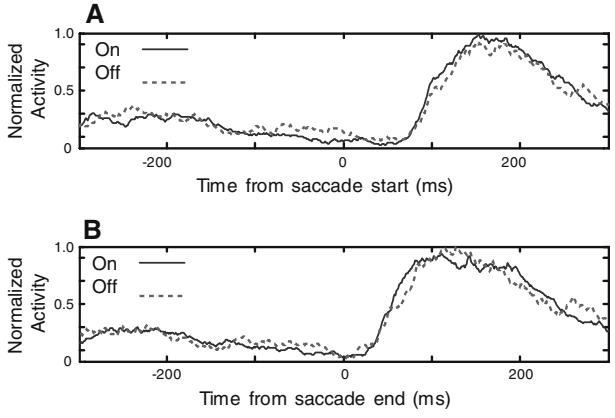

Fig. 8 Activity of LGN ON-center and OFF-center cells that demonstrate significant perisaccadic modulations during spontaneous saccades. **a** Convolved spike density functions for 25 LGN cells (10 ON-center, 15 OFF-center; 5M, 19P, 1K) that demonstrated significant saccadic suppression and postsaccadic facilitation during spontaneous saccades. Trials are aligned on saccade start (0 ms). **b** Convolved spike density functions for the same 25 LGN cells with trials aligned on saccade end (0 ms) to demonstrate the time course of the postsaccadic facilitation. Spike density functions were normalized to the highest peak-firing rate observed in the two curves

Discussion

On average, 25% of LGN cells of all classes demonstrated significant perisaccadic modulation. This modulation consisted of a suppression of activity that began

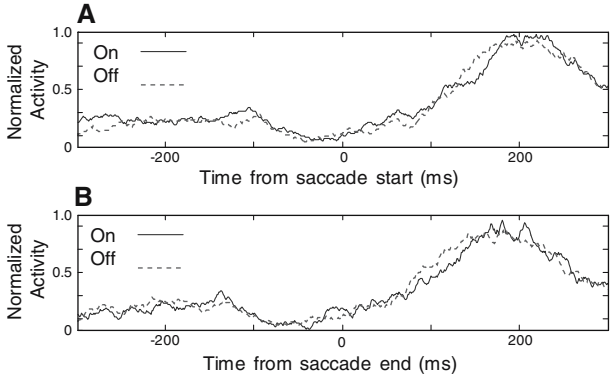

Fig. 9 Activity of LGN ON-center and OFF-center cells that demonstrate significant perisaccadic modulations during visually guided saccades. **a** Convolved spike density functions for 15 LGN cells (3 ON-center, 12 OFF-center; 3M, 9P, 3K) that demonstrated significant saccadic suppression and postsaccadic facilitation during visually guided saccades. Trials are aligned on saccade start (0 ms). **b** Convolved spike density functions for the same 15 LGN cells with trials aligned on saccade end (0 ms) to demonstrate the time course of the postsaccadic facilitation. Spike density functions were normalized to the highest peak-firing rate observed in the two curves

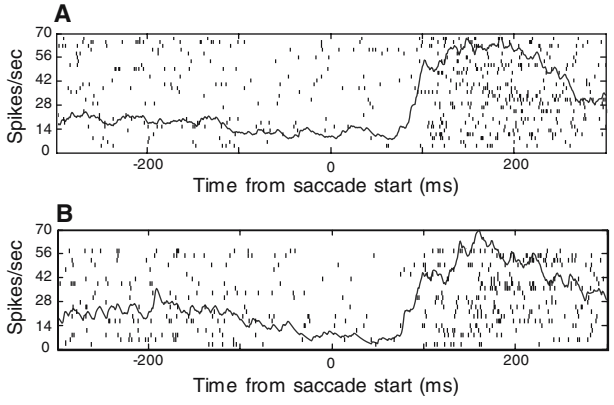

Fig. 10 The effect of saccade direction on LGN cell activity. Saccades illustrated in Fig. 4a were divided into two populations, those with trajectories between 0° and 90° and those with trajectories between 180° and 270°. **a** Corresponding cellular activity aligned to saccade start for saccades having trajectories between 0° and 90°. **b** Corresponding cellular activity aligned to saccade start for saccades having trajectories between 180° and 270°. Modulations (suppression followed by enhancement) present in the spike density function for the entire population of saccades are evident in both saccade subpopulations

well before the saccade, ended after its termination and was followed by a large enhancement of activity after fixation. Since the onset of the modulation preceded saccades by more than 100 ms, it is likely that this modulation reflects higher order motor-planning rather than a corollary of mechanisms in direct control of eye movements themselves. Moreover, the fact that the largest modulation is a postsaccadic enhancement of activity also supports the idea that perisaccadic modulations are designed more for facilitating transfer of information to cortex once the eyes have landed at their new location than for filtering unwanted visual stimuli. Below, we consider these findings in light of results published by others.

Saccadic suppression

Previous studies have made conflicting claims as to whether LGN cells are modulated by saccades and, if so, which classes of LGN cell are modulated and whether activity was enhanced or suppressed. Some studies have suggested that only primate M cells (or cat Y cells) are modulated significantly during saccadic eye movements (Burr et al. 1994; Ross et al. 1996; Ramcharan et al. 2001), while other studies have claimed that both primate M cells (or cat Y cells) and primate P cells (or cat X cells) are modulated significantly (Bartlett et al. 1976; Lee and Malpeli 1998). Most recently, Reppas et al. (2002) provided evidence that saccadic modulations are stimulus-independent for M cells and stimulus-dependent for P cells. All M cells in the latter study demonstrated saccadic suppression and postsaccadic enhancement, while P cells demonstrated only postsaccadic enhancement. No studies to date have investigated saccadic modulation in K cells. Data from the present study using two forms of analysis, however, show that a percentage of all three LGN relay cell classes (M, P and K) are modulated before, during, and after saccades in a stimulus-independent manner. The difference in results between Reppas et al. (2002) and the current study mainly concerns the existence of saccadic suppression in P cells, a difference that could be attributed to stimulus design. Where Reppas et al. (2002) trained monkeys to make visually guided saccades during full-field flash stimulation of various lengths, the current study had monkeys make visually guided saccades to targets presented *outside* the LGN cell's RF. Constant presentations of full-field flashes could artificially raise baseline levels while only driving individual P cells weakly since P cells have strong surrounds (Derrington and Lennie 1984; Irvin et al. 1993; White et al. 2001; Xu et al. 2001). Given that the degree of suppression we detected was always smaller than the degree of enhancement we saw following a saccade, it is possible that Reppas et al. (2002) were unable to detect suppression in P cells due to the combination of a high or variable baseline caused by variably presented bright flashes and a weak, complex response to the onset and offset of the stimulus that occurred at different times relative to saccade initiation. The reason they would have detected suppression in M cells under the same conditions could be explained by the relatively stronger responses M cells exhibit to full-field flashes given that M cells have weak surrounds (Derrington and Lennie 1984; Irvin et al. 1993; White et al. 2001; Xu et al. 2001). This same logic could be applied to other studies that used full-field flash stimuli and were unable to detect suppression but still saw postsaccadic enhancement such as the study in macaque monkeys by Ramcharan et al.

(2001). In cats, Lee and Malpeli (1998) did, however, see evidence for saccadic suppression using comparable stimulus conditions, but they may have overcome the signal to noise problem simply because they averaged across cells and across a large number of trials.

Presaccadic suppression

Psychophysically, a number of studies have provided perceptual evidence that suppression of visual information starts before saccade initiation (Riggs, et al. 1974; Zuber and Stark 1966). In cat LGN, Lee and Malpeli (1998) also showed that suppression of activity preceded saccade initiation. In fact, they showed that the peak of suppression measured in the dark occurred ~100 ms *before* saccade start. This value agrees well with our data in both the spontaneous and visually guided conditions where the modal times of suppression were −121 and −87 ms, respectively, relative to saccade start. In no case of using the Poisson analysis did we observe cells where suppression coincided with the onset of saccades, suggesting that suppression before and during saccades is driven by the same mechanism. The timing of the suppression argues against the theory that the mechanism responsible for the suppression represents a corollary to signals that are tied closely to each eye movement. Rather, the timing suggests that the signal could originate either cortically or subcortically and travel to the LGN via a network of pathways given the long time delay. Since the suppression starts so early relative to the eye movement, it is impossible to predict the exact pathways involved using our experimental design (see also below).

Postsaccadic enhancement

The most consistent modulatory effect seen in our sample of cells, in both conditions, was a postsaccadic enhancement. Under the condition where our monkeys made a saccade to a target the largest effect was a postsaccadic enhancement of activity that began an average of 97 ms after saccade end. These enhancements were observed in every cell class and, in many cases, the enhancement was more than 200% of baseline despite the fact that the RFs were never stimulated. This postsaccadic enhancement was four times as large as the suppression seen before and during saccades and occurred in more cells under both conditions tested than did suppression. These results mirror those in earlier reports (Buttner and Fuchs 1973; Malpeli and Lee 1988; Ramcharan et al. 2001) where full-field flashes were used to stimulate LGN RFs. In cats, Lee and Malpeli (1998) also found that this enhancement was independent of visual stimulation. They report that postsaccadic enhancement was the major change seen for all LGN cells recorded either in complete darkness or under direct stimulation conditions, which agrees with our results in monkeys. It seems clear that the increase in gain observed following saccadic eye movements (particularly when preceded by a depression) would be useful in "resetting" the visual system's frame of reference once visual targets had been captured.

Saccadic modulations and information transmission

If one were to design a visual system that would enhance information at each fixation and, at the same time, decrease the transfer ratio of signals during gaze shifts in order to minimize perceptual irregularities, one could do so using exactly the phenomena we have reported here. The three phases of saccadic modulation (presaccadic suppression, saccadic suppression, and postsaccadic enhancement) outlined above are entirely congruent with a system that is designed to modulate the geniculocortical transfer ratio. The timing of these events suggests that the saccade related suppression is a reflection of a decision to move the eyes (motor plan) and not part of the actual motor command underlying the gaze shift. The fact that the postsaccadic enhancement of activity occurs ~50 ms following saccade end makes sense given that retinal signals require an average of 20–40 ms to impact the firing of LGN cells (Ichida et al. 2003).

Circuits responsible for LGN saccadic modulation

A final issue that is important for understanding the role of saccade related modulation of LGN cells concerns its origin, namely, the potential sources of signals to the LGN responsible for these modulations. Although less information is available in primates than in cats, it is known that macaque monkey LGN receives major inputs from primary visual cortex (V1), the thalamic reticular nucleus and a variety of midbrain and pontine sources (Bickford et al. 2000; see also Casagrande et al. 2005 for review). Since major differences have not been found in basic laminar circuits in primates (Casagrande and Norton 1991; Bickford et al. 2000), one would expect each of these inputs to impact all LGN layers.

The most direct route for suppression in the LGN is via a GABAergic pathway. Three such pathways exist: (1) synaptic input from inhibitory LGN interneurons, (2) input from GABAergic thalamic reticular neurons, and (3) input from pretectal GABAergic neurons. Since all three of these pathways can receive input from any of the extraretinal sources listed above including several cortical sources, the question remains, which extraretinal sources with pathways to the LGN have been identified as carrying saccade related signals? Potential sources include: the superior colliculus, pretectum, pons, central lateral nucleus of the thalamus and primary visual cortex (Casagrande and Norton 1991; Bickford et al. 2000; Sherman and Guillery 2001; Casagrande et al. 2004 for review). Direct input from

the superior colliculus is unlikely since the colliculus sends its main input only to LGN K layers (Harting et al. 1991). An indirect route from the colliculus, however, has been identified in rabbits that could provide the appropriate input (Zhu and Lo 1996). This circuit involves a projection from the deep layers of the superior colliculus to the thalamic reticular nucleus via the central lateral nucleus. Furthermore, the overall time course of activation in the colliculus and suppression in the LGN fit with the time course of saccadic suppression beginning before saccade initiation and ending just prior to saccade end. There are many other pathways that could provide for such signals. Cholinergic input from the pons could also provide these signals but this input has generally been reported to have an excitatory effect on LGN cells (Fitzpatrick et al. 1989). If the timing proves correct, however, cholinergic input could account for postsaccadic enhancement of activity as could input from the visual cortex. Another possible source of saccade-related activity is the pretectogeniculate pathway (Schmidt 1996; Schmidt and Hoffman 1996). Pretectogeniculate cells are excited during saccades (Schmidt 1996). Schmidt and colleagues (Schmidt 1996; Schmidt and Hoffman 1996) have argued that pretectogeniculate cells inhibit LGN interneurons in cats thus facilitating excitation of LGN relay cells. The latter circuit would be entirely appropriate to explain a postsaccadic enhancement of activity given the timing reported. Others, however, have provided evidence in cats that pretectal activity suppresses LGN activity and thus contributes directly to saccadic suppression (Funk and Eysel 1995). Moreover, electron microscopic evidence suggests that the pretectum projects to both interneurons and relay cells in primates allowing for both suppression and enhancement (Feig and Harting 1994). The fact that cells were found that showed postsaccadic enhancement but no suppression during saccades suggests that suppression and enhancement may involve different circuits. It is more likely, however, that we were not able to detect weak suppression in these cells given their low and variable spontaneous level of activity. The fact that there were no other classification differences between cells that showed just enhancement and those that showed both suppression and enhancement reinforces this view. Nevertheless, we cannot rule out the possibility that two populations exist.

Clearly, more information is required before we can identify the particular pathway that provides the LGN with information about saccadic eye movements with confidence. Regardless, the current data and the anatomy of pathways likely to inform the primate LGN of the time course of planned and ongoing saccades support the view that these signals globally inform LGN cells in all layers about the intent to make a saccade, the saccade's time course, and through postsaccadic enhancement could facilitate the transfer of visual information during fixations.

Acknowledgments We are especially grateful to Mary Feurtado for her excellent assistance with animal care and anesthesia. We would also like to thank Jackie Munch and Kathryn Saclarides for help with experiments. Finally, we would like to thank Julia Mavity-Hudson, Yuliya Sokolova, Leanne Boucher, Veit Stuphorn, and Stephanie Shorter-Jacobi for their comments and suggestions on earlier drafts of this article. Supported by 1F31NS44691 (DWR) EY01778 (VAC), IBN-0234646 (VAC) EY08890 (JDS), and core grants EY08126 and HD15052.

References

Bartlett JR, Doty RW Sr, Lee BB, Sakakura H (1976) Influence of saccadic eye movements on geniculostriate excitability in normal monkeys. Exp Brain Res 25:487–509

Beeler GW Jr (1967) Visual threshold changes resulting from spontaneous saccadic eye movements. Vision Res 7(9):769–775

Bickford M, Ramcharan E, Godwin D, Erisir A, Gnadt J, Sherman S (2000) Neurotransmitters contained in the subcortical extraretinal inputs to the monkey lateral geniculate nucleus. J Comp Neurol 424:701–717

Burr DC, Morrone MC (1996) Temporal impulse response functions for luminance and colour during saccades. Vision Res 36:2069–2078

Burr D, Morrone M, Ross J (1994) Selective suppression of the magnocellular visual pathway during saccadic eye movements. Nature 371:511–513

Burr DC, Morgan MJ, Morrone MC (1999) Saccadic suppression precedes visual motion analysis. Curr Biol 9:1207–1209

Buttner U, Fuchs AF (1973) Influence of saccadic eye movements on unit activity in simian lateral geniculate and pregeniculate nuclei. J Neurophysiol 36:127–141

Campbell FW, Wurtz RH (1978) Saccadic omission: why we do not see a grey-out during a saccadic eye movement. Vision Res 18:1297–1303

Casagrande VA, Norton TT (1991) The lateral geniculate nucleus: a review of its physiology and function. In: *The Neural Basis of Visual Function*, Ed. A.G. Leventhal, *Vol. 4 of Vision and Visual Disfunction*, ed. J.R. Cronley Dillon, MacMillan, London, pp 41–84

Casagrande VA, Royal DW, Sáry GM (2005) Extraretinal inputs and feedback mechanisms to the lateral geniculate nucleus (LGN). John Wiley and Sons (in press)

Castet E, Masson GS (2000) Motion perception during saccadic eye movements. Nat Neurosci 3:177–183

Derrington AM, Lennie P (1984) Spatial and temporal contrast sensitivities of neurons in lateral geniculate nucleus of macaque. J Physiol 357:219–240

Diamond MR, Ross J, Morrone MC (2000) Extraretinal control of saccadic suppression. J Neurosci 20:3449–3455

Ditchburn RW (1955) Eye movements in relation to retinal action. Optica Acta 1:171–176

Feig S, Harting JK (1994) Ultrastructural studies of the primate lateral geniculate nucleus: morphology and spatial relationships of axon terminals arising from the retina, visual cortex (area 17), superior colliculus, parabigeminal nucleus, and pretectum of *Galago crassicaudatus*. J Comp Neurol 343:17–34

Fitzpatrick D, Diamond IT, Raczkowshi D (1989) Cholinergic and monoaminergic innervation of the cat's thalamus: comparison of the lateral geniculate nucleus with other principal sensory nuclei. J Comp Neurol 288:647–675

Funk K, Eysel UT (1995) Pharmacological inactivation of pretectal nuclei reveals different modulatory effects on retino-geniculate transmission by X and Y cells. Vis Neurosci 12:21–33

Hanes D, Thompson K, Schall J (1995) Relationship of pressaccadic activity in frontal eye field and supplementary eye field to saccade initiation in macaque: Poisson spike train analysis. Exp Brain Res 103:85–96

Harting JK, van Lieshout DP, Hashikawa T, Weber JTl (1991) The parabigeminogeniculate projection: connectional studies in 8 mammals. J Comp Neurol 305:559

Hochberg Y (1988) A sharper Bonferroni procedure for multiple tests of significance. Biometrika 75(4):800–802
Ichida J, Royal D, Sary Gy, Xu X, Shostak Y, Schall J, Casagrande V (2001) Evidence for suppression of activity in both parvocellular (P) and magnocellular (M) lateral geniculate nucleus (LGN) cells during saccadic eye movements. Abstr SFN 2001
Ichida JM, Royal D, Sary Gy, Schall J, Casagrande V (2003) Are there significant onset latency differences between LGN cells that carry S cone signals compared to those that carry M or L cone signals? Abstr SFN 2003
Irvin GE, Casagrande VA, Norton TT (1993) Center/surround relationships of magnocellular, parvocellular, and koniocellular relay cells in primate lateral geniculate nucleus. Vis Neurosci 10(2):363–373
Jeannerod M, Putkonen PTS (1971) Lateral geniculate unit activity and eye movements: Saccade-locked changes in dark and in light. Exp Brain Res 13: 533–546
Johnson K, Balakrishnan (1994) Continuous univariate distributions, vols I and II, 2nd edn. John Wiley and Sons
Judge SJ, Richmond BJ, Chu FC (1980) Implantation of magnetic search coils for measurement of eye position: an improved method. Vision Res 20:535–538
Khayat PS, Spekreijse H, Roelfsema R (2004) Correlates of transsaccadic integration in primary visual cortex of the monkey. proceedings of the National Academy of Sciences 101:12712–12717
Kim H, Connors B (1993) Apical dendrites of the neocortex: correlation between sodium- and calcium-dependent spiking and pyramidal cell morphology. J Neurosci 13:5301–5311
Latour PL (1962) Visual threshold during eye movements. Vision Res 2:261–262
Lee D, Malpeli JG (1998) Effects of saccades on the activity of neurons in the cat lateral geniculate nucleus. J Neurophysiol 79:922–936
Legendy CR, Salcman M (1985) Bursts and recurrences of bursts in the spike trains of spontaneously active striate cortex neurons. J Neurophysiol 53(4):926–939
Martin PR, White AJR, Goodchild AK, Wilder HD, Sefton AE (1997) Evidence that blue on cells are part of the third geniculocortical pathway in primates. Eur J Neurosci 9:1536–1541
Mason A, Nicoll A, Stratford K (1991) Synaptic transmission between individual pyramidal neurons of the rat visual cortex in vitro. J Neurosci 11:72–84
McKinney B, Lewis E, Wills V, Richards JE (2002) Developmental changes in the main sequence using interesting visual stimuli. International Society for Infancy Studies, Toronto, CA
Mitrani L, Mateeff S, Yakimoff N (1970) Smearing of the retinal image during voluntary saccadic eye movements. Vision Res 10(5):405–409
Montero VM, Robles L (1971) Saccadic modulation of cell discharges in the lateral geniculate nucleus. Vis Res Suppl 3:253–268
Paxinos G, Huang X, Toga AW (2000) The rhesus monkey brain in stereotaxic coordinates. Academic Press
Ramcharan EJ, Gnadt JW, Sherman SM (2001) The effects of saccadic eye movements on the activity of geniculate relay neurons in the monkey. Vis Neurosci 18:253–258
Reppas JB, Usrey WM, Reid RC (2002) Saccadic eye movements modulate visual responses in the lateral geniculate nucleus. Neuron 35:961
Riggs LA, Merton PA, Morton HB (1974) Suppression of visual phosphenes during saccadic eye movements. Vision Res 14:997–1011
Ross J, Burr D, Morrone C (1996) Suppression of the magnocellular pathway during saccades. Behav Brain Res 80:1–8
Sayer RJ, Friedlander MJ, Redman SJ (1990) The time course and amplitude of EPSPs evoked at synapses between pairs of CA3/CA1 neurons in hippocampal slice. J Neurosci 10:826–836
Schmidt M (1996) Neurons in the cat pretectum that project to the dorsal lateral geniculate nucleus are activated during saccades. J Physiol 76:2907–2918
Schmidt M, Hoffman K-P (1966) Physiological characterization of pretectal neurons projecting to the lateral nucleus in the cat. Eur J Neurosci 4:318–326
Schmolesky MT, Wang Y, Hanes DP, Thompson KG, Leutgeb S, Schall JD, Leventhal AG (1998) Signal timing across the macaque visual system. J Neurophysiol 79:3272–3278
Sherman SM, Koch C (1986) The control of retinogeniculate transmission in the mammalian lateral geniculate nucleus. Exp Brain Res 63:1–20
Shioiri S, Cavanagh P (1989) Saccadic suppression of low-level motion. Vision Res 29:915–928
Starr A, Angel R, Yeates H (1969) Visual suppression during smooth following and saccadic eye movements. Vision Res 9(1):195–197
Thilo KV, Santoro L, Walsh V, Blakemore C (2004) The site of saccadic suppression. Nat Neurosci 7:13–14
Volkmann FC (1962) Vision during voluntary saccadic eye movements. J Opt Soc Am 52:571–578
Volkmann F (1986) Human visual suppression. Vision Res 26(9):1401–1416
White AJ, Solomon SG, Martin PR (2001) Spatial properties of koniocellular cells in the lateral geniculate nucleus of the marmoset *Callithrix jacchus*. J Physiol 533:519–535
Wiesel TN, Hubel DH (1966) Spatial and chromatic interactions in the lateral geniculate body of the rhesus monkey. J Neurophysiol 29:1115–1116
Xu X, Ichida JM, Allison JD, Boyd JD, Bonds AB, Casagrande VA (2001) A comparison of koniocellular, magnocellular and parvocellular receptive field properties in the lateral geniculate nucleus of the owl monkey (*Aotus trivirgatus*). J Physiol 531:203–218
Xu XM, Bonds AB, Casagrande VA (2002) Modeling receptive-field structure of koniocellular, magnocellular, and parvocellular LGN cells in the owl monkey (*Aotus trivirgatus*). Vis Neurosci 19:703–711
Zhu JJ, Lo FS (1996) Time course of inhibition induced by a putative saccadic suppression circuit in the dorsal lateral geniculate nucleus of the rabbit. Brain Res Bull 41:281–291
Zuber BL, Stark L (1966) Saccadic suppression: elevation of visual threshold associated with saccadic eye movements. Exp Neurol 16:65–79
Zuber BL, Stark L, Lorber M (1966) Saccadic suppression of the pupillary light reflex. Exp Neurol 14:351–370