

Enhanced Motion Sensitivity Follows Saccadic Suppression in the Superior Temporal Sulcus of the Macaque Cortex

M.R. Ibbotson^{1,2}, N.S.C. Price^{1,4}, N.A. Crowder¹, S. Ono² and M.J. Mustari^{2,3}

¹Visual Sciences, Research School of Biological Sciences, Australian National University, Canberra, ACT 2601, Australia, ²Visual Sciences, Yerkes National Primate Research Center, Atlanta, GA 30322, USA and ³Department of Neurology, Emory University, Atlanta, GA 30322, USA

⁴Current address: Department of Neurobiology, Harvard Medical School, Boston, MA 02115, USA

The responses of neurons in the middle temporal and medial superior temporal areas of macaque cortex are suppressed during saccades compared with saccade-like stimulus movements. We utilized the short-latency ocular following paradigm to show that this saccadic suppression is followed by postsaccadic enhancement of motion responses. The level of enhancement decays with a time constant of 100 ms from saccade end. The speed of ocular following is also enhanced after saccades and decays over a similar time course, suggesting a link between the neural and behavioral effects. There is some evidence that maximum postsaccadic enhancement occurs when cells are stimulated at their optimum speeds. Latencies of motion responses are saccade dependent: 37 ms for saccade-generated motion, 45 ms for motion in the half-second after saccades, and 70 ms with no prior saccades. The finding that saccades alter response latencies may partially explain perceptual time compression during saccades and time dilation after saccades.

Keywords: chronostasis, *Macaca mulatta*, ocular following, saccades, saccadic suppression

Introduction

Primates change their direction of gaze using rapid eye movements called saccades. The perception of visual stimuli, including motion, is attenuated during saccades. This is referred to as saccadic suppression (e.g., Ilg and Hoffmann 1993; Castet and Masson 2000; Park and others 2001; for review, see Ross and others 1996, 2001). Perception of time is compressed during saccades (Morrone and others 2005) and expanded after them (Yarrow and others 2001). Despite these perceptual observations, little is known about the neural basis of these effects, particularly those in the wake of saccades. This postsaccadic period is critical because the visual scene can change dramatically during saccades, particularly if the eyes are directed to moving targets. A clue that modified processing occurs in the visual system after saccades is that the speeds of smooth tracking eye movements are transiently enhanced and they have shorter than normal latencies (Kawano and Miles 1986; Busetini and others 1996). In macaque monkeys and humans, the movement of large-field textured patterns after saccades generates reflexive, short-latency ocular following (Kawano and Miles 1986; Gellman and others 1990; Masson and Castet 2002). The initial eye speeds can be 3–4 times larger after saccades compared with the nonsaccade case. The enhancement is partially reduced without a textured stimulus being present during the saccade, suggesting at least some visual origin to the effect (Kawano and Miles 1986). Busetini and others (1996, 1997) showed that the initial acceleration of vergence eye movements is enhanced if brief horizontal disparity steps or

radial optic flow patterns are presented soon after saccades, with this effect also having some visual origin.

What neural mechanisms lead to the perceptual and behavioral modifications outlined above, and what are the implications for perception during and after saccades? Neural activity in the lateral geniculate nucleus (LGN) of cats (Lee and Malpeli 1998) and macaques (Ramcharan and others 2001; Reppas and others 2002) is enhanced after saccades. In cortical motion-coding areas within the superior temporal sulcus, such as the middle temporal (MT) and medial superior temporal (MST) areas of macaque, responses to saccade-generated visual motion are suppressed compared with the same motion presented during fixation (Thiele and others 2002). Two other studies, both reported in abstract form, show that MT/MST activity was suppressed before and during saccades but enhanced afterward (Bremmer and others 2002; Takemura and Kawano 2004).

Here we test cells in MT/MST for saccadic suppression by comparing responses to saccades with responses to saccade-like displacements of the stimulus pattern during fixation. To assess the motion sensitivity of the system after saccades, we measured responses of the same cells, and the ocular following behavior that depends on that motion analysis (Kawano 1999), using the short-latency ocular following paradigm developed by Miles and others (Kawano and Miles 1986; Miles and Kawano 1986; Miles and others 1986). This task requires monkeys to make saccades to a central target, after which a textured background is moved to initiate ocular following. While monkeys performed this task, we recorded from MT and MST, matching the direction of postsaccadic background motion to the cell's preferred direction. By adjusting the duration between saccade end and the onset of background motion, we were able to compare motion responses with a range of postsaccadic delays.

Materials and Methods

Data were collected from 2 juvenile rhesus monkeys (*Macaca mulatta*). All surgical and experimental procedures were performed in strict compliance with the National Institutes of Health guidelines and protocols approved by the Institutional Animal Care and Use Committee at Emory University. Sterile surgical procedures were carried out under aseptic conditions using isoflurane anesthesia (1.25–2.0%) to stereotactically implant a magnetic resonance imaging (MRI)-compatible head stabilization system (Crist Instruments, MD) and a recording chamber centered above the superior temporal sulcus (lateral 15 mm, posterior 5 mm). A scleral search coil for measuring eye movements was implanted underneath the conjunctiva of one or both eyes. Our recording tracks vertically penetrated the anterior bank of the superior temporal sulcus and entered area MST, then MT after crossing the lumen of the sulcus.

Neurons were assigned to area MT based on their preference for moving stimuli, high direction selectivity, sensitivity to motion only in the contralateral hemifield, and the consistency of receptive field

positions with known retinotopic maps. Prior to arrival in MT, electrode tracks passed through MST where cells were also highly direction selective but had larger receptive fields at a given eccentricity, which often included the ipsilateral hemifield. On most tracks, there was a distinct gap of up to 200 μm between MST and MT, in which it was not possible to isolate cells. Physiological cell types were correlated with their cortical position to develop a depth profile for the MT/MST border. Recording locations were confirmed using MRI (as outlined below).

Visual Stimuli

Monkeys were comfortably seated with the head stabilized in the horizontal stereotaxic plane. They were rewarded with fruit juice every 0.5–1 s for maintaining fixation on a red spot presented on the screen. Visual stimuli were rear projected onto a tangent screen placed 61 cm from the eyes, covering a maximum visual angle of $77^\circ \times 77^\circ$. Stimuli were projected using a Mirage 2000 Digital Light Projector (DLP; Christie Digital, CA) with resolution 1024×1024 pixels, frame rate 96 Hz, and mean luminance 170 cd/m^2 (for issues relating to the DLP, see Price and others 2005).

Stimuli were random texture patterns formed of 0.8° black or white squares, with contrasts of 100% (Fig. 1). Four stimulus protocols were used. 1) We determined the preferred direction and speed of a cell using moving texture patterns while the animal fixated a central, stationary target. 2) We recorded the responses to 10° saccades in a cell's preferred and nonpreferred directions across a stationary texture pattern. 3) We reconstructed the mean eye positions during the saccades and used these to construct the mean speed profile of the retinal slip that occurred during the saccades. Monkeys were then required to fixate the central spot in the stimulus (point X in Fig. 1), while we moved the background stimulus with the same speed profile as during the saccades. 4) Finally, we used an ocular following paradigm. In this task, a stationary texture pattern was presented, and the animal fixated a target spot 10° from the center of the animal's visual field. This peripheral target spot disappeared and was simultaneously replaced by a central target spot, which the animal was required to saccade to and refixate. At an interval of 50–500 ms after the end of the refixation saccade, the central target disappeared, and the background texture simultaneously started to move in the cell's preferred direction at its preferred speed, evoking reflexive tracking eye movements.

Data Collection

Eye position was monitored in 2 dimensions using a magnetic coil system (CNC Electronics, Seattle, WA) and sampled at 1 kHz with 16-bit precision using a Power 1401 (CED, Cambridge, England, UK). Eye velocity $v(t)$ was calculated off-line by differentiation of the eye position traces $p(t)$ using a central difference algorithm: $v(t) = (p(t + \delta t) - p(t - \delta t)) / 2 \times \delta t$, with $\delta t = 2$ ms. Eye acceleration was calculated using the same algorithm, by differentiating the velocity traces.

Unit activity was sampled at 25 kHz using iron-tipped, epoxy-coated tungsten electrodes with impedance 1–4 M Ω prior to iron tipping (Frederick-Haer Corporation, Brunswick, ME). Single-unit action potentials were detected online with a hardware window discriminator or software template-matching algorithm (Alpha-Omega, Nazareth, Israel). In addition, we checked action potential shape and detection off-line using the Wavemark template matching provided in Spike2 (CED). For off-line analysis, neuronal responses were represented as spike density functions (SDFs) with 1 kHz resolution generated by initially convolving a delta function at each spike arrival time with a Gaussian window with $\sigma = 3$ ms. SDFs for stimulus-evoked responses were calculated by averaging responses to individual stimulus presentations, including only portions of the response during which the eye position was within 1° of the desired fixation point.

Data Analysis

Response latencies for stimulus-evoked responses were calculated relative to a synchronization pulse provided by the stimulus generation computer or relative to the time when the eye acceleration first exceeded $100^\circ/\text{s}^2$ for saccade-evoked responses. Each cell's spontaneous rate was averaged from at least 48 periods of 500 ms duration while the monkey fixated a stationary stimulus before motion onset. We calculated a response threshold based on the 99% cutoff from a Poisson

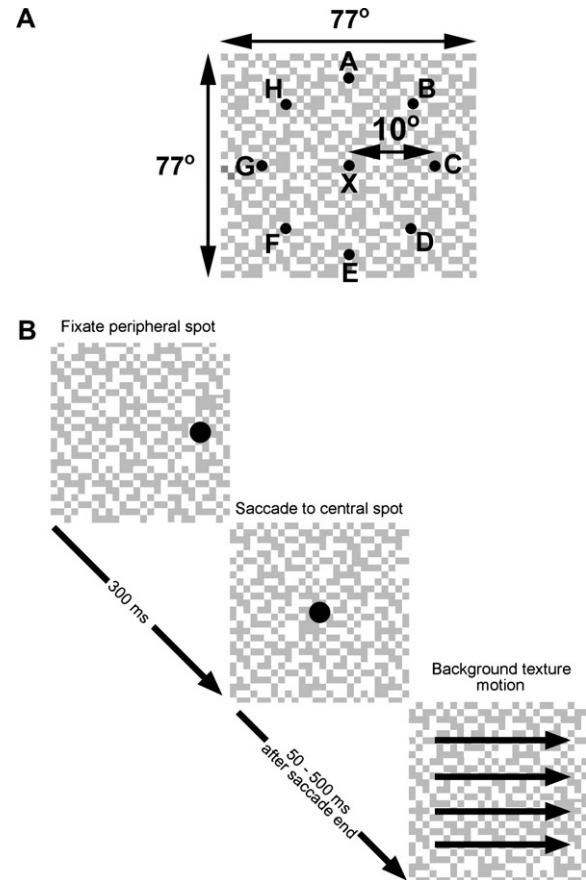


Figure 1. (A) Schematic drawing of the stimulus (not to scale). The texture was $77^\circ \times 77^\circ$ and consisted of random black and white squares with 0.8° sides. Points A–H mark fixation points 10° peripheral to a central point X. (B) For the ocular following paradigm, a saccade was made from a peripheral point to X, then the central fixation point disappeared, and the background texture moved in the cell's preferred direction (see Results for details). The contrast of the stimulus in the figure has been set artificially low for schematic reasons. The texture contrast was 100%, and the fixation marks were red spots.

distribution fitted to the spontaneous firing rate. Response latencies were then taken as the first time when the spiking rate in the SDF exceeded this threshold and stayed above the threshold for the subsequent 25 ms (Price and others 2005). We measured the responses to background generated retinal slip in a 50-ms window starting at the measured latency of the cell.

Saccade onset and the beginning of tracking eye movements during ocular following were defined as the time when the eye acceleration first exceeded $100^\circ/\text{s}^2$. This time was used to align the spiking responses to saccades. Mean responses to saccades were then calculated in the time window from the end of the latent period for a time that equaled the mean duration of the saccade. The initial ocular following speed was calculated from the mean eye speed in the 50 ms starting from the time at which the eye acceleration exceeded $100^\circ/\text{s}^2$.

MRI Reconstruction of Electrode Tracks

Locations of recording sites were confirmed using a Siemens 3-T magnetic resonance imager located at the Yerkes National Primate Research Center. Imaging sessions to acquire 3-dimensional T_1 -weighted images were performed under sedation (ketamine/telezol) and surgical levels of isoflurane (1.0–1.5%). Vital signs including blood pressure, heart rate, body temperature, expired CO_2 , and blood oxygenation were continuously monitored and maintained at physiological levels. Monkeys were held in an MRI-compatible stereotaxic frame (Crist Instruments) during imaging studies. We set the scan parameters to sample 1 mm slices through the entire anterior–posterior

extent of the brain including the recording chambers mounted over MT/MST. We used NeuroLens software to identify regions of interest below our “Cilux” recording chambers (Crist Instruments). We used an adjustable radius and angle positioning device that attached to the recording chamber to precisely place guide tubes and electrodes into MT/MST. This device includes a centering bushing that carries a saline-filled guide tube made of fused silica (Plastics One Inc, Roanoke, VA) for visualization during MRI sessions. The small internal diameter (0.15 mm) of the fused silica probe facilitates accurate localization.

Results

Postsaccadic Enhancement

Neural activity and eye movement behavior were recorded from 2 alert monkeys. After characterization of the neuron’s receptive field location, preferred direction, and preferred speed, the monkeys performed an ocular following task that involved fixating a small spot located 10° peripheral to the center of a large-field pattern (points A–H, Fig. 1A). After fixation for 300 ms (left panel Fig. 1B), this peripheral target disappeared and was simultaneously replaced by a central target, which the animal was required to saccade to and fixate (point X, Fig. 1A; middle panel Fig. 1B). At an interval of 50–500 ms after the refixation saccade, the central target disappeared, and the background texture simultaneously moved in the cell’s preferred direction (right panel Fig. 1B). Except where indicated, the saccades were opposite to the subsequent background motion direction. The eyes remain stationary for approximately 50 ms after background motion onset and then move in the stimulus direction with a characteristic initial jump in eye speed (Fig. 2A). The digital light projector used in the experiments provided steady output whenever the pattern was stationary. Consequently, during the saccades and fixation periods, the stimulus was equivalent to a pattern projected on a screen using steady illumination.

The Initial Eye Speeds during Ocular Following Are Larger for Shorter Postsaccadic Delays

When the postsaccadic delay before motion onset was 50 ms (Fig. 2A; thick line), there is a clear initial jump in eye speed followed by a slower but steady increase in eye speed as it catches up with the stimulus. When the postsaccadic delay was 500 ms (thin line), the initial jump in eye speed was significantly lower, thus delaying the overall increase in eye speed. The difference between the 2 eye speed profiles is highlighted in gray (Fig. 2A). Both monkeys used in this study showed clear enhancement of initial eye speed after saccades. In the period between the removal of the fixation target and the onset of ocular following, the eye almost always remains stationary. This stable eye period is the open-loop phase. Trials in which the eye drifted outside the fixation window during the first 50 ms after background motion started were not included in the analysis if it was clear that the eye movement was not associated with the background motion (e.g., saccades or eye drift in directions orthogonal to the background motion). The retinal slip during the open-loop phase is almost identical to the retinal slip induced by background movement during fixation. Once ocular following begins retinal slip speeds are reduced as the eye tracks the background motion (the closed-loop phase). Importantly, the stability of the eye position during the open-loop phase excludes any explanation of the enhancement effects based on eye movements during that phase.

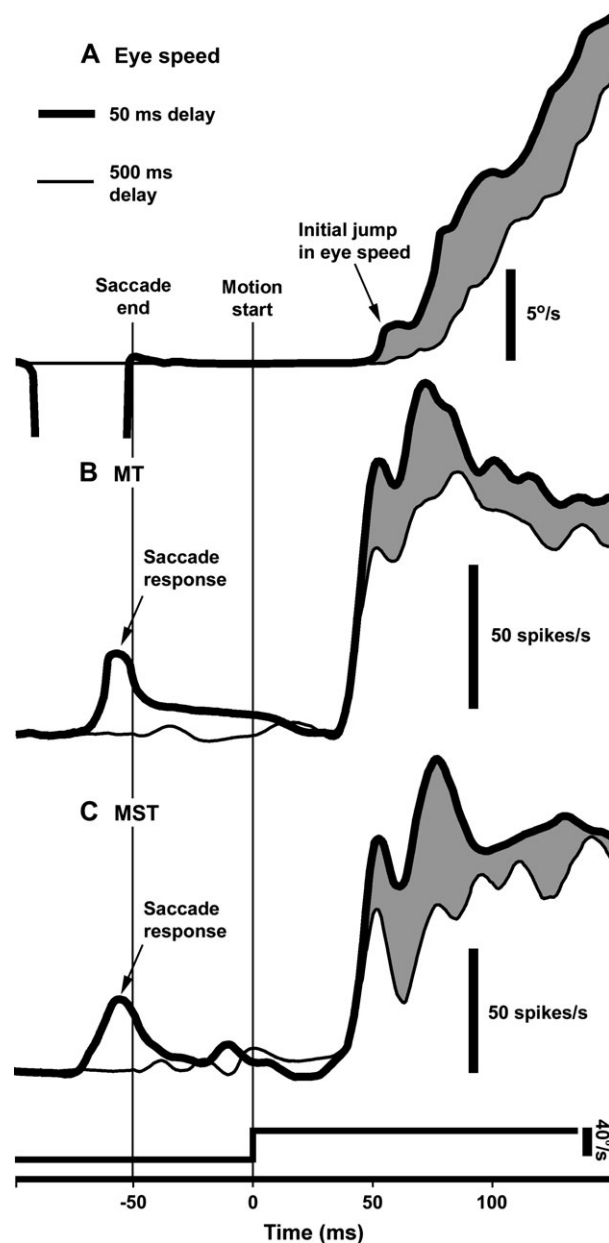


Figure 2. Eye speed and spike rate profiles. (A) Eye speed profiles generated by motion either 50 ms (thick line) or 500 ms (thin line) after saccade end (80 repeats). The gray area shows the difference between the 2 eye speed profiles. The postsaccadic background motion was $40^\circ/\text{s}$. Using the same line conventions, SDFs are shown for single MT (B) and MST (C) neurons stimulated 50 or 500 ms after a saccade.

The Spike Rates of Neurons Are Higher for Shorter Postsaccadic Delays

Fifty-four cells were tested with the ocular following paradigm (12 MT, 42 MST). The open-loop response is taken as the spike rate in the first 50 ms after the neuron’s latent period because firing during that phase of the response arises from visual motion that occurred during the open-loop period. Therefore, responses in that phase represent responses to background stimulus speeds. Figure 2B shows the SDFs for an MT neuron recorded while measuring the eye movements in Figure 2A. The background motion of $40^\circ/\text{s}$ was matched to the cell’s preferred direction and speed. With a 50-ms postsaccadic delay (thick

line), there is a rapid increase in firing rate 41 ms after background motion begins. However, when the delay between saccade and motion onset was 500 ms, the open-loop response is significantly lower (thin line, *t*-test, $P < 0.01$). The difference between the 2 spiking rates is highlighted in gray.

Figure 2C uses the same line conventions to show the responses of an MST neuron to background motion at 40°/s. This cell was optimally tuned to a speed of 20°/s. There is a significant enhancement in spiking rate when the saccade is 50 ms prior to the background motion (*t*-test, $P < 0.01$). The open-loop responses for both postsaccadic delay conditions consist of an initial transient burst of firing (40–55 ms), then a significant rebound effect where mean firing rates are transiently lower (around 60 ms). In the short postsaccadic delay condition, the firing rate is enhanced throughout the first 100 ms after the response begins. In both postsaccadic delay conditions, the response to background motion ramps up over time. This is because the ocular following starts to slow the retinal slip speed toward the optimum for the cell (20°/s), thus increasing the spike rate toward the end of the time window. Note that the enhancement generated by the prior saccade persists in the closed-loop period (shown up to 150 ms). It is clear that the neural enhancement cannot be due to the differences in ocular following speeds because the eyes do not start to move until 10 ms after the first neural responses, and the neural responses are delayed by the cell's latency.

The enhancement effect of a preceding saccade is shown for 54 cells by plotting the mean responses obtained from the 500-ms postsaccadic delay condition against those for the 50-ms delay (Fig. 3A). Forty-six cells (9 MT, 37 MST) have lower spike rates for the 500-ms delay, that is, they fall below the line of equality. Thirty-six cells (7 MT, 29 MST) had responses that were significantly lower (*t*-test, $P < 0.01$). Spiking latencies for the 500-ms delay condition were at least 2 ms longer for 13 cells, at least 2 ms shorter for 3 cells, and very similar for 38 cells (i.e., between ± 2 ms), showing that latency is not greatly affected by the 2 stimulus regimes (Fig. 3B).

For Postsaccadic Delays between 50 and 500 ms, There Was a Gradual Decline in Initial Response Magnitude

Figure 3C shows the responses of a representative MT and MST cell (dashed lines) and the mean response decline for all cells (solid line). Responses were normalized to the value measured for a 50-ms postsaccadic delay and fit with an exponential curve:

$$R = (R_{\max} - R_{\min})e^{-t/\tau} + R_{\min},$$

where R_{\max} is the peak normalized response, R_{\min} is the asymptotic normalized response, τ is the time constant of decay, and t is postsaccadic delay time. The time constants of decay for the individual cells in Figure 3C were both 100 ms (R^2 : 0.87 MT; 0.97 MST). The decay time constant for the mean response decline is 99 ms (Fig. 3C), and the goodness of fit was excellent ($R^2 = 0.95$). The mean latencies for all cells are also plotted as a function of postsaccadic delay (Fig. 3D). Latencies showed no significant change for any of the tested delays (analysis of variance [ANOVA], $P > 0.05$).

For comparison, we looked at the decay of the initial speed of ocular following. We used the mean eye speed in the first 50 ms from the time that eye acceleration exceeded 100°/s². When the postsaccadic delay was 50 ms the speed trace had a consistent initial jump in eye speed with a latency of 52–56 ms,

whereas at delays of 500 ms, the initial eye speed was smaller (e.g., Fig. 2A). Figure 3E shows the mean relationship between the initial eye speed and the postsaccadic delay for both our monkeys (solid and dashed lines) and the mean decay derived previously from 6 monkeys (Kawano and Miles 1986) (Fig. 3E, thick solid line). In all cases, the data points have been normalized to the value at a postsaccadic delay of 50 ms. Initial eye speed decays steeply in the first 100 ms after the saccade, then levels out at around 300 ms. Exponential functions fitted to the data (see above formula) have time constants of 74 ms (monkey QP), 58 ms (monkey HZ), and 60 ms (Kawano and Miles 1986). The R^2 values for the fits to QP and HZ are 0.99 and 0.98, respectively. Mean ocular following latencies were 50–55 ms for the 2 animals for postsaccadic delays ≤ 400 ms. For the 500-ms case, a sharp increase in ocular following latency to approximately 70 ms was observed in both animals (Fig. 3F).

Speed Tuning of Neural Enhancement

For comparison with the ocular following data, speed-tuning functions for all neurons were measured during fixation (speed range 4–192°/s). Responses were measured in the first 50 ms from the end of the latent period. Figure 4A shows the speed-tuning function of an MST neuron (optimum speed 64°/s). After obtaining the full speed-tuning function, we ran the ocular following tests with 50 and 500 ms postsaccadic delays at 3 background speeds (16, 64, and 128°/s). For all speeds, the response magnitudes with the 500-ms delay were not significantly different to those during fixation (gray dots, Fig. 4A). With a 50-ms delay to motion start, responses to 16 and 128°/s were also not significantly different to the fixation condition. However, when the postsaccadic test speed was 64°/s, the cell's response was significantly enhanced (Fig. 4A, *t*-test, $P < 0.01$). This cell shows the trend found in all cells tested with multiple speeds that the greatest enhancement occurred at the cell's optimum speed.

We tested 13 cells using the ocular following paradigm at multiple speeds. Speed-dependent enhancement across the population was calculated with the following formula:

$$E = \frac{R_{50} - R_{500}}{R_{50} + R_{500}},$$

where R_{50} is the response when the postsaccadic delay was 50 ms and R_{500} is the response when the postsaccadic delay was 500 ms. As each cell had a unique speed-tuning function, it was not possible to use standardized speeds, so E values are shown for stimuli at the cell's preferred speed and at least one octave below the preferred speed (Fig. 4B). At low speeds, the responses were slightly enhanced ($E = 0.025$), but for speeds at the optimum, the responses were greatly enhanced ($E = 0.114$). The amount of enhancement was significantly higher than the nonsaccade response only for optimum speeds (*t*-test, $P < 0.001$). Within the limits of this experiment, we provide evidence that neurons in MT/MST show postsaccadic enhancement at preferred speeds but not at speeds that are far below or above the preferred speed. However, a stimulus regime utilizing a greater range of speeds will be required to show the precise relationship between speed and postsaccadic enhancement.

Influence of Saccade Direction

Up to this point, we have only described the effects of saccades made opposite to the cell's preferred direction, thus generating retinal slip in the preferred direction. As is clear from Figure 2,

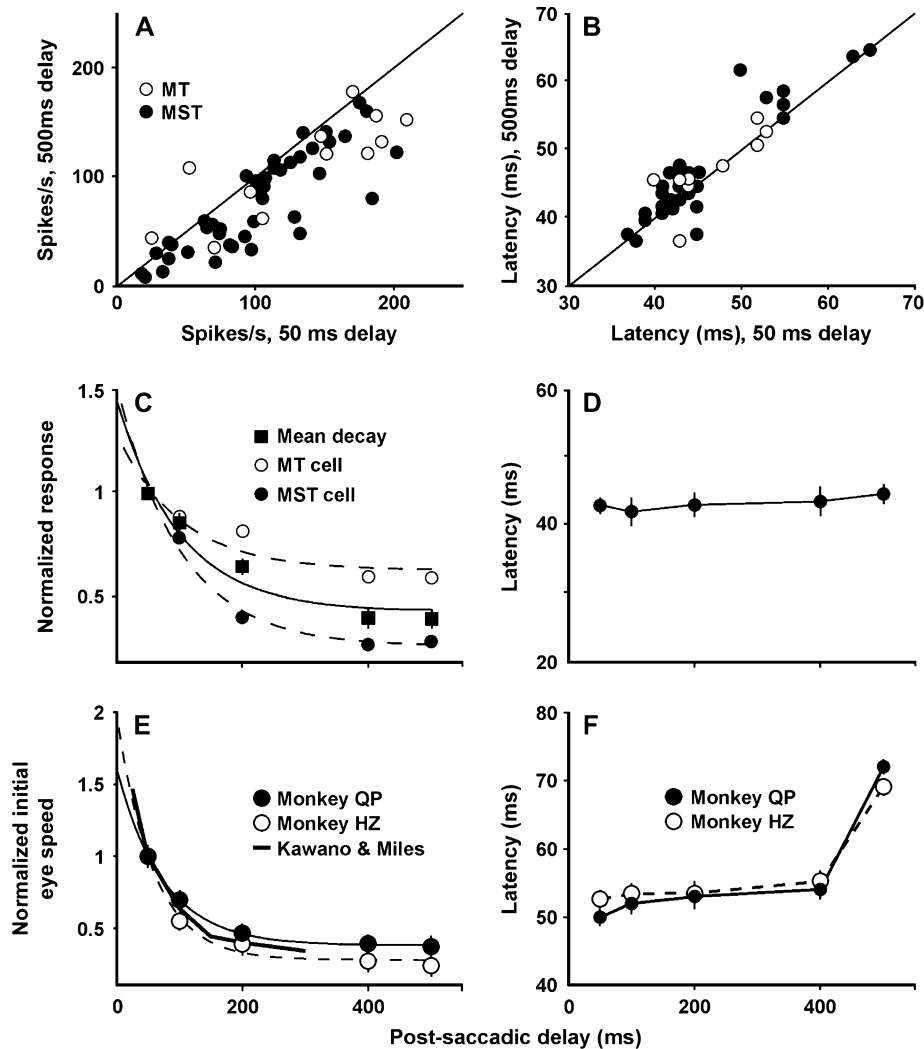


Figure 3. Responses to motion after saccades. The mean spike rates (A) and neuronal latencies (B) of 12 MT and 42 MST neurons generated by preferred direction motion starting 50 or 500 ms after saccades. (C) Decay functions for the mean response amplitudes of all tested cells (squares, solid line) and for 2 individual cells (circles, dashed lines). The lines are fitted exponential functions. Error bars are standard error (SE). Data for individual cells are based on 16 repetitions. (D) Response latencies of all tested cells plotted against postsaccadic delay. Error bars are SE. (E) Mean initial eye speeds for 2 monkeys plotted against postsaccadic delay. The data points have been fit with exponentials. The thick line shows data from Kawano and Miles (1986). (F) Latencies of ocular following plotted against postsaccadic delay for the 2 monkeys.

the neurons usually generated quite clear responses to the saccades, as has been shown previously in MT and MST (Thiele and others 2002; Price and others 2005). Now we look at a situation where the background motion still moved in the neuron's preferred direction, but the preceding saccade was not necessarily in the antipreferred direction. Figure 5A shows eye traces from monkey HZ for ocular following after saccades in both directions along the preferred motion axis (solid or dashed lines). The speed of ocular following to the subsequent background motion is virtually identical following saccades in either direction. Figure 5B shows the responses of a neuron during the same eye movement sequence. It is clear that the cell responds strongly to the postsaccadic background motion and that the amplitudes of those responses are not significantly different for the 2 saccade directions. The independence of saccade direction and the amplitudes of subsequent motion responses are shown for the population by plotting the size of the initial response to preferred direction motion 50 ms after saccades in the same (ordinate) and opposite (abscissa) directions (Fig. 6A). In no cells were the postsaccadic response

amplitudes significantly different for opposite directions of prior saccades (*t*-test, $P > 0.05$). We were able to test the influence on postsaccadic motion responses of saccades in 8 directions in 14 cells (see Fig. 1). In all cells, the response to the background motion was not significantly different following any saccade direction (ANOVA, $P > 0.05$), as shown for one MT neuron (Fig. 6B). All cells responded in a direction-selective manner to retinal slip induced by the saccades themselves. An example of the directional tuning to saccades in 8 directions is shown for the same MT cell (Fig. 6C). In conclusion, despite significant differences in firing rates evoked by different saccade directions, there were no significant differences in responses to subsequent background motion in the preferred direction.

Saccadic Suppression

In addition to the ocular following tests, the response magnitudes of all cells were measured during real saccades and saccade-like background motion. The experiments consisted of monkeys making 10° saccades between target spots positioned along the preferred motion axis for each cell. Monkeys made

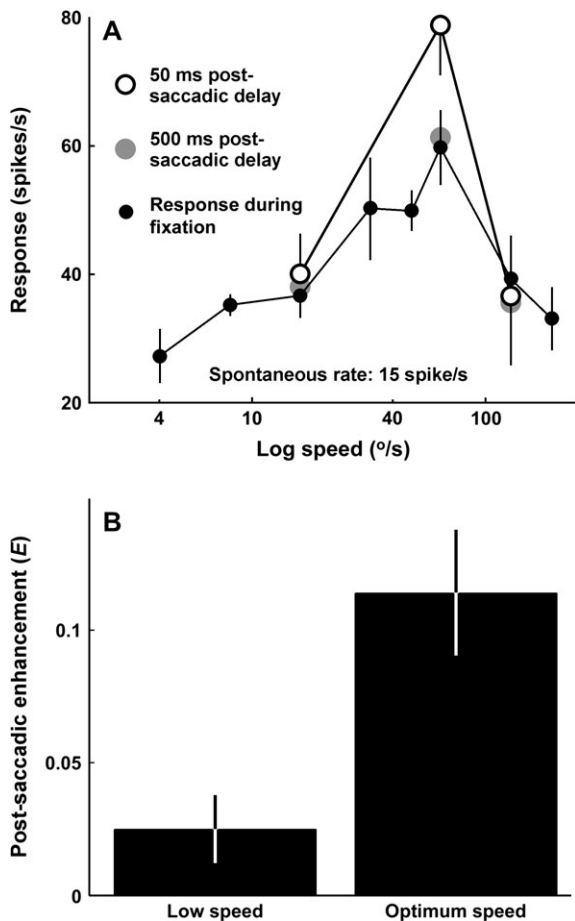


Figure 4. Speed dependence of enhancement. (A) Speed-tuning function of an MST neuron (small filled symbols, $n = 10$). The large gray symbols show responses to motion 500 ms after saccade end ($n = 16$). Open symbols show the responses of the same neuron to 3 test speeds presented 50 ms after saccades ($n = 16$). The responses are significantly enhanced only at optimum test speeds (t -test, $P < 0.01$). Error bars are standard error (SE). (B) Bar graphs of the postsaccadic enhancement (E , see Results) at low and optimal speeds. Error bars are SE ($n = 13$).

repeated saccades back and forth between the targets. Spike rates were then taken in the period from response onset for time periods equal to the durations of the saccades.

All eye movements were recorded, and the average saccade-related eye position profile was used to reconstruct a background image movement that replicated that during the saccades. During these saccade-like background movements, monkeys were required to fixate a central spot, and the background pattern was moved back and forth along the cell's preferred motion axis in a saccade-like manner with the same frequency as during the real saccades. Figure 6D plots the mean firing rates for saccades (ordinate) against responses to saccade-like background movements (abscissa). In all cases, only responses to preferred direction image motion are shown, and all spike rates have had the mean spontaneous activity subtracted. From the 54 cells, 45 (83%) show responses that are smaller for motion during real saccades (below the dashed, diagonal line of equality), confirming the existence of saccadic suppression in MT/MST (Thiele and others 2002).

Latencies of Neural Responses

The population data across the 54 cells reveal stimulus-dependent differences in latencies between the various types

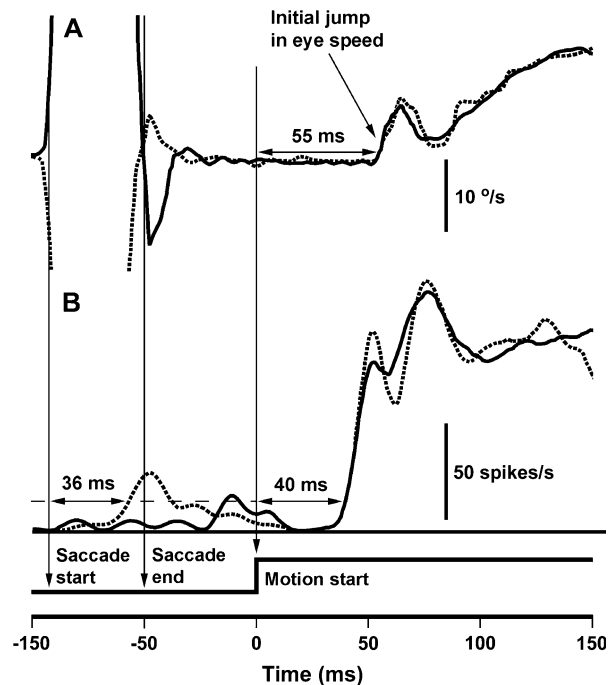


Figure 5. Eye speed and spiking response profiles. (A) Eye speeds showing leftward (dotted) and rightward (solid) saccades (saccades are truncated) and subsequent ocular following responses to rightward motion at 40%/s (80 repetitions). Thin vertical arrows indicate saccade onset, saccade end, and stimulus onset, respectively. (B) Spike density functions from an MST neuron during the eye movements shown above. The neuron responded optimally to rightward motion during fixation (not shown) and to leftward saccades (dotted line, latency 36 ms). Regardless of the saccade direction, the neuron responded with identical latency and similar amplitude. The horizontal dashed line represents significant spiking.

of stimulation. The results from MT and MST are combined because cells from both areas showed the same trends. Responses to preferred direction retinal slip during saccades usually had very short latencies (median 37 ms, Fig. 7A). For ocular following with a postsaccadic delay of 50 ms, the median latency was 45 ms (Fig. 7B). In the preliminary speed-tuning tests collected during fixation, the latencies were more variable as a population and have a median latency of 70 ms (Fig. 7C).

Discussion

Our results confirm the findings from 2 previous investigations that showed that responses of MT/MST neurons are suppressed during saccades (Bremmer and others 2002; Thiele and others 2002). The attenuation of motion sensitivity during saccades fits with the general concept of saccadic suppression (Ross and others 2001). It can be hypothesized that signals originating in the saccade-planning areas inhibit the activities of areas MT/MST, thus reducing the overall perception of motion during saccades. Neurons in both areas are known to have a role in motion perception (Newsome and Pare 1988; Celebrini and Newsome 1994, 1995).

One of the novel findings reported here is that saccadic suppression of MT/MST responses is followed by the enhancement of responses to background motion. This enhancement occurs over a similar time course to the postsaccadic enhancement of ocular following speeds (Kawano and Miles 1986). The physical speed at which the eyes move during saccades prevents observers monitoring changes in the visual scene.

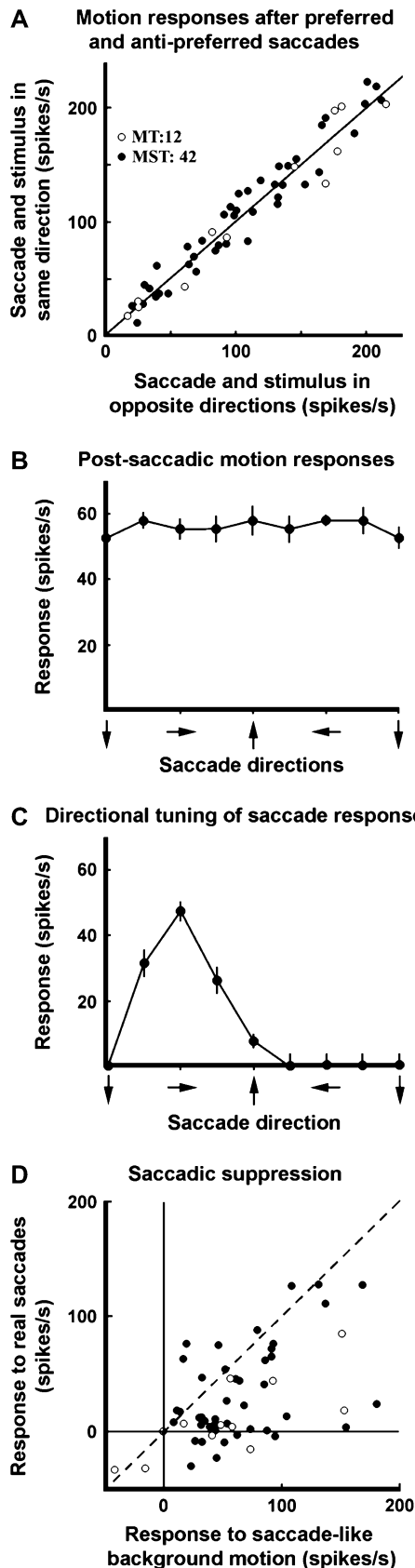


Figure 6. Directionality of saccade and postsaccade responses. (A) Responses to preferred direction background motion 50 ms after saccade end. The ordinate shows responses when the saccade and subsequent motion were in the same direction. The abscissa shows responses when the saccade and motion were in opposite directions.

Therefore, there is a need for a short-latency, high-gain motion detecting system after saccades to rapidly reassess the visual environment. For example, after making a saccade to a large moving object, such as a moving bus, the system must rapidly detect the direction and speed of the visual movement. This information is important perceptually for planning future actions such as avoiding the bus and for initiating ocular following so the eyes remain on target. Presumably, a mechanism that enhances the responsiveness of cortical neurons provides a transiently high-gain input to the visual and visuo-motor pathways that drive perceptions, action planning, and ocular following (Lisberger 1998). Our results have implications for motion perception after saccades. It is well established that visual sensitivity is reduced during saccades (Ross and others 2001), but based on our results it would be interesting to look for modulations of motion perception to large-field patterns soon after saccades.

Psychophysical observations demonstrate that compressions of time occur during saccades (Morrone and others 2005). If observers are asked to compare the time interval between consecutively presented flashed bars, the perceived time interval is 30–70 ms less than normal around saccade start. Conversely, increases in the time period for which objects are perceived to be present occur after saccades, that is, chronostasis (Yarrow and others 2001). A possible neural correlate for perceived perisaccadic time compression and postsaccadic chronostasis is found in the reduced response latencies that we report. Figure 7D shows that the same interstimulus interval would generate responses closer together in time before a saccade but more distant in time following a saccade. Thus, assuming that a downstream internal clock remains unaffected by saccades, our observations provide a possible mechanism to explain the perceptual time modulations that occur during and after saccades.

Neural Origins of Postsaccadic Enhancement

The neural activity that occurs during saccades does not influence the spike rates to subsequent motion, suggesting that enhancement is not driven by saccade-related spiking activity in the cells themselves. We conclude that the enhancement occurs afferently to MT but that its influence is dependent on the cell's speed tuning. There are 3 candidates for the neural origins of the enhancement: 1) efference copy of saccade-planning signals; 2) attentional mechanisms; and 3) rapid visual processing of saccade-induced motion.

First, the influence of signals from areas involved in saccade planning, such as the superior colliculus and frontal eye fields (e.g., Fuchs and others 1985; Scudder and others 2002), is suggested by the changes in response latencies of the MT/MST

The responses do not differ significantly in the 2 conditions, showing that saccade direction did not influence the amplitude of subsequent motion responses. The diagonal line is the line of equality. (B) Responses of an MT neuron to preferred direction background motion 50 ms after saccades in 8 different directions. The arrows below the graph indicate the directions of the saccades. Error bars are standard error (SE) ($n = 16$). (C) Responses to saccades in 8 directions for the same cell. It was tuned for leftward retinal slip (rightward saccades). The arrows below the graph indicate the direction of the saccades. Error bars are SE ($n = 16$). (D) Responses are smaller during saccades (ordinate) than to saccade-like background motion presented to the animal while it fixates (abscissa), as shown by the majority of points falling below the dashed line of equality. Spontaneous rate has been normalized to zero (solid vertical and horizontal lines). Symbols as in (A).

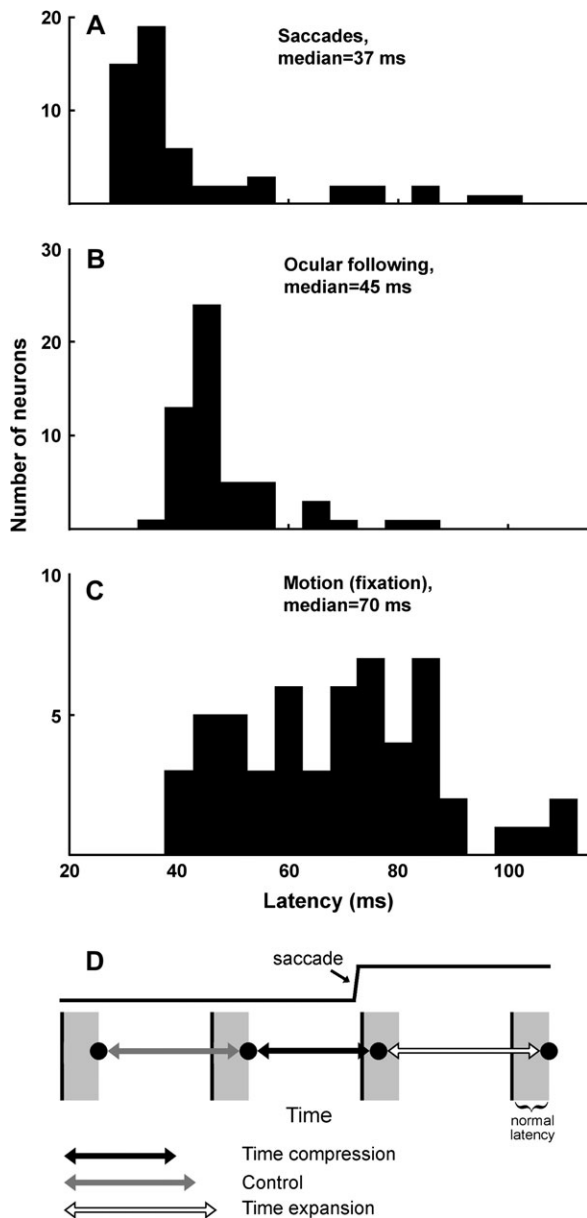


Figure 7. Histograms of cell number against latency ($n = 54$). (A) Latencies of responses to saccades. (B) Latencies generated by motion stimulation 50 ms after saccade end. (C) Latencies of the same neurons to their preferred speeds while the monkeys fixated stationary targets. (D) The decreased latency following saccades could explain perceptual time compression before saccades and time dilation following saccades. Black vertical lines indicate presentation times of a flashed stimulus with equal interstimulus intervals, and gray regions indicate the neuronal latency elicited by that stimulus during fixation. Dots represent observed latency. As summarized in (A–C), latency is shorter around the time of saccades. Time between neuronal responses during maintained fixation serves as the control measure (gray arrow). The decreased latency generated by the saccade reduces the time between responses if consecutively presented stimuli are flashed well before, then just after the saccade (black arrow). Conversely, the reduced response latency causes an increase in time between responses if the stimuli occur just after the saccade, then well after the saccade (empty arrow).

cells. Latencies are very short in response to saccade-generated visual motion, suggesting that a mechanism is adjusting cell sensitivity such that depolarizing membrane potentials reach spiking threshold very rapidly. Importantly, we have previously shown that saccades made in the dark do not directly modulate MT spiking activity, so the short-latency saccade responses do

not appear to be motor related (Price and others 2005). In a catch-up saccade task, which is quite different from the short-latency ocular following task used in this study, Lisberger (1998) showed that eye speeds during smooth pursuit were enhanced following saccades. Furthermore, prior saccade direction had no influence on the enhancement of the speeds of smooth pursuit eye movements. Lisberger suggests that the smooth pursuit system has a component with a nonretinal origin that controls the gain of neural transmission through the visuomotor pathways. We suggest that a similar component could partially control the gain of the ocular following system. Links between the neural control of smooth pursuit and ocular following have been noted (for review, see Ilg 1997), so a general gain control mechanism of nonvisual origin could underlie all visually driven slow eye movements.

Enhancing influences from nonretinal sources may impact directly on neurons in MT/MST or at earlier stages in the visual pathway. All cortical visual regions receive their input ultimately via the LGN or pulvinar (for review, see Maunsell and van Essen 1983; Felleman and Van Essen 1991; Merigan and Maunsell 1993; Born and Bradley 2005). The LGN is a complex nucleus containing feedforward and feedback neurons to the cortex and intrinsic modulatory interneurons (Sherman and Guillery 1996; Casagrande and others 2005). In macaques, Ramcharan and others (2001) showed that most magnocellular neurons in the LGN showed enhancement of activity during and after saccades. Reppas and others (2002) showed biphasic effects where weak suppression was followed by strong enhancement, and this pattern was the same in cat LGN (Lee and Malpeli 1998). In the cat, most of the effect observed arises from nonretinal sources, but the impact of visual stimuli could not be totally excluded. Thus, it is plausible that the postsaccadic effects reported here in MT/MST are inherited from the LGN.

The second possible source of nonretinal influences arises from attentional mechanisms. Response enhancement of neurons in MT has been demonstrated for visual stimuli when an animal is attending to the spatial location of a neuron's receptive field (Seidemann and Newsome 1999; Treue and Maunsell 1999) or to a preferred feature of the stimulus, such as direction (Treue and Martinez Trujillo 1999). Spatial attention would be directed to the saccade target prior to the saccade in our short-latency ocular following task (Madelain and others 2005). However, Treue and Martinez Trujillo (1999) demonstrated that spatial attention increased MT responses to stimuli in a multiplicative fashion, so it is unlikely that attention is responsible for the apparent "speed-specific" enhancement observed in MT/MST.

The third potential source for postsaccadic enhancement is from signals generated by the visual motion produced by saccades. Kawano and Miles (1986) showed that removing the textured stimulus during the saccade reduced the magnitude of postsaccadic enhancement of ocular following speeds and that saccade-like displacements of the visual scene without eye movements generated enhancement. Busetini and others (1996) found similar results for the acceleration of vergence eye movements after saccades.

We have already flagged the LGN as an area that could receive nonretinal signals that generate postsaccadic neural enhancement. Could there be visual factors producing postsaccadic enhancement of LGN activity? Sudden movements in the periphery well beyond the limits of centrally located LGN (and retinal) cell receptive fields are known to enhance stimuli

presented centrally (Kruger and others 1975; Kruger 1977). The large-field stimuli that we used could activate such mechanisms. Our results clearly show that any visual signal related to postsaccadic enhancement is not direction dependent. Large-field, nondirectional visual neurons optimally sensitive to saccade-like image speeds have been identified in the pretectum (Hoffmann and Distler 1989; Schmidt 1996; Price and Ibbotson 2001), with some proven to project to the LGN and hypothesized to account for reduced latencies associated with saccade-generated retinal slip (Fischer and others 1996, 1998). These inputs have a complex interaction with local LGN interneurons and relay cells to the cortex. Such neurons could provide a visually driven component that adds to the nonvisual gain control mechanism in the visuomotor system suggested by Lisberger (1998). Orban and others (1985) showed that neurons in LGN are tuned to visual speed. To our knowledge, nobody has investigated whether the enhancement of LGN activity after saccades is related to retinal slip speed, but this would be interesting as we found such a relationship in MT/MST.

Notes

We thank Dr Valabh E. Das and Tracey Broznya for assistance with data collection and invaluable discussion. This work was supported by the Australian Research Council Centre of Excellence in Vision Science (CE0561903), an ARC International Linkage Grant (IX0349251), and grants from the National Institutes of Health (EY06069; RR0165). *Conflict of Interest:* None declared.

Address correspondence to Michael R. Ibbotson, PhD, Visual Sciences, Research School of Biological Sciences, Australian National University, Canberra, ACT 2601, Australia. Email: Michael.Ibbotson@anu.edu.au.

Funding to pay the Open Access publication charges for this article was provided by the Australian Research Council Centre for Excellence in Visual Science.

References

- Born RT, Bradley DC. 2005. Structure and function of visual area MT. *Annu Rev Neurosci* 28:157-189.
- Bremmer F, Kubischik M, Hoffmann KP, Krekelberg B. 2002. Neural dynamics of saccadic suppression. *Soc Neurosci Abstr Program No. 57.2. Abstract Viewer/Itinerary planner*. Washington D. C. Online.
- Busettini C, Masson GS, Miles FA. 1997. Radial optic flow induces vergence eye movements with ultra-short latencies. *Nature* 390(6659):512-515.
- Busettini C, Miles FA, Krauzlis RJ. 1996. Short-latency disparity vergence responses and their dependence on a prior saccadic eye movement. *J Neurophysiol* 75(4):1392-1410.
- Casagrande V, Royal D, Sary G. 2005. Extraretinal inputs and feedback mechanisms to the lateral geniculate nucleus (LGN). In: Kremers J, editor. *The primate visual system: a comparative approach*. Chichester, UK: Wiley. p 191-206.
- Castet E, Masson GS. 2000. Motion perception during saccadic eye movements. *Nat Neurosci* 3(2):177-183.
- Celebrini S, Newsome WT. 1994. Neuronal and psychophysical sensitivity to motion signals in extrastriate area MST of the macaque monkey. *J Neurosci* 14(7):4109-4124.
- Celebrini S, Newsome WT. 1995. Microstimulation of extrastriate area MST influences performance on a direction discrimination task. *J Neurophysiol* 73(2):437-448.
- Felleman DJ, Van Essen DC. 1991. Distributed hierarchical processing in the primate cerebral cortex. *Cereb Cortex* 1(1):1-47.
- Fischer WH, Schmidt M, Hoffmann KP. 1998. Saccade-induced activity of dorsal lateral geniculate nucleus X- and Y-cells during pharmacological inactivation of the cat pretectum. *Vis Neurosci* 15(2):197-210.
- Fischer WH, Schmidt M, Stuphorn V, Hoffmann KP. 1996. Response properties of relay cells in the A-laminae of the cat's dorsal lateral geniculate nucleus after saccades. *Exp Brain Res* 110(3):435-445.
- Fuchs AF, Kaneko CR, Scudder CA. 1985. Brainstem control of saccadic eye movements. *Annu Rev Neurosci* 8:307-337.
- Gellman RS, Carl JR, Miles FA. 1990. Short latency ocular-following responses in man. *Vis Neurosci* 5(2):107-122.
- Hoffmann KP, Distler C. 1989. Quantitative analysis of visual receptive fields of neurons in nucleus of the optic tract and dorsal terminal nucleus of the accessory optic tract in macaque monkey. *J Neurophysiol* 62(2):416-428.
- Ilg UJ. 1997. Slow eye movements. *Prog Neurobiol* 53(3):293-329.
- Ilg UJ, Hoffmann KP. 1993. Motion perception during saccades. *Vision Res* 33(2):211-220.
- Kawano K. 1999. Ocular tracking: behavior and neurophysiology. *Curr Opin Neurobiol* 9(4):467-473.
- Kawano K, Miles FA. 1986. Short-latency ocular following responses of monkey. II. Dependence on a prior saccadic eye movement. *J Neurophysiol* 56(5):1355-1380.
- Kruger J. 1977. The shift-effect in the lateral geniculate body of the rhesus monkey. *Exp Brain Res* 29(3-4):387-392.
- Kruger J, Fischer B, Barth R. 1975. The shift-effect in retinal ganglion cells of the rhesus monkey. *Exp Brain Res* 23(4):443-446.
- Lee D, Malpeli JG. 1998. Effects of saccades on the activity of neurons in the cat lateral geniculate nucleus. *J Neurophysiol* 79(2):922-936.
- Lisberger SG. 1998. Postsaccadic enhancement of initiation of smooth pursuit eye movements in monkeys. *J Neurophysiol* 79(4):1918-1930.
- Madelain L, Krauzlis RJ, Wallman J. 2005. Spatial deployment of attention influences both saccadic and pursuit tracking. *Vision Res* 45(20):2685-2703.
- Masson GS, Castet E. 2002. Parallel motion processing for the initiation of short-latency ocular following in humans. *J Neurosci* 22(12):5149-5163.
- Maunsell JH, Van Essen DC. 1983. The connections of the middle temporal visual area (MT) and their relationship to a cortical hierarchy in the macaque monkey. *J Neurosci* 3(12):2563-2586.
- Merigan WH, Maunsell JH. 1993. How parallel are the primate visual pathways? *Annu Rev Neurosci* 16:369-402.
- Miles FA, Kawano K. 1986. Short-latency ocular following responses of monkey. III. Plasticity. *J Neurophysiol* 56(5):1381-1396.
- Miles FA, Kawano K, Optican LM. 1986. Short-latency ocular following responses of monkey. I. Dependence on temporospatial properties of visual input. *J Neurophysiol* 56(5):1321-1354.
- Morrone MC, Ross J, Burr D. 2005. Saccadic eye movements cause compression of time as well as space. *Nat Neurosci* 8(7):950-954.
- Newsome WT, Pare EB. 1988. A selective impairment of motion perception following lesions of the middle temporal visual area (MT). *J Neurosci* 8(6):2201-2211.
- Orban GA, Hoffmann KP, Duysens J. 1985. Velocity selectivity in the cat visual system. I. Responses of LGN cells to moving bar stimuli: a comparison with cortical areas 17 and 18. *J Neurophysiol* 54(4):1026-1049.
- Park J, Lee J, Lee C. 2001. Non-veridical visual motion perception immediately after saccades. *Vision Res* 41(27):3751-3761.
- Price NS, Ibbotson MR. 2001. Pretectal neurons optimized for the detection of saccade-like movements of the visual image. *J Neurophysiol* 85(4):1512-1521.
- Price NS, Ibbotson MR, Ono S, Mustari MJ. 2005. Rapid processing of retinal slip during saccades in macaque area MT. *J Neurophysiol* 94(1):235-246.
- 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(2):253-258.
- Reppas JB, Usrey WM, Reid RC. 2002. Saccadic eye movements modulate visual responses in the lateral geniculate nucleus. *Neuron* 35(5):961-974.
- Ross J, Burr D, Morrone C. 1996. Suppression of the magnocellular pathway during saccades. *Behav Brain Res* 80(1-2):1-8.
- Ross J, Morrone MC, Goldberg ME, Burr DC. 2001. Changes in visual perception at the time of saccades. *Trends Neurosci* 24(2):113-121.
- Schmidt M. 1996. Neurons in the cat pretectum that project to the dorsal lateral geniculate nucleus are activated during saccades. *J Neurophysiol* 76(5):2907-2918.

- Scudder CA, Kaneko CS, Fuchs AF. 2002. The brainstem burst generator for saccadic eye movements: a modern synthesis. *Exp Brain Res* 142(4):439-462.
- Seidemann E, Newsome WT. 1999. Effect of spatial attention on the responses of area MT neurons. *J Neurophysiol* 81(4):1783-1794.
- Sherman S, Guillery R. 1996. The role of the thalamus in the flow of information to the cortex. *Philos Trans R Soc Lond B Biol Sci* 357:1695-1708.
- Takemura A, Kawano K. 2004. The role of the cortical area MST in modulating the gain of ocular following responses. *Soc Neurosci Abstr Program No. 186.9*. Abstract Viewer/Itinerary planner. San Diego. Online.
- Thiele A, Henning P, Kubischik M, Hoffmann KP. 2002. Neural mechanisms of saccadic suppression. *Science* 295(5564):2460-2462.
- Treue S, Martinez Trujillo JC. 1999. Feature-based attention influences motion processing gain in macaque visual cortex. *Nature* 399(6736):575-579.
- Treue S, Maunsell JH. 1999. Effects of attention on the processing of motion in macaque middle temporal and medial superior temporal visual cortical areas. *J Neurosci* 19(17):7591-7602.
- Yarrow K, Haggard P, Heal R, Brown P, Rothwell JC. 2001. Illusory perceptions of space and time preserve cross-saccadic perceptual continuity. *Nature* 414(6861):302-305.