J Neurophysiol 94: 235–246, 2005.
First published March 16, 2005; doi:10.1152/jn.00041.2005.

Rapid Processing of Retinal Slip During Saccades in Macaque Area MT

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Submitted 12 January 2005; accepted in final form 10 March 2005

Price, N.S.C., M. R. Ibbotson, S. Ono, and M. J. Mustari. Rapid processing of retinal slip during saccades in macaque area MT. *J Neurophysiol* 94: 235–246, 2005. First published March 16, 2005; doi:10.1152/jn.00041.2005. The primate middle temporal area (MT) is involved in the analysis and perception of visual motion, which is generated actively by eye and body movements and passively when objects move. We studied the responses of single cells in area MT of awake macaques, comparing the direction tuning and latencies of responses evoked by wide-field texture motion during fixation (passive viewing) and during rewarded, target-directed saccades and nonrewarded, spontaneous saccades over the same stationary texture (active viewing). We found that MT neurons have similar motion sensitivity and direction-selectivity for retinal slip associated with active and passive motion. No cells showed reversals in direction tuning between the active and passive viewing conditions. However, mean latencies were significantly different for saccade-evoked responses (30 ms) and stimulus-evoked responses (67 ms). Our results demonstrate that neurons in area MT retain their direction-selectivity and display reduced processing times during saccades. This rapid, accurate processing of peri-saccadic motion may facilitate postsaccadic ocular following reflexes or corrective saccades.

INTRODUCTION

Saccades repeatedly interrupt our stable view of the world, yet we rarely notice the rapid retinal slip that they generate. Despite the well-characterized phenomenon of saccadic suppression (Burr et al. 1982, 1994; Diamond et al. 2000; Shioiri and Cavanagh 1989), we retain sensitivity to motion direction during saccades (Castet and Masson 2000). This may assist the generation of subsequent corrective saccades (Eggert et al. 1999) or contribute to the enhancement of ocular following reflexes (Kawano and Miles 1986). These behaviors require the rapid, accurate analysis of visual motion. At the population level, neurons located in macaque middle temporal area (MT) provide information about motion direction and speed and have a role in motion perception (Britten et al. 1992; Maunsell and Van Essen 1983b; Nichols and Newsome 2002). Most studies of area MT have focused on the effects of passive retinal slip, generated by stimulus movements during fixation or anesthesia. Some studies have examined the responses of area MT to active retinal slip generated by eye movements between specific targets (Bair and O'Keefe 1998; Krekelberg et al. 2003; Thiele et al. 2002). However, no studies have systematically examined the responses to saccades with a range of speeds and directions or the differences between rewarded saccades made to specified targets and nonrewarded spontaneous saccades to targets chosen by the animal. We studied the responses of single cells in area MT of awake macaques, comparing the direction tuning and latencies of responses evoked by wide-field texture motion during fixation (passive viewing) and during rewarded and spontaneous saccades over stationary texture (active viewing).

The most-detailed studies of the interactions between direction and speed tuning in area MT used bar stimuli, which provide localized motion signals consistent with object motion (Lagae et al. 1993; Rodman and Albright 1987). These studies suggest separability of a neuron's direction and speed tuning: the preferred direction is constant across all speeds. Cells in area MT have receptive field surrounds that either antagonize or facilitate their responsiveness to small stimuli presented in the receptive field center. Cells with antagonistic or facilitatory surrounds are anatomically segregated (Born and Tootell 1992). Although direction and speed tuning may be separable for small stimuli, it is not clear how direction and speed tuning interact when using wide-field stimuli that cover a cell's entire receptive field, including any inhibitory surround region. Because eye movements play a significant role in primate vision, assessing if and how MT distinguishes active from passive retinal slip will help us understand the role of MT in processing visual information during natural viewing. Our work adds to previous studies assessing the ability of area V1, the ventral intraparietal area (VIP) and medial superior temporal area (MST), to discriminate active and passive motion. In V1, which projects to MT (Maunsell and Van Essen 1983a), neurons are unable to discriminate active and passively generated retinal slip (Ilg and Thier 1996). However, in VIP and MST, which receive efferents from MT, many motion-processing cells can distinguish active from passive motion (Erickson and Thier 1991; Gabel et al. 2002; Thiele et al. 2002).

Small saccades during fixation affect the responses of individual MT neurons to moving stimuli in a direction-selective manner (Bair and O'Keefe 1998). Saccades may affect receptive field properties, contributing to the perisaccadic mislocalization of objects (Krekelberg et al. 2003). At the population level, psychophysical and functional imaging studies have also suggested that saccades suppress the magnocellular pathway (Burr et al. 1994) and specifically human area MT+ (Kleiser et al. 2004). Interestingly, some neurons in area MT have been reported to reverse their preferred direction when stimulated with saccade-generated rather than stimulus-generated retinal slip, which has been suggested as a physiological mechanism underlying saccadic suppression (Thiele et al. 2002).

We recorded from single cells in area MT and compared their responses to passive retinal slip generated by wide-field

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stimulus movements and active retinal slip generated by saccades. We found that the responses to active and passive motion showed similar direction tuning but had significant differences in latency. This suggests that temporal processing in area MT, or its input pathways, can be modified by eye movements.

METHODS

Surgical procedures

Behavioral and single unit data were collected from four normal juvenile rhesus monkeys (*Macaca mulatta*), weighing 3–6 kg. All surgical and experimental procedures were performed in strict compliance with National Institutes of Health guidelines, and the protocols were reviewed and 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 stereotaxically implant a stainless steel head stabilization post (Crist Instruments, Hagerstown, 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 locations in MT were verified using standard histological methods. Briefly, at the end of our single-unit recording studies, monkeys were sedated with ketamine-hydrochloride and deeply anesthetized with pentobarbital (80–100 mg/kg) prior to transcardial perfusion with physiological saline and 4% buffered paraformaldehyde. Frozen sections were cut in the coronal stereotaxic plane at 50 μm, mounted on slides, and stained for Nissl substance or myelin (Distler et al. 2002). Our recording tracks vertically penetrated the anterior bank of the superior temporal sulcus and entered area MT after crossing the lumen of the sulcus (Fig. 1). By relating the depth of neurons as indicated on the microdrive with histological features associated with representative electrode tracks, all neurons were assigned to area MT.

Behavioral paradigms and visual stimulation

During all experiments, monkeys were comfortably seated with the head stabilized in the horizontal stereotaxic plane. Monkeys were rewarded with fruit juice every 0.5–1 s for maintaining fixation on a red spot presented in the center of the screen. Visual stimuli were rear projected onto a tangent screen placed 61 cm from the eyes, covering a visual angle of 92 $\times$ 79°. Stimuli were projected using a Mirage 2000 digital light projector (DLP; Christie Digital, CA) with resolution 1,024 $\times$ 820 pixels, frame rate: 120 Hz and mean luminance: 170 cd/m^2. The DLP technology implemented in the Mirage 2000 does not suffer from typical refresh and scanning problems associated with CRT or LCD displays. Images are projected by light reflected from an array of micromirrors, with each pixel's color controlled by blending red, green, and blue light from three independently controlled mirrors. Each mirror can be set ON to project light to the screen or OFF to absorb the light. Thus the luminance of each pixel is controlled by the proportion of the time that each mirror is set to the ON position. Because each mirror can be moved to an ON or OFF position hundreds of times per frame and each pixel in a frame is updated simultaneously, there are no problems with phosphor luminance decay or scanning and refresh flicker as seen with CRT and LCD displays. To create continuous motion, stimuli could be moved with galvanometers, however, these can only be moved finite distances, thus high speeds can only be presented for very short durations. The DLP offers maximum flexibility when stimuli of high contrast, long duration, and high speeds are desired. Further, human observers were able to distinguish motion from flicker, and could accurately state the motion direction for speeds up to the highest tested speed of 1,280°/s.

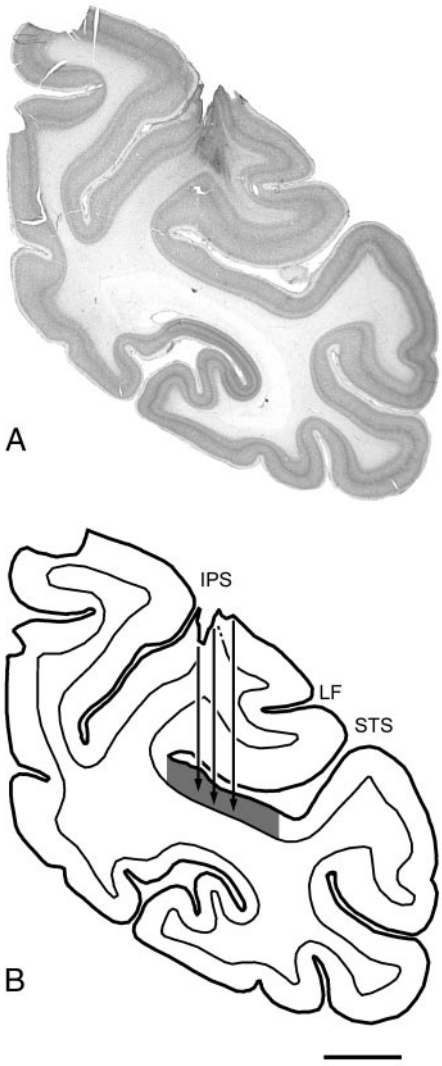

FIG. 1. Histological reconstruction of representative recording sites in middle temporal area (MT). Nissl-stained section cut in the coronal stereotaxic plane (*A*) and corresponding line drawing (*B*) showing the path of our electrodes entering cortex near intraparietal sulcus (IPS), traveling through MSTd before reaching MT cortex. Shaded section on B indicates the approximate borders of MT. Most of our recordings were obtained within 2 mm of this level. The damaged region immediately below the IPS was caused by the guide tubes used to penetrate the dura. LF, lateral fissure; STS, superior temporal sulcus. Scale bar = 5 mm.

During recording, cells were assigned to area MT based on their preference for moving stimuli, their high direction-selectivity, and their sensitivity to motion only in the contralateral hemifield. Prior to arrival in MT, electrode tracks typically passed through another area containing direction-selective neurons, shown by histological reconstructions to be area MST. On many tracks there was a distinct gap of $\leq$200 μm between MST and MT, in which it was not possible to isolate cells.

Stimuli were random texture patterns formed of 0.8° black or white squares. Studies of spatiotemporal frequency tuning in area MT suggest that nonlinear mechanisms tighten the speed tuning bandwidth of neurons when multiple spatial frequencies are present (Priebe et al. 2003). The random texture pattern contains broad spatial frequency content and provides continual motion information within the cell's receptive field. Bar stimuli cannot provide continual motion stimulation because the time they spend in a cell's receptive field is inversely proportional to their speed.

In two monkeys, we compared physiological responses of 111 cells to retinal slip generated by spontaneous saccades over the same stationary texture (active viewing) and retinal slip generated by steps in stimulus speed during fixation (passive viewing). In both conditions, texture was presented in an aperture of 60° diam centered in front of the monkey. For passive viewing conditions, the texture pattern was moved for 500 ms in eight directions, separated by 45°, with constant speeds ranging from 5 to 240°/s. Each stimulus condition was tested 8–16 times. A synchronization pulse at the start of the first frame of each stimulus condition was used to align stimulus presentation, eye movements and action potential timing. Between each passive test the texture pattern remained stationary for 1–5 s and monkeys were free to move their eyes. In this way, we were able to record responses to retinal slip during active viewing associated with spontaneous saccades with a range of amplitudes and directions. When making saccades, there were no specified targets for the monkeys and they were not trained or rewarded for making saccades of any size. Consequently, these saccades were self-motivated or spontaneous (Mohler and Wurtz 1976).

In addition, in 71 cells from two different monkeys we compared physiological responses to retinal slip generated by rewarded 10° saccades (active viewing) with a recreation of these saccades during fixation (passive viewing). The recreated saccade had a position profile averaged from 20 rewarded saccades that displayed no overshoot. Eye position traces during each rewarded saccade were initially sampled at 1 kHz, then averaged after being aligned at the time of saccade onset (speed >10°/s). Subsequently the position trace was subsampled at the projector frame rate to recreate an average 10° saccade profile. For active and passive viewing conditions, texture was presented over the entire screen covering 92 × 79°. For the directed saccades, a fixation point presented 5° from the center of the animal's visual field disappeared and immediately reappeared 10° away on the opposite side of the center of the visual field.

Eye movement collection and analysis

Eye position was monitored in two-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, UK). Eye velocity $v(t)$ was calculated off-line by differentiation of the eye position traces $p(t)$ using a central difference algorithm: $v(t) = [p(t + \delta t) - p(t - \delta t)]/2*\delta t$, with $\delta t = 2$ ms. Eye acceleration was calculated using the same algorithm by differentiating the velocity traces. Saccades were initially identified from peaks in the eye velocity traces that were >10°/s. Resting pre- and postsaccadic eye positions were calculated by averaging eye position in the windows 200–50 ms prior to and 100–200 ms after the velocity peak. If the eye position in these two periods exceeded 0.25° in range, or 0.2° in SD, saccades were rejected. Saccade start and end were identified as the time when eye speed first exceeded and dropped <10°/s. Additionally, saccades were rejected if their mean speed did not exceed 10°/s, their total displacement was <0.5°, or they were separated by <170 ms. This prevented the inclusion of corrective saccades. Finally, saccades were clustered into eight direction groups of 45° bandwidth, speed groups based either on the peak or mean eye speed during the saccade and displacement groups based on the total displacement of the eyes from start to end of the saccade.

Action potential collection and analysis

Unit activity was sampled at 25 kHz using iron-tipped, epoxy-coated tungsten electrodes with impedance 0.3–0.8 MΩ (Frederick-Haer, Brunswick, ME). Single-unit action potentials were detected on-line 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 (SDF) with 1-kHz resolution generated by initially convolving a delta function at each spike arrival time with a Gaussian window with $\sigma = 3$–10 ms. SDFs for stimulus-evoked responses were then calculated by averaging responses to individual stimulus presentations, including only portions of the response during which the eye position was within 3° of the desired fixation point. In addition, we ignored responses 100 ms after the eye position re-entered the desired 3° fixation window. SDFs for saccade-evoked responses were calculated by averaging the response to saccades of similar direction and speed or displacement, aligned on the time at which eye speed exceeded 10°/s.

Response latencies for stimulus-evoked responses were calculated relative to a synchronization pulse provided by the stimulus generation compute or relative to the time when the eye speed first exceeded 10°/s for saccade-evoked responses. We calculated a response threshold based on the ±3 SD of the neuron's spontaneous firing rate. The spontaneous rate was averaged from ≥48 periods of 500-ms duration while the monkey fixated a stationary stimulus prior to testing the responses to passive motion. 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. This represents a more rigorous implementation of a protocol used previously to measure latencies in area MT (Raiguel et al. 1999). Relative delays in the stimulus trigger, window discriminator and software template matching system used to identify action potentials were always <1.6 ms, thus they do not significantly affect the latency calculations.

We measured the responses to saccade-evoked retinal slip in a 50-ms window starting 25 ms after saccade onset, which spanned the period in which responses were elevated above the spontaneous level. As will be seen in RESULTS, the latencies for passive motion were typically 30 ms longer than for active motion. Consequently, for passive responses a 50-ms window was used that began at response onset, which provided a reliable transient response. For direct comparison with saccadic responses, a 50-ms window was used that started 25 ms after stimulus onset but this only included responses in the last 10–15 ms due to the longer latency. As a result, responses from this time period are not shown as they were unreliable and did not provide a realistic comparison with the active responses.

Measures of direction selectivity

We quantified direction selectivity using three measures. First the direction index (DI) is given by: $\mathrm{DI} = 1 - (R_{\mathrm{anti}}/R_{\mathrm{pref}})$. R_{pref} and R_{anti} represent the responses to motion in the preferred and antipreferred directions relative to the spontaneous firing rate (R_{spont}). DI values close to 0 indicate poor direction selectivity, while values close to 1 indicate strong direction selectivity. Values >1 indicate strong direction selectivity with inhibition in the antipreferred direction (Baker et al. 1981).

Second, we used the selectivity index (SI), which takes into account responses to motion in all directions (Vogels and Orban 1994)

$$\mathrm{SI} = \frac{\sqrt{\left[\sum_{i=1}^{n} R_i \sin(\theta_i)\right]^2 + \left[\sum_{i=1}^{n} R_i \cos(\theta_i)\right]^2}}{\sum_{i=1}^{n} R_i}$$

where n is the number of equally spaced stimulus directions and R_i is the response evoked by motion in direction θ_i. Values of SI lie between 0 and 1, with values close to 0 indicating poor direction selectivity and high values indicative of strong direction selectivity.

Finally, we fitted von Mises curves to the responses in each direction

$$\mathrm{VM}(\theta) = A*\exp([B*\cos(\theta - \theta_{\mathrm{pref}})] + C$$

Where $\mathrm{VM}(\theta)$ is the fitted response in direction θ; θ_{pref} is the cell's preferred motion direction; A affects the fit amplitude, B affects the

tuning width, and C is an offset. Least-squares fitting of a von Mises curve was chosen because it has been shown to give the best fits to physiological orientation tuning curves (Swindale 1998). Fits were judged using a F test to compare the goodness of fit between the data and a von Mises curve or a straight line. For cells the responses of which were significantly better fit by von Mises curves ($P < 0.05$), bandwidths were calculated from the von Mises curve, otherwise the cell was not included in the data set or its bandwidth was set to 360°. From the fitted curves we calculated the full-width at half-maximum direction bandwidth (BW) by finding the range of directions whose responses exceeded half the difference between R_{Spont} and R_{pref}. Smaller bandwidths indicate tighter direction selectivity.

RESULTS

We tested 182 cells in 4 monkeys. In the following sections, we describe the responses of 111 cells tested with unrewarded spontaneous saccades and passive retinal slip. The size and shape of the receptive fields of these cells were not explicitly mapped; however, we did determine their visual field locations. All were contralateral and within 30° of the fovea. Of the 111 tested with spontaneous saccades, 17 were unresponsive to saccades and gave unreliable responses to passive motion. These cells were excluded from the data presented here. Of the 94 sensitive to saccadic motion, 84 gave significant responses to both saccades and passive retinal slip such that latency information could be collected. Only 77/84 cells had sufficient data collected so that direction tuning at a range of active and passive speeds could be compared. Finally, of these 77, only 67 gave significantly direction-selective responses, with DI >0.5 for both active and passive retinal slip. In the following text, we describe the responses of a further 71 cells recorded in two different monkeys, which were tested with rewarded, target-directed saccades. The size and shape of the receptive fields of these cells were mapped. Their centers were never more than 15° from the fovea and their excitatory regions were never more than 20° diameter. Cells with obviously inhibitory surrounds were not included in this analysis.

Eye movements during saccades

During saccades, the eyes follow predictable trajectories, with the peak speed and duration of a saccade related primarily to the magnitude of the eye displacement. Figure 2, *A–C*, plots mean eye position, speed, and acceleration as functions of time for 1,258 spontaneous leftward saccades (directed ±22.5° from the horizon). The saccades have been divided into five groups according to their peak speed: 20–40, 40–80, 80–160, 160–320, and 320–640°/s. The mean eye traces for each speed group demonstrate that increases in the peak speed of a saccade are correlated with increases in duration, displacement, mean speed, and peak acceleration.

A scatter plot of saccade displacement against the peak speed during the saccade shows a clear linear relationship (Fig. 2*D*, $R^2 = 0.90$), consistent with the "main sequence" (Bahill et al. 1975). Plotting saccade displacement against duration shows a more scattered relationship (Fig. 2*E*, $R^2 = 0.66$); however, increases in saccade displacement are typically accompanied by increases in saccade duration. Saccade displacement was measured as the distance between the stable eye positions identified 200–50 ms prior to and 100–200 ms after the time when the eye speed reached a maximum. Saccade duration was measured as the time from when the eye speed exceeded 10°/s to when it dropped <10°/s. Eye speeds always increased monotonically from the time at which the 10°/s threshold was reached to the time of peak eye speed and then decreased monotonically.

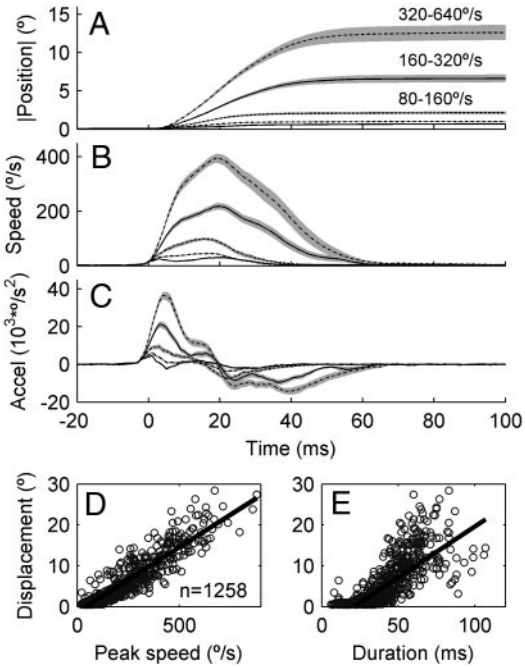

FIG. 2. *A–C*: eye position, speed, and acceleration traces during spontaneous saccades. Saccades were directed leftward (±22.5° from the horizon) and divided into 5 groups based on their peak speed (20–40, 40–80, 80–160, 160–320, and 320–640°/s; shown from lower to upper trace in each window). The mean of each group is presented as a black line embedded in a gray area representing ±1 SE. Traces are plotted relative to the time of saccade onset, defined as the time when the eye speed first exceeds 10°/s. Scatter plots showing the relationship between saccade displacement and peak speed (*D*) and duration (*E*) are shown for 1,258 leftward saccades made while recording from 1 cell. Solid black lines indicate the linear regression (*D*, $R^2 = 0.88$; *E*, $R^2 = 0.61$).

Responses to spontaneous saccades

SACCADE-EVOKED ACTIVITY IS DIRECTION SELECTIVE. We recorded responses from MT neurons while the monkeys made spontaneous saccades over a stationary textured background. Saccade-evoked activity in MT is clearly direction selective with the preferred direction of retinal slip during saccades matching that for retinal slip during fixation. Figure 3 shows eye traces, rasters, and the average spike density functions (SDF) measured from an MT cell when stimulated by left or rightward saccades with displacements of 4–8°. Saccades with motion directions of 157.5–202.5° were grouped together because they produce retinal slip predominantly in the cell's preferred direction (Fig. 3, *A* and *B*). Similarly, saccades with directions of −22.5–22.5° were grouped together because they produce retinal slip in the cell's antipreferred directions (Fig. 3, *C* and *D*). The responses to the two saccade directions show a clear asymmetry in the magnitude of saccade-evoked activity, illustrative of strong direction selectivity. We studied 17 cells while eye movements were made in complete darkness. For this, all light sources were removed, door jambs were

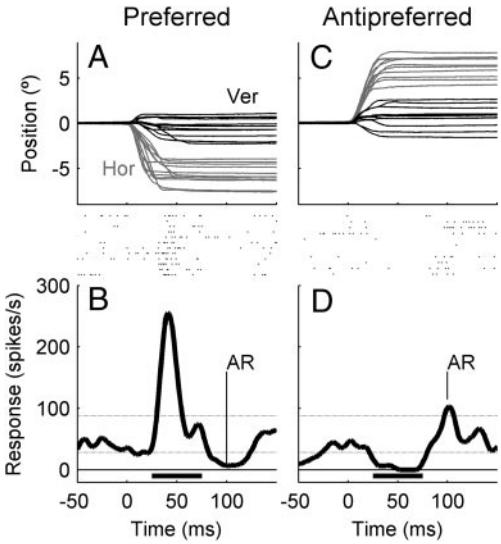

FIG. 3. Horizontal (gray) and vertical (black) eye position traces aligned on saccade onset (*A* and *C*) and the associated response rasters and spike density functions (*B* and *D*) recorded from 1 cell (*cell 63*) during 12 spontaneous horizontal saccades directed leftward (*A*) or rightward (*C*). Saccades were within ±22.5° of the horizon, with peak speeds of 200–360°/s and displacements of 4–8°. Horizontal dotted lines in *B* and *D* indicate the spontaneous spiking rate and threshold used to determine cell latency. Solid horizontal lines indicate the time window 25–75 ms after saccade onset used to calculate the cell's response to each saccade.

sealed, and the projector was switched off. In all of these cells, visual input was necessary to generate the saccade-related responses, with no cell's saccade-induced responses exceeding a threshold level of 2 SD above the mean spontaneous level of firing. Responses in the time window 25–75 ms after onset of saccades in the preferred direction were measured and converted to a *Z* score using the mean and SD of the cell's spontaneous activity measured in darkness. Across the population of 17 cells studied, the mean *Z* score was 0.34, indicating the very small effect of saccades in the dark on cell firing.

We generated direction-tuning curves for saccades grouped into eight direction categories with 45° bandwidth and five peak speed categories with one octave speed bandwidth (e.g., 40–80°/s, 80–160°/s). Each point in the direction-tuning curves represents the average response evoked by ≥4, but on average 11 saccades. In total, we analyzed the responses to 49,400 spontaneous saccades made while recording from 111 cells.

The directional and speed tuning properties of two cells to saccades of varying direction and peak speed are summarized in Fig. 4. Across a range of speeds, the responses evoked by retinal slip during saccades are direction selective. Some cells show strong excitation for preferred motion and no responses for antipreferred motion (Fig. 4, *left*). Others are motion opponent, with strong excitation in one direction and a reduction in the ongoing background activity for antipreferred motion (Fig. 4, *right*). No cells showed bidirectional excitation during the 25- to 75-ms response window. However, many cells showed opposite-sign after-responses in the immediate wake of this response window. That is, after preferred-direction motion spontaneous activity was transiently inhibited (Fig. 3*B*) and after antipreferred motion firing rate was transiently increased (Fig. 3*D*).

Speed dependence of motion responses

Many cells showed strong responses to saccades with very high peak speeds (320–640°/s). Importantly, this does not imply that they are sensitive to these high speeds because they are most likely responding to the slower speeds as the eye accelerates.

We determined the DI, SI, bandwidth, and response latency. These measures are shown averaged across the population of 94 cells for five speed ranges (Fig. 5, *A–D*) and as histograms of the values measured at each cell's preferred speed (*E–H*). Responses did not vary significantly with peak saccade speed. The mean DI ranged from 0.94 to 1.07 across the different speeds and was 1.06 ± 0.22 when using the response at each cell's preferred speed. Similarly, at the cell's preferred speed the mean SI was 0.55 ± 0.20 and mean bandwidth 79 ± 32°. The spike in the distribution at bandwidths of 45° (Fig. 5*G*) is due to a large number of cells that responded to saccades in only one direction group, giving no response to saccades in any other direction groupings. For the fitted von Mises curves used to determine bandwidth, the median R^2 at the preferred speed was 0.90. All these measures indicate strong direction selectivity, independent of the peak saccade speed.

Response latencies to stimuli in the preferred direction also did not vary significantly with speed (Fig. 5*D*). Mean latencies ranged from 29.1 ms for the 40–80°/s group to 37.2 ms for the

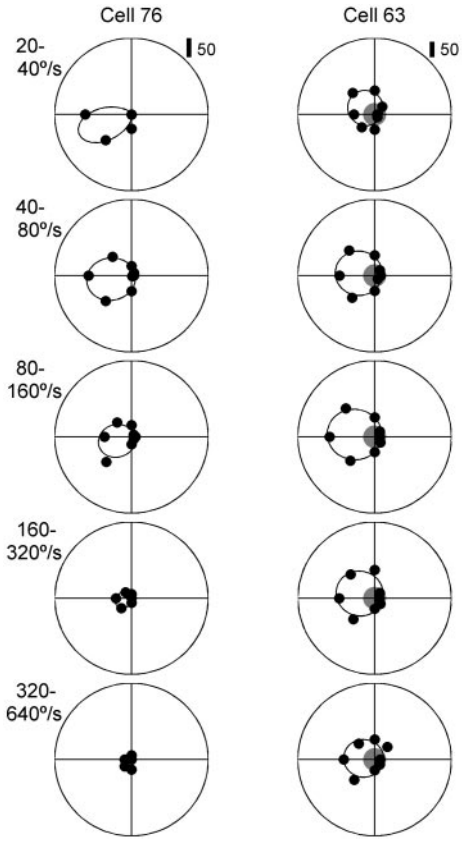

FIG. 4. Speed and direction tuning for spontaneous saccades. Each column shows the responses of 1 cell to saccades clustered into 8 direction groups and 5 speed groups based on the peak saccade speed. The speed ranges for each group are shown to the left of each plot. Measured responses (●) are average spiking rates in the window 25–75 ms after saccade onset. The fitted curves show the best fit von Mises function for each speed group. ◍, the spontaneous activity (*cell 76*: 1.9 spikes/s; *cell 63*: 36 spikes/s).

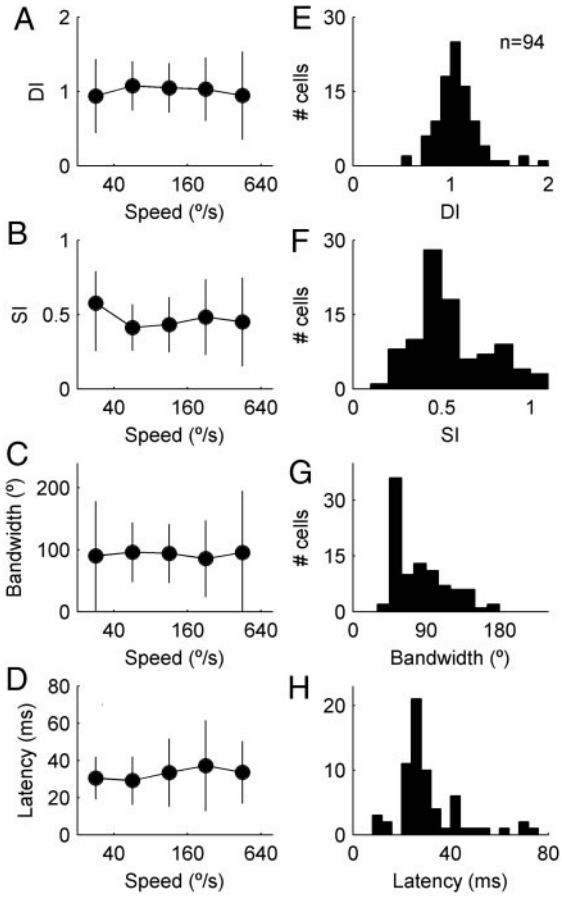

FIG. 5. Direction tuning indices and latencies for responses to spontaneous saccades. Mean ± SD values of direction index (DI, *A*), selectivity index (SI, *B*), and bandwidth (BW, *C*) are shown for each range of saccade peak speeds, plotted at the logarithmic center of each group, e.g., 56.6°/s for the speed group 40–80°/s. The corresponding histograms (*E–G*) show the distribution of DI, SI and BW for the same cells tested at their preferred speed. The mean ± SD latencies for each speed group and latency distribution at the preferred speed are shown in *D* and *H*.

120–320°/s group with the latency to each cell's preferred speed having a mean of 34.6 ± 19.9 ms (Fig. 5*H*).

Accuracy of response latencies

Figure 6 shows the eye position, speed, and acceleration averaged from 16 horizontal saccades. The horizontal solid line in Fig. 6*B* shows the threshold level of 10°/s used to find saccade onset, defined as the first point at which eye speed exceeds threshold (vertical solid line, Fig. 6). This measure of saccade-start was chosen because it provides a reliable measure of saccade onset allowing a high degree of automation. Lower thresholds can lead to errors in saccade timing if the eye is slowly drifting prior to a saccade, or if it shows dynamic overshoot or undershoot (Bahill et al. 1975). While a 10°/s threshold provides a reliable measure of saccade start, it could be criticized because the saccade may actually start some time before the eye speed reaches 10°/s.

To examine this possibility we also used a lower threshold of 4°/s. This was limited by random eye movements, which generate average speeds during fixation of 2°/s (Fig. 6*B*). This speed is associated with a change in eye position of only 0.008° over the 4 ms used by the central difference algorithm to calculate speed. These position changes are below the resolution of the eye-movement recording system. A criterion for correctly identifying saccade start was that the eye position and speed had to increase monotonically from the time the speed threshold was met to the time at which peak eye speed occurred. This presented no problems if a speed threshold of 10°/s was used; however, with the lower 4°/s threshold, any nonmonotonic changes in position of eye speed caused the saccade to be rejected. Similar problems were encountered when we used an eye-acceleration threshold of $<2{,}000°/s^2$. In cases where saccades were accepted using the 4 and 10°/s thresholds and no slow eye drift prior to saccade onset was obvious, we found that the increase in latency was 3.0 ± 0.43 (SD) ms. The averaged eye trace shown in Fig. 6 shows a typical example where the 4°/s threshold advances the saccade onset time by only 3 ms relative to the standard 10°/s threshold.

Comparison of MT responses during spontaneous saccades and passive motion

We compared the responses of 77 cells from two monkeys for which we characterized responses to saccades and step-changes in stimulus speed during fixation. Figure 7 shows superimposed direction tuning curves for 12 cells during spontaneous saccades (●) and steps in stimulus speed (○). In each case, the direction tuning curve at the cell's preferred speed is shown. For every cell, the preferred directions are closely aligned. We compared the preferred directions for 67 cells that showed strong direction-selectivity (DI > 0.5) for active and passive retinal slip (Fig. 8, *A* and *E*). Across this subpopulation,

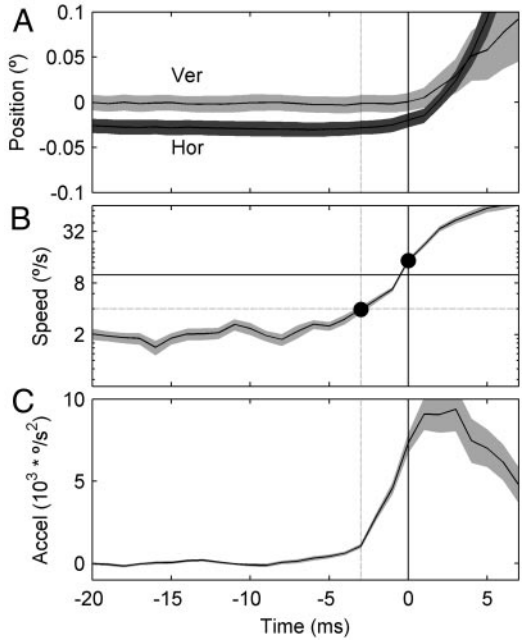

FIG. 6. Mean eye position, speed, and acceleration traces showing only the time around saccade onset (gray shading ± SE). Note that the vertical axis in *B* has a log scaling. The solid horizontal line in *B* indicates the 10°/s speed threshold used to define saccade onset, with the vertical solid lines in all panels marking the associated time of saccade onset to the nearest millisecond. The dotted lines show the time of saccade onset found using a 4°/s speed threshold. Traces are averages from 16 leftward saccades within ±22.5° of the horizon and with peak speeds of 80–160°/s.

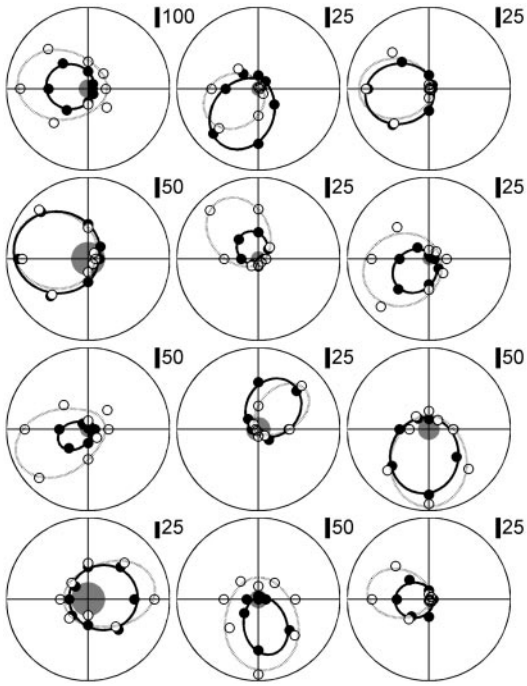

FIG. 7. Direction tuning curves for the responses of 12 cells to active (●) and passive (○) retinal slip. The fitted curves show the best fit von Mises function. ◎, the spontaneous activity for each cell, measured while fixating a stationary texture pattern.

close similarity in the preferred directions was found with the mean difference in preferred direction being 0.4 ± 32°. Linear regression of the preferred directions in the two cases produces a fit with a slope of 0.97 ($R^2 = 0.92$).

To compare direction tuning between spontaneous saccade-evoked (●) and stimulus-evoked responses (○), we used the DI, SI, and BW for a range of saccade peak speeds and stimulus speeds. A detailed analysis of latencies in these stimulus conditions is presented in the next section. Figure 8, *B–D*, shows the direction tuning measures for a range of speeds averaged across the population of 77 cells. For saccade-evoked responses, the values are plotted at the center of each speed range. Stimulus-evoked responses show strong direction-selectivity at speeds of 10–80°/s, but as a population, the direction selectivity decreases at higher speeds (160–240°/s), indicated by the increase in BW and reduction in DI and SI (○). Across the population, the majority of cells were highly direction selective for all speeds to which they were sensitive. However, 15% of cells were highly direction selective only for speeds of 20–80°/s but became omnidirectional for higher speeds, responding significantly and equally to motion in all directions. There was no observed correlation between a cell's preferred speed and its likelihood of becoming omnidirectional at high speed. As seen in Fig. 5, the saccade-evoked responses show similar direction-selectivity for all peak speed ranges, with values of DI, SI, and BW close to those seen for responses to passive stimulus speeds of 20–80°/s (●).

Figure 8, *F–H,* shows scatter plots of the DI, SI, and BW for 77 cells tested with passive motion at 40°/s and saccades with peak speeds in the range 20–40°/s. No strong correlation was observed between the measures for passive and active motion; however, in 88% of cells for both stimulus conditions, DIs were >0.5 and BWs were <180°, indicative of good direction selectivity.

Responses to rewarded saccades and passive recreation of saccadic profiles

Up to this point the responses to saccades have been compared with those to step-like changes in passive stimulus speed. To provide more precise comparison between active and passive motion, we trained two monkeys to make rewarded 10° saccades between fixation targets. The responses to these rewarded saccades were compared with responses to passive motion in which the stimulus moved in a saccade-like fashion. Of 71 cells tested with this protocol, 50 gave significant, direction-selective responses from which latencies could be measured. Figure 9 shows the responses of a cell to the passive recreation of vertical saccades (Fig. 9*A*) and to real vertical saccades (Fig. 9*B*). Solid black lines show responses to retinal slip in the same direction (the preferred direction), whereas dotted gray lines show responses to the opposite, antipreferred,

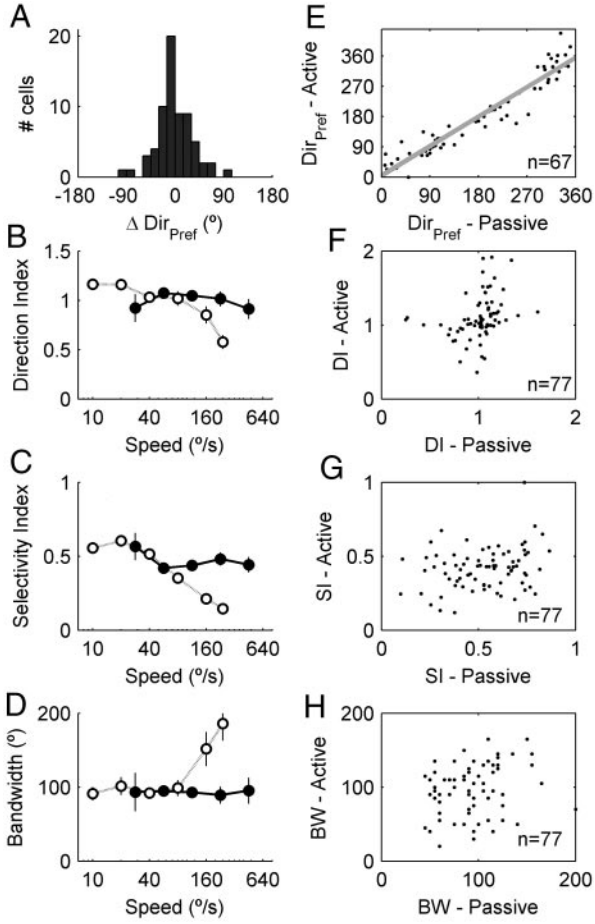

FIG. 8. Comparison of direction tuning for active and passive retinal slip. *A* and *E*: the difference in preferred direction for 67 cells that gave highly direction-selective responses (DI > 0.5) to active and passive motion. The gray line in *E* shows the linear regression to the data ($R^2 = 0.92$). *B–D*: mean ± SD values of DI (*A*), SI (*B*), and BW (*C*) for each range of saccade peak speeds (●) and for each constant step in passive stimulus speed (○). *F–H*: the DI, SI, and BW measured for 77 cells tested with a 40°/s passive stimulus or saccades producing peak speeds of 20–40°/s. R^2 values for the linear regression between active and passive measures of DI, S,I and BW were 0.10, 0.06, and 0.05, respectively.

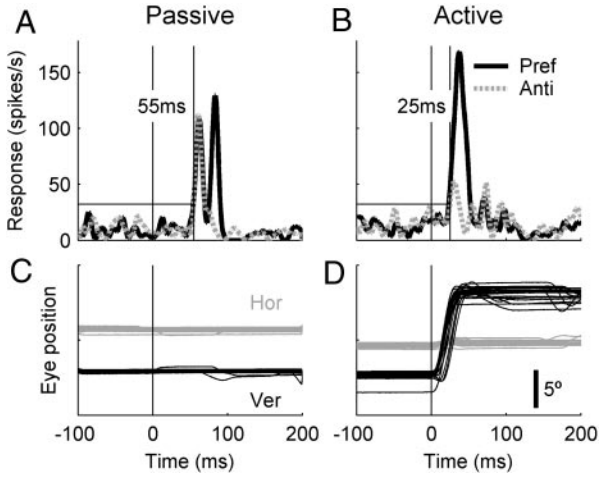

FIG. 9. Responses to 32 passive simulations of a 10° saccade (*A*) and responses to 24 rewarded 10° saccades (*B*) in the preferred and antipreferred directions. Solid lines in the 2 panels indicate responses to motion producing retinal slip in the cell's preferred direction, which was the same for both active and passive conditions. The corresponding eye movements during fixation (*C*) and the saccades (*D*) are shown. Horizontal solid lines in *A* and *B* indicate the threshold level (mean +3SD of spontaneous activity) used to determine response latency. The resulting latencies of 55 and 25 ms and *time 0* ms are marked with vertical lines.

direction. Thin horizontal lines indicate the threshold used to calculate response latency. This threshold represents the mean plus 3 SD of the spontaneous activity recorded while fixating the stationary pattern. Eye traces while recording from the cell in Fig. 9, *A* and *B*, are shown during fixation while viewing the passive stimulus (Fig. 9*C*) and around the time of saccade execution (Fig. 9*D*).

Responses to rewarded saccades were consistently direction-selective during the 25–75ms response window (Fig. 9*B*). There were no obvious differences between responses to these rewarded saccades and the responses to nonrewarded spontaneous saccades measured in the previous section of this paper (Fig. 3, *B* and *D*). For passive stimulation with a reconstruction of the saccade profile, cells showed nondirectional onset transients followed by a highly directional response (Fig. 9*A*). The nondirectional onset transient may be of the same origin as a low latency omnidirectional motion response reported when bicuculline methiodide was used to reduce the activity of local inhibitory circuits within MT (Thiele et al. 2004). In all cells recorded, the preferred direction of retinal slip generated by rewarded saccades and passive stimulus motion were the same.

We measured the response magnitude associated with responses to active and passive motion in the 50-ms window after the end of the latent period. The strength of responses elicited by rewarded saccades and passive recreation of these saccades was weakly correlated ($R^2 = 0.76$). However, there was no correlation between response strength and latency under active or passive conditions (passive: $R^2 = 0.09$; active: $R^2 < 0.01$). These results show that response magnitudes generated by retinal slip during active and passive motion were similar and that response magnitude cannot by itself be responsible for differences in response latency.

We compared the response latencies for passive motion, spontaneous saccades (e.g., Fig. 3) and rewarded saccades. As described previously, response onset was measured by the time when the neuron response exceeded a threshold of 3 SD above the mean spontaneous firing rate. This time was measured relative to the frame when the stimulus was first displaced (passive motion) or when the eye speed first exceeded 10°/s. As an example, the latency to passive stimulation for the cell in Fig. 9, *A* and *B*, was 55 ms, starting 30 ms later than the response to a rewarded saccade. Across the entire population, rewarded saccades had a mean latency of 30 ± 5 ms (Fig. 10) and had significantly shorter latencies than responses to the passive recreation of the same motion (67 ± 15 ms, $P < 0.001$, Wilcoxon rank-sum test). Spontaneous saccades had response latencies that were not significantly different to those measured for rewarded saccades (37 ± 17 ms; Fig. 10*B*). These had significantly lower latency than responses to step-like passive motion ($P < 0.001$, Wilcoxon rank-sum test), which had mean latencies of 63 ± 25 ms. Even if we add 5 ms to the latencies associated with saccades to overcompensate for our use of a 10°/s eye-speed threshold, the significant difference in latencies to passive and active motion remains.

DISCUSSION

We present here the first detailed analysis of the sensitivity of neurons in area MT to spontaneous saccades with a range of speeds and directions. We compare this and responses to rewarded saccades to specified targets with the responses to passive retinal slip associated with steps in stimulus speed and passive recreations of saccadic profiles.

Direction tuning

We found that all cells give direction-selective responses across a wide range of speeds in accordance with previous

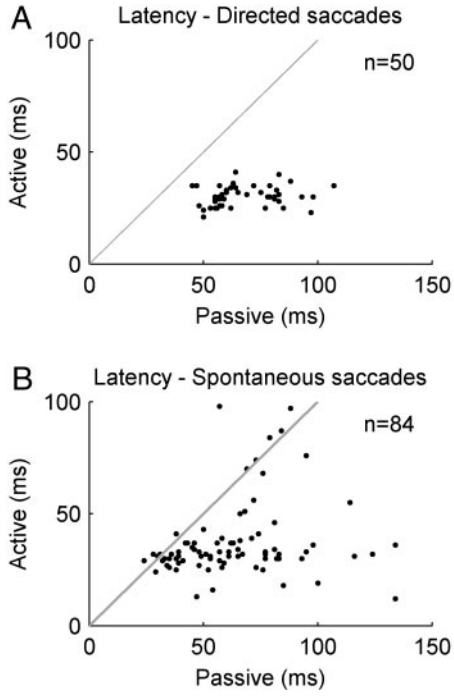

FIG. 10. *A*: comparison of response latencies associated with active retinal slip produced by rewarded 10° saccades and passive retinal slip produced by recreating the average retinal slip profile during a 10° saccade. *B*: comparison of latencies associated with spontaneous saccades and passive retinal slip produced by a step change to the cell's preferred speed.

studies using moving bars in area MT (Lagae et al. 1993; Rodman and Albright 1987) and V1 (Orban et al. 1981, 1986). We compared the direction tuning associated with retinal slip generated by spontaneous saccades and stimulus motion during fixation. Of 67 cells with strong responses and direction indices >0.5 for both conditions, 41 (61%) had preferred directions within $\pm 22.5°$, and the distributions of preferred directions were not significantly different. Further, no cells showed reversals in their preferred direction.

At high speeds of passive motion, the direction selectivity of the population of neurons declined, evidenced by a decline in mean direction index and increase in mean bandwidth. This is attributable to the omnidirectional responses observed in 15% of neurons at high speeds and may be due to temporal aliasing of the stimulus at high speeds. The fact that 85% of neurons retain their direction selectivity at high speeds demonstrates that temporal aliasing did not affect all neurons. Broader direction tuning is also predicted by psychophysical studies, which have found greatly reduced orientation sensitivity at high speeds, tending toward a complete loss of orientation tuning (Kelly and Burbeck 1987; Snowden 1992). Omnidirectional responses are not expected to emerge for saccades with high peak speeds because all saccades contain some low speed information, thus direction-selective responses to this low-speed retinal slip should be retained.

Previous studies have compared the direction selectivity of neurons in V4, MT, MST, and VIP when tested with active motion generated by smooth pursuit with constant speeds of $\leq 20°/s$ and identical passive motion generated by stimulus motion during fixation (Erickson and Thier 1991; Gabel et al. 2002). Areas VIP and MST receive direct inputs from MT (Maunsell and Van Essen 1983a). Cells in V4 and MT have similar preferred directions for active smooth pursuit and passive motion; however, in MST, many cells respond preferentially to active motion (Erickson and Thier 1991). Like area MT, VIP contains cells that show strong direction selectivity and sensitivity to high speeds (Colby et al. 1993); however, the cells in VIP show no systematic correlation between the preferred directions for active and passive motion (Gabel et al. 2002). The differences in preferred directions for active and passive motion have been used as evidence to argue that cells in MST and VIP incorporate information about head and eye movements into their analysis of visual motion, helping to disambiguate object and observer motion.

Thiele et al. (2002) recorded from cells in MT and MST as monkeys made 10° saccades or as they fixated while viewing saccade-like image displacements. In area MST, they found 68% of cells showed significant differences in activity between active and passive motion, with 39 of 116 cells showing reversals in direction tuning. This is consistent with studies of active and passive direction-tuning in area MST using lower stimulus speeds (Erickson and Thier 1991). However, unlike Erickson and Thier, Thiele and colleagues found that 20 of 51 MT cells showed reversals in directional tuning. None of the 67 highly direction-selective cells in our experiments showed these reversals.

The consistency in preferred direction that we found for responses to active and passive motion matches the findings of Erickson and Thier but differs from many of the cells described by Thiele et al. What could explain the discrepancies? Our measures of direction selectivity relied on the responses to passive motion in eight directions and saccades in all directions, whereas Thiele et al. used active and passive motion in only four directions. Although this reduces the precision of their measured preferred directions, it cannot account for reversals in direction tuning. We suggest four possible reasons for the different results: the projection method, the viewing distance, the stimulus size, and the time window used to measure responses.

First we used a projector with a refresh rate of 120 Hz, whereas Thiele et al. used a galvanometer-based system with continuous projection. Aliasing associated with our refresh rate may have contributed to differences in direction tuning; however, the fact that we found reliable, direction-selective responses at a range of speeds suggests that aliasing was not a problem. Further, human observers were able to accurately state motion direction for all tested speeds of passive motion and for the recreated saccades, showing that motion perception was not affected by the projector's refresh rate.

Second, different viewing distances used in the three studies may affect the responses. Our viewing distance (61 cm) and that of Erickson and Thier (80 cm) are both beyond grasping reach for a monkey. In contrast, Thiele et al. used a viewing distance of 38 cm, which is within grasping reach and increases the vergence angle required for fixation by a factor of ~ 1.6 relative to our study. Responses of neurons in area VIP are highly dependent on the viewing distance, preferring stimuli presented in near space or within grasping distance (Colby et al. 1993). If responses in area MT are similarly depth or vergence dependent, it is possible that the differences in viewing distance could account for the different results found in the three studies.

Third, in our study of spontaneous saccades, the stationary texture was only presented in an aperture of 60° diam. Thus a 10° saccade could move a receptive field over the boundaries of this texture. However, in later tests on 50 cells with rewarded saccades and passive recreation of these saccades, none showed reversals in their preferred direction. These tests were done with a stimulus subtending $92 \times 79°$, which covered the receptive field of all cells. This suggests that the different stimulus conditions used when comparing spontaneous saccades with passive motion are unlikely to account for the lack of direction reversal.

Finally, the reversal in preferred direction observed by Thiele et al. evolved ~ 100 ms after saccade onset and peaked 150 ms after saccade onset. Earlier responses did not show the reversal in preferred direction. It is possible that the later activity could actually represent responses to motion cessation. In many motion-sensitive neurons, activity is transiently inhibited or raised following preferred and anti-preferred direction motion, respectively (Barlow and Hill 1963; Hammond et al. 1986). These have been referred to as opposite-sign after-responses (Price and Ibbotson 2002). Such after-responses were very evident in our own data from MT (e.g., Fig. 3), but we did not include them in the analysis as our data focused entirely on the responses evoked during the period of motion (having corrected for latency).

Comparing latencies with previous studies

Latencies to retinal slip generated by spontaneous and rewarded saccades are ~ 30 ms shorter than for retinal slip

generated by stimulus movement while a monkey is fixating a stationary target. What could cause these discrepancies? Problems associated with measuring the time of saccade and response onset are unlikely to be a factor. Response onset was determined using a threshold of 3 SD above the mean background firing rate. This value was consistent for all stimuli (saccade or stimulus motion), so it could not have introduced a relative bias. Pushing back the time of saccade onset by using a saccade onset-threshold of 4°/s only extended the latency by a further 3.0 ms, which can only account for a small fraction of the latency differences. Similarly the use of a projector with a 120-Hz refresh rate could account for up to an 8.3-ms error in latencies; however, this cannot account for the long latency differences we observed. In control experiments, we observed no time-locking of neuron action potentials to the projector refresh rate, and cell response magnitudes and latencies to apparent motion steps of one and two frame durations were not significantly different (unpublished observations). Therefore at the frame rates used, we believe that the cells were responding to smooth motion rather than a sequence of displacements every 8.3 ms, supporting the comparison of active and passive motion we make here. We suggest that the latencies we report reflect real differences between saccade- and stimulus-evoked MT responses.

Thiele et al. (2002) recorded from 51 MT neurons while monkeys made 10° saccades and compared those responses to similar stimulus movements while the monkeys fixated a stationary target. Seventeen cells showed no significant differences in response magnitude 30–190 ms after motion onset, with 12 of these having different latencies for the two conditions (active: 57 ms; passive: 64 ms). Of the 34 cells that showed significant differences in response, 20 cells, classed as extra-retinal, showed reversals in preferred direction for active versus passive viewing. However, there were no significant differences between the latencies for MT cells that showed reversals and those that did not.

There are differences between the latencies measured by Thiele et al. (2002) and those measured by us. Moreover, the differences become even more pronounced if one considers the methods for calculating latency. Thiele and colleagues identified saccade-start as the time at which speed was 10% of the maximum saccade-speed. In our study and earlier reports (Fuchs 1967), 10° saccades have peak speeds of 250–600°/s, so saccade onset would be recorded at speeds of 25–60°/s. This would lead to latencies 5–10 ms shorter than those measured by us. Consequently, if they used our latency criteria their passive and active latencies would be extended to around 60–75 ms, although ours are 30 ms (directed saccades), 37 ms (spontaneous saccades) and 67 ms (passive).

The difference in stimulus patterns is the most likely source of the different latencies measured for passive stimulation in the two studies. However, this cannot readily explain the difference that we observed between passive and active viewing. Thiele et al. (2002) used a background noise pattern with a very low mean luminance of 1.7 cd/m^2 and an RMS contrast of 0.31. We used a stimulus with a mean luminance of 170 cd/m^2 and a Michelson contrast of 0.96. Therefore our stimulus was 2 orders of magnitude brighter and had square-wave contrast edges, which provide very robust stimulation for MT cells (Priebe et al. 2003). Moreover, it is well established that lower contrasts lead to longer latencies in macaque cortical neurons. For example, in macaque, V1 neurons have latencies of around 41 versus 52 ms for contrasts of 1.0 and 0.3, respectively (Reich et al. 2001). Behavioral responses dependent on cortical motion processing also show contrast-dependent latencies. For example, short-latency ocular following in macaques shows significant differences between response latencies at different contrasts (Miles et al. 1986).

Comparison of active versus passive latencies

What could account for the differences we observed between saccade latencies and stimulus-evoked latencies? Area MT receives most of its retinal input signal via a thalamocortical pathway, with additional inputs arriving via the superior colliculus and pulvinar (Maunsell and Van Essen 1983a). Inputs arriving via the pulvinar have longer latencies than inputs via the LGN (Azzopardi et al. 2003), suggesting this pathway is unlikely to account for the short latencies observed in area MT during saccades. Neuron latencies in the thalamocortical pathway are sufficiently low to account for the latencies we observed during saccades. In awake macaques, the shortest latencies of V1 cell responses to grating flashes are 20–31 ms (Maunsell and Gibson 1992; Mazer et al. 2002). In area MT, mean response latencies of 33 ms have been reported for flashed bars (Petersen et al. 1985), and 36 ms for the shortest latencies associated with moving bars (Raiguel et al. 1989, 1999). Thus passive motion information can reach area MT in well under 40 ms.

We suggest two explanations for the different latencies found for saccade- and stimulus-evoked responses: direct modulation of activity in area MT and modulation of activity along the input pathway. Saccade planning areas such as the frontal eye fields project to area MT (Leichnetz 1989) and become active ≤200 ms before saccade initiation (Bruce and Goldberg 1985). These oculomotor signals could modulate MT activity directly, accounting for the shorter latencies of saccade-evoked responses. However, the absence of presaccadic activity or responses to saccades in the dark suggests that saccadic responses in MT are primarily of retinal origin.

Saccadic modulation of the LGN is evident as decreased responsiveness around the time of saccade onset followed by increased responsiveness by the time the saccade ends (Lee and Malpeli 1998; Reppas et al. 2002). Further, cat LGN X- and Y-cell responses to saccades are on average 7.1 and 15.2 ms shorter, respectively, than responses to saccade-like motion during fixation, with latency differences of ≤40 ms reported (Fischer et al. 1996). This reduced latency has been attributed to GABAergic projections carrying oculomotor and visual information from the pretectum, which may provide a disinhibitory role as they synapse on inhibitory interneurons in the LGNd (Cucchiaro et al. 1993; Fischer et al. 1998).

Rapid responses to retinal slip during saccades may help calibrate eye movements and assist in postsaccadic gaze stabilization. The initial speed of ocular following reflexes is enhanced following saccades, which has been attributed largely to the retinal slip generated during the saccade (Kawano and Miles 1986). To ensure a low delay between motion onset and the start of ocular following eye movements requires rapid analysis of visual motion, as is mediated by area MT and passed to the oculomotor system. That this delay is reduced following saccades suggests that processing of retinal

slip must be accelerated around the time of saccades. Alternatively, rapid analysis of visual motion during saccades may help prevent degradation of acuity by postsaccadic retinal slip (glissades) or may be used to calibrate eye movements. While it is not clear if visual feedback during saccades improves their accuracy (Klier and Crawford 1998; Quaia et al. 2000), visual information sampled during the deceleration phase of saccades affects the latency of subsequent corrective saccades (Eggert et al. 1999).

Saccadic suppression

Despite differences in the responses to saccade- and stimulus-generated retinal slip, we found no evidence in area MT for saccadic suppression in the form of reduced responsiveness, motion sensitivity or spontaneous activity. Previous studies have identified presaccadic modulation in the LGN (Lee and Malpeli 1998; Reppas et al. 2002), V1 (Super et al. 2004) and MT/MST (Krekelberg et al. 2003). A separate study suggested that perceptually, saccadic suppression may be mediated by the saccade-related reversal in direction preference evident in some cells in area MT (Thiele et al. 2002). However, given that psychophysically saccadic suppression is evident prior to saccades (Ross et al. 2001), we think that this direction reversal may be a result of, rather than the cause of, saccadic suppression. Psychophysical testing also suggests that despite a general reduction in motion perception during saccades (Burr et al. 1982; Shioiri and Cavanagh 1989), direction selectivity is maintained (Castet and Masson 2000). Despite this, it has been argued that perception of second-order motion can be supported without explicit activity in areas MT or MST (Ilg and Churan 2004). We suggest that area MT plays an important role in analyzing the retinal slip that occurs during saccades. This may contribute to the perception of saccadic motion or may facilitate visual processing or eye movements after the saccades end.

ACKNOWLEDGMENTS

We thank T. Broznya and Dr. Valahb E. Das for assistance.

GRANTS

This work was supported by grants from the Australian Research Council (LX0349251), Sangora Educational Foundation and the National Institutes of Health Grants EY-06069 and RR-0165.

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