Role of the Oculomotor Vermis in Generating Pursuit and Saccades: Effects of Microstimulation

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Krauzlis, R. J. and F. A. Miles. Role of the oculomotor vermis in generating pursuit and saccades: effects of microstimulation. *J. Neurophysiol.* 80: 2046–2062, 1998. We studied the eye movements evoked by applying small amounts of current (2–50 μA) within the oculomotor vermis of two monkeys. We first compared the eye movements evoked by microstimulation applied either during maintained pursuit or during fixation. Smooth, pursuitlike changes in eye velocity caused by the microstimulation were directed toward the ipsilateral side and occurred at short latencies (10–20 ms). The amplitudes of these pursuitlike changes were larger during visually guided pursuit toward the contralateral side than during either fixation or visually guided pursuit toward the ipsilateral side. At these same sites, microstimulation also often produced abrupt, saccadelike changes in eye velocity. In contrast to the smooth changes in eye velocity, these saccadelike effects were more prevalent during fixation and during pursuit toward the ipsilateral side. The amplitude and type of evoked eye movements could also be manipulated at single sites by changing the frequency of microstimulation. Increasing the frequency of microstimulation produced increases in the amplitude of pursuitlike changes, but only up to a certain point. Beyond this point, the value of which depended on the site and whether the monkey was fixating or pursuing, further increases in stimulation frequency produced saccadelike changes of increasing amplitude. To quantify these effects, we introduced a novel method for classifying eye movements as pursuitlike or saccadelike. The results of this analysis showed that the eye movements evoked by microstimulation exhibit a distinct transition point between pursuit and saccadelike effects and that the amplitude of eye movement that corresponds to this transition point depends on the eye movement behavior of the monkey. These results are consistent with accumulating evidence that the oculomotor vermis and its associated deep cerebellar nucleus, the caudal fastigial, are involved in the control of both pursuit and saccadic eye movements. We suggest that the oculomotor vermis might accomplish this role by altering the amplitude of a motor error signal that is common to both saccades and pursuit.

INTRODUCTION

The neural coordination of pursuit eye movements involves widespread regions of the cerebral cortex, brain stem, and cerebellum. The best understood pathways involve projections from areas of the cerebral cortex to specific nuclei within the basilar pontine gray, which in turn project to specific portions of the cerebellum. One of these pathways includes cortical areas near the superior temporal sulcus, such as the middle temporal and the medial superior temporal areas (Dürsteler and Wurtz 1988; Dürsteler et al. 1987; Newsome et al. 1985), whereas a second pathway includes the frontal eye fields (Keating 1991, 1993; MacAvoy et al. 1991). These different cortical areas tend to project to different portions of the pontine nuclei; the areas near the superior temporal sulcus project primarily to the dorsolateral edge (Glickstein et al. 1980; May and Andersen 1986), whereas the frontal eye fields project more strongly to the dorsomedial portion (Stanton et al. 1988). These regions of the pontine nuclei in turn project to different regions of the cerebellum known to be important for pursuit, including the ventral paraflocculus and so-called oculomotor vermis (Brodal 1979, 1982; Glickstein et al. 1994; Langer et al. 1985; Yamada and Noda 1987). These corticopontocerebellar connections therefore provide alternative descending pathways by which visual information can access the motor pathways for pursuit.

The functional significance of these alternative pathways is not yet established. In part, this uncertainty is due to the fact that, although specific processing steps for pursuit have been suggested for the pathway through the ventral paraflocculus, none have been proposed for the pathway through the oculomotor vermis. Lesions of the ventral paraflocculus and flocculus produce severe, permanent deficits in smooth eye movements (Zee et al. 1981), and microstimulation of the ventral paraflocculus elicits smooth eye movements (Belknap and Noda 1987; Lisberger and Pavelko 1988). The output of the ventral paraflocculus, conveyed by Purkinje cells, exhibits a tonic modulation during maintained pursuit as well as a transient modulation whenever eye speed changes (Krauzlis and Lisberger 1994; Miles et al. 1980; Stone and Lisberger 1990). These two components of Purkinje cell activity have been interpreted as encoding the motor command needed to maintain pursuit eye speed and the visual inputs needed to change pursuit eye speed, respectively. In contrast, the role of the oculomotor vermis in pursuit eye movements is much less clear. Some results are qualitatively similar to those obtained in the ventral paraflocculus; lesions of the oculomotor vermis produce deficits in pursuit eye movements, and Purkinje cells in the oculomotor vermis are modulated during pursuit eye movements (Kase et al. 1979; Keller 1988; Suzuki and Keller 1983, 1988a,b; Takagi et al. 1996). However, unlike Purkinje cells in the ventral paraflocculus, those in the vermis are also modulated during the presentation of moving visual stimuli when no eye movements occur (Sato and Noda 1992a; Su-

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zuki and Keller 1988b; Suzuki et al. 1981). Furthermore, whereas electrical microstimulation within the oculomotor vermis has been shown to elicit saccadic eye movements and to modify the metrics of visually guided saccades (Fujikado and Noda 1987; Keller et al. 1983; McElligott and Keller 1984; Noda and Fujikado 1987a,b), it has not been shown to produce smooth eye movements. Indeed, it has been argued that the oculomotor vermis is involved only in the control of saccadic eye movements and that pursuit-related regions might lie in adjacent folia of the cerebellum (Sato and Noda 1992a).

This study reexamines the effects of microstimulation within the oculomotor vermis on pursuit eye movements. In contrast to previous studies, in which microstimulation was applied while the animal fixated a stationary spot or was in the midst of making a saccade, we additionally applied microstimulation during visually guided pursuit eye movements. We found that, although microstimulation during fixation produces only small smooth eye movements, microstimulation during pursuit can produce much larger changes. We also found that, depending on the current eye movement behavior and the strength of microstimulation, both pursuit and saccadic eye movements can be evoked. Based on a new method for classifying eye movements, we suggest that one function of this cerebellar region is to regulate the amplitude of a motor error signal that is common to both saccades and pursuit.

METHODS

Data were collected from two adolescent rhesus monkeys (*Macaca mulatta*), weighing 4–9 kg. All experimental protocols were approved by the Institute Animal Care and Use Committee and complied with Public Health Service Policy on the humane care and use of laboratory animals. Under isoflurane anesthesia and aseptic conditions, the head of each monkey was fitted with a pedestal and secured to the skull with titanium screws and dental acrylic, which allowed the head to be fixed in the standard stereotaxic position during experiments. A scleral search coil was implanted around each eye by using the technique of Judge et al. (1980). The coils were used to monitor eye position with the electromagnetic induction technique (Fuchs and Robinson 1966). The AC voltages induced in the search coils were routed to a phase detector circuit that provided separate DC voltage outputs proportional to horizontal and vertical eye position (CNC Engineering). These outputs were low-pass filtered (6-pole Bessel, −3 dB at 180 Hz) and then sampled at 1 kHz (A/D converter, National Instruments). The coil output voltages were calibrated with respect to eye position by having the animal fixate small light-emitting diode (LED) targets at known eccentricities along the horizontal and vertical meridia.

After the animals were well trained on the behavioral paradigms, they were prepared for microstimulation in a second surgery, again by using isoflurane anesthesia and aseptic conditions. We trephined a hole in the midline of the skull, just in front of the occipital ridge. A chamber was placed over the craniotomy and affixed to the skull with dental acrylic and additional titanium screws. In the first monkey, the chamber was tilted back 35° from stereotaxic vertical and aimed at the interaural line. In the second monkey, the chamber was tilted back 25° and aimed at a point 4 mm posterior of the interaural line.

Electrical microstimulation

Microstimulation was applied through tungsten microelectrodes (Frederick Haer) with impedances of 0.2–1 MΩ. Electrodes were advanced through stainless steel guide tubes (23 gauge) with a micromanipulator (Narishige) mounted on top of the recording chamber. The guide tubes were held fixed in the chamber with a grid system (Crist et al. 1988). Microstimulation was applied as biphasic pulses (pulse width 0.2 ms) at frequencies of 50–500 Hz for a duration of 50 ms; currents used for microstimulation ranged from 2 to 50 μA. Sites likely to be concerned with eye movements were identified by recording multiunit activity and by testing the effects of microstimulation at regular intervals ($\sim$200 μm) along an electrode track. For each site at which we elicited changes in eye position we determined the minimum current that produced a just noticeable effect on eye velocity. For behavioral paradigms, the current was generally set at twice this minimum value to produce an appreciable effect on smooth eye velocity without always eliciting a saccade. Consistent with previous studies (Fujikado and Noda 1987; Noda and Fujikado 1987a,b), sites that required the lowest currents were also associated with high-frequency multiunit activity characteristic of white matter.

Behavioral paradigms

The monkeys viewed LED stimuli that were projected as 0.1° spots onto a translucent tangent screen located 1 m in front of the animal. The monkeys were trained to maintain fixation of a target spot until it moved or was extinguished. During this fixation period, which had a randomized duration of 1,000–1,500 ms, the monkeys were required to remain within 2° of the central target and to refrain from making saccades, which were detected on-line by the computer as any velocity >48°/s. If these requirements were not met, the target was extinguished and the paradigm reverted to the fixation period after a 2-s delay. If the fixation requirements were met, the target could remain stationary (''fixation trial''), step horizontally to an eccentric location on the tangent screen (''saccade trial''), or step horizontally to a new location and start to move at a constant speed of 15°/s back toward the center of the screen (''pursuit trial''). The amplitude of the step on pursuit trials was adjusted to reduce the need for catch-up saccades (Rashbass 1961). On fixation trials, the monkey was required to keep its eyes within 2° of the target position. On saccade and pursuit trials, the monkey was required to foveate the target within 450 ms and thereafter keep its eyes within 4.5° of the target position for an additional 500 ms. If eye position strayed outside of these windows, the trial was aborted and followed by a new randomly selected trial. Controlled movements of the target spot were achieved with an X-Y mirror galvanometer system under negative feedback control (General Scanning, CCX101). The monkey was given a liquid reward at the end of each correctly performed trial. The luminances of the target and background were 3.8 and 0.04 cd/m^2, respectively.

During individual experiments conducted at each site, trials including microstimulation were randomly interleaved with trials without microstimulation, such as those described previously. On fixation trials with microstimulation, a single 50-ms train of microstimulation was applied as the animal fixated the stationary target. On pursuit trials with microstimulation, the microstimulation was applied during maintained pursuit, defined as 450–700 ms after the onset of target motion. On saccade trials with microstimulation, the microstimulation was triggered by the onset of the saccade, detected by the computer as any eye velocity exceeding 48°/s. Because the effects of microstimulation in the vermis were known to depend on eye position, additional conditions were added to provide the necessary controls. In experiments at some sites, the target was initially centered on the screen for pursuit and saccade trials so that the target and eye were at slightly eccentric positions ($\sim$6°) when microstimulation was applied. For these sites, we used additional fixation conditions at ±6° along the horizontal meridian. At other sites, the target was initially placed at an eccentric position

that compensated for its subsequent motion so that the target and eye were roughly centered when the microstimulation was applied. These two experimental condition produced essentially identical results.

Data collection and analysis

The presentation of stimuli, application of microstimulation, and the acquisition, display, and storage of data were controlled by a personal computer using REX, a real-time experimentation software package (Hays et al. 1982). Voltage signals encoding the horizontal and vertical components of eye position and the horizontal and vertical mirror position provided by transducers in the galvanometer systems were low-pass filtered (6-pole Bessel, −3 dB at 180 Hz) and then digitized to a resolution of 12 bits, sampling at 1 kHz (A/D converter, National Instruments). All data were stored on disk (Wren Runner II SCSI disk) during the experiment and later transferred to a Unix-based system for subsequent analysis with Silicon Graphics workstations.

An interactive analysis program was used to filter, display, and make measurements from the data. Signals encoding horizontal and vertical eye velocity were obtained by applying a 29-point finite impulse response filter (−3 dB at 54 Hz) to the signals encoding horizontal and vertical eye position, respectively. Most of the measurements reported in this paper were taken directly from these individual traces of eye position and velocity. The interval used for these measurements was based on the observed latency of the evoked eye movements (slightly longer than 10 ms) and the duration of the microstimulation (50 ms). Consequently, we obtained three measurements from the raw traces of eye position and velocity over the interval 10 to 60 ms after the onset of microstimulation: *1*) the change in eye position over the 50-ms interval of microstimulation, *2*) the peak change in eye velocity over the 50-ms interval of microstimulation, and *3*) the change in eye position up to the time of the peak change in eye velocity. For reasons explained in RESULTS, these last two measurements were also obtained from pursuit and saccadic eye movements evoked by movements of the target spot. Statistical significance of differences between the effects of microstimulation obtained under different behavioral conditions was assessed with either a Mann-Whitney test for single comparisons or a Kruskal-Wallis test for multiple comparisons. As described more fully in RESULTS, some of the data were also subjected to a linear discriminant analysis. Statistical tests were performed with commercially available statistical software (BMDP and SigmaStat).

To document the effects of microstimulation on smooth eye velocity, in the first part of the analysis we also extracted traces of the average smooth eye velocity portions of the recorded eye movements. These average ''desaccaded'' traces were obtained by marking those segments of individual trials containing saccadic eye movements by using an algorithm described previously (Krauzlis and Miles 1996). Average eye velocity was computed by aligning the responses to the same target motion on the onset of the target motion and computing the mean and SD of eye velocity for each millisecond sample of the data. Within each millisecond sample, eye velocity values marked as residing within a saccade were not included in the calculation of mean and SD of smooth eye velocity. However, we documented the occurrence of such marked intervals by computing the frequency with which each millisecond of the trial was included in such an interval; this computation provided the ''freq(sacc)'' traces shown in RESULTS. To analyze the smooth eye velocity traces, we subtracted the desaccaded eye velocity obtained on trials without microstimulation from the desaccaded eye velocity obtained on trials with microstimulation to generate traces of changes in smooth eye velocity caused by microstimulation. From these traces, we made three measurements: *1*) the average change in smooth eye velocity over the 50-ms interval of microstimulation, *2*) the average direction associated with this change in smooth eye velocity, and *3*) the latency of this change in smooth eye velocity. Latency measurements were obtained with a linear regression algorithm described previously (Krauzlis and Miles 1996). Statistical significance of differences between the effects of microstimulation obtained under different behavioral conditions was again assessed with either a Mann-Whitney test for single comparisons or a Kruskal-Wallis test for multiple comparisons.

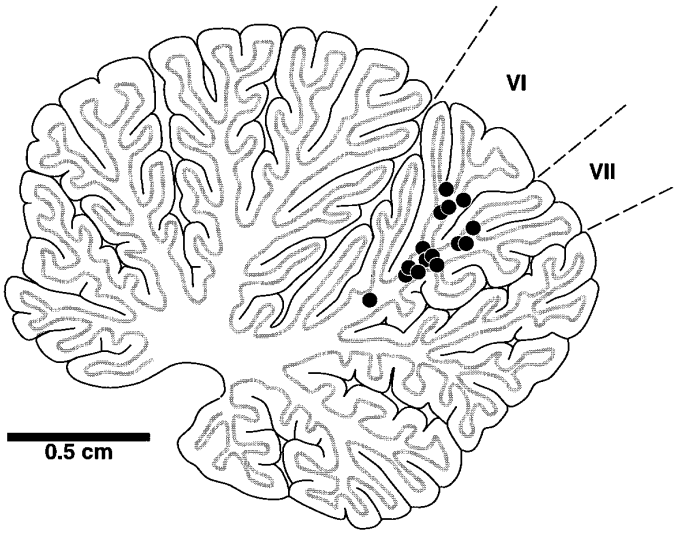

FIG. 1. Sites in oculomotor vermis at which microstimulation elicited eye movements; sample data from *monkey 1*. Drawing shows a single parasaggital section, 1 mm from the midline in the left cerebellar hemisphere. ●, individual sites of stimulation; – – –, primary, posterior superior, and prepyramidal fissures, from *top left* to *bottom right*. Scale bar indicates ½ cm.

Histology

After completion of the set of experiments on each monkey, we made small marking lesions (50 μA, 10 s) at several of the sites at which eye movements were evoked with microstimulation. The monkeys were then deeply anesthetized with pentobarbital sodium and perfused through the heart with saline followed by 10% glutaraldehyde. The cerebellum and brain stem were embedded in celloidin and cut into 50 μm parasaggital sections. The sections were stained with cresyl violet and the marking lesions were identified to help reconstruct the electrode penetrations.

RESULTS

We stimulated a total of 112 sites in the ''oculomotor vermis'' (Fujikado and Noda 1987; Noda and Fujikado 1987a,b) of two monkeys. The sites were located in lobule VII and the posterior portion of lobule VI, within 2–3 mm of the midline, as indicated by the sample of sites shown for *monkey 1* in Fig. 1. A minority of sites was located within the cerebellar cortex; most sites were located in the underlying white matter where the thresholds for evoking eye movements were lower. Microstimulation at these sites produced both pursuitlike smooth changes in eye velocity and saccadelike abrupt changes in eye position. We first describe the pursuitlike effects and show how these were facilitated during pursuit eye movements. We then compare these effects with the saccadelike effects. Finally, we de-

scribe a quantitative method for rigorously classifying the effects as pursuit or saccadelike.

Enhancement of pursuitlike effects during ongoing pursuit

Electrical microstimulation could produce pursuitlike smooth changes in eye velocity that were larger when the stimulation was applied during ongoing pursuit than when it was applied during fixation. At some ties, microstimulation during pursuit produced smooth changes in eye velocity directed toward the ipsiversive side (with respect to the site of microstimulation), regardless of whether the pursuit movement was in the ipsiversive or contraversive direction. The results obtained from stimulating at one such site are shown in Fig. 2, *A*–*F*. As indicated by the cartoons atop the figure, microstimulation was applied in the left oculomotor vermis (50 μA, 300 Hz) as the monkey maintained fixation of a stationary target (Fig. 2*A*) or pursued the target as it moved at a constant speed of 15°/s to the right (Fig. 2*B*) or to the left (Fig. 2*C*). The microstimulation was applied for the 50-ms interval indicated by the filled bar; data from this period of the trial are shown on an expanded time scale in Fig. 2, *D*–*F*. When microstimulation was applied while the monkey was fixating (Fig. 2*D*), it caused a small leftward (i.e., ipsiversive with respect to the side stimulated) deflection in horizontal eye velocity (thick trace, $\dot{E}_{HOR}$), and also increased the likelihood of saccades immediately afterward [freq(sacc)]. In contrast, when the microstimulation was applied while the monkey pursued a target moving toward the right (contraversive) side (Fig. 2*E*), it caused a more vigorous and abrupt ipsiversive change of horizontal eye velocity (i.e., a deceleration), relative to the eye velocity recorded during this same period of the trial in the absence of microstimulation (''control''). Similarly, when the microstimulation was applied while the monkey pursued toward the left (ipsiversive) side (Fig. 2*F*), it again caused an ipsiversive change in eye velocity (i.e., an acceleration).

At many sites, however, microstimulation with fixed parameters produced pursuitlike change in eye velocity during pursuit toward the contralateral side but produced small saccadelike eye movements during pursuit toward the ipsilateral side. Sample data from one such site are shown in Fig. 2, *G*–*I*. Microstimulation applied while the monkey was fixating (Fig. 2*G*) caused a small upward deflection in horizontal eye velocity and an increase in the frequency of saccades. When the microstimulation (15 μA, 300 Hz) was applied during contraversive pursuit (Fig. 2*H*), it caused a predominantly ipsiversive change in smooth eye velocity and a small change in the frequency of saccades. However, when the microstimulation was applied during ipsiversive pursuit (Fig. 2 *I*), it caused a change in smooth eye velocity that was not much different from that observed during fixation but produced a large increase in the frequency of ipsiversive saccadelike eye movements. These saccadelike eye movements were typically accompanied by changes in smooth eye velocity but occurred at slightly longer latencies. The occurrence of these saccadelike eye movements is indicated by the abrupt increase in saccadic frequency 25–100 ms after the onset of the microstimulation (Fig. 2*I*). Although we did not systematically examine the latency of evoked saccades, we noted that their timing was influenced by the stimulation parameters; lower currents evoked saccades that followed the evoked changes in smooth eye velocity (e.g., Fig. 2), whereas higher currents evoked saccades in <20 ms that superceded any evoked changes in smooth eye velocity.

Before considering how these saccadelike eye movements interacted with the effects on pursuit, we first documented the effects of microstimulation on smooth eye velocity alone. Across our sample of sites, we found that the changes in smooth eye velocity caused by microstimulation tended to be larger during pursuit than during fixation (Fig. 3). At 63% (53/84) of the sites, the amplitude of the changes in smooth eye velocity obtained during contraversive pursuit was significantly different from that obtained during fixation; all of these differences were due to the effects being larger during pursuit (Fig. 3*A*). At 33% (26/78) of the sites, the amplitude of the effects during ipsiversive pursuit was significantly different from those obtained during fixation; 62% (16/26) of these differences were due to enhanced effects during pursuit (Fig. 3*B*). Thus, although larger effects on smooth eye velocity were observed during pursuit in general, the enhancement was more pronounced during contraversive than during ipsiversive pursuit.

Pursuit eye movements altered not only the amplitude but also the direction of the smooth eye movements evoked by microstimulation. During fixation, microstimulation produced changes in eye velocity that were mostly directed upward, as indicated by the vectors plotted in Fig. 4, *C* and *G*. As summarized by the open bars in Fig. 4, *D* and *H*, the distribution for evoked eye movements during fixation forms a single peak centered near upward (mean 81.7 and 85.6° in Fig. 4, *A* and *B*, respectively). In contrast, during pursuit, microstimulation produced changes that included a significant horizontal component, especially during contraversive pursuit, when the evoked eye movements acquired a substantial ipsiversive component (Fig. 4*A*). The distribution of directions during contraversive pursuit (solid bars in Fig. 4*B*) contains a single large peak for directions ipsilateral and slightly upward (mean 42.2°) while retaining an extended tail that includes the upward directions evoked at these same sites during fixation. During ipsilateral pursuit, the horizontal component was only slightly larger and not always directed toward the ipsilateral side (Fig. 4*E*). Consequently, the distribution of directions during ipsiversive pursuit (shaded bars in Fig. 4F) is lower and flatter (mean 70.2°).

Despite the differences in the metrics of the smooth eye movements evoked by microstimulation during fixation and pursuit, all pursuitlike eye movements were evoked at similar short latencies. At a majority of sites (80/103), microstimulation produced changes in smooth eye speed that were large enough to allow us to estimate their latency. The majority of these evoked eye movements had latencies of 10–20 ms, regardless of whether the microstimulation was applied during fixation (mean 14.1 ms), contraversive pursuit (mean 12.6 ms), or ipsiversive pursuit (15.5 ms).

Saccadelike effects evoked by microstimulation during pursuit

In the analysis described previously (Figs. 3 and 4), the segments of eye movements containing saccades were removed to document the effects on pursuit. However, in addition to changes in smooth eye velocity, microstimulation

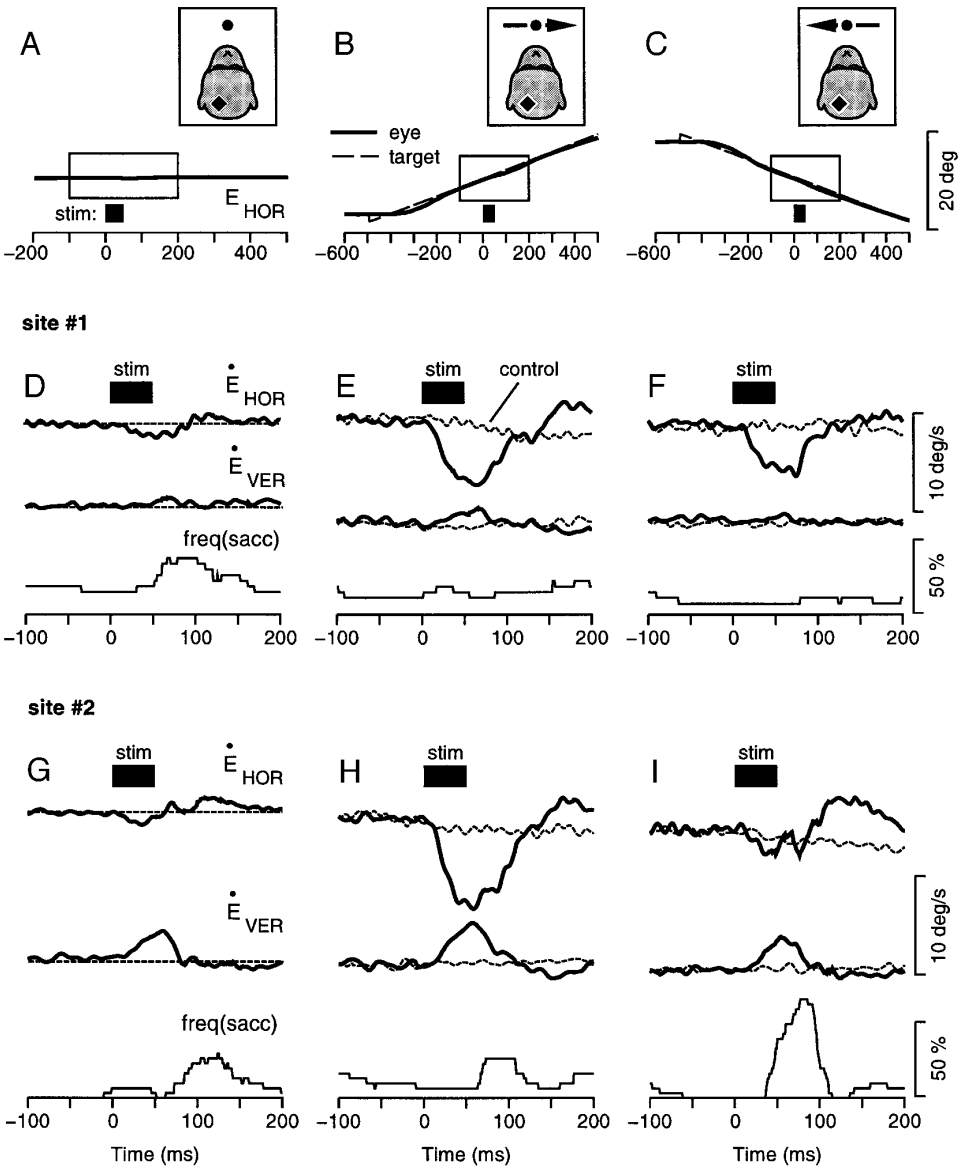

FIG. 2. Pursuitlike eye movements evoked by microstimulation at 2 sites (*monkey 2*). *A*, *D*, and *G*: microstimulation applied during fixation. *B*, *E*, and *H*: microstimulation applied during pursuit toward the contralateral side. *C*, *F*, and *I*: microstimulation applied during pursuit toward the ipsilateral side. Black horizontal bars indicate the 50-ms intervals during which stimulation was applied. Traces in *A–C* show average horizontal eye position over 26 trials (E_{HOR}) and horizontal target position (dashed line) recorded during microstimulation (50 μA, 300 Hz) at site 1. Eye position traces were not desaccaded. Similar traces (not shown) were recorded during microstimulation (15 μA, 300 Hz) at site 2. Boxes in *A–C* indicate the peristimulation intervals that are shown on an expanded time scale below. Traces in *D–I* show average desaccaded horizontal eye velocity ($\dot{E}_{HOR}$), average desaccaded vertical eye velocity ($\dot{E}_{VER}$), and percentage of trials on which a saccade occupied each millisecond of the trial [freq(sacc)]. Thicker eye movement traces show averages on trials with microstimulation; dashed traces indicate either 0 (*D* and *G*) or average eye velocity on control trials without microstimulation (*E*, *F*, *H*, and *I*). For the horizontal eye velocity traces, 0 eye velocity lies below the dashed control traces in *E* and *H* but above the dashed control traces in *F* and *I*.

often elicited saccadic eye movements (e.g., Fig. 2, *G–I*). Thus the observation that the effects of microstimulation were larger during contraversive than ipsiversive pursuit holds true for the smooth eye velocity component but not necessarily for the entire evoked eye movement. We therefore analyzed data from each site again without removing those segments identified as containing saccades. These measurements were taken from the same temporal interval as the measurements of smooth eye velocity (Fig. 3) but were made from individual traces of eye position rather than average traces of smooth eye velocity. The results are summarized by the graph in Fig. 5*A*, in which each data point represents a single site and indicates the amplitude of the change in eye position evoked during ipsiversive pursuit as a function of the amplitude evoked during contraversive pursuit. The data mostly lie along the line of unity slope, indicating that the net changes in eye position were generally comparable during ipsiversive and contraversive pursuit. In

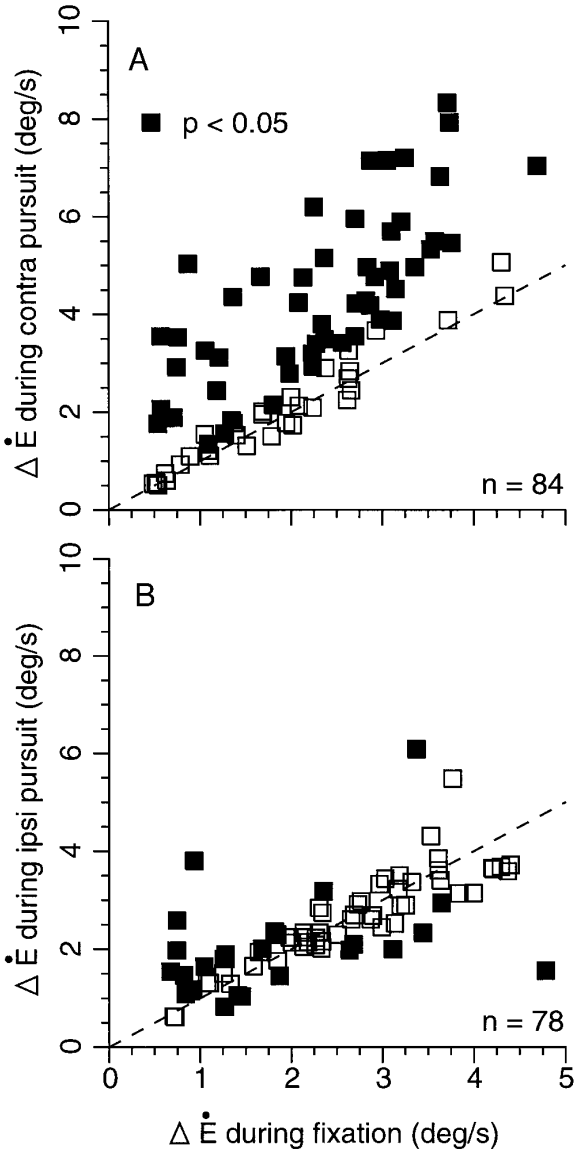

FIG. 3. Amplitudes of pursuitlike eye movements evoked by microstimulation during pursuit and fixation. Graphs plot the average change in smooth eye velocity evoked during pursuit as a function of the average change evoked during fixation for contraversive (*A*) and ipsiversive (*B*) pursuit. Individual symbols represent single sites; different symbol types are used to indicate sites for which the 2 measurements were (■) or were not (□) significantly different ($P < 0.05$, Kruskal-Wallis). – – –, *both graphs*, unity slope. The amplitude of the evoked movement was measured as the average change in eye speed in the interval 10–60 ms after the onset of the microstimulation minus the change over this same interval on trials without stimulation, using desaccaded traces of eye velocity.

fact, at sites where larger eye movements were evoked, there was a tendency for the effects to be larger during ipsiversive than during contraversive pursuit (arrow).

The discrepancy between the effects on smooth eye velocity (Fig. 3) and the effects on eye position (Fig. 5*A*) is due to differences in the numbers of saccades elicited during the two directions of pursuit. We summarized this aspect of our data by determining the change in saccade frequency caused by electrical microstimulation during contraversive and ipsiversive pursuit. For each site, we measured the fraction of trials on which a saccade occurred within the interval 10–60 ms after the onset of microstimulation, minus the fraction of trials on which a saccade occurred in this interval without microstimulation. At almost all of the sites (68/71, 96%), microstimulation increased the occurrence of saccades. This increase tended to be larger during pursuit toward the ipsiversive side (mean increase 24%) than during pursuit toward the contraversive side (mean increase 14%). A direct comparison of these increases during ipsiversive and contraversive pursuit, on a site by site basis, is shown in Fig. 5*B*. A majority of the data points (47/71, 66%) lie above the dashed line of unity slope, indicating that microstimulation was more likely to evoke saccades during ipsiversive than during contraversive pursuit.

In addition, by varying the parameters of the microstimulation, we were able to manipulate the relative fraction of pursuit and saccadelike effects evoked at single sites. As illustrated by data from one site (Fig. 6), higher stimulation frequencies evoked larger eye movements. For example, dur-

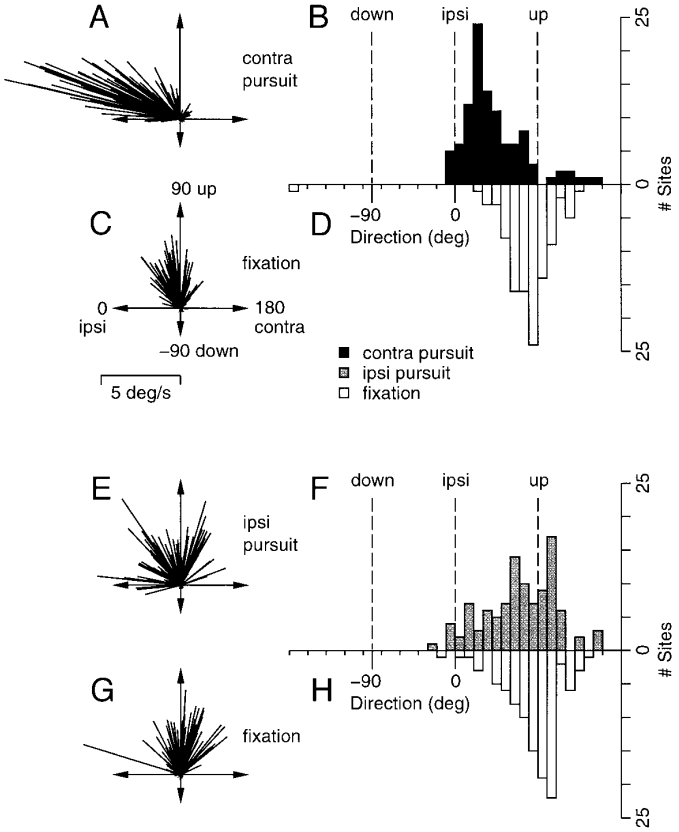

FIG. 4. Direction of pursuitlike eye movements evoked by microstimulation during pursuit and fixation in both monkeys. *A*, *C*, *E*, and *G*: polar plots indicate the size and direction of evoked smooth eye velocity during contraversive pursuit (*A*), ipsiversive pursuit (*E*), and during fixation (*C* and *G*). Each line summarizes the effects obtained at one site and represents a vector pointing in the direction of the evoked eye movement with a length proportional to the amplitude of the evoked eye movement. *B*, *D*, *F*, and *H*: histograms summarize the distributions of the directions of evoked smooth eye velocity during contraversive pursuit (solid bars), ipsiversive pursuit (shaded bars), and fixation (open bars). Direction of evoked eye movement was taken as the average direction of desaccaded eye velocity over the interval 10–60 ms after the onset of microstimulation. Two sets of measurements are shown for fixation because for some sites 2 different fixation points were used to provide comparisons with measurements taken from the 2 directions of pursuit.

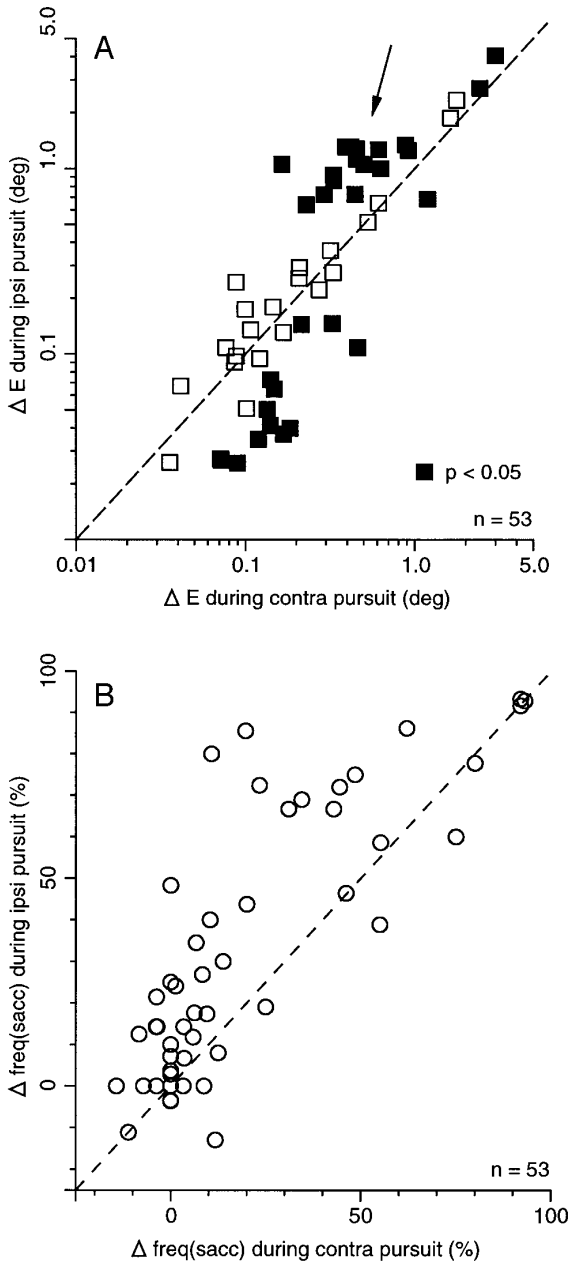

FIG. 5. Combined pursuit- and saccadelike effects evoked by microstimulation during pursuit. *A*: comparison of the amplitudes of eye movements evoked by microstimulation during contraversive and ipsiversive pursuit. Each datum point shows the net change in eye position over an interval 10–60 ms after the onset of microstimulation during ipsiversive (ordinate) and contraversive (abscissa) pursuit. Measurements were made from individual trials without removing saccades. Different symbol types are used to indicate sites for which the 2 measurements were (■) or were not (□) significantly different ($P < 0.05$, Kruskal-Wallis). Arrow indicates cluster of data points for which evoked movements were larger during ipsiversive pursuit than during contraversive pursuit. *B*: comparison of the relative numbers of saccades evoked by microstimulation during contraversive and ipsiversive pursuit. The graph plots the percentage of trials on which microstimulation elicited a saccade during ipsiversive pursuit against the corresponding fraction of trials during contraversive pursuit. Each symbol represents measurements from a single site, taken from the interval 10–60 ms after the onset of the microstimulation. Values <0 correspond to sites at which microstimulation decreased the number of saccades compared with control trials. The dashed lines have a slope of one.

ing contraversive pursuit, the 500-Hz microstimulation (Fig. 6*E*) produced a larger change in the average smooth (i.e., desaccaded) eye velocity than the 300-Hz microstimulation (Fig. 6*B*) and also led to an increase in the frequency of saccades. During ipsiversive pursuit, the higher frequency of microstimulation led primarily to an increase in the frequency of saccades without a marked effect on average smooth eye velocity (Fig. 6, *C* and *F*). Similar effects were observed at 14 sites; increasing the frequency of microstimulation increased the size of the evoked eye movements, either by increasing the amplitude of the evoked smooth eye movements or the evoked saccades, or both.

Classifying evoked eye movements as pursuit- or saccadelike

To document the range of eye movements evoked by microstimulation, it was necessary to develop a novel method for classifying eye movements as either pursuit or saccadelike. The method we devised is a modification of the main sequence used to analyze the metrics of saccadic eye movements (Bahill et al. 1975; Boghen et al. 1974). The main sequence for saccades is obtained by plotting the peak velocity of each saccade as a function of its amplitude for a set of saccades spanning a range of amplitudes. This type of measurement procedure cannot be applied directly to pursuit because the continuous nature of pursuit eye movements makes measurement of their amplitude arbitrary. Our modification of this procedure is illustrated in Fig. 7. For saccades (Fig. 7*A*), we measured the peak velocity of the saccade (96.7°/s) but compared this with the displacement of the eye at the time of the peak (0.98°, indicated by the dashed vertical lines) rather than to the total displacement over the entire saccade. The advantage of defining the end of the measurement interval based on the peak velocity is that an analogous procedure can be applied to pursuit. As shown in Fig. 7*B*, we measured the peak velocity reached during the initiation of pursuit (21.6°/s) and compared this with the displacement of the eye at the time of the peak (1.14°). These measurements document the fact that, for our sample eye movements of similar amplitude (0.98 vs. 1.14°), the saccade moved the eyes much faster (96.7 vs. 21.6°/s).

We summarized similar measurements taken from a large number of saccadic and pursuit eye movements with the graph shown in Fig. 7*C*. Each square plots the peak velocity of a saccade against the displacement at the time of this peak for a single eye movement. Saccades were either elicited by stepping a target to an eccentric location or occurred spontaneously between periods of steady fixation. Each triangle plots analogous measurements for the smooth eye movements on a single trial. Pursuit eye movements were evoked by using step-ramp stimuli with several different target velocities (5, 10, 15, 20, and 25°/s). The segregation of the two sets of data in the graph illustrates that these measurements provided a clear demarcation between the metrics of saccadic and pursuit eye movements. To obtain an objective distinction between the two classes of eye movements based on these measurements, we performed a linear discriminant analysis on the two data sets. This provided the linear function shown by the dashed oblique line in Fig. 7*C*. The discriminant function correctly classified

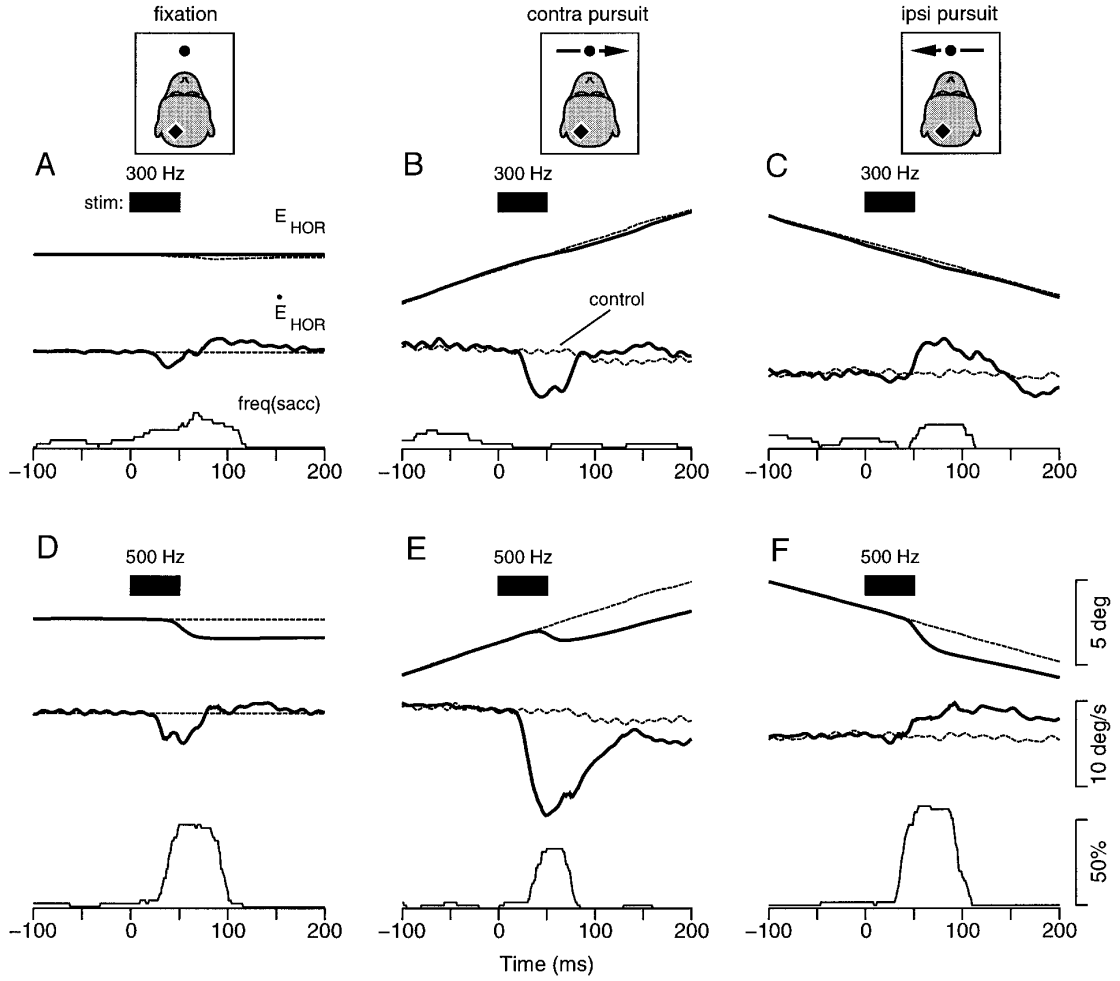

FIG. 6. Changes in pursuit and saccadelike eye movements caused by changing the frequency of microstimulation. Each panel shows the effect of applying microstimulation (30 μA) during fixation (*A* and *D*), contraversive pursuit (*B* and *E*), or ipsiversive pursuit (*C* and *F*) at either 300 (*A–C*) or 500 Hz (*D–F*). Traces in each panel show average horizontal eye position (E_{HOR}, not desaccaded), average desaccaded horizontal eye velocity ($\dot{E}_{HOR}$), and percentage of trials on which a saccade occupied each millisecond of the trial [freq(sacc)]. Thicker eye movement traces show averages on trials with microstimulation; dashed traces indicate either 0 (*A* and *D*) or average eye position and velocity on control trials without microstimulation (*B*, *C*, *E*, and *F*). Each average is based on 50 single trials. Black horizontal bars indicate the 50-ms intervals during which stimulation was applied.

99.9% of the sample data points (n = 1,284), ''incorrectly'' classifying only one eye movement.

We used this same analysis to classify the eye movements evoked with different strengths of microstimulation at single sites. The data in Fig. 8 provide three examples of measurements obtained at the same site shown in Fig. 6; these examples were chosen to show the range of evoked eye movements that we measured. The superimposed traces in Fig. 8, *A–C*, compare the eye position and velocity on single trials with microstimulation (solid line) with the average eye position and velocity on the 54 control trials without microstimulation (dashed line). The three sets of traces are shown on the same vertical scale to illustrate the range of amplitudes evoked by microstimulation. The panels in Fig. 8, *D–F*, show data from these same trials but plotted as the difference between the stimulation and control traces. For these difference traces, the vertical scale was changed (as indicated in parentheses) to compensate for differences in amplitudes across the three cases. For each evoked eye movement, we measured the peak change in eye velocity caused by the microstimulation (3.4, 7.3, and 230°/s) and the change in eye position at the time of this peak eye velocity (0.05, 0.17, and 1.5°).

After making similar measurements for each of the individual eye movements evoked by microstimulation at a site, we plotted the data just as we plotted the data obtained from visually guided saccadic and pursuit eye movements. Because the effects of microstimulation depended on the current eye movement behavior of the animal, we made separate plots for eye movements evoked during fixation (Fig. 9, *A* and *B*), contraversive pursuit (Fig. 9, *C* and *D*), and ipsiversive pursuit (Fig. 9, *E* and *F*). We then compared these measurements with the discriminant function (indicated by the dashed oblique lines) obtained by analysis of visually guided saccadic and pursuit eye movements (i.e., Fig. 7). Those evoked eye movements that provided measurements lying above this line were classified as saccadelike (crosses) and those lying below this line were classified as

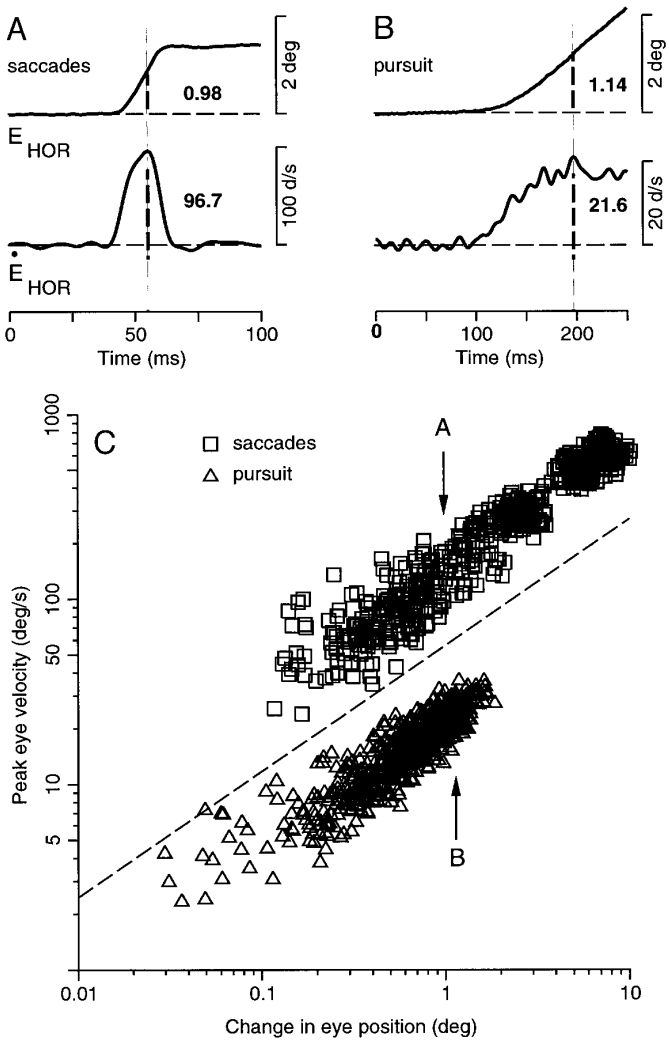

FIG. 7. Method for classifying visually guided eye movements as pursuit or saccadelike. *A*: example of measurements made from eye position and eye velocity records during a visually guided saccade. *B*: example of analogous measurements made from records during a single trial of visually guided pursuit. The target motion was a 20°/s step–ramp. *C*: plot of peak eye velocity against change in eye position for 640 saccadic (□) and 644 pursuit (△) eye movements. – – –, discriminant function used to demarcate the 2 classes of eye movements. Arrow A indicates location of sample saccade measurements; arrow B indicates location of sample pursuit measurements.

pursuitlike (circles). In addition, the shaded regions in the graphs reiterate the measurements obtained from visually guided saccades and pursuit. That most of the data points lie over these shaded regions indicates that the metrics of eye movements evoked with electrical microstimulation were similar to those of normal saccades and pursuit.

The classification of evoked eye movements helps resolve the apparent contradiction in our observations that microstimulation produced larger effects on smooth eye velocity when applied during contraversive pursuit (Fig. 3), whereas the overall changes in eye position were similar during ipsiversive pursuit (Fig. 5*A*). As shown by the circles in the *bottom left regions* of the plots in Fig. 9, the smallest evoked eye movements—those caused by lower microstimulation frequencies—were always classified as pursuitlike. In contrast, as shown by the crosses in the *top right regions* of the plots in Fig. 9, the largest evoked eye movements were always classified as saccadelike. For evoked eye movements of intermediate amplitudes, there was a transition between pursuit and saccadelike effects. This transition occurred within a much narrower range than the overlap evident in the measurements obtained from visually guided pursuit and saccades (shaded regions). In addition, the location of this transition depended on the eye movement behavior of the animal. When the animal was fixating (Fig. 9, *A* and *B*) or pursuing in the ipsilateral direction (Fig. 9, *E* and *F*), the transition occurred for smaller eye movements than when the animal was pursuing in the contraversive direction (Fig. 9, *C* and *D*). Thus the basis for our observation that the largest smooth eye movements occurred only during contraversive pursuit appears to lie in how the placement of this transition point was affected by the behavioral context.

To quantify the transition point between pursuit and saccadelike effects, we converted scatter plots of data such as those shown in Fig. 9 into frequency plots such as those shown in Fig. 10. First, we replaced each measurement of peak velocity shown in Fig. 9 with a ''0'' if it was classified as pursuitlike or with a ''1'' if it was classified as saccadelike. We then placed these scores into equally spaced bins (in log coordinates) based on the amplitude of the change in eye position associated with each data point. Next, we determined the fraction of data points within each bin that were classified as saccadelike, the result from each bin producing one of the circles plotted in Fig. 10. Thus for the smallest evoked eye movements the data points in each graph lie near zero (bottom dashed horizontal lines), whereas for largest evoked eye movements the data points lie near one (top dashed horizontal lines). Finally, we fitted these transformed data points with the function

$$P = 1 - \delta \exp\left[-(\Delta E/\alpha)^{\beta}\right]$$

where P is the probability of a saccade, ΔE is the amplitude of the change in eye position, α is the change in eye position corresponding to a ''threshold'' probability of 82%, β is the slope of the function, and δ determines how far less than one the probability can drop. The primary purpose of fitting the data with this function was to provide an objective measure of the transition point, which we defined as the value of α for each fitted function (shown within each graph in Fig. 10). These thresholds quantify the point made graphically by the plots of the raw measurements in Fig. 9. For the horizontal component of the eye movement, the transition between pursuit- and saccadelike effects at this site occurred at larger eye movements (Fig. 10*C*, 0.59°) during contraversive pursuit than during either fixation (Fig. 10*A*, 0.09°) or ipsiversive pursuit (Fig. 10*E*, 0.19°).

Similar results were obtained at a total of 14 sites at which several microstimulation frequencies were applied to evoke eye movements with a range of amplitudes. For the data from each site, we applied the method described above for locating the transition point between pursuit- and saccadelike effects. A summary of these results is shown by the histograms in Fig. 11. During fixation, the horizontal component of the evoked horizontal eye movements almost always qualified as saccadelike, as indicated

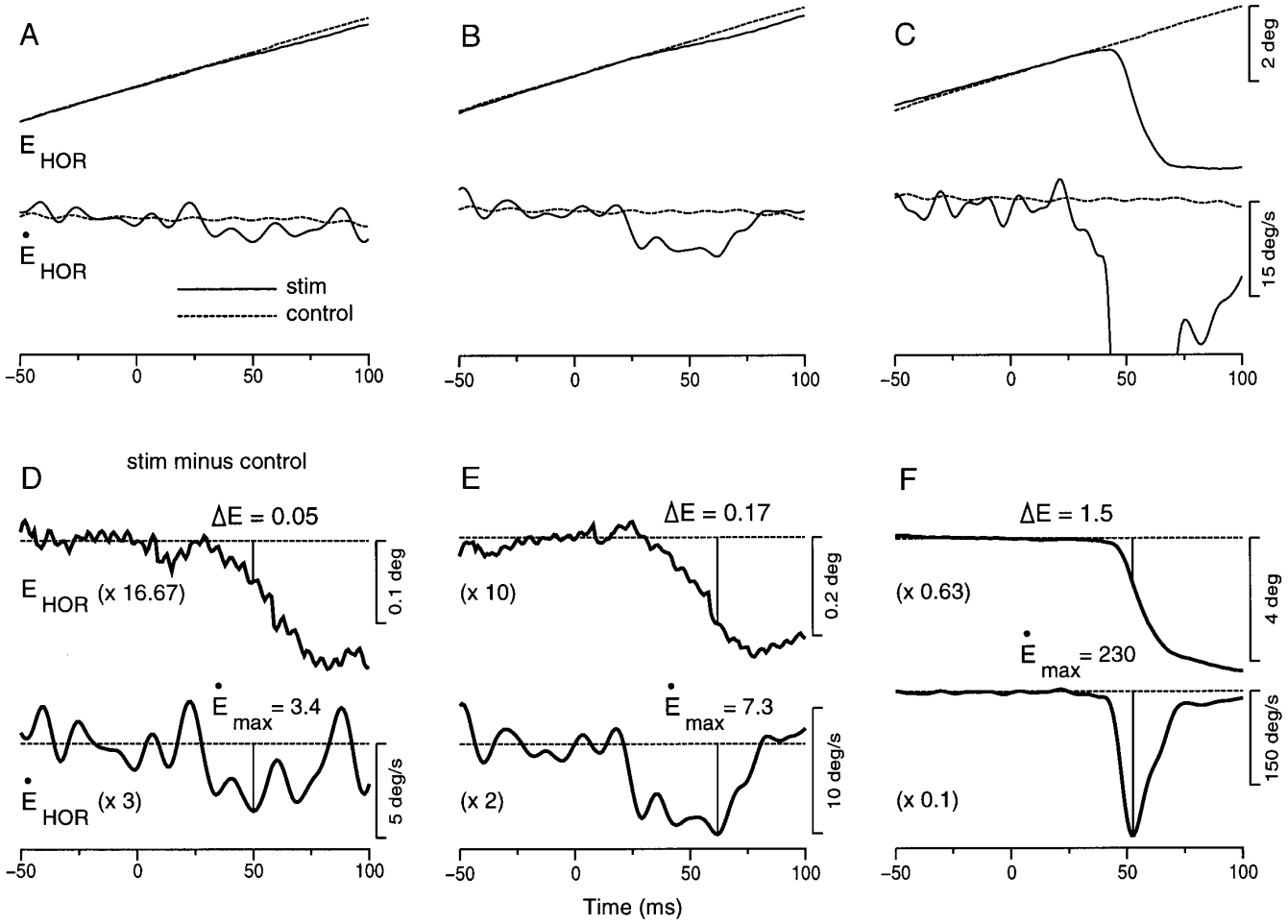

FIG. 8. Measuring eye movements evoked by microstimulation on single trials. *A–C*, *top traces*, horizontal eye position (E_{HOR}) recorded either on single trials with microstimulation (——) or average (not desaccaded) recorded on 54 control trials without microstimulation (– – –); *bottom traces*, horizontal eye velocity ($\dot{E}_{HOR}$) either on single trials with microstimulation (——) or average desaccaded eye velocity on control trials without microstimulation (– – –). All 3 panels are shown with the same vertical scaling. *D–F*, ——, differences between the pairs of superimposed traces in *A–C*; – – –, 0. Vertical scaling is increased in *D* and *E* and decreased in *F* compared with scaling in *A–C* by factor indicated in parentheses. Vertical bars and numbers in *D* and *E* indicate measurements of peak eye velocity and change in eye position derived from these traces.

by the small amplitudes at which the transitions occurred (mean 0.13°). In contrast, during contraversive and ipsiversive pursuit, the transitions occurred at larger amplitudes (means 0.43 and 0.28°, respectively). These data indicate that the type of eye movement evoked by microstimulation as well as its amplitude depended on the current eye movement behavior.

DISCUSSION

Our results demonstrate that microstimulation of the oculomotor vermis can modify smooth pursuit as well as saccadic eye movements. Previous studies have examined the effects of electrical microstimulation within this region but have done so only during either fixation (Fujikado and Noda 1987; McElligott and Keller 1984; Noda and Fujikado 1987a,b) or the execution of visually guided saccades (Keller et al. 1983). Consistent with these previous studies, we found that microstimulation applied under these conditions produces predominantly saccadelike effects. However, we also found that, if microstimulation is applied during ongoing pursuit, it can modify smooth eye velocity. These findings support the suggestion that this structure may contribute to both pursuit and saccadic eye movements. We first discuss the possible mechanisms for these effects before considering the functional circuits that include this structure.

Possible mechanisms underlying the microstimulation effects

Because our electrodes were always located in either the cerebellar cortex or the immediately underlying white matter, the effects we observed were caused by either the orthodromic activation of Purkinje cell axons or the antidromic activation of mossy fiber inputs. Two aspects of our data argue that the effects were due predominantly to orthodromic activation of Purkinje cells. First, the direction of the evoked effects is consistent with the activation of Purkinje cells. Injections of muscimol into the caudal fastigial nucleus, the

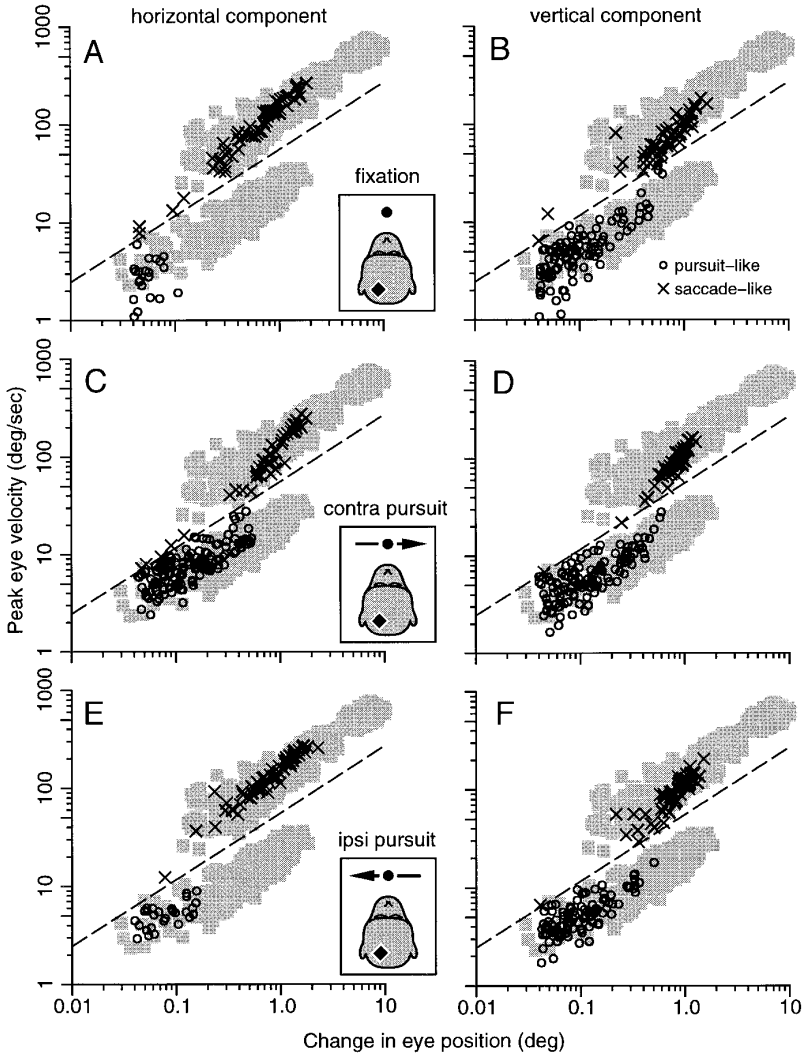

FIG. 9. Classifying eye movements evoked by microstimulation as pursuit or saccadelike. Panels show eye movements evoked by microstimulation (30 μA) at a range of frequencies (100–500 Hz) during fixation (*A* and *B*), contraversive pursuit (*C* and *D*), and ipsiversive pursuit (*E* and *F*) from one site in *monkey 2*. Each datum point shows measurements taken from a single evoked eye movement and plots the peak change in eye velocity against the change in eye position. The dashed line indicates the same discriminant function shown in Fig. 7. Eye movements whose measurements lie above this line were classified as saccadelike (crosses); those lying below were classified as pursuitlike (circles). Shaded regions in each graph reproduce the individual datum points obtained from the visually guided pursuit and saccades shown in Fig. 7.

target of vermal Purkinje cells, results in saccadic eye movements that are hypermetric toward the ipsilateral side and hypometric toward the contralateral side (Robinson et al. 1993) and produces a similar pattern of effects on eye acceleration during pursuit (Robinson et al. 1997). The ipsiversive eye movements we evoked were therefore consistent with a transient inhibition of the caudal fastigial nucleus. Because Purkinje cells in the oculomotor vermis have an inhibitory effect on target neurons in the caudal fastigial nucleus, the effects we observed with microstimulation were likely mediated, at least in part, by activation of Purkinje cell axons. Second, Noda and Fujikado (1987a) showed that the ability to elicit saccadic eye movements with low currents applied in the white matter underlying the vermis was eliminated by injecting kainic acid into the cortex, demonstrating that the evoked eye movements required intact Purkinje cells. Because the location and parameters of microstimulation in our experiments essentially duplicate those of Noda and Fujikado (1987a), it seems likely that the effects we observed also required Purkinje cells. Nonetheless, we cannot rule out the possibility that some of the effects we observed were caused by antidromic activation of inputs to the oculomotor vermis.

Involvement of the oculomotor vermis in saccades and pursuit

The importance of this region for the control of saccades in monkeys was initially established by the observations that ablation of this structure altered the metrics of saccades (Aschoff and Cohen 1971; Optican and Robinson 1980; Ritchie 1976) and that electrical microstimulation elicited saccadic eye movements (Ron and Robinson 1973). It was subsequently shown that, although saccades can be evoked or modified by microstimulation within folia V to VII (Keller et al. 1983; McElligott and Keller 1984), saccades can be evoked with low currents ($<$10 μA) only within a more restricted region referred to as the ''oculomotor vermis,'' consisting of lobule VII and the posterior one-half of lobule VI (Fujikado and Noda 1987; Noda and Fujikado 1987a,b). The mossy fibers and Purkinje cells within these folia both exhibit bursts correlated with the onset and duration of saccadic eye movements (Kase et al. 1980; Sato and Noda 1992a).

Several studies have also provided evidence that the oculomotor vermis may contribute to pursuit eye movements. During sinusoidal pursuit, the firing rate of many Purkinje

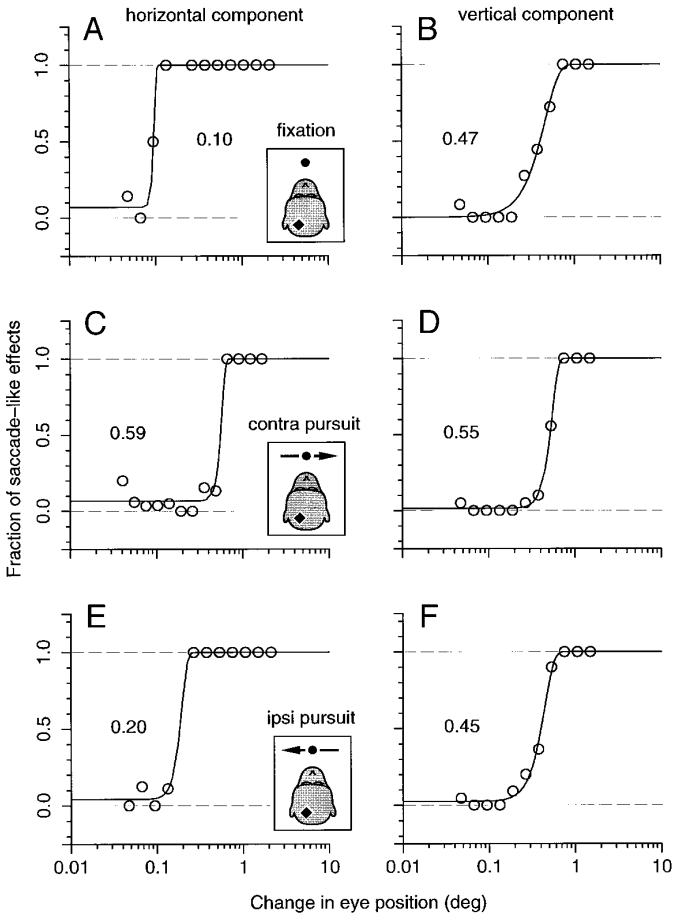

FIG. 10. Estimating the transition points between pursuit and saccadelike evoked eye movements. Panels show frequency plots of eye movements classified as saccadelike during fixation (*A* and *B*), contraversive pursuit (*C* and *D*), and ipsiversive pursuit (*E* and *F*). Each datum point shows the fraction of recorded eye movements at each amplitude that were classified as saccadelike from the data in the corresponding panel in Fig. 9. The numbers in each panel indicate the change in eye position at which the fitted function reached a value of 0.82, defined as the transition point from pursuit- to saccadelike effects.

cells in the vermis is modulated approximately in phase with eye velocity (Kase et al. 1979; Suzuki and Keller 1988b; Suzuki et al. 1981). Because these cells often also respond to retinal image movement and to head movement, it was suggested that their activity encodes a neural correlate of target velocity that could act as a guide for pursuit eye movements (Kase et al. 1979; Suzuki and Keller 1982, 1988a,b; Suzuki et al. 1981). Some support for a vermal contribution to pursuit was also provided by ablation studies. Lesions of the ventral paraflocculus, the primary cerebellar region involved in pursuit, produce severe but incomplete deficits in smooth eye movements (Zee et al. 1981). In contrast, complete cerebellectomy produces a more nearly complete deficit (Westheimer and Blair 1974), indicating that other cerebellar regions such as the oculomotor vermis also contribute to pursuit. However, only brief reports were made on the effects of vermal lesions on pursuit eye movements (Keller 1988; Suzuki and Keller 1983; Takagi et al. 1996). Furthermore, although previous studies often demonstrated the effects of microstimulation on saccadic eye movements, they have not documented effects on smooth eye movements.

Our results provide evidence for a causal role of the oculomotor vermis in the control of pursuit by demonstrating that microstimulation in this structure can modify pursuit eye movements. The changes in eye movements occurred within 10–15 ms, indicating a direct link between activity in the vermis and the output pathways for pursuit. This class of effect had not been observed in previous studies because microstimulation was applied during fixation rather than during ongoing pursuit. In addition, previous studies (e.g., Keller et al. 1983; McElligott and Keller 1984; Ron and Robinson 1973) typically used currents that were much larger ($\leq$300 μA) than those used in our current study (2–50 μA). In contrast, using smaller currents ($<$10 μA) applied during fixation, Noda and Fujikado (1987b) evoked saccadic eye movements with small amplitudes in the same range that we found ($<5°$). The facilitation of evoked smooth eye movements during ongoing pursuit that we observed in the oculomotor vermis has also been found at other sites in the pathways for pursuit. For example, microstimulation within the foveal portion of the middle temporal area (area MT) or the lateral portion of the medial superior temporal area (area MST) can cause changes in pursuit eye speed during ongoing pursuit, but microstimulation during fixation is ineffective (Komatsu and Wurtz 1989). A similar conditional effect has been observed for microstimulation applied in the dorsolateral pontine nuclei (May et al. 1985), a region to which areas MT and MST both project (Glickstein et al. 1980) and which in turn provides inputs to the oculomotor vermis (Brodal 1979; Yamada and Noda 1987).

The involvement of the oculomotor vermis in the control of both saccades and pursuit raises an important question. How does this region contribute to these two very different eye movements? One possibility is that saccades and pursuit involve different portions of the oculomotor vermis. The Purkinje cells that discharge during saccades are found almost exclusively within lobule VII and the posterior folia of lobule VI, whereas Purkinje cells that discharge during pursuit are found not only in the oculomotor vermis but also in lobule VIII (Sato and Noda 1992a). In addition, Purkinje cells sensitive to head rotation are found primarily in lobule VIII but not in the oculomotor vermis (Sato and Noda 1992a). If one defines pursuit-related neurons as those sensitive to both eye and head motion, a combination that holds true in the ventral paraflocculus (Lisberger and Fuchs 1978; Miles et al. 1980), then one can argue that pursuit involves lobule VIII but not the oculomotor vermis (Sato and Noda 1992a). However, if one defines pursuit-related neurons simply as those neurons that are modulated during pursuit eye movements, the majority of both pursuit- and saccade-related neurons lie within the oculomotor vermis. In fact, of the relatively few of these neurons that were tested during both saccades pursuit, approximately one-half are responsive to both types of eye movements (Sato and Noda 1992a; Suzuki and Keller 1988b).

Consistent with the results from these single-unit studies, we found that microstimulation at sites within the oculomotor vermis could produce either pursuit or saccades. In particular, microstimulation with one set of parameters often evoked pursuitlike eye movements when applied during con-

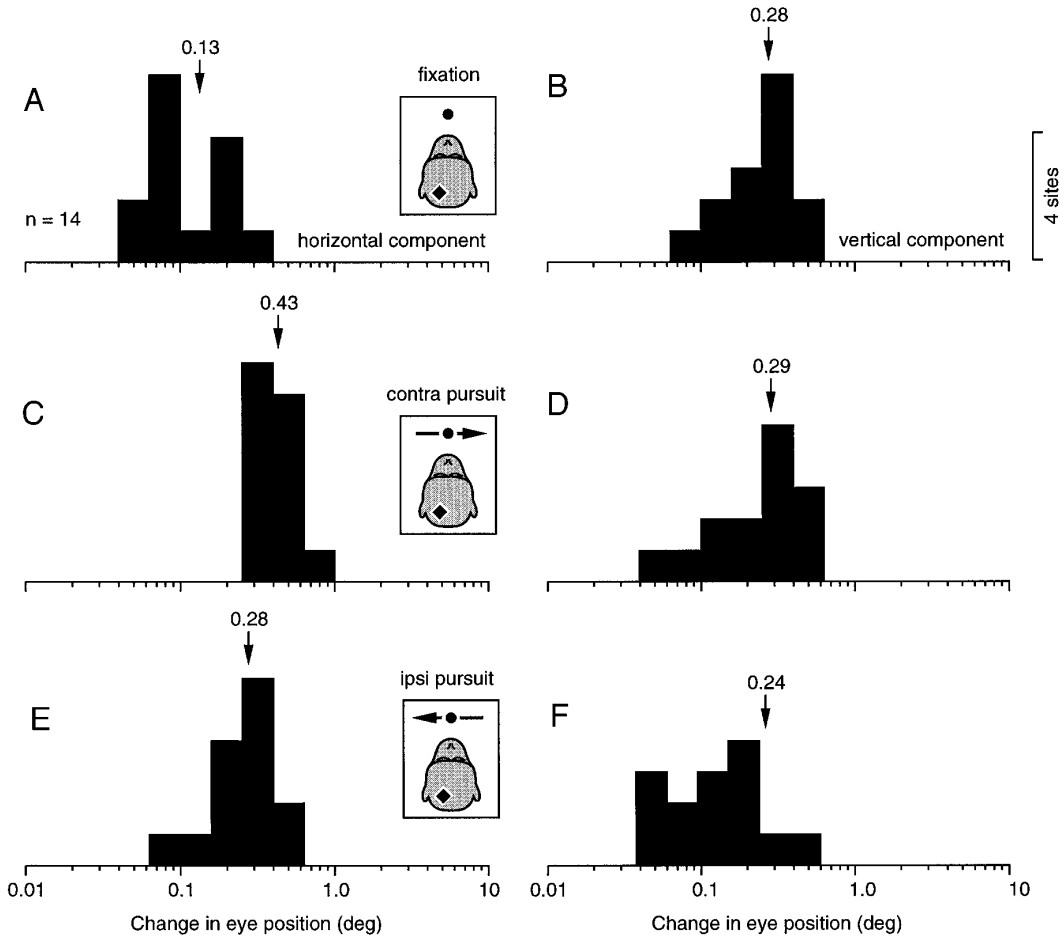

FIG. 11. Summary of transition points from 14 stimulation sites in both monkeys at which multiple frequencies were applied. Panels show distributions of transition points obtained during fixation (*A* and *B*), contraversive pursuit (*C* and *D*), and ipsiversive pursuit (*E* and *F*). Numbers in each panel indicate the means of the distributions.

traversive pursuit but evoked saccadelike eye movements when applied during ipsiversive pursuit. This suggests that recruitment of a single set of fibers could influence both saccades and pursuit, as would be true if pursuit- and saccade-related functions were colocalized within the oculomotor vermis. However, the final determination of whether and how single Purkinje cells contribute to both saccades and pursuit can only be achieved by additional experiments. In particular, it would be informative to study the responses of Purkinje cells during eye movements of different amplitudes and to examine their pursuit responses with ramp target motions that have been useful in dissociating the different phases of Purkinje cell activity in the ventral paraflocculus (Krauzlis and Lisberger 1994; Stone and Lisberger 1990).

The prospect of functional overlap between the pursuit and saccadic systems within this region of the cerebellum is also supported by the properties of the fastigial oculomotor region (FOR), which receives an exclusive projection from the oculomotor vermis (Yamada and Noda 1987). Disruption of activity in the FOR has been shown to cause both dysmetric saccades (Robinson et al. 1993; Sato and Noda 1992b; Vilis and Hore 1981) and deficits in smooth eye movements (Kurzan et al. 1993; Robinson et al. 1997). Accordingly, the firing rate of neurons in the FOR is modulated during both saccades (Fuchs et al. 1993; Helmchen et al. 1994; Ohtsuka and Noda 1991) and smooth eye movements (Büttner et al. 1991; Fuchs et al. 1994). In the one study that tested both eye movements, 29% of the pursuit-related neurons also exhibited a burst during saccades (Fuchs et al. 1994). This degree of overlap between pursuit- and saccade-related activity is not substantially different from that observed on Purkinje cells in the oculomotor vermis (Sato and Noda 1992a; Suzuki and Keller 1988b).

Possible mechanisms for the vermal contribution to saccades and pursuit

The known anatomy provides multiple sites at which the oculomotor vermis might affect the motor commands for saccades and pursuit. For saccades, the output from the oculomotor vermis could directly change the firing rate of burst neurons responsible for saccades because the caudal fastigial nucleus projects to both the paramedian pontine reticular formation (PPRF) and the rostral interstitial nucleus of the medial longitudinal fasciculus (Noda et al. 1990). Alternatively, the caudal fastigial nucleus could indirectly affect the burst by acting on the pause neurons that are also located in the PPRF. An additional possibility is provided by the projection from the caudal fastigial nucleus to the intermediate layers of the superior colliculus (May et al. 1990). For

pursuit, the most direct path might seem to be the projection from the caudal fastigial nucleus to the vestibular nuclei (Noda et al. 1990), but this projection does not include the medial or superior divisions that contain neurons modulated during pursuit. A less direct path could be provided by projections to the nucleus reticularis tegmenti pontis (NRTP), which recent data suggests may also be involved in pursuit (Suzuki et al. 1990; Yamada et al. 1996). Interestingly, the anterograde pattern of termination in the NRTP after caudal fastigial injections (Fig. 2 of Noda et al. 1990) is similar to the retrograde pattern seen after injections of the ventral paraflocculus (Fig. 13 of Langer et al. 1985), suggesting that this nucleus might act as a relay between these two cerebellar regions. The projection to the PPRF is also a possibility because, in addition to burst and pause neurons, this region also contains eye position-related neurons (Keller 1974; Luschei and Fuchs 1972), and microstimulation in the vicinity of these neurons evokes smooth eye movements (Cohen and Komatsuzaki 1972; Keller 1974). Finally, the recent observation that eye movement-related neurons in the rostral superior colliculus are modulated during pursuit eye movements as well as during fixation and small saccades (Krauzlis et al. 1997) suggests that the fastigial projection to the rostral superior colliculus (May et al. 1990) might also affect pursuit.

Does the contribution of the oculomotor vermis to saccades pursuit represent two separate functions or a single function? Several observations lead us to favor the latter possibility. First, as described above, the pursuit- and saccade-related neurons are located within the same portions of the oculomotor vermis and caudal fastigial nucleus, and many individual neurons respond during both types of eye movements. Second, the bursts exhibited by these neurons show the same directional preferences and occur during comparable phases for the two types of eye movements. For example, it was suggested that neurons in the caudal fastigial help to accelerate contraversive eye movements and decelerate ipsiversive eye movements, for both saccades (Fuchs et al. 1993) and pursuit (Fuchs et al. 1994). Finally, the results we obtained with microstimulation indicate that altering the output of the oculomotor vermis can produce both pursuit and saccadelike eye movements. The abrupt, but orderly, transition between these two types of effects (Fig. 9) suggests the presence of some coordinating influence.

These observations lead us to propose a simple model. This model (Fig. 12) is not intended to provide a complete description of the processing underlying pursuit and saccades (e.g., it does not include visual motion inputs for pursuit) but illustrates one simple mechanism that can account for the major features of our results. The basic assumption of the model is that a single feedback loop (Fig. 12*A*, *left side*) modulates a position error signal that contributes to both saccades and pursuit. The model incorporates two critical features. First, the caudal fastigial nucleus (FN) is placed within a feedback loop that calibrates the estimate of motor error (ME) by subtracting the current estimate from the value of position error (PE). The oculomotor vermis changes the gain of this feedback loop by decreasing the output of the FN. Second, eye movements can be produced by either *1*) directly applying the motor error as an input to the final common pathway, consisting of the neural integrator (NI) and a simple model of the plant, or *2*) using motor error to trigger a burst generator circuit, which then provides an additional input to the final common pathway.

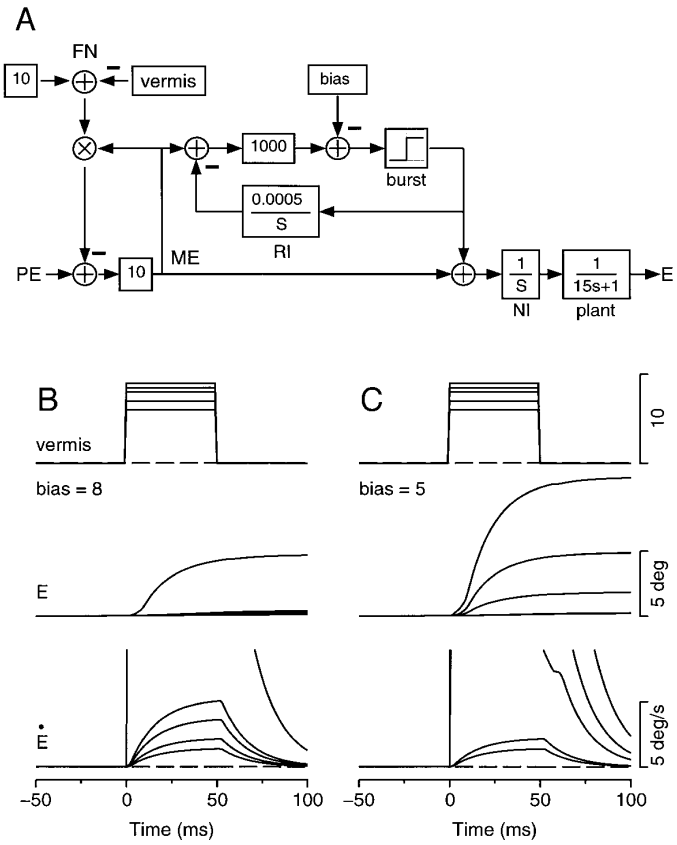

FIG. 12. Model that can account for our pattern of results. *A*: diagram of the model. Numbers in boxes are gains; other boxes contain transfer functions expressed in Laplace notation. Output of box labeled burst is 10 if input is >0; otherwise, output is 0. Changes in value of vermis increment or decrement the output of the fastigial nucleus (FN), which is then multiplied by the value of motor error (ME) in a negative feedback loop. Position error (PE) was set equal to 0.01° in all simulations. Simulations were run on a millisecond timescale, so that the effective time constant of the ''plant'' was 15 ms. *B*: simulations of the model that produce primarily pursuitlike effects. *Top traces*: output of vermis increased from 0 to 6, 7, 8, 8.5, or 9 for 50 ms and then returned to 0. Bias was set to a constant value of 8. *Middle traces*: Small deflections in eye position (E) result from all but the largest change in vermis. *Bottom traces*: small pursuitlike evoked eye movements are seen more clearly in traces of simulated eye velocity. High velocities of the largest evoked eye movement are clipped. *C*: simulations of the model that produced primarily saccadelike effects. Bias was set to a constant value of 5. Other conventions same as in *A*.

Because the ''vermis'' decreases the gain of the feedback loop that calibrates motor error, increases in the output of the vermis amplify the motor error produced by a given position error. If motor error does not exceed some critical value, such increases in the output of the vermis produce deflections in smooth eye velocity. For example, in Fig. 12*B*, pulsing vermis briefly from 0 to 6, 7, 8, or 8.5 produces a graded series of pursuitlike deflections in smooth eye velocity. However, if motor error becomes large enough (e.g., when vermis is pulsed from 0 to 9), a saccadelike eye movement is evoked. These simulations mimic our data with microstimulation applied during contraversive pursuit, which exhibited the largest changes in smooth eye velocity. The

transition from pursuit- to saccadelike effects is determined by the value of ''bias,'' which sets the threshold for triggering the burst generator circuit. When this threshold is lowered by decreasing the value of bias from 8 to 5, saccadelike eye movements are now produced by pulsing vermis from 0 to 8 and 8.5 as well as to 9 (Fig. 12*C*). These simulations mimic our data with microstimulation applied during ipsiversive pursuit and during fixation, which exhibited smaller changes in smooth eye velocity and more frequent saccades. Finally, in simulations not shown in Fig. 12, we allowed the value of the bias to change over time (e.g., change from 8 to 5 over the 50-ms interval of vermal stimulation). This manipulation allowed the model to reproduce those instances when stimulation evoked a smooth change in eye velocity followed by a saccade on the same trial.

The simplicity of this scheme ensures that it will have shortcomings not seen in more sophisticated models of the vermis (e.g., Dean 1995). For example, we do not assign anatomic labels to all parts of the model. In part, this is because little is known about the brain stem pathways for pursuit eye movements. The bias and burst nonlinearity are a simplified version of the omnipause and burst neuron circuit identified in models of saccade generation (e.g., Van Gisbergen et al. 1981), but because of these simplifications we have not identified these parts of the model as specific anatomic structures. However, the bias in the model could be related to omnipause neurons because both increases in the bias of the model and stimulation of the omnipause region prevent the occurrence of saccades (Westheimer and Blair 1973). Because our simulations required changes in the value of the bias signal to reproduce the eye movements evoked during ipsiversive and contraversive pursuit, this predicts that the activity of omnipause neurons should be altered by smooth eye velocity.

Another question raised by the model is the use of an internal feedback loop for pursuit. One well-known model invokes an internal feedback loop for pursuit (Robinson et al. 1986), which has the advantage of providing a possible mechanism for the demonstrated plasticity in the pursuit system (Kahlon and Lisberger 1996; Optican et al. 1985). Other studies indicate that this particular model cannot accurately account for several critical features of pursuit (Goldreich et al. 1992), but this does not necessarily invalidate all models containing internal feedback loops (Ringach 1995). An additional consideration is that the signals in the feedback loop of Robinson et al. (1986) are velocities, whereas the signals in the feedback loop for saccades have been traditionally viewed as positions (Robinson 1975; Van Gisbergen et al. 1981). The discrepancy between position and velocity domains could be resolved if the sensory inputs to the premotor circuits were converted into equivalent prospective motor commands rather than handled in their native format as so-called ''retinal position'' or ''visual motion'' errors. For example, the conversion of inputs for pursuit and saccades into a common ''eye velocity'' motor format could explain how a single population of neurons, such as that recorded in the caudal fastigial nucleus (Fuchs et al. 1994), might accelerate or decelerate both saccades and pursuit.

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