Modulation of Pursuit Eye Movements by Stimulation of Cortical Areas MT and MST

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SUMMARY AND CONCLUSIONS

1. Many cells in the superior temporal sulcus (STS) of the monkey that represent the foveal region of the visual field discharge during pursuit eye movements. Damage to these areas produces a deficit in the maintenance of pursuit eye movements when the target moves toward the side of the brain with the lesion. In the present experiments, we electrically stimulated these areas to better localize and understand the mechanisms underlying this directional pursuit deficit.

2. Monkeys were trained to pursue a moving target using a step-ramp task in which the target first stepped to an eccentric position and then moved smoothly across the screen. Trains of stimulation were applied after the monkey had begun to pursue the target to study stimulation effects on maintenance of pursuit.

3. Stimulation during pursuit frequently produced eye acceleration toward the side of the brain stimulated. Eye speed increased during pursuit toward the side stimulated and decreased during pursuit away from the side stimulated. This increase in velocity toward the side of the brain where stimulation presumably activated cells is consistent with the decrease in pursuit velocity toward the side of the brain after cells were removed by chemical lesions.

4. The increase or decrease in pursuit speed following stimulation produced a slip of the target on the retina. The pursuit system seemed to be insensitive to this slip during the period of stimulation, however, since the effect of stimulation during pursuit of a stabilized image (open-loop condition) was similar to that resulting from stimulation under normal pursuit conditions (closed-loop). This insensitivity to visual motion during stimulation suggests that the stimulation substitutes for that visual input.

5. The separation of eye and target position that resulted from stimulation did produce catch-up saccades. This provides added evidence that alteration of middle temporal area (MT) and medial superior temporal area (MST) modifies visual-motion but not visual-position information.

6. Stimulation that produced eye acceleration during pursuit produced only a slight effect during fixation of a stationary target. The effectiveness of the stimulation also increased as the speed of the pursuit increased between 5 and 25%. These observations, which show that pursuit velocity altered the effect of stimulation, suggest that the stimulation acted on visual motion processing before information about the pursuit movement itself is incorpo-

rated. Since this stimulation produces directional pursuit effects, we hypothesize that the directional bias for pursuit originates in the visual signal conveyed to the pursuit system.

7. Stimulation during pursuit with a vertical component reduced eye speed for either upward or downward pursuit, and usually also produced a slight acceleration in the horizontal component toward the side of the stimulation. This decrease in vertical eye speed (along with other observations) is consistent with the hypothesis that the effect of stimulation on pursuit results from two factors: a decrease in pursuit velocity in all directions and an increase in pursuit velocity toward the side of the brain stimulated.

8. Sites within the gray matter of MT and MST, where stimulation most frequently produced acceleration of pursuit, were concentrated in the foveal region of MT (MTf) and in the lateral-anterior region of MST (MSTL). This clarifies the conclusions derived from chemical lesion studies that MST is involved in the maintenance of pursuit, that MTf is also, that the dorsal-medial region of MST (MSTD) is not, and that the floor of the STS (FS) is probably not.

9. In some experiments, we applied stimulation before a saccade had been made to the target in the step-ramp task. This led to an increase in saccadic latency of over 100 ms. This increased latency resulted from stimulation both at points that did and points that did not modify pursuit which suggests that the underlying mechanism of the two effects is different.

10. Stimulation before the onset of pursuit in the step-ramp task also led to a slowing of the initial pursuit speed after the saccade to the target and a failure of the amplitude of the saccade to compensate for target motion. This stimulation effect was observed at sites representing the extrafoveal visual field in MT, but only when the target was moving in the contralateral visual field represented by the visual receptive fields of cells at the recording site. This stimulation effect mimicked the deficit following chemical lesions within extrafoveal MT.

INTRODUCTION

Both single-cell recording experiments and chemical lesion experiments have led to the conclusion that restricted regions of the superior temporal sulcus (STS) are related to the initiation and maintenance of pursuit eye movements in old world monkeys. Cells in the middle temporal area (MT) within the STS have receptive fields in the contralateral visual field and are sensitive to the motion of a visual stimulus within a given range of directions and speeds (Albright et al. 1984a; Baker et al. 1981; Peperman and Kass 1984; Maunsell and Van Essen 1983a,b; Rodman and Albright 1987; Saito et al. 1986; Tanaka et al. 1986; Zeki 1974a,b, 1980). Chemical lesion of these cells produces a deficit in a monkey's ability to initiate pursuit to a target moving in the contralateral visual field, and since the deficit is limited to the area of the retinotopic map within MT related to the damaged cells, the deficit has been referred to as a retinotopic deficit (Newsome et al. 1985).

Cells in the adjacent medial superior temporal area (MST) also show directional selectivity to moving visual stimuli (Desimone and Ungerleider 1986; Tanaka et al. 1986; Komatsu and Wurtz 1988a), and many cells receive
in addition an input related to the generation of the pursuit eye movement itself (Newcombe et al. 1988). Damage to a subregion of MST, centered on the lateral-anterior area of MST (MSTI), produces a deficit in the maintenance of pursuit in addition to the initiation of pursuit (Dursteler and Wurtz 1988). This deficit in maintenance is a directional one: pursuit speed toward the side of the brain with the lesion is reduced whereas that away from the lesion is unimpaired. A similar directional pursuit deficit results from lesions that centered on the foveal region of MT (MTI) which lies adjacent to MST (Dursteler et al. 1987).
FIG. 4. Reduced sensitivity to visual motion (retinal slip) during periods of electrical stimulation. A: comparison of the effects of stimulation during pursuit of a moving target (normal closed-loop pursuit) with the effects of stimulation during position stabilization of that target (open-loop pursuit). Electrical stimulation (150 µA, 0.4-s duration, biphasic) began at the arrow (labeled stim on) after the monkey had begun to pursue the target moving at 15°/s to the left. Eye traces show mean and standard error of horizontal velocity. Solid lines are for the trials under the normal closed-loop condition without stabilization of the target and dashed lines for trials with stabilization (which started at 100 ms before stimulation). Eye acceleration was nearly the same under both conditions; the slight difference in the eye velocity in these 2 conditions was also observed without stimulation. B: effect of superimposing a constant slip velocity in the direction opposite to that of pursuit (in the absence of electrical stimulation). Position stabilization was present throughout the record of pursuit to the left and a velocity step 27°/s to the right was added at the arrow labeled stab on. Solid lines indicate the mean and standard error for pursuit with velocity stabilization and dashed line the baseline pursuit without velocity stabilization. The eye velocity was reduced by this "slip" to the right. Time (100 ms) and velocity scale (15°/s) apply to both A and B.

In the present experiments, we electrically stimulated these areas within the STS during pursuit in an attempt to understand the mechanism underlying the directional pursuit deficit. Stimulation allows comparison of effects to be made between many discrete sites in the same hemisphere, in contrast to even a punctate chemical lesion that allows only one test and that usually damages cells over a large area. In addition, use of stimulation in conjunction with other behavioral tests offers the possibility of determining where in the sequence of visual to visual-motor processing the directional effect emerges. We were encouraged in this attempt to use stimulation in the cortex by recent studies that showed significant stimulation effects in what is presumably the next step in the pathway related to pursuit, the dorsolateral pontine nuclei (May et al. 1985). These pontine nuclei receive a prominent projection from the STS (Glickstein et al. 1980), cells in this region discharge during pursuit eye movements (Maister et al. 1986; Suzuki and Keller 1984; Thier et al. 1988), and chemical lesions produce a directional deficit (May et al. 1988).

We have found that stimulation is effective at limited sites in MT and MST, and that the effect, like that of the lesions in this area, is frequently a directional one: stimulation produces an acceleration toward the side of the brain stimulated. Other characteristics of the stimulation suggest that it acts on the visual motion processing before this information is combined with extraretinal information about the ongoing pursuit.

Brief reports of these experiments have appeared previously (Komatsu and Wurtz 1987a,b).

METHODS

Three monkeys (Maccaca mulatta) were trained to fixate or to pursue a small visual target to obtain a reward. During experiments, monkeys were seated in a primate chair that faced a tangent screen 86 cm in front of them. The head was restrained so that the eyes, when in primary position, were directed toward the center of the screen. Eye movements were recorded using the magnetic search coil technique (Robinson 1963; Fuchs and Robinson 1966). Details of these methods have been previously presented previously (Komatsu and Wurtz 1988a).

We trained the monkeys using a step-ramp or a step paradigm. A trial began with the onset of a small light at the center of the screen which the monkey had to fixate for a variable length of time. When this light went out, a second light appeared simultaneously, and the monkey had to move its eyes to this second light to receive a reward. The reward was given for detecting a dimming of the target or for keeping the eye position in a window surrounding the target. In step trials the second target usually came on 10° away from the fixation point, remained stationary, and the monkey then made a rapid (saccadic) eye movement to the target. In step-ramp trials, the second target came on 10° from the fixation point and moved at 15°/s (unless otherwise indicated) toward the fixation. This step size was adjusted depending on target speed. Target motion was usually in the horizontal direction, but vertical and oblique directions were also used.

We tested the effects of electrical stimulation applied to areas MT, MST, and surrounding areas on eye movements in five hemispheres of three monkeys (1 hemisphere in me, 2 in ge and te). We identified areas MT and MST physiologically by the depth profile of recordings from gray matter and white matter along the penetration and by the visual and pursuit related properties of the cells. The recording procedures and criteria for identification of these areas have been described (Komatsu and Wurtz 1988a). For the stimulation, electrical pulse trains were generated by a stimulator (Berl 220B) and were applied through the same electrode as that used for neuronal recording. Stimulation frequency was always 500 Hz. Pulses were biphasic with a duration of 0.2 ms for each of the pulses, or monophasic negative going with a duration of 0.2 ms. Stimulus train duration ranged between 0.2 and 0.4 s. Stimulation current was varied from 25 to 200 µA (usually 100 µA) and was monitored as the voltage drop across a resistance (300 Ω) inserted in series between the stimulator and electrode.

In two hemispheres (me left (L), ge right (R)), the effect of
FIG. 5. Comparison of stimulation during pursuit of a moving target (A) and fixation of a stationary spot (B). The top traces show eye positions (---) for six different trials and representative target position (---). The target moved at 15°/s to the right (A) or was 10° to the right of primary position during fixation (B). Upper position traces are without stimulation; lower traces are with stimulation. The vertical line indicates onset of stimulation. Parameters of stimulation were the same as in Fig. 1. Bottom traces show mean and standard error of eye velocity. Solid lines are for the trials with stimulation and broken lines for trials without stimulation. In both eye position and velocity traces, upward deflection represents rightward movement.

FIG. 6. Comparison of the magnitude of the directional stimulation effect during pursuit and fixation. Upper three bars show the effect of stimulation during several fixation conditions (no-pursuit): during fixation of a stationary spot (fixation), during blink of the stationary spot during fixation (blink), and during the fixations interspersed with spontaneous eye movements in intertrial intervals (ITI). To minimize the contamination of spontaneous saccades and eye drift, stimulation was started 100 ms after the occurrence of the first saccade during ITI. Lower three bars show effect of stimulation during horizontal pursuit (H-pursuit) at the target speeds indicated. Change in horizontal eye speed (with leftward positive) is the mean speed 100–200 ms after stimulation for ITI or the difference between this value and the mean of that during the same period without stimulation for the other conditions. For fixation and blink data, we averaged the effect during fixation at points 10° to the right and to the left of the center of the screen since we observed no difference in the effect of stimulation at different eye positions. The means for the pursuit conditions includes stimulation effects during both rightward and leftward pursuit. Means include at least 5 trials under each condition—stimulation, no stimulation, left or right. Same stimulation site and parameters as in Fig. 5.

stimulation was tested throughout area MT, MST, and surrounding areas by making electrode penetrations in a regular grid pattern. In a given penetration, after the characteristics of the cells were recorded, stimulation was applied at intervals of 400 or 500 μm. In the other three hemispheres (1 in ge, 2 in te), we concentrated the penetrations in certain areas of MT and MST by using guide tubes directed toward these areas. In one of these hemispheres (tel), stimulation effects were tested in smaller steps (200–250 μm) along some penetrations, and these penetrations were marked by small electrolytic lesions (10 μA for 60 s). These penetrations were then reconstructed based on the sequence of neurons recorded and the marks made.

Stimulation applied during pursuit usually started 400 ms after the initial saccade to the moving target and lasted for 400 ms. Stimulation applied before onset of pursuit was given 100 ms after the start of target motion and lasted 100–150 ms. In the step trials, the timing of stimulation was the same as above except that the target was stationary after the initial step. The direction of the target motion and type of trial (step or step-ramp) were randomized from trial to trial, and trials without stimulation were also randomly intermixed as controls.

In one set of experiments, we measured the effect of stimulation during pursuit of a target that was stabilized on the retina. This position stabilization was achieved by using the monkey’s eye position instead of the usual stimulus ramp to drive the mirror galvanometers. We allowed the monkey to initiate pursuit under normal conditions, but once it was pursuing the target, we stabilized the image on the fovea. With our system the latency for mirror movement following eye movement was at most 15 ms. In another set of experiments, we measured pursuit of a target whose velocity was stabilized on the retina. This velocity stabilization was achieved by first stabilizing the position of the target on the retina and then adding a fixed velocity of target motion. The general procedures used in the stabilization experiments have been described previously (Dursteler et al. 1987). We sampled and stored horizontal and vertical eye position and mirror position on each trial for a period beginning 150 ms before target onset and lasting throughout the trial. The horizontal and
FIG. 7. Influence of horizontal eye velocity on the stimulation effect. The ordinate shows the difference in the mean eye velocity during the period 100–200 ms after stimulation onset and that during the equivalent period without stimulation. Positive on the ordinate indicates increased eye velocity toward the stimulated hemisphere. The abscissa shows eye velocity during horizontal pursuit without stimulation for rightward (positive number) and leftward pursuit (negative number) with target motion at 5, 15, or 25°/s. In A, the effects obtained during pursuit toward and away from the stimulated hemisphere are shown separately. The equation for the linear regression line on the left is $y = -0.24x + 0.12$ ($r = -0.81$) where $x$ is the horizontal eye velocity and $y$ is the horizontal eye velocity change. The equation for the linear regression line on the right is $y = -0.032x + 1.82$ ($r = -0.12$). The linear regression lines were derived using the method of least squares. In B, the effect for the 2 directions of horizontal pursuit were averaged together. For the linear regression line, $y = 0.11x + 1.00$ ($r = 0.61$). The results obtained from 9 different stimulation sites are shown using different symbols. Stimulation parameters for the data shown by circles with lines are the same as in Fig. 1. Those for the remaining 8 sites are the same as in Fig. 4.

Vertical eye position signals were low pass filtered at 250 Hz (−3 dB) and then digitized at 500 Hz. The entire system had a resolution of 0.1°. The storage and display of data as well as the behavioral tests were controlled by a real-time experimental system (REX) developed by Hazs, Richmond, and Optician (Hays et al., 1982), which was run on a PDP 11/73 computer.

For quantitative analysis of the eye movements, we used an off-line program that digitally filtered (13.6 Hz, −3 dB) the stored position information and computed eye velocity and acceleration of the eye, as described previously (Dursteler et al., 1987). The experimenter then specified certain velocity and acceleration criteria by which the program automatically identified the beginning and the end of saccadic eye movements in each trial in the data file. These criteria were initially selected so that the identification of saccadic and pursuit eye movements looked reasonable to the experimenter; once selected the same criteria were used for analysis of all data. This method allowed us to identify the beginning and end of the saccade made to acquire the moving visual target and to remove saccades before averaging the velocity traces. We used an acceleration criteria to identify saccades. The beginning and end of the saccade was identified as the point where eye acceleration exceeded or dropped below 80% of the maximum, respectively.

At the end of the experiments, monkeys were deeply anesthetized with pentobarbital sodium and were perfused with saline followed by 10% Formalin. Paraffin sections were made of the posterior one-half of the brain, and these were stained for either cell bodies (creal violet) or fibers (Gallyas 1979). MT was identified by the dense myelination on the posterior bank of the STS. In four hemispheres we identified individual electrode tracks by their location relative to other tracks and from the recorded depth profile and cell characteristics of the penetration. In one hemisphere (tel), each stimulation site was identified on the histological sections using the electrolytic marking points as well as the points listed above.

RESULTS

Smooth pursuit eye movements can be modified by electrical stimulation within the regions of MT and lateral-anterior MST that represent the fovea. We will first describe the effects of stimulation during pursuit, then describe the anatomic location of points producing the effects, and finally show the effect of stimulation before pursuit begins.

Directional stimulation effects during pursuit

STIMULATION DURING HORIZONTAL PURSUIT. Figure 1 shows an example of the effects we observed during pursuit following stimulation in the lateral-anterior area of MST. In the step-ramp paradigm that we used, the monkey made a saccade to the target and was pursuing it before we applied stimulation. In normal pursuit, eye velocity matched target velocity as can be seen in the position records (Fig. 1, A and C) and in the velocity records (Fig. 1, B and D) for no stimulation trials. When we applied stimulation in the left hemisphere during rightward pursuit (between the vertical tick marks, Fig. 1, A and D), the eye started to lag behind the target and eye velocity decreased sharply to ~7°/s. Eye velocity remained low during the period of stimulation, but the resulting position error was corrected by a catch-up saccade (between the stimulation tick marks). When we applied stimulation during leftward pursuit (Fig. 1, C and D), eye velocity increased sharply up to 21°/s. This increase moved the eye ahead of the target and a saccade returned the eye nearer to the target, but increased velocity was not maintained throughout the stimulation period. Although this pursuit was over a light background, stimulation during pursuit against a dark background produced similar effects.

We measured the latency between onset of stimulation and change in eye velocity by using records filtered with a higher cutoff frequency (34 Hz, −3 dB) than that used for the traces shown in Fig. 1. For this purpose, we took the start of the stimulation effect to be the point at which eye velocity exceeded the mean eye velocity in the 100 ms before stimulation by two standard deviations. This latency was 20–25 ms.
Figure 2 shows quantitatively these effects of stimulation during pursuit to the right (top) and to the left (bottom). The graph shows means and standard errors of eye velocity averaged over 100-ms periods before and after stimulation onset (0 on the abscissa). For pursuit in both directions, eye velocity during stimulation was clearly different from that without stimulation in the 0-100- and 100-200-ms periods after pursuit started.

The effect was consistent from trial to trial and illustrates the relation between the side of the brain stimulated and the change in velocity seen during pursuit. In this experiment, stimulation of the lateral-anterior MST in the left hemisphere during pursuit to the left resulted in an increase in eye velocity, effectively a leftward acceleration. Stimulation during pursuit to the right produced a decrease in pursuit velocity which is also an acceleration to the left. Therefore, for both directions of pursuit, stimulation in the left hemisphere resulted in an acceleration to the left, toward the hemisphere being stimulated. We observed a similar acceleration in all five hemispheres studied, and we will refer to this as a directional stimulation effect.

Although the direction of the effect with respect to the stimulated hemisphere was the same for all cases, the time course and magnitude of the effects were not the same. In the experiment shown in Figs. 1 and 2, the effects were more transient for pursuit away from the side of the stimulated hemisphere. In other hemispheres, the time course was the same for both directions of pursuit, but the relative magnitude of the effects was different for the two directions of pursuit. In all hemispheres, however, stimulation during pursuit away from the side of the stimulated hemisphere produced a stronger and more consistent effect than during pursuit toward the stimulated hemisphere.

Current intensity had a predictable effect on eye speed as is illustrated in Fig. 3. At the five stimulation points shown, we increased the current from 25 to 200 μA, while keeping other stimulation parameters constant. The magnitude of the acceleration toward the side of stimulation increased as the current intensity increased. In the two hemispheres in which we tested the effect of current strength, stimulation produced an effect on eye speed with current at ~50 μA in one hemisphere (geR), and between 25 and 50 μA in the other hemisphere (tel). The difference in threshold may be related to the polarity of the current; we used biphasic pulses for hemisphere geR and monophasic negative only pulses for hemisphere tel. Subsequent experiments were done at the higher current levels (usually 100-150 μA), and no attempt was made to study the effect of stimulation near threshold.

VISUAL FEEDBACK DURING STIMULATION. The change in eye velocity resulting from stimulation during pursuit also produced both motion of the target on the retina (a velocity error) and a difference in the position of the eye and target (a position error). If visual feedback were operating during the period of stimulation, this visual information should operate to reduce the errors in eye velocity and position.

As can be seen in Figs. 1 and 2, the velocity error often persisted throughout the period of stimulation, and this raised a question about the effectiveness of visual motion...
In contrast, Fig. 4B shows the change in pursuit velocity that we would expect to occur as a result of the error introduced by the stimulation in Fig. 4A. Here we introduced during normal leftward pursuit without electrical stimulation a 2°/s velocity error to the right under stabilized condition (open-loop). A clear reduction of eye velocity followed within ~100 ms. The difference in the effect of the velocity error in the experiments shown in Fig. 4, A and B, suggests that electrical stimulation largely prevented the normal functioning of visual motion feedback during the period of stimulation.

On the other hand, catch-up saccades occurred which corrected the position error resulting from the increased eye velocity produced by electrical stimulation. This indicates that position feedback is functioning normally during stimulation and can be dissociated from the abnormal functioning of motion feedback during such stimulation.

**COMPARISON OF STIMULATION DURING FIXATION AND PURSUIT.** While Fig. 4 shows the relative lack of sensitivity to visual input during electrical stimulation, Figs. 5 and 6 show that the state of the pursuit system does alter the effect of stimulation. Figure 5 shows the effect of stimulation in the left hemisphere both during rightward pursuit of a target and during fixation of a stationary target. Stimulation during pursuit produced an increase in leftward eye velocity (Fig. 5A) but during fixation it produced only very weak motion in the same direction, to the left (Fig. 5B). The comparison of the stimulation effect during pursuit and during fixation was done at 22 stimulation sites, and the results were consistent with those described above. We conclude that stimulation is much more effective during pursuit than during fixation of a visual target.

During fixation either the presence of the stationary fixation target or the "effort of fixation" required to keep the eye from drifting might have reduced the effect of stimulation. To test the influence of the fixation spot, we blinked off the fixation spot for several hundred milliseconds before and during stimulation. As can be seen by comparing the two top panels of Fig. 6, there was little difference in the effect of stimulation with and without the fixation spot. To test the influence of active or attentive fixation, we applied stimulation in the intertrial interval (ITI) during periods of fixation between saccades when no particular point was being fixated. Since this experiment was done in a dark room in the absence of any visual targets, no attentive fixation on a visual target could occur. In this condition virtually no change in eye speed was observed throughout the entire 400 ms of stimulation. This suggests that active fixation is a necessary condition for the stimulation to produce even the small eye accelerations seen with stimulation during fixation, but we have done this experiment at very few sites.

The lower half of Fig. 6 shows that at this stimulation site the magnitude of the stimulation effect increased as pursuit speed increased. Even at the lowest speed tested (5°/s), the stimulation effect was greater than during fixation. Figure 7 shows the effect of pursuit velocity on the stimulation effects using the data obtained from nine stimulation sites and confirms that stimulation produced, with only a few exceptions, acceleration toward the side of stimulation. For
pursuit away from the stimulated hemisphere (Fig. 7A, left), the higher the eye velocity, the larger the effect. For the pursuit toward the stimulated hemisphere (Fig. 7A, right), however, no clear relationship between eye velocity and the stimulation effect is evident.

As we will consider in the DISCUSSION, we think that there are two superimposed stimulation effects during pursuit: an increase in velocity toward the side of stimulation and a decrease of eye velocity regardless of the direction of pursuit. To obtain a better estimate of the influence of the eye velocity on the net directional effect, we took the average of the effect of stimulation for pursuit toward and away from the stimulated hemisphere as shown in Fig. 7B. There was a tendency for higher eye speeds to be accompanied by larger accelerations. In net, the speed of the pursuit, like the presence of pursuit itself, changes the effectiveness of the electrical stimulation.

STIMULATION DURING VERTICAL AND HORIZONTAL PURSUIT... When we stimulated during vertical pursuit, we found clear effects on both vertical and horizontal pursuit components as shown in Fig. 8. In this case we stimulated in MST of the right hemisphere during vertical pursuit, and we saw a change in vertical eye velocity. This was a decrease in downward eye speed during downward pursuit, (Fig. 8, A and B) and a decrease in upward eye speed during upward pursuit (not shown in Fig. 8). We also saw an increase in the rightward component of horizontal eye movement (Fig. 8, C and D). When stimulation was applied during horizontal pursuit at this site, we observed the directional stimulation effects in the horizontal component of the eye movement, (an increase in eye velocity to the right) but no effect in the vertical component of eye movement.

Figure 9 shows the effect of vertical pursuit velocity on the stimulation effect at the same nine points shown for horizontal pursuit in Fig. 7. In Fig. 9A, which shows the effect on the horizontal component of eye velocity during vertical pursuit, the positive value on the ordinate represents horizontal acceleration toward the side of stimulation. The positive values of acceleration for this series of stimulation sites emphasizes the acceleration toward the side of stimulation, but reveals only a weak tendency for higher vertical eye velocities to be associated with larger horizontal eye accelerations. Figure 9B shows larger changes in vertical eye velocity with stimulation during vertical pursuit.

The stimulation effects on both the horizontal and vertical components of the eye movement can be better shown on a two-dimensional plot as in Fig. 10. This figure summarizes the effect of stimulation during pursuit in eight directions and at three speeds, and the scale in Fig. 10D shows that radii of the circle indicate direction of pursuit and that eccentricity indicates speed. Each point indicates
pursuit velocity with (●) and without (○) stimulation, and each pair of circles is connected by a solid line and represents the effect of stimulation in a vectorial manner. In the example in Fig. 10d (same stimulation site as in Fig. 8) stimulation during horizontal pursuit produced the expected decrease in velocity for pursuit away from the side of stimulation that is much clearer than the increase during pursuit toward the side of stimulation. Stimulation during either upward or downward pursuit also produced a decrease in vertical eye speed together with an increase in horizontal velocity toward the side of stimulation. The stimulation effects obtained during oblique pursuit were qualitatively similar to those observed during vertical pursuit. Figure 10b shows the effect of stimulation obtained at a different site in the same hemisphere (but with no oblique pursuit). We again obtained nearly pure horizontal effects during horizontal pursuit and a combination of horizontal and vertical effects during vertical pursuit, and the overall pattern of the effects is very similar to that in Fig. 10a. However, in Fig. 10d, acceleration toward the side of stimulation during horizontal pursuit was only clear during pursuit away from the side of stimulation. In Fig. 10b, such acceleration was clear during horizontal pursuit both toward and away from the stimulated hemisphere.

At 11 sites we collected data comparing at least 12 combinations of target directions and speeds (3 different speeds in at least 4 different directions). Nine of these showed the same pattern as the examples in Fig. 10, A and B, namely an acceleration toward the side of stimulation during horizontal pursuit and a decrease of vertical eye speed together with an acceleration to the side of stimulation during vertical pursuit. (The results of these 9 sites were used for Figs. 7 and 9). In two other cases, the pattern was different in that we did not observe a consistent decrease of vertical velocity with downward pursuit, but instead a weak but consistent downward acceleration with both upward and downward pursuit as illustrated in Fig. 10c. We obtained this pattern of results at one site in lateral-anterior MST and at one other site in the white matter. We also obtained a similar pattern of results at several sites in the white matter where we did less extensive comparison of different parameters of pursuit. In these cases, we first observed the patterns like those in Fig. 10, A and B, and then with stimulation at deeper sites, the decrease in the vertical speed gradually became less consistent and the pattern finally became like that in Fig. 10c.

**Anatomic localization of active stimulation sites**

We mapped the effective sites of stimulation during pursuit throughout areas MT and MST and adjacent areas in two hemispheres of two monkeys. We made penetrations on a 1-mm grid in one hemisphere and 1.5 mm in the other, and stimulation was usually applied every 400 or 500 μm in the STS and surrounding areas. To identify the areas and the subregions within these areas physiologically, we first determined the receptive-field properties of the cells. We then identified the regions active to vertical velocity as described in detail in our previous paper (Komatsu and Wurtz 1988a). We identified MT by the directional selectivity of the cells, by the size of their receptive fields, by their position on the posterior bank of the STS, and by the generally orderly retinotopic organization. We identified MST into a foveal (MTI) and an extravefoval region (MTTe). MST was also identified by the large proportion of directionally selective cells but was distinguished from MT by its position within the STS and by its larger receptive-field sizes. Within MST we identified a dorsal-medial region of MST that we have referred to as MSTd where cells have large receptive fields that include the fovea, respond best to large-field stimuli, and frequently discharge during pursuit eye movements. A second region within MST is a lateral-anterior area that we have referred to as MSTI where cells have a mixture of large and small receptive fields that include the fovea, and that also frequently discharge continuously during pursuit. An intermediate area between MSTI and
and MSTd has extrafoveal receptive fields, and we have suggested previously that this MSTi area might represent the extrafoveal region of MSTi and that together they might be regarded as one area (MSTL as opposed to MSTD) (Komatsu and Wurtz 1988a). An area that has fewer directionally selective cells lying on the floor of the STS was also identified (FST), as was an area higher up on the anterior bank (PP).

Because our major purpose was to localize the directional stimulation effect, we concentrated on horizontal pursuit, and we used only 15°/s target motion in most cases. We classified the stimulation sites as producing a directional stimulation effect, as described previously, any other effect, or no effect. Of a total of 746 stimulation sites, 432 were in the gray matter, and 314 were in the white matter.

Figure 11 shows the areas within the STS (Fig. 11A4) and the frequency of stimulation effects at the 432 points in the gray matter (Fig. 11B). There was a clear localization of the directional stimulation effect (Fig. 11B, left column). The highest incidence was in MT where nearly 80% of stimulation sites were associated with the directional effect. The next highest incidence was MSTi where about one-half of the sites yielded the directional stimulation effect. Stimulation at some points in MT and FST also showed the directional stimulation effect, but these active points were close to MT and we think spread of current might be responsible for the effects in these two regions. Other areas showed a much lower incidence of the directional stimulation effect.

At a small number of stimulation sites in gray matter (n = 23), we obtained different types of effect (Fig. 11B, center column). These include 17 cases which showed a decrease in the horizontal eye speed during pursuit toward the side of stimulation, although almost one-half of these showed a decrease in eye speed with stimulation during pursuit in both horizontal directions.

We also observed the directional effect following stimu-
FIG. 13. Delay of saccades to a visual target with stimulation of MT in the right hemisphere. Eye positions on 6-8 trials are superimposed for step (A and B) or step (C and D) trials. Upward in each segment represents rightward motion. Short vertical bars are the onset and offset of electrical stimulation at 150 μA for 150 ms. Other conventions as in Fig. 1. Latencies without stimulation were (in ms): A, 203 ± 10 (mean ± SE), B, 193 ± 22, C, 185 ± 17, and D, 184 ± 7. Latencies with stimulation were (in ms): A, 320 ± 21, B, 313 ± 6, C, 310 ± 10, and D, 327 ± 8 (for all cases, P < 0.005 in Student's t test). There was also a slight decrease in the amplitude of the saccade with stimulation, but we have not investigated this factor.

FIG. 14. Example of an overshoot of the saccade to the moving target and reduced pursuit velocity with electrical stimulation after target onset but before the saccade was made (A). Superimposed eye positions of 8 trials are for step and ramp trials with stimulation (bottom) and 6 trials without stimulation (top). Upward in the figure is rightward motion. (150 μA, biphasic, 0.2-s pulse train). Electrical stimulation started 100 ms after the target step. Other conventions as in Fig. 1. A and C: mean and standard error of the eye speed 30 ms after the initial saccade to the target from primary position for trials with stimulation (a) and without stimulation (o). B: rightward pursuit. C: leftward pursuit. Abscissa in each graph represents target step size (degree); positive numbers are for rightward step (ipsilateral field) and negative number for leftward step (contralateral field). White matter underlying right MT was stimulated.
lation in the white matter but almost exclusively in the white matter underlying the posterior bank of the STS. These effective stimulation sites in the white matter did not necessarily underlie MT or MSTI cortex although the sites were close to these areas.

Figure 12 shows comparison of the effects of stimulation with receptive fields and anatomic location at a series of sites along reconstructed penetrations. In this hemisphere (te), instead of mapping the entire MT and MST, we concentrated on certain areas and recorded the effect of stimulation with stimulation sites much closer together than during the mapping study. Figure 12A shows an example of a penetration through MST. The penetration entered the posterior bank of the STS in extrafoveal MT (MTe), and the receptive field of the cell was in the upper visual field, separated from the fovea by ~3° (labeled 1 in Fig. 12A). After about a 500-μm interval with vague visual responses, receptive fields consistently included the fovea but were much larger than those of foveal MT cells, which we took as an indication that the electrode had entered MST. The receptive fields then rapidly increased in size and within 1 mm, they covered an entire quadrant or almost an entire hemifield (receptive fields 7-10). The transition of this extrafoveal to foveal receptive field corresponded well to the border of the dense myelination determined from subsequent histological sections. A very clear directional stimulation effect was obtained in MST 1 mm from the MT-MST border, and a second smaller peak was observed ~700 μm deeper. Around these peaks, acceleration to the side of stimulation (leftward) was clear during both rightward and leftward pursuit. However, the effect during leftward pursuit disappeared 1.2 mm deeper than the first peak, and even the direction of the effect reversed at deeper sites so that decrease of eye speed in either direction of pursuit occurred. Current spread at 100 μA used in this study is estimated to be ~450 μm in radius (Stone et al. 1968), and this value corresponds well to the one-half width of the peak. If we apply this estimate of the current spread to the present results, there may actually be one or two effective sites corresponding to the peak in the profile of the stimulation effect. Weaker effects obtained around these peaks may result from current spread to these more localized effective sites.

Figure 12B shows an example of a penetration passing through MT. The penetration entered the posterior bank of the STS within the densely myelinated area, the receptive fields were in the fovea, and the small size was indicative of MT. The penetration remained in MT as indicated by the constant visual properties of the cells. Like the example in Fig. 12A, there was a peak in the directional stimulation effect even within the small MT area.

There is a tendency in Fig. 12A for the cells at the point of effective stimulation to have a preferred direction for visual motion toward the side of the brain stimulated. We did not find this to be generally true, however, as indicated in Fig. 12B, where the most effective stimulation point is at the site of a cell whose preferred direction could not be determined. We did not, however, systematically investigate the effect of threshold stimulation that would be more likely to reveal any relationship of individual cell groups to the effect of stimulation on pursuit.

**Stimulation during pursuit initiation**

When we stimulated before the onset of pursuit in the step-ramp task, we saw two types of effects that differ from the directional stimulation effect during pursuit. One was an increase in the latency of the saccade to the pursuit target; the other was a disruption of pursuit initiation to targets in the contralateral visual field.

**SACCADIC LATENCY.** Figure 13 shows an example of the increased saccadic latency following stimulation applied for 150 ms starting 100 ms after the step of the target that then moved (Fig. 13, A and B) or was stationary (Fig. 13, C and D). For trials without stimulation, the latency of the saccade to the target was ~200 ms. However, when the stimulation was applied before the saccade, this latency was significantly prolonged to ~320 ms. Stimulation at this point also produced a directional effect during pursuit, but the increase of saccadic latency occurred regardless of the horizontal direction of the saccade or whether the target was moving or stationary (a symmetrical increase in latency). This type of increase was observed at more than one-half of the effective stimulation sites (19/35). For the rest of the effective sites (16/35), the increase of saccadic latency was clearer for one direction of saccade than for the other (an asymmetrical increase). Of these latter 16 sites, 9 sites showed an increase in latency of saccades to the ipsilateral field, 7 an increase in latency to the contralateral field. All increases in latency occurred with either moving or stationary targets.

In one hemisphere we mapped throughout MT, MST, and surrounding areas the effects of stimulation on the latency of saccades. We found the clearest increase in latency in MT and MST where the increase in saccadic latency was symmetrical. These stimulation sites frequently produced a directional pursuit effect as well. Stimulation in FST, which is adjacent to MT and MST, also produced delay of saccades with high incidence (9/13, 7 symmetrical, 2 asymmetrical). Stimulation of other areas occasionally produced mild asymmetrical increases in latency. The frequency of increased saccadic latency following FST stimulation, where we rarely obtained directional stimulation effects, suggests that the localization of this latency effect can be separated from that of the direction al effect. Furthermore, the tendency to see the increased latency in areas within the STS where the fovea is represented, MT, MST, and FST, suggests that the latency effect is specifically related to alteration of foveal visual processing.

**DISRUPTION OF PURSUIT INITIATION.** Another effect we found when we applied stimulation after the target step but before the eye started to move was an error in the utilization of motion information in the contralateral visual field, similar to the effects of chemical lesions of MT on pursuit shown previously (Newsome et al. 1985). Figure 14A shows an example of such effects. In this experiment, when stimulation was applied 100 ms after the target onset, the initial saccade to the target failed to compensate for target motion and showed considerable amplitude overshoot, and eye speed following the initial saccade was lower than target speed. Both of these effects are consistent with the stimulation acting to disable the use of information on target mo-
tion. We observed the deficit only with target motion in the contralateral visual field, but the direction of target motion was irrelevant; the effect was retinotopic, not directional. Figure 14, B and C, shows the means and standard errors of eye speed measured 50 ms after the initial saccade on the step-ramp trials with steps to different locations on the horizontal meridian. In Fig. 14B, the direction of target motion after the step was rightward, and in Fig. 14C, it was leftward. A clear reduction in postascadic eye speed occurred for trials with target steps in the contralateral visual field regardless of the direction of target motion (P < 0.005 in Student's t-test for all steps into the contralateral field). No significant effect was observed for trials with target steps in the ipsilateral visual field.

We observed this retinotopic effect with stimulation of extrafoveal regions of MT and the white matter underlying it, and occasionally by stimulation of MSTi and MSTi as well. The effective sites for this retinotopic effect represented more peripheral visual fields than the sites where we obtained the delay of the initial saccade. However, at some stimulation sites, stimulation produced a combination of both effects.

**Discussion**

We have been able to alter the maintenance of smooth pursuit eye movements by electrical stimulation of subregions within the STS. We think our observations contribute to the localization of the directional pursuit mechanisms in cerebral cortex, and to a better understanding of the sequence of visual and visual-motor events in cortex occurring during the maintenance of pursuit. We will discuss each of these points and then consider the effects of stimulation on the initiation of pursuit.

**Localization of the directional bias in pursuit maintenance**

Previous studies have shown that chemical lesions in the region of MSTi or MT produce a deceleration of pursuit eye movements made toward the side of the lesion (Dursteler et al., 1987, 1988). In these experiments, alteration of pursuit was produced by removing neuronal activity; ibotenic acid injected into limited areas of MT or MST killed cells around the injection sites. In the present stimulation experiments, the stimulation of MSTi or MT produced an acceleration of pursuit toward the side of stimulation. This deceleration was produced by activating rather than removing cells around the stimulation site. Therefore, the opposite effects of deceleration and acceleration obtained fit well with the two techniques used, removal and activation of neurons.

The previous chemical injections, while limited in extent compared to most lesion experiments, still spread to include substantial areas of MT, MST, or both areas. The stimulation effects in gray matter of cortex probably affect a more limited area, and allow better localization of the effects. Estimates based on stimulation of motor cortex of the monkey (Stoney et al. 1968) put the area activated by stimulation currents of 50--100 μA (with other stimulation parameters) between 300 and 450 μM. Such a limited area of activation (<0.5 mm) allows more restricted localization than the chemical lesions of 2--3 mm radius. This allows clarification of several issues of localization if we make the assumption that the lesion and stimulation act on the same underlying mechanisms. First, lesions within MST (Dursteler and Wurtz 1988) had indicated that directional pursuit deficits resulted from damage to MSTi but not to MT, and the ease of producing acceleration during pursuit by stimulating MSTi as compared to MT (as summarized in Fig. 11) fully confirms this distinction. Second, the lesion experiments indicated that damage to MSTi in the absence of damage to MT produced a directional deficit and the clear eye acceleration produced by stimulation within MSTi also confirmed this point. Third, the lesion experiments left unresolved the issue of whether the direction effect also could be related to activity in MT. Since stimulation at many sites within MT modified pursuit, this issue would seem to be resolved in favor of a role for MT in producing a directional pursuit deficit comparable to that of MSTi. Finally, there was an indication from the lesion experiments that damaging FST might contribute to the directional pursuit deficit, but there is no confirmation of this from studies because stimulation of FST usually did not produce eye acceleration. In net, based on both the lesion and stimulation experiments, we conclude that a directional bias in the maintenance of pursuit results from activity in MT and MST, but not MT, and probably not FST.

**Relation of the directional effect to visual processing**

The effect of the electrical stimulation can be regarded as combining with the ongoing neuronal activity or substituting for this neuronal activity. For the STS areas that we stimulated, we think that the pursuit system behaved as if the stimulation substituted for the normal neuronal signal. The experiments on which we base this conclusion compared the effects of stimulation in the normal (closed-loop) condition where visual feedback was operating with those in the open-loop condition where such feedback was experimentally reduced. In the closed-loop condition, stimulation produced an increase in eye speed that was visual slip of the target in the direction opposite to that of the eye movement. Slip in this direction should act to reduce eye speed if the system were sensitive to visual motion during the period of stimulation. It did not, but instead, the pursuit stayed about the same as in the open-loop condition, when such slip was minimized because the target was stabilized on the retina. This indicates that the pursuit system was insensitive to the visual motion feedback signal even in the closed-loop condition, suggesting that the visual motion feedback loop was opened by the stimulation itself. This suggests that the stimulation substituted for the normal neuronal signal by cutting out the visual feedback signal and injecting an artificial signal. Up to this point we have referred to the neuronal signal related to pursuit maintenance, but in previous experiments (Newsome et al., 1988), we found at least two types of neurons related to pursuit. One type of neuronal signal was a visual one representing motion of the target and we saw neurons with such signals in both MT and MST. The other signal was a combination of visual and extraretinal input. The extraretinal input became evident during pur-
suit when the visual input was experimentally removed, and we found this visual-motor signal in MST but not MT.

The visual pathway related to pursuit can therefore be separated into two parts: one before the summation with extraretinal input and the other after that summation. We think that our experimental evidence suggests that the stimulation acts on the visual signal before it is combined with extraretinal input. The first piece of evidence that the stimulation acts here instead of after the visual and extraretinal signals are combined is the effectiveness of the stimulation in MT. Since there is no clear indication of an extraretinal signal in MT at the single-cell level (Newcombe et al. 1988), and since the stimulation here produces a directional effect, this directional stimulation effect must result from modification of a purely visual signal.

Another indication that the electrical stimulation acts primarily on a visual processing stage, is the interaction of stimulation with the presence and speed of ongoing pursuit. Stimulation was more effective during pursuit than during visual fixation, and the higher the eye speed during pursuit (within the range of 5–25°/s), the larger the acceleration toward the side of stimulation. The system acted as if the artificial stimulation signal were modified by a signal related to the presence and speed of pursuit. Therefore any input related to pursuit must have entered the system after the point of stimulation in the pursuit pathway. This is consistent with the argument that the effect of stimulation was to replace the visual input.

In fact, the effect of eye speed was less clear at some stimulation sites than others and this may imply that the interaction of visual and extravisual signals takes place in MST over a number of steps. This may correspond to the observation that the relative contribution of the visual and extravisual signal on the pursuit discharge is graded among the population of pursuit cells in MST (Newcombe et al. 1988).

This interpretation of a directional stimulation effect acting on visual processing is consistent with recent observations on the directional pursuit deficit resulting from chemical lesions in MST. In these experiments an increase in target velocity was superimposed on the pursuit velocity under stabilized conditions. In the normal monkey this velocity step produced an increase in pursuit velocity, indicating that the pursuit system responded to the visual slip signal. After a chemical lesion of MST, pursuit was less sensitive to such a velocity step when the step was toward the side of the brain with the lesion regardless of the direction of pursuit; the deficit followed primarily the direction of target motion, not the direction of pursuit. This lesion experiment, like the present ones, suggests that it is the alteration of visual processing that is related to the directional pursuit effects.

Two components of the stimulation effect

We think that the effect of electrical stimulation can best be understood as acting on separate groups of visual neurons. Neurons in these areas respond to different directions of visual motion (Albright 1984a,b; Baker et al. 1981; Felleman and Kaas 1984; Maunsell and Van Essen 1983a,b; Saito et al. 1986; Tanaka et al. 1986; Zeki 1974a,b, 1980). Since different groups of cells are coding different directions of visual motion, we can regard these at least conceptually as multiple directional channels for the visual input to pursuit. If we can activate one of these channels, we can expect an acceleration in the direction indicated by that channel to be superimposed on the ongoing pursuit. However, if channels for different directions coexist within a small spatial region, stimulation of a single channel particularly with suprathreshold stimulation would be unlikely. If we assume that electrical stimulation activates all channels, all directions of pursuit would be invoked. If we assume further that each channel has an equal influence on the pursuit response, activation of all channels would produce no net input to the pursuit system, and the effect could be a reduction of pursuit speed in all directions. Such a reduction would be consistent with the decrease in vertical pursuit speed since if stimulation activated all channels, it would have activated upward and downward channels equally.

This argument would lead us to expect a similar reduction of speed in the horizontal component of velocity as well. We usually did not observe such a reduction but instead an acceleration during pursuit toward the side of stimulation and a deceleration during pursuit away from the side of stimulation. However, if we add the assumption that certain channels are more frequently represented, and if these channels represent visual directions toward the side of the stimulated hemisphere, the acceleration to the side of stimulation becomes understandable. The effect of stimulation would then be greater on these more frequently represented channels and would produce a greater effect in these directions. So far we have not found clear indication of any bias toward the side of stimulation in the population of cells studied (Komatsu and Wurtz 1988a), but the sample studied has been small. The bias may also result from the strength of the connections of cells with different preferred directions of motion rather than in the frequency of the cells themselves.

If stimulation acts by producing these separate effects, the behaviorally observed result of stimulation would be the interaction of these two effects: the reduced pursuit in all directions and the increased pursuit in the one horizontal direction toward the side of the brain stimulated. This is shown schematically in Fig. 15 by the summation of the two effects. For horizontal pursuit, the two effects subtract from each other during pursuit to the side of stimulation so that the resulting acceleration is smaller than it would have been if the directional bias acted alone. For pursuit away from the side of the stimulation, the two effects add to each other so that the resulting deceleration is larger than the directional stimulation effect would have produced alone. For vertical pursuit, the combination of the reduction in vertical velocity and the acceleration to the side of stimulation would produce an oblique effect on the eye velocity, as was frequently the case (Fig. 10d).

Previous studies of the effects of stimulation on pursuit

The STS projects heavily to the dorsolateral pontine nuclei where cells have been shown to discharge during pur-
suit eye movements. May et al. (1985) modified pursuit by electrical stimulation of the dorsolateral pontine nuclei, with parameters of stimulation very similar to those used in our experiments. They found that stimulation increased pursuit velocity when it was applied during ongoing pursuit, but not when it was applied during fixation. Their observations are generally similar to ours since while we saw an acceleration during fixation it was less than that during higher pursuit speeds. However, this is a difficult result to explain, if we consider that the interaction between visual and extraretinal signals take place in MST.

We would have expected the stimulation in the pons to be effective regardless of whether pursuit was occurring or not, because the next stage of the system after our hypothesized stimulation point within the STS should already have incorporated the presumed extraretinal signal indicating that pursuit is ongoing. If stimulation at this point replaces the neuronal activity, as we have argued that it does within the STS, whether pursuit is occurring or not should have no effect. One interpretation of this conundrum is that the transition from purely visual to visual and extraretinal signals develops over several steps, both in MT-MST and in the pons. May et al. (1985) also did not report a bias for acceleration toward the side of the brain stimulated, as was the case in the STS, so that a number of points of fact and interpretation remain to be resolved before the relation of MT-MST and the dorsolateral pons can be specified.

There is little information about the effects on pursuit of stimulating other areas of the cerebral cortex. Stimulation of the posterior parietal cortex has recently been reported to produce pursuit eye movements while the monkey fixated on a target (Kurzly and Skavenski 1987). Stimulation of selected points within the frontal eye fields (FEF) also produced smooth pursuit eye movements during fixation (Bruce et al. 1985). Thus at both of these points stimulation did not require ongoing pursuit to be effective, and this suggests that these structures lie beyond the visual stage hypothesized for the STS cells in our earlier discussion. The direction of the pursuit produced with parietal stimulation was toward the side of the brain stimulated, as was the case in the STS. At the 12 points stimulated in the FEF, cells had no reported directional selectivity for visual motion, but they did tend to discharge with pursuit in the same direction produced by stimulation of the site. Whether this might also be the case for stimulation in the STS must await a comparison of cellular activity with electrical stimulation closer to threshold than the suprathreshold stimulation used in this study.

**Stimulation effects on pursuit initiation**

When we stimulated before the start of pursuit, we disrupted pursuit initiation. Stimulation of the extrafoveal representation within MT or the underlying white matter led to an inability of the monkey to use motion information when the moving target appeared in the contralateral visual field. This deficit is identical to that observed after chemical lesion of extrafoveal MT (Newsome et al. 1985). The lesion effect almost certainly results from the loss of visual information normally carried by the missing cells, and stimulation should have activated cells not removed them. We think that the discrepancy can be explained by the same arguments we used to interpret the slowing of vertical pursuit by stimulation. If we simultaneously stimulate many channels of visual information, each with different preferred directions, and none of them occur more frequently than any other, the signals carried by individual channels would act to cancel each other and no net directional signal would result. The effect of stimulation would then effectively be the same as removing the cells by the chemical lesion. This hypothesis is similar to that for the reduction in pursuit in all directions following foveal stimulation but is in contrast to our explanation of the directional stimulation effects in the fovea resulting from the larger representations of certain directions of movement.

**Stimulation effects on saccade latency**

Stimulation applied to FST, MTF, and part of MST in the time between target onset and the start of the saccade to the target increased the latency of that saccade. This increase occurred whether the target was moving or stationary. We also obtained similar latency increases when we stimulated sites on the posterior bank of the intraparietal sulcus (IPS). Similar increases in latency have been re-
ported for stimulation of the lateral part of the FEF (Azuma et al. 1986). It is worth noting that the neuronal activity in these areas has several characteristics in common. First, the cells in these areas represent the central visual field. The cells at effective stimulation sites in MT, FST, and the lateral FEF all have small receptive fields within the fovea. The receptive fields of cells in MST and IPS are larger but still include the fovea. Second, we frequently observed a tonic discharge during fixation of a small spot in MTF (marked by a T on the location section of Fig. 12) and in FST (unpublished observations), and such activity was also observed in the lateral FEF (Suzuki and Azuma 1977). Therefore, we suggest that these areas are involved in maintaining fixation on the target and in inhibiting a shift of fixation to peripheral stimuli. Since pursuit also maintains the target on the fovea, and since the increase in latency was frequently observed at the same sites that produced the directional stimulation effect, it might seem as if the latency effect results from stimulation of the pursuit system. However, the lack of selectivity of this effect for saccades to moving targets, as well as the lack of a stimulation effect on pursuit with stimulation of FST suggests that this is not the case. It seems more likely that the functional system involved in the maintenance of fixation and pursuit can be distinguished. A similar conclusion, based on entirely different types of evidence, was reached by Robinson et al. (1986).

Finally, it is clear that not all areas where the central visual field is represented are involved in the maintenance of fixation. Even though many cells in MSTD include the fovea in their receptive field, we did not see an effect on saccadic latency as a result of stimulation of this area. As we have reported in a previous study (Komatsu and Wurtz 1988a), cells in MSTD prefer large textured stimuli, such as random dot patterns, rather than small well localized stimuli like spots. Cells in MTF and FST prefer spots as do many MSTD cells (Komatsu and Wurtz 1988a,b), and cells in FEF respond well to small spots (Suzuki and Azuma 1983). Therefore, a preference for small spots might be a common characteristic of systems involved in the maintenance of fixation. It would be interesting to know whether stimulation of the foveal representation of the superficial layers of the superior colliculus would increase the latency of saccades, because the cells in that area have the characteristics observed at the effective sites we have considered.

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