Directional Pursuit Deficits Following Lesions of the Foveal Representation Within the Superior Temporal Sulcus of the Macaque Monkey

MAX R. DÜRKSTELEL, ROBERT H. WURTZ, AND WILLIAM T. NEWSOME

Laboratory of Sensorimotor Research, National Eye Institute, National Institutes of Health, Bethesda, Maryland 20892

SUMMARY AND CONCLUSIONS

1. Ibotenic acid lesions of the middle temporal visual area (MT) have previously been shown to impair a monkey's ability to initiate smooth pursuit eye movements to targets moving in the extraneous visual field (30). This is a retinotopic deficit: pursuit is impaired in all directions within the affected portion of the contralateral visual field.

2. In the present experiments we analyzed the effects of lesions of the foveal representation of MT on the maintenance of foveal pursuit. Injections of ibotenic acid were directed toward the representation of the fovea within MT but spread into extraneous regions of MT and adjacent visual areas within the superior temporal sulcus.

3. Chemical lesions of the foveal representation produced a directional deficit in the maintenance of pursuit: the monkey failed to match eye speed to target speed when pursuing a target that moved toward the side of the brain with the lesion. This deficit was evident regardless of the part of the visual field in which target motion began, and pursuit at higher target speeds was more severely affected. The directional deficit was qualitatively similar to pursuit deficits observed in human patients following large parietal-occipital lesions.

4. Extension of the lesions into extraneous regions of the contralateral visual field representation also resulted in retinotopic deficits for pursuit initiation: the monkey was unable to match the speed of its pursuit eye movement to that of a target or to adjust the amplitude of its saccade to compensate for target motion. The errors in pursuit speed and saccade amplitude for initiation of pursuit into the contralateral visual field were linearly related, which supports the hypothesis that both deficits arise from damage to the same underlying visual motion processing mechanism.

5. The selectivity of the retinotopic deficit for motion information was also investigated by reducing retinal motion through the use of a stabilized image. After the lesion, the monkeys continued normal pursuit when a position error was present during stabilization, supporting the view that the deficit was related to loss of motion but not position information.

INTRODUCTION

The middle temporal area (MT) lies in the depths of the superior temporal sulcus (STS) (44, 54) and has been identified as an area involved in the processing of visual motion information. MT contains a preponderance of neurons that encode the direction of visual motion (2, 6, 25, 52, 53), and different directions of motion are represented in a system of columns similar to that for orientation in striate cortex (1). In addition to directional selectivity, MT neurons are also selective for speed and binocular disparity (2, 25, 26).

MT probably represents only one of several cortical areas devoted primarily to visual motion processing. Direct projections connect MT to adjacent regions within the STS (43, 46) and to areas within the intraparietal sulcus (27, 42). The adjacent regions within the STS, originally named MST for the medial superior temporal area (27, 46), also have a substantial proportion of cells that show directional se-
lectivity to stimulus motion (27, 29, 39, 46). MST has recently been further divided into subregions based on differences in physiological properties between these areas (5, 34).

We have recently found that damage to the first of these areas devoted to motion processing, MT, markedly alters a monkey's ability to use motion information for accurate initiation of smooth pursuit and saccadic eye movements (30). In these experiments we injected a neurotoxic chemical, ibotenic acid, into an area of MT that represented an extrafoveal region of the contralateral visual field, and the resulting deficits were confined to targets moving within the damaged portion of the visual field. Saccades to stationary targets were unaffected by these lesions, suggesting that this retinotopic deficit affected the monkey's use of motion, but not position, information.

In the present experiments, we have turned our attention toward the maintenance phase of pursuit that occurs after foveal pursuit has been established. Since during pursuit maintenance the target image falls on the fovea, we directed ibotenic acid injections toward the representation of the fovea in MT, although adjacent areas of MST, which also are related to the fovea, were almost certainly affected. We observed the retinotopic deficit as in our previous experiments, but we also observed an additional, directional, deficit in which pursuit maintenance was impaired for all target motion toward the side of the lesion. Similar deficits have been observed following large parietal-occipital lesions or hemispherectomy in man (21, 40), and the present observations may shed light on the cortical pathways underlying this clinical deficit.

Since the lesions in the present experiment also produced the previously reported retinotopic deficit for pursuit initiation to extrafoveal targets, we took the opportunity to investigate the dependence of that deficit upon target speed and the relation of recovery rate to lesion size. We also investigated further the differential effect of the lesions on the use of extrafoveal motion as opposed to position information. We reduced motion information during pursuit by stabilizing the image on the retina, and introduced a position error by displacing the target from the fovea, a condition that induces pursuit eye movements (16, 19, 31). The monkey's pursuit of this stabilized image was normal following the lesion, lending further support to the conclusion that the deficit is related to the loss of motion, not position, information for ocularmotor control.

Brief reports have been published previously (7, 48).

METHODS

Behavioral task

Two monkeys were trained to fixate, or to follow with their eyes, a small visual target presented on a tangent screen at a distance of 36 cm (1.5 rad) in front of them. Each monkey was seated in a primate chair and had an unobstructed view of the screen out to 40° eccentricity. 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 (13, 32).

The same step-ramp paradigms were used as in our previous study (30). These paradigms allowed us to study the initiation of pursuit and the maintenance of pursuit after the monkey moved his fovea near to the target. 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, the monkey moved his eyes to a second light that appeared simultaneously at one of several possible locations on the horizontal meridian. In step trials the second target remained stationary and the monkey made a rapid (naccadic) eye movement to the target. In step-ramp trials, the second target moved at 16°/s away from or toward the fovea along the horizontal meridian. The step for a given trial was usually 0, 2, 5, 10, or 15° in amplitude and could occur in either horizontal direction from primary position. The size and direction of the target step, the direction of target motion following the step, and the trial condition (step or step-ramp) were randomized from trial to trial. A block of trials usually consisted of 240 trials with 10 each for the 8 step locations along the horizontal meridian and 3 for each condition at each step location (step, step-ramp away, step-ramp toward).

During the initial training period, all monkeys were rewarded with a drop of water for fixating a spot of light and detecting the dimming of that spot (47). For three experiments (M1, M2, T7) this task was used only to calibrate the eye position at the beginning of each experimental day, but during the step-ramp task the monkeys were rewarded for keeping their eyes inside an electronically defined window centered on the target (38). At the beginning of each of these trials, the fixation light came on and the monkey was required to move his eye into the position around the fixation light within 500 ms. This fixation window was usually 0.7–1.5° in width. The fixation light remained on for 500–
2,500 ms (after the monkey's eye entered the window) and then went out as the target light came on. The monkey was allowed a grace period of 360 ms to shift his gaze to the target light. After this time the position window was centered on the target and moved with the target. The monkey was rewarded if he maintained eye position within that window until the end of the trial (350–600 ms). The trial was terminated if eye position moved out of the window. During pursuit of the moving target, the position window varied in size (1.0–5.0°) from trial to trial depending upon the eccentricity of the target on the screen and the consequent difficulty of the task.

The visual target was relatively small (0.2° in diameter) and bright (38 cd/m²) against a dim background (0.13 cd/m²). The target was produced by projecting a spot of light from a tungsten filament bulb onto mirrors mounted on galvanometers. The fixation point was a spot 0.05° in diameter produced by projecting a beam from a light-emitting diode.

In a second set of experiments, we measured pursuit of a stabilized image. 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 the monkey was pursuing the target we stabilized the image on the fovea or with a known position offset from the fovea. With our system the latency for mirror movement following eye movement was, at most, 15 ms. In one of the experiments, T2, the monkey was required to both keep its eye in the window around the target and to detect the dimming of the target, in order to obtain a reward. This condition produced better pursuit under the stabilized image conditions.

We did not attempt to elicit pursuit initiation with the stabilized image; in our experience, saccadic rather than pursuit eye movements occurred when the monkey attempted to track a stabilized target that was stationary and then moved. In fact, we exploited this behavior to align eye and target position during initial calibration procedures, as suggested to us by S. G. Lisberger. After allowing the monkey to fixate for 360 ms, we stabilized the fixation point 1° to the left of the fovea and compared the amplitude of the saccades the monkey made attempting to "catch" the stabilized image with the saccade amplitude when the step was 1° to the right. We then adjusted the eye position so that saccades sizes in the two directions were equal. This ensured that the fixation point and the eye position were aligned and that the target was displaced by the proper amount during stabilization trials.

Throughout the training and the experiments, the monkey's weight was checked each day and supplementary water or fruit were provided if needed. Monkeys sat in a primate chair during experiments but were returned to their home cages each day after the experimental session.

Injection of ibotenic acid

Under general anesthesia (pentobarbital sodium) monkeys were implanted with a headholder for restraint of the head during the experiments, with a stainless steel cylinder for microelectrode recording (10, 11) and with an eye coil for measurement of eye position (15). A detailed description of these procedures can be found in a previous report (17). Following surgery, monkeys were given analgesia for several days and allowed to recover for at least a week before experiments began.

We first located MT using single-cell recording and then implanted a guide tube so that the tip was within 3 mm of the foveal representation of the horizontal meridian within MT. We confirmed the guide tube location by recording neuronal receptive fields with a fine tungsten electrode (Hart) introduced through the guide tube. We then obtained pretension data usually over a period of several days with the guide tube already in place. Following this behavioral testing we inserted through the guide tube the needle of a 1- or 5-μl syringe (Hamilton), which in some cases had been insulated to allow recording of multiple cell activity and which allowed us to confirm the visual field location where the ibotenic acid was released. We then injected ibotenic acid (15 μg/μl in a basic saline solution), a neurotoxin that we used in order to kill cells while avoiding damage to white matter (30). Monkeys exhibited no signs of discomfort during or after the injection.

Since there was recovery of function following the injections, we repeated the same procedure in the second hemisphere of each monkey. We designated the first injection in each monkey as experiment M1 and T1, the second as M2 and T2. There was some variation in the injection procedures from experiment to experiment. We injected 1 μl of 15 μg/μl ibotenic acid on two successive days in experiments M1 and T1; postinjection behavioral results were from the data following the second injection. In experiment T2 we injected 2 μl of 15 μg/μl ibotenic acid on one day. In experiments M2 we made a series of injections, as described above, as well as infusing using an osmotic pump (Alzet 2001). For the infusion we connected the osmotic pump filled with 10 μg/μl ibotenic acid to a syringe needle inserted into the guide tube and ending in foveal MT. The pump was placed in the saline-filled implanted cylinder. The quantity of ibotenic acid infused was estimated from the manufacturer's specifications for the osmotic pump. Records from this experiment (M2) are shown following an initial infusion of ~23 h (18 μl of ibotenic acid).

Following completion of experiments, monkeys were deeply anesthetized and perfused with saline
and 10% formalin. Parasagittal sections through the occipital and parietal lobes were stained with cresyl violet for cell bodies and with a modified silver stain (14) for myelinated fibers. The extent of the lesions was determined by examination of the histological sections, and the data were displayed on flattened maps of the cortex created by the method of Van Essen and Maunsell (45).

Data analysis

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 for ~1 s. The horizontal and vertical eye position signals were low-pass filtered at 250 Hz 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 Hayes, Richmond, and Optican (15), which was run on a PDP 11/40 computer.

For quantitative analysis of the eye movements, we used an off-line program that digitally filtered the stored position information and computed eye velocity and acceleration of the eye as described previously (30). 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.

RESULTS

Two types of pursuit deficits

Following isotonic acid injection into the foveal representation within the STS, we observed changes in pursuit behavior that appear to result from two distinct deficits: a retinotopic deficit for initiation of pursuit to extrafoveal targets moving in any direction within one region of the contralateral visual field and a directional deficit for maintenance of foveal pursuit when the target was moving toward the side of the lesion. The directional deficit has not been seen previously following injections limited to the STS and it will be described first. The retinotopic deficit was similar to that reported previously following injections into the extrafoveal representation of MT (30) and will be described only briefly. Although the size and extent of the chemical lesions varied following injections in the four experimental hemispheres (referred to as M1, M2, T1, T2), we made the same basic observations in all four cases. We will first illustrate these deficits for one monkey (M1).

The directional deficit is illustrated in Fig. 1. Figure 1A shows rightward pursuit of a target whose motion began after a 5° step into the ipsilateral hemifield. The schematic drawing in the upper left corner of Fig. 1A indicates the motion of the target (16°/s, rightward motion is up) and its relation to the visual hemifield affected by the lesion in the right hemisphere (shaded). The top of Fig. 1A shows 10 superimposed traces of eye position recorded before the injection (pre). The monkey first made a saccadic eye movement, which brought the fovea near the target, and it then followed the target with smooth-pursuit movements for the duration of the trial. Small catch-up saccades were evident, indicating that even in the normal monkey, eye velocity lagged pursuit velocity during the initial several hundred milliseconds of pursuit.

As shown by the schematic drawing in the upper left of Fig. 1A, the injection in this monkey was in the right hemisphere, which we would expect from our previous experiments (30) to affect the left (contralateral) visual field but spare the right (ipsilateral) field. Pursuit of targets in the ipsilateral visual field would be expected to show no deficit. The lower set of traces in Fig. 1A, obtained 24 h after the injection of isotonic acid, shows that this is not the case. Following the saccade to the target, the eye speed lagged target speed, and repeated catch-up saccades were made to keep the fovea near the target. Figure 1B shows the mean and standard error of eye speed plotted against time for the same records shown in Fig. 1A. Although the eye speed before injection approached target speed (16°/s), the eye speed after the injection usually remained below 16°/s during the rest of the trial. Thus maintenance of pursuit toward the side of the lesion was impaired. Although the most prominent deficit is maintenance of pursuit, it should be noted that the reduced speed is evident immediately after the end of the sac-
FIG. 1. Directional pursuit deficit in a monkey with a right hemisphere lesion. *A:* position traces for target steps 5° into the right visual field with motion at 16°/s away from the fovea. The step was into the hemifield ipsilateral to the lesion (as shown in the schematic drawing—top left). Position traces show 10 superimposed eye movements pre- and postinjection. *B:* eye speed, as a function of time, shows mean and standard error for the trials illustrated in *A.* Interruptions in the traces occur where saccades have been removed. The records in *A* and *B* show a reduction in the pursuit speed for the duration of the trial (the directional deficit). *C* and *D:* position and speed records for target steps 5° into the right (ipsilateral) visual field but with target motion to the left, away from the side of the brain with the lesion. No deficit in pursuit is evident. All records are from experiments M1, as indicated in the lower right corner.

cade to acquire the target so that initiation of pursuit is affected as well.

Pursuit maintenance was entirely normal when the target stepped into the ipsilateral visual field but moved away from the side of the lesion. Figure 1, *C* and *D* show traces for position and speed following a target step of 5° into the right visual field with target motion to the left. This motion away from the side of the lesion was not accompanied by any deficit in pursuit initiation or maintenance. We therefore refer to the pursuit deficit in the ipsilateral visual field as a directional one; the eye fails to match target speed when the target motion is in one direction, toward the side of the lesion.

As shown in Fig. 2, pursuit toward the side of the lesion was also impaired when the initial target step was into the contralateral hemifield. Following the 5° step to the left (Fig. 2A), the monkey's pursuit speed failed to match that of the rightward moving target (Fig. 2B), for the entire trial. What is particularly striking in Fig. 2, however, is the retinotopic deficit related to pursuit initiation that we have reported previously following MT lesions (30). Before the injection, recording of multiple cells at the injection site yielded receptive fields that were within 1° of the fovea in the contralateral visual field. Following the injection, however, the impaired region of the visual field included extrafoveal regions in the contralateral visual field, presumably due to spread of the ibotenic acid. This inclusion of extrafoveal regions resulted in the two components of the retinotopic deficit that can be seen in the first 200
ms of the pursuit response in Fig. 2A. The first is a decrease in pursuit speed just after the monkey made the saccade to acquire the moving target. As shown in the speed traces in Fig. 2B, eye speed remained below 5°/s for the first 200 ms following a saccade to the target. The second component of the retinotopic deficit is a failure to reduce the amplitude of the saccade to the target to compensate for motion of the target; the saccade overshoots the target (Fig. 2A). Pursuit of targets moving to the left following a step into the contralateral field also showed a retinotopic deficit during initiation, but did not show a directional deficit during pursuit maintenance (Fig. 2C and D).

This distinction between the directional deficit and the retinotopic deficit can be seen by comparing Figs. 3 and 4. Figure 3A shows eye speed during the first 100 ms of pursuit for rightward motion (toward the side of the lesion) initiated at seven different locations in the visual field. Postlesion performance was impaired in each case. The impairment in the right hemifield was due to the directional deficit alone. The more severe impairment in the left hemifield resulted from the summed effects of the directional and retinotopic deficits. In contrast, leftward pursuit (away from the side of the lesion) is illustrated in Fig. 4A. Pursuit initiation was impaired in the left hemifield as expected from the retinotopic deficit. However, pursuit was entirely normal in the right hemifield, which was subject only to the directional deficit.

Figure 3B illustrates data in the same format as Fig. 3A obtained at successive times after
FIG. 3. Spatial extent of the deficit for pursuit toward the side of the lesion. A: pursuit deficit during the first 100 ms after the initial saccade. Abscissa shows the step size from the primary position (rightward steps, positive; leftward steps, negative). Ordinate shows horizontal eye speed, which is the mean of 10 trials. Eye speed for each trial was averaged over the 100 ms following the initial saccade to the target. The means and standard errors were derived from 10 pursuit responses obtained at each step location before and 24 h after the injection. The centers of the multi-cell receptive fields recorded through the syringe needle at the injection site were within 1° of the fovea. In this and subsequent figures, a double asterisk indicates a significance level, $P < 0.001$, as determined by a Student's $t$ test, and a single asterisk indicates $P < 0.01$. The decreased eye speed after the lesion reflects both the field specific deficit and direction specific deficit in the left hemifield and the directional deficit alone in the right hemifield. The deficit was
the end of the saccade made to acquire the target. The first graph on the left of Fig. 3B for the period 0–100 ms is, in fact, the same as that shown in Fig. 3A. Figure 3B shows that rightward pursuit was impaired for the entire duration of the trials regardless of the point in the visual field where pursuit initiation occurred. In contrast, pursuit away from the side of the lesion (Fig. 4B) was impaired only in the contralateral hemisphere and only during the first 100–300 ms following the initial saccade. Pursuit was normal in all cases by 400–500 ms after the saccade to the target. In other words, leftward pursuit was subject only to the retinotopic deficit for pursuit initiation; the maintenance phase was unaffected.

Three other injections produced a similar pattern of deficits in pursuit speed, and Fig. 5 condenses the results of these experiments (M2, T1, T2) into summary graphs like those shown for experiment M1 in Figs. 3B and 4B. The left column of Fig. 5 shows results for pursuit away from the side of the injection, the right column illustrates pursuit toward it. Note that the side of the injection (shown schematically) varies with different experiments. Pursuit away from the lesion showed the retinotopic deficit for pursuit initiation during the first 200 ms after the initial saccade to the target moving in the contralateral visual field. The monkeys established normal pursuit within 300–500 ms after the initial saccade. Pursuit toward the side of the lesion showed a retinotopic deficit in the contralateral visual field with the directional deficit superimposed throughout the visual field. The directional deficit resulted in reduced eye speed long after the acquisition of the target (300–to 500-ms periods), although by 500 ms the effect was slight in T2 and nearly gone in T1. Even for targets in the ipsilateral visual field, the directional deficit in pursuit speed just after the initial saccade was greater than it was 400–500 ms later.

We previously reported that retinotopic deficits in pursuit initiation were accompanied by errors in saccades to moving targets as well. In contrast, the directional deficit seems only to affect pursuit, leaving saccades to moving targets unimpaired. Figure 6 shows a quantitative measure of saccade amplitude for the same experiment illustrated in Figs. 1–4. Saccade error, the difference between eye and target position at the end of the saccade, is plotted against size of the step. For steps into the left (contralateral) visual field, saccadic amplitudes were consistently short for motion away from the side of the lesion (Fig. 6A) and were consistently long for motion toward the side of the lesion (Fig. 6B). In contrast, saccades to stationary targets (Fig. 6C) were largely unaffected by the lesion. This pattern of results is typical of the retinotopic deficit (30).

As can be seen in Fig. 1A, however, the directional deficit in pursuit was not accompanied by deficits in saccades. Note that the amplitudes of the initial saccades were normal (Fig. 1A), even though initial pursuit speeds were clearly reduced (Fig. 1B). This lack of reduction in saccadic amplitude is shown quantitatively for steps into the right visual field in the graphs in Figs. 6, A and B. Thus, in contrast to the retinotopic deficit, the directional deficit appears to involve mechanisms specifically related to the execution of pursuit eye movements.

In summary, the directional deficit for pursuit maintenance was observed when target motion was toward the side of the lesion, regardless of the visual field in which target motion began. This directional deficit did not include changes in the amplitude of saccades made to acquire targets. The retinotopic deficit, on the other hand, was apparent for initiation of pursuit to targets moving in the contralateral visual field, regardless of direction of target motion. Effects of the retinotopic deficit highly significant (P < 0.001) at all tested locations in the visual field. B: pursuit deficit throughout the visual field at successive times after the monkey acquired the pursuit target. Axes show the target step, the eye speed, and the time after the end of the saccade at which the speed was compared. The 0–to 100-ms range is shown to the left of the graph in the middle of the time range so that 0–100 ms, for example, is plotted at 50 ms. Where standard error bars are not shown, they are less than the size of the symbol. Postlesion pursuit speed failed to reach prelesion levels at any point during the 500-ms period following the end of the saccade, indicating that maintenance of pursuit toward the side of the lesion (the directional deficit) was reduced independently of the specific visual field (retinotopic) deficit.
FIG. 4. Spatial extent of the deficit for pursuit away from the side of the lesion. Same conventions as in Fig. 3.4: pursuit deficit during the first 100 ms following the initial saccade to the target moving at 16°/s. Following the injection, significant deficits were observed at 1°, 5°, and 10° in the left (contralateral) visual field but not the right (ipsilateral) visual field. B: pursuit deficit at successive times after the monkey had acquired the target. The deficit gradually became less pronounced from the first period (0–100 ms) to the last (400–500 ms). This is consistent with the interpretation that this deficit is a retinotopic one and that it results from inadequate information about target motion that occurred in a specific part of the visual field before the monkey acquired the target.
FIG. 5. Pursuit deficits observed following three additional injections (M2, T1, T2). For all monkeys, regardless of the side of the lesion, the pursuit is away from the side of the lesion in the left column and toward the side of the lesion in the right column. The left column shows the retinotopic deficit (primarily in the 0- to 100-ms period after the arcade), and the right column shows an additional directional deficit that persisted throughout the period of tracking. Same conventions as in Fig. 3. Note that the scale of the axis for target step size is different in B compared with A and C.
FIG. 6. Effects of the lesion on the amplitude of saccades. Ordinate is saccadic error, defined as the difference between eye position and target position at the end of the initial saccade. The sign of the saccadic error indicates whether the monkey overshot (+) or undershot (−) the target. Absolute is amplitude of target step (+ to the right, − to the left) as in Fig. 3. The mean and standard errors are for 10 saccades obtained at each step location before and 24 h after the injection. A: saccades to targets moving away from the side of the lesion. Postlesion saccades were hypometric for target motion in the left (contralateral) hemifield in the same part of the visual field that produced reduced pursuit speed (−1, −5, −10; see Fig. 3d). The values for zero target steps were for saccades made to targets moving away from the center of gaze without any initial target step. B: saccades to targets moving toward the side of the lesion. Again, significant saccadic errors occurred in the visual hemifield contralateral to the lesion (−10, −5). C: saccades to stationary targets were normal. Saccadic errors were associated only with the retinotopic deficit in the contralateral visual field.
included reduced initial pursuit speed and altered amplitude of the saccade made to the target.

**Speed dependence of deficits**

Our use of a single target speed (16°/s), raises the question of how the predictable speed influenced the monkey's pursuit. We therefore examined pursuit for different target speeds for one injection in each monkey. In a given block of trials the target speed varied randomly from trial to trial between two speeds (8 or 32, 12 or 24) in addition to moving toward or away from the center of gaze following different step sizes. The pursuit deficit was proportional to the speed of target motion for both monkeys, and results from one of the monkeys are shown in Fig. 7. The retinotopic deficit (Fig. 7A—for steps into the contralateral visual field, for target motion away from the side of the injection, and for pursuit just after the saccade to the target) was more pronounced at higher speeds. The deficits described above for target speeds of 16°/s in fact have been near minimal ones. The slope of the regression lines shows the change in eye speed with change in target speed, which is the gain of the pursuit. Before the injection of ibotenic acid the gain was 1.0, whereas after injection it dropped to 0.18. This substantial decrease in gain resulted from the largest lesions made (as we consider subsequently in Fig. 14).

The directional deficit (Fig. 7B—for steps into the ipsilateral visual field, for target motion toward the side of the lesion, and for a later period of pursuit after target acquisition) was also somewhat larger at higher speeds. The effect of the lesion on the gain of pursuit was not as great; the reduction was from 1.0 to 0.7.

The saccadic error associated with the retinotopic deficit was also proportional to target speed. Figure 8A shows saccadic error for targets stepped into the contralateral visual field moving away from the side of the lesion. The error increased with increasing speeds. In contrast, saccades to targets in the ipsilateral field, where pursuit showed a directional deficit, did not show a deficit at any speed (Fig. 8B).

For the retinotopic deficit, as target speeds increased, the magnitude of the impairment in pursuit speed and the size of the saccadic error both increased. Figure 9 shows the relation between these increases by plotting the eye speed error (difference in eye speed and target speed derived from results in Fig. 7A) against saccadic error (from Fig. 8A). The triangles show the postinjection results and the dashed line the regression line through the points. The correlation coefficient for the regression line is 0.92, indicating that the magnitude of the impairment in the speed of pursuit and the amplitude of the saccades to moving targets are closely related. This relationship provides further evidence that the two effects of the retinotopic deficit arise from the same basic impairment in visual motion processing.

**Pursuit of stabilized images**

Another method for testing the selectivity of the retinotopic deficit for visual motion processing is to measure pursuit generated in response to a position error rather than to a motion error. Pursuit eye movements can be maintained by the position error of an image stabilized on the retina in the absence of any retinal motion of the target (16, 19, 31). If the retinotopic deficit related to extraretinal targets is dependent on velocity and not position errors, pursuit of an image displaced from the fovea and stabilized on the retina should be largely unaffected by the lesion. By stabilizing the image in the parafoveal visual field affected by the injection, we were able to test this prediction. Unfortunately we were unable to study pursuit initiation under these conditions, since in our monkeys we found that stabilized images elicited a series of saccades rather than pursuit (see METHODS). Instead we studied the effect of displacing and stabilizing the target in the parafovea after pursuit had begun.

Figure 10 shows an example of a trial with pursuit of a stabilized image. The monkey initiated pursuit in the step-ramp task, as described previously, and we then stabilized the image on the retina by using eye position rather than the usual ramp input to drive the mirror galvanometer. In Fig. 10, the target stepped to the left and then moved smoothly to the right at 16°/s. The image was then stabilized and displaced 1.0° off the fovea in the direction of the monkey's pursuit at the time indicated by the vertical line on the abscissa. In this experiment the monkey was required to keep his eye within the window around the target (which he can not avoid during stabilization) and to detect a dimming of the target in order to receive a reward. Adding the re-
Fig. 7. Gain of pursuit derived using different speeds of target motion. Target speeds were 8, 12, 16, 24, and 32°/s. Circles indicate speeds before injection, triangles 24 h after injection. A: effect of varying target speed on the retnitopic deficit. Mean eye speed during the first 100 ms after the end of the initial saccade. Target motion was away from the side of the lesion for targets stepping 3° and 9° into the right visual field. Since both points were in the region of the right hemifield affected by the lesion, the data from the two points were combined. Regression lines were fitted using the method of least squares with correlation coefficients for the pre- and postlesion data of $r = 0.99$ and $r = 0.87$. The slope of the lines, the gain of the pursuit, went from 1.0 before to 0.18 after the lesion. B: effect of varying target speed on the directional deficit. Mean eye speed in the period 300–400 ms after the end of the initial saccade. Pursuit is toward the side of the lesion for steps 3° and 9° into the left visual field, which had no retnitopic deficit. The slopes of the regression lines (gain) went from 1.0 precision to 0.67 postlesion. Correlation coefficients were $r = 0.99$ and $r = 0.97$, respectively.
FIG. 8. Saccadic error for targets moving at different speeds. Same target steps and trials as in Fig. 7 for the retinotopic deficit (A) and the directional deficit (B). Saccadic error is the difference between eye position and target position at the end of the initial saccade. Saccadic error for target motion associated with the retinotopic deficit (A) increased with target speed, whereas saccadic error for target motion associated with the directional deficit (B) remained negligible at all speeds. Target locations are the same as in Fig. 7. Slopes of regression lines for pre- and postinjections are 0.07 and −0.13 in A and 0.73 and 0.76 in B. Correlation coefficients in A were 0.87 and −0.92 and in B 0.73 and 0.76.

requirement to detect the dimming produced better pursuit particularly late in the stabilization period.

For the experiment illustrated in Fig. 11, the injection was in the right hemisphere and affected the left visual field. Once the target was acquired, the target was stabilized 1° to the left of the fovea at the time indicated by the vertical dashed line on the abscissa. This target displacement moved the target into the
parafocal visual field affected by the lesion, as shown schematically on the drawing at the top of the figure. That the target displacement was into the affected field is shown on the graph in Fig. 5C, left, which shows that the visual field deficit for this injection extended at least 5° into the left visual field. Figure 11B shows mean eye speed before and after injections of ibotenic acid. The period of interest after stabilization is indicated by the horizontal dashed line. This line starts 100 ms after onset of stabilization to allow for the latency of the pursuit response (20, 23) and ends when saccades intrude in the prelesion records. During this 250-ms period, the monkey continued pursuit with little acceleration, and the pursuit after the lesion (dashed traces) was comparable to that before. This indicates that pursuit to a position error was not noticeably degraded by the lesion.

Figure 12 shows pursuit of stabilized targets for displacements to the left of 1.0° (Fig. 12A), 0.5° (Fig. 12B), 0.0° (Fig. 12C), and to the right of 0.5° (Fig. 12D); in the direction opposite to the direction of the initial pursuit). Pursuit speed in the period of interest following stabilization (dashed line along the abscissa) did not decrease after the lesion. The only case showing a significant decrease in pursuit speed during stabilization was the step backward (Fig. 12D); deceleration of ~20°/s). The step here was into the ipsilateral field and would not be expected to show any change related to the retinotopic deficit.

We examined pursuit of stabilized images following three other injections; pursuit with a position error under stabilized conditions was unaffected in all cases. However, since the monkeys in these additional experiments were not rewarded for detecting the dimming of the target, their pursuit tended to slow at the end of the stabilization period, presumably because their performance had no effect on the probability of obtaining a reward.

For all these lesions the position error under stabilized conditions failed to produce a deficit in pursuit, although a velocity error in the same area of the visual field did produce a deficit. To further emphasize this difference in position and velocity effects, we added a velocity error under stabilized conditions, as shown in Fig. 13 for the same experiment shown in Figs. 11 and 12. In Fig. 13A, the monkey with a lesion on the right initiated pursuit to a target moving to the left. After the monkey acquired the target with its fovea, the target was stabilized (time 0 on the abscissa), and a ramp of ~6.7°/s was added to the stabilized target. This produced an acceleration in the pursuit response in the normal monkey (solid lines in Fig. 13A, top) but no such acceleration after the injection (dashed lines in

![Graph showing comparison of pursuit and saccadic errors for the retinotopic deficit.](image)

**Fig. 9.** Comparison of magnitude of pursuit and saccadic errors for the retinotopic deficit. Speed error on the ordinate is the difference in eye speed and target speed from data in Fig. 7A, and saccadic error on the ordinate is derived from data in Fig. 8A. Triangles and the regression line (slope = 0.18, r = 0.92) are for pursuit data, circles are for prelesion data. The linear relation between speed error and saccadic error indicates that the lesion affects both pursuit and saccades to moving targets to the same degree.

![Graph showing eye position and time relationship.](image)

**Fig. 10.** Example of a step-ramp trial with image stabilization in a normal monkey. The target (dashed line) stepped to the left after the start of the trial and the subsequent saccade brought eye position (solid line) close to target position, the target position was stabilized and displaced 1° forward at the time of the left vertical line on the abscissa and remained stabilized until the time of the right vertical line. Initial target speed was 16°/s.
Fig. 11. Lack of a pursuit deficit during pursuit maintained by a position error under stabilized conditions. A: position traces before and after a lesion in the right hemisphere that produced a retinotopic deficit in the left visual field. The target stepped $10^\circ$ to the right and moved at $16^\circ/s$ to the left. The target was stabilized $1^\circ$ ahead of eye position at the time indicated by the vertical line on the abscissa. The target was therefore displaced into the hemifield with the retinotopic deficit, as shown by the schematic drawing. B: speed traces (mean and standard error) for the position traces shown in A. Solid lines are mean and standard error of speed before the injection, dashed lines are after the injection. The period of interest following stabilization is indicated by the dashed line on the abscissa. Because of the latency of the pursuit system, the line begins 100 ms after stabilization; it continues until saccades disrupt the precision speed record. Dotted lines indicate the hypothetical target position (A) or speed (B) if stabilization had not been used. Pursuit speed in the stabilized condition was as good or better after the lesion as it was before.
FIG. 12. Permit of a stabilized image with different position errors. Same experiment as in Fig. 11. The target stepped 10° to the right and moved leftward at 16°/s (as shown in Fig. 11,i) with stabilization starting at the dashed vertical line on the abscissa. The image was stabilized 1.0° (A) and 0.5° (B) ahead of eye position (to the left), on the front (C), and 0.5° behind (D), to the right. Posterior pursuit of the stabilized image was similar to pretension performance for all conditions tested.
FIG. 13. Pursuit of a stabilized image to which a fixed velocity error was added at the time of stabilization (6.7%/s in upper panels of A and B; 12.5%/s in lower panels). Stabilization occurred at 0 on the time scale of the abscissa and continued until the end of the trace. Eye acceleration is shown on the ordinate. A: velocity error in the direction of pursuit, into the hemifield with the retinotopic defect. Before the lesion (solid lines—mean and standard error) the monkey responded to the velocity error with a clear acceleration of the eye. This acceleration is absent following the lesion (dashed lines), indicating that response to velocity error under stabilized conditions was affected by the lesion. Periods during which the records are significantly different at $P < 0.01$ (Student’s $t$ test) are indicated by the solid line above the time scale. Saccades occurred later in the traces and are removed from the record. B: velocity error in target motion in the direction opposite to pursuit but still into the visual field with the retinotopic defect. The deceleration seen before the lesion was replaced following the lesion. Note that pursuit in this case is toward the side of the lesion.

Fig. 13, top). An attempted saccade ended the record. The acceleration produced by a 12.5%/s ramp (Fig. 13, bottom) also was eliminated by the lesion. The deceleration was also reduced when the same ramp velocities were subtracted from target speed (Fig. 13B). Thus addition of a velocity error, rather than a position error, during stabilization reveals a pursuit deficit. It should be noted, however, that these velocity steps are large in comparison to the position steps, as indicated by the accelerations they produce—a deceleration of $\sim 200^{\circ}/s^2$ in Fig. 13B, compared with one of $\sim 20^{\circ}/s^2$ in Fig. 12D.

Recovery

Both the retinotopic and directional deficits described were studied within $\sim 24$ h after the injection. On subsequent days there was a gradual recovery of both deficits until, after a week, little or no deficit remained. In one case, $T_2$, recovery was nearly complete 2 days after the injection. Recovery for both the retinotopic and directional deficits occurred roughly...
in parallel. Figure 14.4 shows the recovery in experiment M2 for target motion toward the side of the lesion. The records for target steps into the left visual field show the directional deficit, and those into the right field show an additional retinotopic deficit (line with solid circles compared with line with solid triangles in Fig. 14.4). The directional deficit and the retinotopic deficit both recovered (line with solid diamonds in Fig. 14.4).

One hypothesis advanced to explain a similar recovery following injections into extrafoveal regions of MT (30) was that cells in the region near to the injection were reversibly damaged and subsequently recovered full function. An exploration of this issue was attempted in experiment M2. The first question was whether a larger injection of ibotenic acid would lead to more prolonged deficits. The results for M2 shown in Figs. 5A, 7, and 8 followed a much larger injection than the 1-2 μl usually used. Eighteen microliters of ibotenic acid (10 μg/μl) were infused over a 24-h period using an osmotic pump. Recovery still occurred as shown in Fig. 14.4. To test whether an injection at the same location would rein-

Fig. 14. Recovery of pursuit following large and repeated injections of ibotenic acid into one location. The graph shows pursuit speed for target motion toward the side of the injection that was in the left hemisphere. In A, the p675 lesion records were obtained 1 day after an infusion for 23 h of ~18 μl of 10 μg/μl ibotenic acid; recovery was 4 days later. A second infusion over 2 days and an injection of 3 μl partially reinstated the deficit, as shown by the repeat line in A obtained on the day after the 3-μl injection. The deficit was maximal at this time. A total of 6 infusions and/or injections were made. B shows pursuit before the last infusion/ injection (ibotenic acid infused over 3 days with minimal effect followed by an injection of 7.5 μl) and the day after the injection. Recovery was slow but complete and is shown 24 days after the last injection.
state the deficit we then did a second infusion of ibotenic acid over a period of 3 days. The deficit was only partially reinstated (compare lines with solid diamonds and open triangles in Fig. 14A). The effect of the second injection, however, is of uncertain origin; it could have resulted from toxic effects on remaining cells in the originally affected area or from a spread of the ibotenic acid to adjacent areas. A subsequent series of infusions and injections (for a total of 6) produced a deficit following each that was not as pronounced as the original one but that recovered more slowly. Figure 14B shows the effect of the last of the series, an infusion of ibotenic acid over 3 days (which had only a slight effect) and a single injection of 7.5 nl ibotenic acid, all made ~6 wk after the original injection. The deficit was reinstated (compare the line with the circles with the line with the triangles in Fig. 14B), but recovery was complete 3 wk after the injection (line with diamond in Fig. 14B). Again, the deficit was less than the original one but recovery was slower. Although this series of experiments indicates that a longer-lasting behavioral deficit follows multiple injections, we do not know whether this results from repeated damage to the same area or invasion of adjacent regions.

**Histological reconstruction**

The extent of the cortical damage following the injections is shown on the two-dimensional cortical maps of Fig. 15. We used outlines of

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**FIG. 15.** Extent of cortical damage from ibotenic acid injections. Two-dimensional reconstructions of the cortex for the four hemispheres in this study (A–D) were created by the method detailed by Van Essen and Maunsell (45). For ease of comparison, maps made from right hemispheres were mirror reversed so that all four maps appear in the left hemisphere format. The light gauge _solid lines_ are the "unfolded" layer IV contours from which the maps were made. The _dashed lines_ indicate the fundus of the sylvian fissure (SF), the lunate sulcus (LS), and the superior temporal sulcus (STS), and the _heavy solid lines_ show the boundaries of the sulci. Anterior is to the right and medial is upward in the maps. The boundaries of MT were determined from myelin-stained sections and are shown in _heavy solid lines_. The _shaded area_ along a border of MT indicates a region of histological uncertainty in determining the border. MST (not drawn, since it is not anatomically identifiable) lies along the floor and anterior bank of the STS. The _solid black areas_ indicate ibotenic acid damage in all cortical layers and the _striped areas_ indicate regions of partial laminar destruction. The experiment designation is given at the _lower right-hand corner_ of each map. See text for other abbreviations.
histological sections to construct these maps centered on the STS using the method of Van Essen and Maunsell (45). The fundi of the sulci are shown by dashed lines, the edge of gyri by heavy solid lines. All plots are shown with posterior, including the lunate sulcus, to the left and the anterior to the right. MT was identified on myelinated stained sections (14), and the extent of the area was outlined; areas of transition or uncertainty at the edge are indicated by shading. MST is adjacent to MT and occupies part of the floor of the STS and extends onto the anterior bank. Areas of cell damage from the injection of ibotenic acid were determined from adjacent Nissl-stained sections with damage through all cortical layers indicated in black and damage to only some layers indicated by stripes. The central area of cell loss in all cases was centered in the lateral part of MT, as would be expected from the location of the foveal representation (46).

In experiments M1 and T1 two injections of 1 µl of 15 µg/µl ibotenic acid were made on successive days, whereas in T2 only one injection of 2 µl of the ibotenic acid was used. The only injection in which cell loss was limited to MT was that in experiment T1 (Fig. 15C). Injection M1 (Fig. 15A) spread slightly onto the anterior bank of the STS, T2 (Fig. 15D) was partially outside of MT onto the floor of the STS, whereas M2 (with a series of injections as described above—Fig. 14A) had extensive damage to the anterior bank of the STS and the lunate sulcus.

The area of damage in T2 (Fig. 15D) is particularly small and is the result of one 2-µl injection. Since the other lesions resulted from 1-µl injections on each of two successive days (M1 and T1) or of a series of injections (M2), this suggests that successive injections of ibotenic acid are more effective in killing cells than is one larger injection, but this would need to be confirmed with a more extensive parametric study. The minimal neuronal damage in T2 coupled with the very clear behavioral deficit following this injection (see Fig. 5C) raises the possibility that the behavioral deficit is, in part, due to a toxic but not lethal effect of ibotenic acid on cells. This in turn suggests that the area affected by the injection might be somewhat larger than indicated by the area of cell death found in histological sections. Since this injection is at the edge of MT, the toxic effect on surrounding cells would certainly have acted on cells in MST, which lies on this same edge of MT. In our previous experiments with injections placed well within the borders of MT (30), such spread was probably not critical, but in the present experiments with injections at the edge of MT, such a toxic halo almost certainly extends the effects of the injection into adjacent MST.

The large area of neuronal damage in M2 (Fig. 15B) followed the series of injections described above. This injection clearly includes damage to the anterior bank of the superior temporal sulcus, which includes part of MST. Backflow along the guide tube also produced damage in the lunate sulcus.

**DISCUSSION**

Injection of ibotenic acid into the foveal representation of the visual field within the STS has produced what we interpret as two deficits. The first is a directional deficit in the maintenance of pursuit, and the second is a retinotopic deficit in the initiation of pursuit and saccadic eye movements.

**Directional deficit**

We observed a directional pursuit deficit following damage to the foveal representation within the STS that we had not seen after lesions of the extrafoveal representation in MT. When the monkey pursued a target moving toward the side of the brain in which the foveal region was damaged, the monkey's pursuit speed lagged behind that of the target. Catch-up saccades returned the eye nearer to the target, but again eye speed was less than target speed, and the eye lagged behind. In contrast to the retinotopic deficit, which was limited to the contralateral visual field, the directional deficit was present regardless of whether in the ipsilateral or contralateral visual field the target initially appeared. The directional deficit affected pursuit speed but did not alter the amplitude of initial saccades to the target.

Our punctate lesions in the STS produced the same directional deficit that follows massive ablation of the cerebral hemisphere or the parietal-occipital cortex in monkeys (24, 41). The directional deficit in monkeys also resembles the neurological deficit typically observed
in man following cortical lesions. The clinical defect was first observed as a direction-specific reduction in gain of the slow phase of optokinetic nystagmus (3, 4, 12) and was subsequently observed for pursuit as well (21, 22, 37, 40). Like the defect in the monkey, that in man is independent of the visual hemifield in which the target moves and is not associated with a deficit in the amplitude of saccades to a moving target (21, 40). The defect in the monkey, however, results from very circumscribed cortical damage, whereas that reported in man has followed extensive loss of neocortex. In both man and monkey the deficit is an extraordinary one in that it is not a sensory field deficit, nor is it an inability to move a part of the body. Instead, it is a deficit of movement in response to motion in a given direction. What is also striking is that it affects movement toward the ipsilateral side and not the contralateral side.

The identification of this deficit in the monkey following localized damage to neocortex opens the possibility for an analysis of its neuronal basis. Cells in the foveal representation within MT show directional selectivity for motion (9), but why damage to these cells should produce the directional deficit is not obvious. Since the eye typically lags the target somewhat during pursuit, the target is generally moving in the visual field ipsilateral to the lesion for pursuit toward the side of the lesion. If a visual field deficit were the basis of the pursuit deficit, one would expect a deficit for target motion away from the side of the lesion (contralateral visual field). Furthermore, substantial asymmetries in the preferred directions of foveal MT neurons that might account for a directional deficit have not been observed (9). Finally, while foveal MT cells discharge vigorously during pursuit eye movements, removing the visual slip of the target by either blinking off the target or stabilizing the image of the target on the retina reduces the discharge of the cells (49), which suggests that these cells convey information about retinal slip, not about eye movement. In short, nothing currently known about the neuronal responses in the foveal representation of MT suggests a possible mechanism for a directional pursuit deficit.

The deficit may, however, involve areas within the STS other than MT. While the injections were placed within the representation of the fovea in MT, examination of the histology has shown that all injections with the exception of T2 invaded the adjacent area referred to as the medial superior temporal area, MST (27). As discussed previously, injection T2 indicates that the deficit may result, in part, from a toxic effect of the biotinic acid from which cells do not die but instead recover. If this is true then even the most limited injection within MT (T7) might have involved spread beyond the borders of MT.

MST is a reasonable candidate for a role in the cortical control of pursuit. MST receives direct projections from MT (27, 43), and several subregions have been identified (5). Many cells in MST show directional selectivity to moving stimuli, and the visual receptive fields of MST cells frequently include the fovea (5, 29, 46). Many MST receptive fields cross the vertical meridian and could thus convey information about visual motion in the ipsilateral visual field. However, the visual properties of these cells, like those in foveal MT, do not provide an obvious explanation for the directional deficit.

A subpopulation of MST cells discharges during pursuit eye movements even if the pursuit is against a dark background (29). These pursuit-related cells almost certainly overlap the population of pursuit cells studied previously by Sakata and his collaborators (35) that discharge in relation to pursuit movements in given directions. Furthermore, while many of these cells also have a visual response, it has recently been shown that the discharge of these cells continues even after the pursuit target is blinked off (35, 49) or the image is stabilized on the retina (49). These cells, in contrast to foveal MT cells, discharge in relation to the pursuit movement even in the absence of visual stimulation. The pursuit-related cells in MST may be the most relevant ones for understanding the directional deficit because their discharge is tied to the direction of pursuit movement, not to the visual stimulation within the contralateral visual field.

Two pieces of information would support this involvement of MST in the directional deficit. First, damage to MST alone should produce the deficit, and recent experiments indicate that injections centered on MST and sparing MT do produce a directional deficit.
(8). Second, a bias in the preferred directions of pursuit cells in MST would provide a possible basis for the directional deficit. A slight bias (10 of 16 cells) for movement toward the contralateral was obtained by Sakata (35), and recent work in our laboratory has also revealed a slight bias toward the contralateral side (Komatsu and Wurtz, unpublished observations).

Thus the pursuit-related cells of MST appear to be the most likely candidate for involvement in the directional pursuit deficit, but the actual mechanisms underlying the deficit remain to be determined.

Retinotopic deficit

The retinotopic deficit for pursuit initiation seen with chemical lesions of the extrafocal representation of MT (30) is also evident following injection of ibotenic acid directed toward the foveal representation of MT. The deficit in pursuit initiation is restricted to the contralateral visual field extending out from the fovea, and both the initial speed of pursuit and the amplitude of the saccade made to acquire a moving target are impaired. The deficit accompanies target motion in either horizontal direction. This pursuit deficit for target motion in the contralateral visual field is similar to that seen following unilateral ablation of striate cortex (36) or large bilateral occipital lobectomy (50, 51).

We have been able to further define the nature of the retinotopic deficit with several additional observations. We have found that the pursuit deficit for higher target speeds (24°/s to 32°/s) is more severely degraded than for the speed we have studied most intensively (16°/s). The pronounced deficit at higher speeds is consistent with the observation that many MT cells respond to speeds up to 128°/s (25) and that they maintain directional selectivity at such speeds (28, 33).

Measurement of pursuit at different speeds allows estimation of the gain of pursuit (eye speed/target speed), and for a large lesion, gain was reduced from 1.0 before the lesion to 0.18 after the lesion. Furthermore, the magnitude of the deficits in initial pursuit and in saccade amplitude grew in parallel as target speed increased, suggesting that both the deficits in pursuit and saccades result from damage to the same underlying mechanism. These results taken together strengthen the previous conclusion that the pursuit deficit following damage to MT results from an inability to determine the speed of target motion (30).

Our hypothesis has been that while MT provides information on velocity error it does not do so for position error. This idea is based on the observation that saccades to moving targets are affected by MT lesions, whereas saccades to stationary targets are not (as illustrated in Fig. 6 for the current experiments). The distinction between velocity and position is supported by the present experiments that introduce a position error during pursuit. In this case, pursuit was maintained under stabilized image conditions, so that no velocity information was present. Damage to an area that conveys velocity information, such as MT, should have minimal effect on this type of pursuit, and this was found to be the case. Generation of pursuit by a position error therefore would appear to be mediated by areas other than those affected by our lesions of the STS. Damage to striate cortex, for example, does reduce a monkey's ability to pursue a stabilized image (36). Our understanding of the function of MT in relation to oculomotor control seems to be strengthened: MT appears to provide information about motion, but not position errors. Our new observations lend further support to the more general argument that MT is selectively related to visual processing of motion information.

Recovery of function

We have found rapid recovery of the directional deficit just as we previously found rapid recovery for the retinotopic deficit (30). Several aspects of the present experiments may shed light on the nature of the recovery.

One question from the previous experiments was whether recovery of neuronal function in and around the injected region contributes to the recovery of pursuit. The minimal neuronal damage shown by the histology in experiment T2 and the rapid recovery in this case suggests that the smaller the number of cells killed, the more rapid the recovery. At the other end of the continuum, repeated injections in experiment M2 that led to cell loss over a large area, led to slower recovery. The problem with this interpretation—more cell loss and slower recovery—is that the larger injections invade several ana-
tomically and physiologically distinct areas as we have already discussed. Part of this slowed recovery could be due to damage to adjacent areas not just damage to more cells in the same area. Current experiments in which damage from ibotenic acid injections is primarily in the adjacent areas such as MST also frequently show rapid recovery of pursuit function (Dürsteler and Wurtz, unpublished results) so that invasion of this area alone probably cannot explain the more persistent effect with larger injections.

Much larger unilateral lesions produced surgically and including all of the occipital and much of the parietal cortex (41) show eventual recovery but over a period approaching a year rather than a week. Monkeys with unilateral surgical ablation limited to striate cortex show only limited recovery when tested over a year after the ablation (36). Thus these studies show that either through direct damage to prestriate areas (41) or massive deafferentation (36), extensive damage leads to dramatically slower recovery. What remains a question, however, is whether the more extensive damage produces more lasting deficits because it eliminates a critical area or because it eliminates a number of equally important areas. Selective lesions within different prestriate areas might help to resolve this issue of localization, but, in part, because of the uncertainty of the borders between areas outside of MT, this will be a difficult task. The question is an important one, however, not only for understanding the neural mechanisms related to pursuit eye movements, but for determining the degree of functional localization within cerebral cortex as well.

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PRESENT ADDRESSES: M. R. Dürsteler, Neurologische Universitätsklinik Frankfurt, Rämistrasse 100, CH-6000 Zürich, Switzerland; W. T. Newsome, Dept. of Neurobiology and Behavior, State University of New York, Stony Brook, NY 11794.

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