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Pursuit and Optokinetic Deficits Following Chemical Lesions of Cortical Areas MT and MST

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SUMMARY AND CONCLUSIONS

1. Previous experiments have shown that punctate chemical lesions within the middle temporal area (MT) of the superior temporal sulcus (STS) produce deficits in the initiation and maintenance of pursuit eye movements (10, 34). The present experiments were designed to test the effect of such chemical lesions in an area within the STS to which MT projects, the medial superior temporal area (MST).

2. We injected ibotenic acid into localized regions of MST, and we observed two deficits in pursuit eye movements, a retinotopic deficit and a directional deficit.

3. The retinotopic deficit in pursuit initiation was characterized by the monkey's inability to match eye speed to target speed or to adjust the amplitude of the saccade made to acquire the target to compensate for target motion. This deficit was related to the initiation of pursuit to targets moving in any direction in the visual field contralateral to the side of the brain with the lesion. This deficit was similar to the deficit we found following damage to extrafoveal MT except that the affected area of the visual field frequently extended throughout the entire contralateral visual field tested.

4. The directional deficit in pursuit maintenance was characterized by a failure to match eye speed to target speed once the fovea had been brought near the moving target. This deficit occurred only when the target was moving toward the side of the lesion, regardless of whether the target began to move in the ipsilateral or contralateral visual field. There was no deficit in the amplitude of saccades made to acquire the target, or in the amplitude of the catch-up saccades made to compensate for the slowed pursuit. The directional deficit is similar to the one we described previously following chemical lesions of the foveal representation in the STS.

5. Retinotopic deficits resulted from any of our injections in MST. Directional deficits resulted from lesions limited to subregions within MST, particularly lesions that invaded the floor of the STS and the posterior bank of the STS just lateral to MT. Extensive damage to the densely myelinated area of the anterior bank or to the posterior parietal area on the dorsal lip of the anterior bank produced minimal directional deficits.

6. We conclude that damage to visual motion processing in MST underlies the retinotopic pursuit deficit just as it does in MT. MST appears to be a sequential step in visual motion processing that occurs before all of the visual motion information is transmitted to the brainstem areas related to pursuit. The directional deficit, on the other hand, might reflect damage to a directional visual signal found in MST but not in MT.

7. Deficits in optokinetic nystagmus (OKN) were also produced by the chemical lesions in MST. The slow buildup of OKN velocity was reduced for drum motion toward the side of the lesion. This deficit tended to be associated with the injections producing the clearest directional pursuit deficits. Lesions in MST also reduced the initial rapid rise in OKN. This reduction was for stimulus motion both toward and away from the side of the lesion, with initial OKN for motion toward the lesion showing the greatest reduction. This deficit tended to be associated with those lesions that showed a prominent retinotopic deficit.

8. We conclude that the areas within the STS included within our chemical lesions

make two contributions to the optokinetic system, one via the indirect pathway to the velocity storage mechanism of OKN, the other via a direct pathway shared with the pursuit system. Areas within MST and MT are likely to be differentially related to these two contributions.

INTRODUCTION

Two deficits in pursuit eye movements have been identified following lesions limited to the superior temporal sulcus (STS) of the old world monkey. A retinotopic deficit for initiation of pursuit has been identified following injection of ibotenic acid into the middle temporal area (MT): the monkey is unable to match the speed of its pursuit eye movements to that of a target or to adjust the amplitude of its saccades to compensate for target motion (34). This retinotopic deficit is limited to pursuit initiation for targets moving in the visual field contralateral to the side of the brain with the damaged MT. The direction of target motion is unimportant, but the area of the visual field in which the target moves is critical. A directional deficit in maintenance of pursuit follows injection into the foveal representation of MT and the adjacent medial superior temporal area (MST): the monkey is unable to maintain pursuit after the fovea has been brought near the target when the target moves toward the side of the brain with the lesion (10). The area of the visual field in which the target moves is unimportant, but the direction of motion is critical.

The retinotopic deficit can be readily understood if it is assumed that the lesion affects visual motion processing in area MT. The restriction of the deficit to the same contralateral visual field as the visual receptive fields of MT cells, and the alteration of both pursuit and saccadic movements when they are dependent on motion information is consistent with this interpretation. The directional deficit is not so easily understood. One hypothesis, however, is that damage to the adjacent area, MST, leads to this directional deficit (10). MST receives a direct projection from MT (30, 51) and has cells like those in MT that show directional selectivity (1, 8, 20, 46, 53). MST also has cells that discharge during pursuit eye movements, and because this discharge is in many cases independent of the visual stimulation from retinal slip occurring during pursuit, it seems to result from an extraretinal source (35, 41).

One way to test this hypothesis that MST is related to the directional deficit is to selectively damage this area while sparing MT. We have made such lesions in MST using a neurotoxin, ibotenic acid, and have found directional deficits. We also found that lesions of MST, unlike those of MT, frequently produce retinotopic deficits affecting a large portion of the contralateral visual field.

Another type of oculomotor response that is dependent on visual motion is the optokinetic response that aids in producing a stable image on the retina in spite of motion of the head and body. The large size of the visual field affected by our lesions suggested that optokinetic nystgamus (OKN), which is dependent on large field stimulation, might also be altered by the lesion. We found two distinct deficits in OKN: a decrease in the slow buildup of OKN velocity and a decrease in the initial rapid rise in OKN.

A brief report has appeared previously (9).

METHODS

Experiments were performed on five monkeys (*Macaca mulatta*) and consisted of three methodological steps: behavioral training, injection of ibotenic acid, and data analysis.

Behavioral task

The step-ramp paradigm for initiation and maintenance of pursuit was identical to that described in a previous report (34), and our procedures need only be outlined here. In each trial, after the monkey began to fixate on a small spot of light in the center of the tangent screen in front of it, this light went out and a second light appeared at one of several possible locations on the horizontal meridian. This target light either remained stationary (step trial) and the monkey then made a rapid or saccadic eye movement to the target, or the target moved away from or toward the fovea along the horizontal meridian (step-ramp trial) and the monkey made a saccade followed by pursuit of the target. The size and direction of the target step, the direction of target motion, and whether the trial was a step or step-ramp were randomized. The monkey was required to keep its eye within an electronic window around the target and to detect the dimming of the target in order to obtain a liquid reward. In one experiment (*W2*) the reward was given just for keeping the eye within the window.

The stimulation conditions were also identical to those used previously (10). In addition to the usual dark background, we also used a patterned background that consisted of random dots (taken from Ref. 16) that was back-projected onto the screen in front of the monkey. The smallest dots subtended about 0.5° and luminance in the light areas was 0.2 cd/m^2.

We tested optokinetic nystagmus (OKN) by placing the monkey in a drum with a radius of 34 cm that had alternating black and white stripes at 1.6 or 15.6°/cycle. Blocks of five trials at a given speed and in a given direction were usually run with alternation of clockwise and counterclockwise rotation. A 5-s period of recording in the dark was followed by 30 s of exposure to the rotating drum in the light, to observe buildup of OKN, and then by another 60–120-s period in the dark for recording optokinetic after-nystagmus (OKAN). The drum rotated continuously during periods of darkness, and visual stimulation was started or stopped by turning on or off the light. To maintain a roughly constant level of alertness, we rewarded the monkey every 0.5 s if he kept his eyes within 10° of the horizontal meridian. We found that such a reward was essential for obtaining consistent records. To study the effect of stimulation of one eye, a patch was attached to the monkey's head holder which allowed occlusion of one or the other eye during a series of tests.

The monkey's weight was checked each day and supplementary water and fruit were provided if needed throughout both the training and experimental periods. The monkeys sat in primate chairs during experiments, but each day after the experimental session they were returned to their home cages.

Injection of ibotenic acid

We used the same fairly standard procedures described in a preceding paper (20) for implantation of a headholder, recording cylinder, and magnetic search coil. These procedures were performed under general anesthesia (pentobarbital sodium) following pretreatment with ketamine hydrochloride. After surgery, monkeys were given analgesia for several days and allowed to recover for at least 1 wk before beginning experiments.

Before doing injections we first located area MT within the STS and then explored areas on the anterior bank and floor of the sulcus. Penetrations were made in the stereotaxic plane which meant the electrode entered the parietal cortex, advanced through the anterior bank of the STS, and then into the posterior bank or floor of the sulcus. Once the area was localized we implanted a guide tube so that the tip was 3 mm above the area of interest. We confirmed this location of the guide tube by recording neuronal activity with a tungsten electrode (Haer) introduced through the guide tube and confirmed the location again at the time of injection by recording through a fine wire within the barrel of the 10-μl syringe (Hamilton) used for injection.

Prelesion data were obtained over a period of several days after the guide tube was in place; any effect due to implantation of the guide tube was included in the prelesion observations. Following this behavioral testing we injected 2–5 μl of 10–15-μg/μl ibotenic acid in a basic saline solution. The monkeys showed no signs of discomfort during or after injection. In one case (*W2*) we made only one injection, in four cases (*B1, B2, P, N1*) we followed the injection on one day by a second injection on the next day, and in two cases (*W1, Y*) we made a third injection on the following day. Multiple injections were used to insure a histologically clear lesion. In one monkey (*B2*) we inserted two guide tubes in order to produce a lesion over a larger area, and we made injections in each of these tubes on each of 2 days. All postlesion results reported were taken after the last of the series of injections.

Recovery of function followed all of the injections and was largely complete in ~2 wk, as had been the case in our previous experiments with ibotenic acid lesions in the STS (10, 34). In the present experiments, we concentrated on localizing deficits within the STS, we did no further experiments on the nature of the recovery, and we will consider recovery no further. The recovery did, however, allow us to use the second hemisphere of each monkey for a second series of experiments after recovery from the first injection was complete. In monkeys in which injections were made in both hemispheres, the first injection was designated as *experiment 1*, the second as *experiment 2*. A total of seven injections were made, and they were designated as *W1, W2, B1, B2, N1, Y*, and *P*.

At the end of the experiments, the monkeys were deeply anesthetized and perfused with saline and 10% Formalin. Maps of the STS region of cortex were created based on the method of Van Essen and Maunsell (52) using sagittal sections as described previously (20). The extent of the lesions were drawn on these maps.

Data analysis

Horizontal and vertical eye position, recorded using the magnetic search-coil technique, were filtered using a Bessel filter with a bandpass of 0–200 Hz (−3 dB). These channels were then digitized at 500 Hz; the system had a resolution of 0.1°. Eye and mirror position were also available for on-line display. Mirror position was also digitized and stored. The behavior of the monkey, stimulus presentation, as well as the storage and display of the data, were controlled by a real-time experimental system (REX), developed by Hays et al. (14), which ran on a PDP-11/40 computer.

Analysis of eye movement records was done off-line. The position record was first differentiated using a finite impulse response filter (37) with a high-frequency cutoff of 13.6 Hz (−3 dB). To obtain acceleration the position record was filtered once but differentiated twice. An acceleration criterion was used to identify saccades. If acceleration exceeded $450° \cdot s^{-1} \cdot s^{-1}$ and if within a 100-ms period acceleration reached a second criterion ($550° \cdot s^{-1} \cdot s^{-1}$), onset of a saccade was recognized. We adopted these dual criteria to reduce false positives due to noise. The beginning of the saccade was marked where the initial eye acceleration reached 80% of the maximal saccadic acceleration. The end of the saccade was marked where the final acceleration fell below 80% of the maximal acceleration. These criteria for the beginning and end of the saccade were used for identifying both the initial saccade to a moving target, and the subsequent catch-up saccades. The error in amplitude of a saccade to a target, the saccadic error, was taken as the difference between the eye position at the end of the saccade and the target position at that time.

Once the saccade had been identified, a time period of 28 ms was eliminated before and after the saccade to insure that the influence of the saccade on the digital filter was not included in the remaining records. The velocity of pursuit after a saccade was then averaged over successive 100-ms periods, out to 500 ms beyond the initial saccade. Records from trials were eliminated if the saccade to the step-ramp target occurred before the target came on, or if the monkey made a saccade in the opposite or a grossly inappropriate direction. In addition, data from trials were not stored in the experiment if the monkey's eye did not arrive in the window around the step-ramp target within 350 ms after the target came on. No other records were removed from the data.

Analysis of the records obtained during optokinetic nystagmus experiments used the same differentiation procedures to produce velocity and acceleration records. Records were stored in 500-ms epochs and the analysis program removed all eye movements that were not part of the slow phase of OKN. The acceleration criteria used for keeping slow phase records was therefore set deliberately low: eye acceleration had to remain below $300° \cdot s^{-1} \cdot s^{-1}$ for at least 100 ms. For display of the OKN records the slow phase periods were broken up into 100-ms periods (or leftover periods > 50 ms) and the mean velocity taken for each period. Means and standard errors were then computed for each of the periods over 5–10 OKN trials.

RESULTS

Pursuit deficits

We found two deficits following injections of ibotenic acid into area MST that we have described previously (10, 34): a retinotopic deficit and a directional deficit. We will first define these two deficits briefly by illustrating them with one experiment, then show how the deficits vary with the location and extent of the lesion within the STS, and finally briefly describe several additional observations.

RETINOTOPIC AND DIRECTIONAL DEFICITS. We used a step-ramp task that allowed us to place the moving target on successive trials at different points on the horizontal meridian depending on the size and direction of the step. We then tested pursuit with targets moving either to the left or to the right. Figure 1 shows an example of the deficits observed following an injection into the right hemisphere of a monkey with target motion towards the side of the injection. The 10 superimposed eye position records at the top of Fig. 1 show the saccades made to the target and the subsequent pursuit of the target both before (pre-) and 24 hours after (post-) the injection of ibotenic acid into MST. The step placed the target in the visual field contralateral to the hemisphere with the lesion (referred to subsequently as the contralateral visual field). Pursuit speed decreased as shown by the mean and standard error of speed in the lower section of Fig. 1. The decrease in speed was evident just after the saccade (which is removed from the record and appears as a gap in the speed trace). In this experiment the decrease in pursuit speed was small but still statistically significant as indicated by the lines above the *abscissa* showing the times when differences between pre- and postlesion speed had a $P \leq 0.01$. In prelesion trials pursuit began before the saccade to the target, and this pursuit was also abolished. Furthermore the amplitude of the saccade did not compensate for the motion of the target; compared to prelesion trials, the saccade was too long. These deficits in pursuit initiation (speed and saccadic amplitude) for targets moving in the contralateral visual field we have referred to as retinotopic pursuit deficits (10, 34).

We would expect the retinotopic deficit in Fig. 1 to decrease rapidly after the monkey made the saccade to the target since the saccade moved the target from the contralateral visual field onto the fovea. Only the first 100- to 200-ms period after the saccade should be relevant for this deficit; because of the latency of the pursuit system, pursuit at this time is

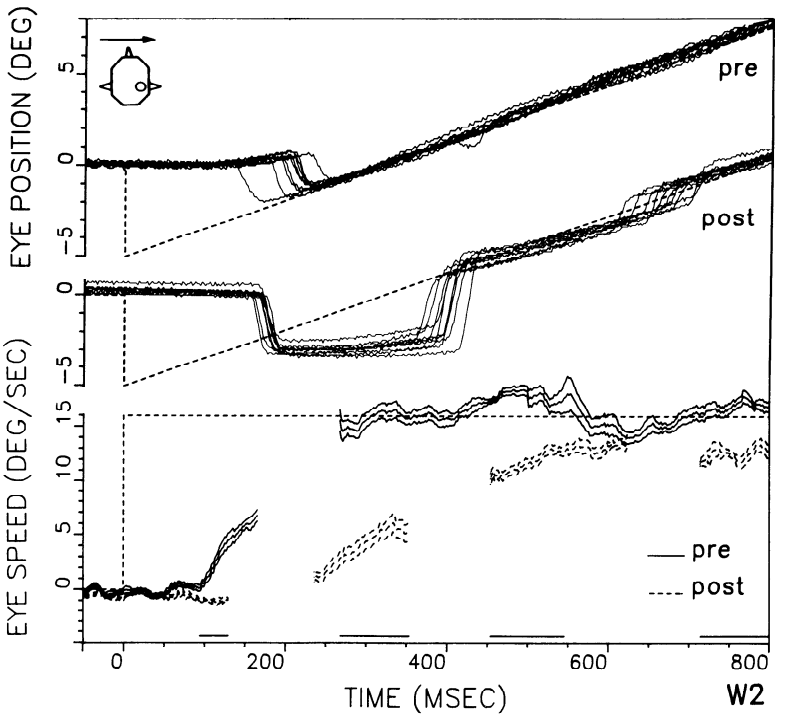


FIG. 1. Retinotopic and directional pursuit deficits. Records show sample position (*top*) and speed (*bottom*) traces for a lesion in the right hemisphere as indicated by the schematic drawing in the *upper left*. The target stepped 5° into the left (contralateral) visual field and moved back toward the fovea at 16°/s. Top eye position trace (pre-) shows 10 superimposed trials; *dashed line*: target position. The lower set of position traces shows 10 superimposed trials 24 h after injection of ibotenic acid (post-). The eye speed traces in the *lower part* of the figure show the mean and standard error for the same position traces pre- and postlesion. Where the mean speed before and after the lesion was significantly different at the $P \leq 0.01$ level (using a Student's t test) a *solid line* was drawn just above the *abscissa*. There was an initial deficit in pursuit after the target was acquired in the visual field contralateral to the lesion (largely a retinotopic deficit) and a continuing failure to match eye speed to target speed (directional deficit). In this and subsequent figures, the experiment number is indicated in the *lower right corner*.

dependent upon target motion at least 80 ms earlier (23, 28). However, in Fig. 1 the eye did not match target speed even several hundred ms after the saccade to the target. This failure to match eye and target speeds occurred only when the target moved toward the side of the lesion, but it persisted even with target steps into the ipsilateral visual field where no retinotopic deficit was observed. We have referred to this deficit as a directional pursuit deficit (10).

These deficits can be more clearly defined if we consider different directions of motion following different size steps of the target into the ipsilateral and contralateral visual fields. Figure 2*A* shows eye speed for target motion to the left (away from the side of the lesion) in order to illustrate the retinotopic deficit. The *abscissa* shows pursuit speed for a series of steps into the contralateral (left) and ipsilateral (right) visual fields for the same experiment shown in Fig. 1. The mean and standard error for eye speed averaged over the first 100 ms after the saccade to a target are shown for 10 prelesion (*filled circles*) and 10 postlesion trials (*filled triangles*). Eye speed decreased significantly after steps of 1 and 5° into the contralateral visual field, whereas speeds following steps beyond 5° were minimally affected. Figure 2*B* shows the change in pursuit speed over time after the initial saccade to the target and shows that the retinotopic deficit in the left visual field has disappeared by the 200–300-ms period after the saccade to the target. Thus we found a small retinotopic deficit in the first few hundred ms after the target was acquired when the step placed the target in a limited region of the contralateral visual field. We have illustrated the retinotopic deficit for motion away from

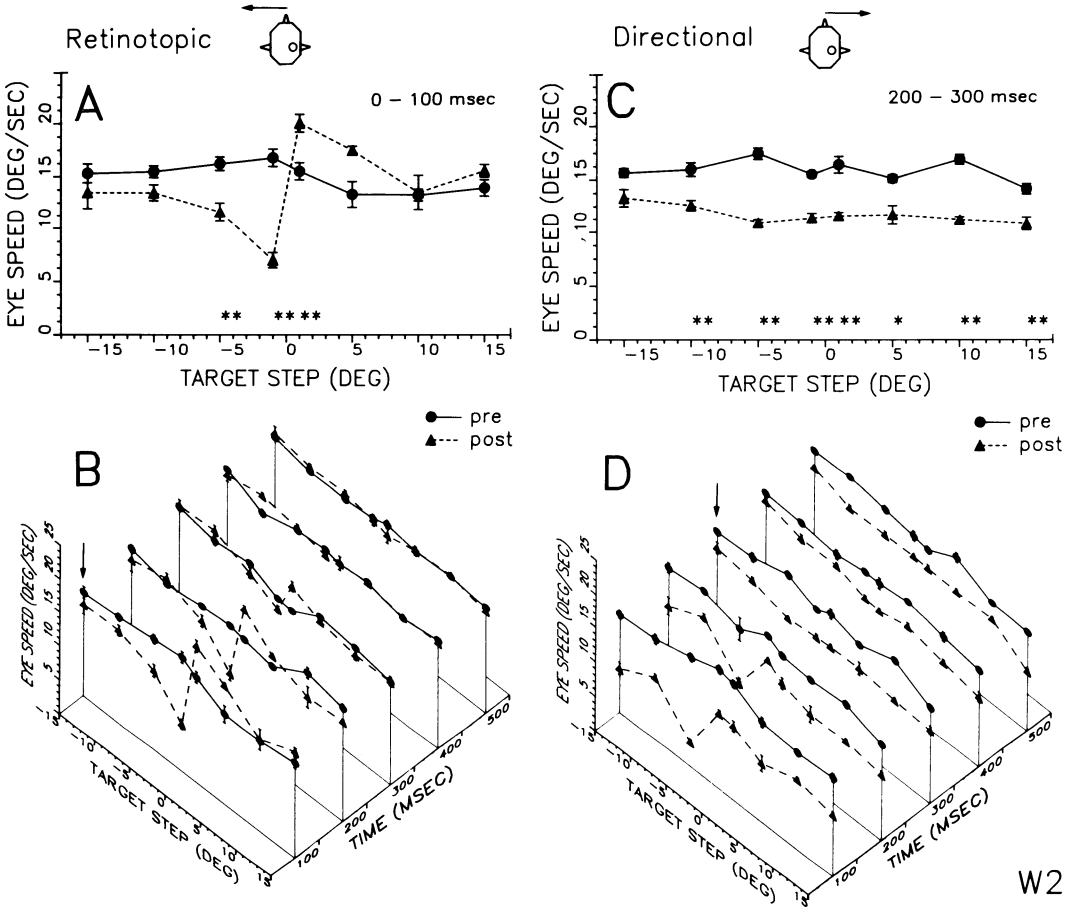


FIG. 2. Visual field specificity of the retinotopic and directional pursuit deficits. *A*: deficit for pursuit away from the side of the lesion. Target motion was to the left at 16°/s. *Abscissa*: step size from the primary position with rightward steps positive, leftward steps negative. *Ordinate*: horizontal eye speed averaged over the first 100 ms following identification of the end of initial saccade. In this and subsequent figures, the means and standard errors for each point were derived from 10 pursuit responses for each step location before (pre-) and 24 h after (post-) the injection. ** *along the abscissa*: significance level of $P \leq 0.001$ as determined by a Student's t test, *: level of $P \leq 0.01$. The retinotopic deficit was small: 1 and 5° on the left and was accompanied by a significantly increased pursuit speed at 1° on the right. *B*: pursuit deficit throughout the visual field at successive time periods after the initial saccade to the pursuit target. Pursuit was always away from the side of the lesion as in *A*. *Axes*: target step, the mean and standard error of eye speed, and the time after the end of the saccade at which eye speed was computed. The 0–100-ms range shown (indicated by the *arrow*) is the same as that in *A*. The mean of any time range on the graph is plotted at the middle point so that the 0–100-ms mean is plotted at 50 ms. The retinotopic deficit (in the left visual field contralateral to the side of the lesion) disappeared by the 200–300-ms period. *C*: pursuit deficit for target motion towards the side of the lesion. Pursuit speed is for the 200–300-ms period after the monkey acquired the target moving to the right. The decreased pursuit speed was clear for target steps extending throughout the visual field, was significant for all points but one, and illustrates the directional pursuit deficit. *D*: eye speed for different target steps at different times after the monkey made saccades to a target moving toward the side of the lesion. The 200–300-ms period on this graph (indicated by the *arrow*) is the same as the record in *C*. At all times the eye speed for this direction was reduced, but the amount of decrease clearly declined as time after the initial saccade increased.

the side of the lesion, but the deficit was present for motion toward the side of the lesion as well, as long as that motion was in the contralateral visual field.

Figure 2*B* also shows a tendency to increase pursuit speed in the visual field ipsilateral to the lesion above what it was before the lesion. This increase in speed might reflect an attempt by the monkey to compensate for the pursuit deficit by turning up the gain of the pursuit system in general. A compensatory increase in gain of the pursuit system has re-

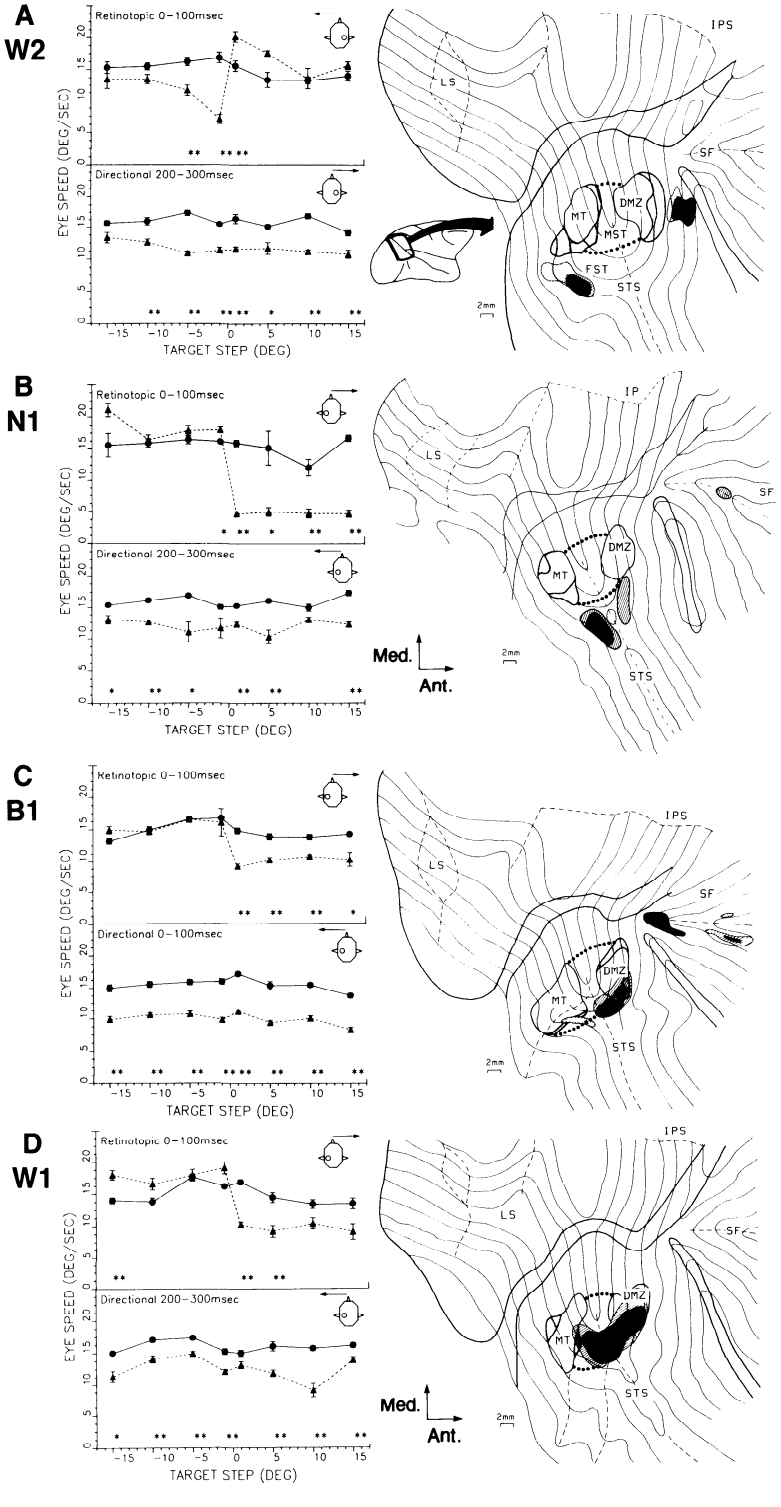
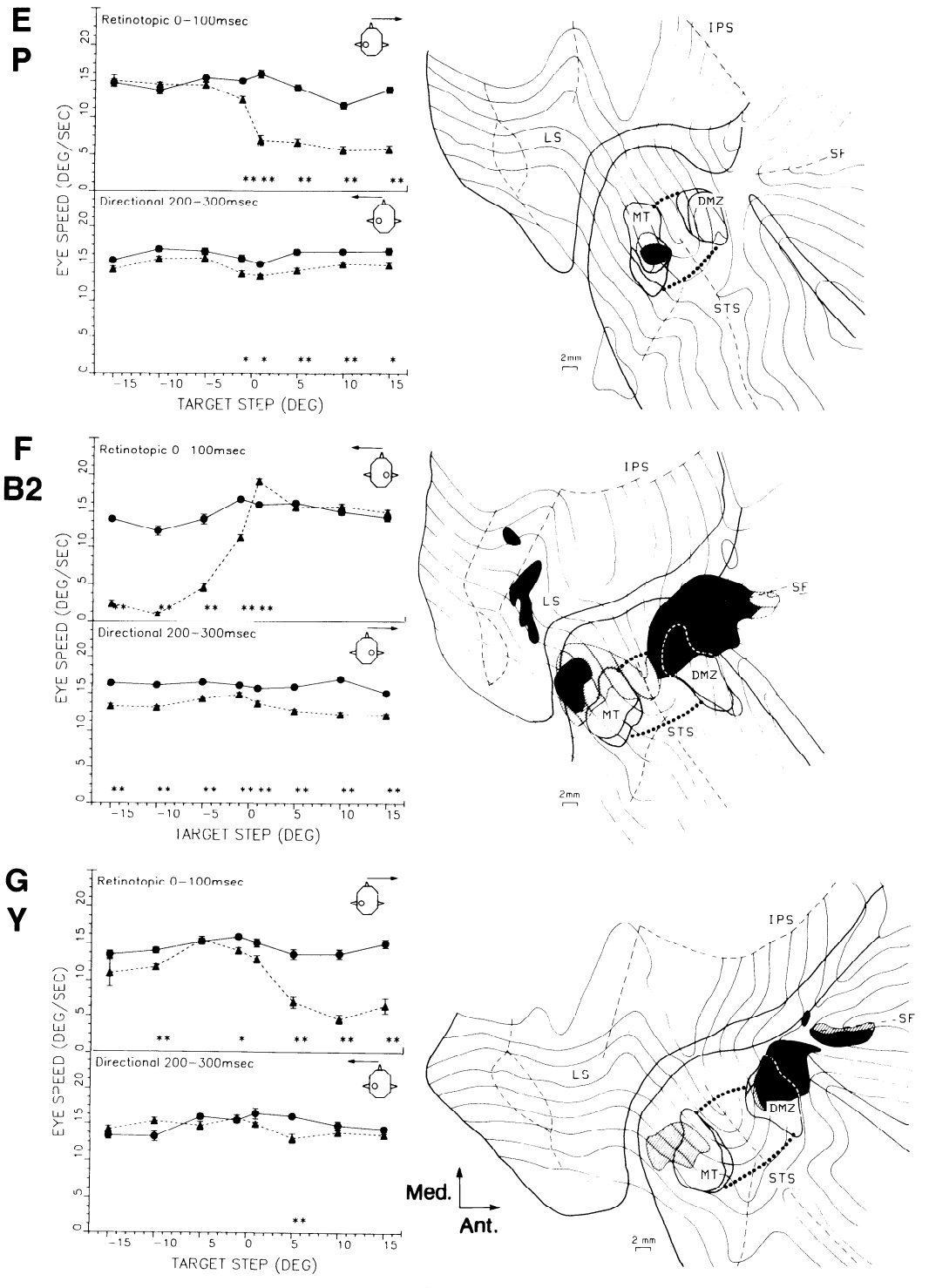


FIG. 3. Comparison of retinotopic and directional deficits with location and extent of the ibotenic acid lesion in the superior temporal sulcus. On the *left* of each section of the figure, the *upper graph* shows the retinotopic deficit in the 0–100-ms period following the initial saccade to the target moving away from the side of the lesion, and the *lower graph* shows the directional deficit for a target moving towards the side of the lesion with the mean time taken 200–300 ms after the initial saccade (with the exception of *C* where time is 0–100 ms). Same conventions as in Fig. 2, *A* and *C*. The *right* side of the figure shows the areas within the superior temporal sulcus and the damage done by the injections of the ibotenic acid. The brain area has been mapped using the procedures described in METHODS. The drawings show the superior temporal sulcus (STS), the lunate sulcus (LS), the intraparietal sulcus (IPS), and the



sylvian fissure (SF). *Dashed lines*: fundi of sulci including the expansion of the fundus to become the floor of the STS as in *C–G*. *Solid lines*: crests of the sulci. Regions damaged throughout all cortical layers are in *solid black*; regions with only partial damage in some layers are indicated by *hatching*. MT is outlined with uncertainty at the edges shown by *fine stippling*. The area of uncertainty at the lateral edge of MT on the floor of the STS is, if anything, somewhat exaggerated to make certain the area of MT is adequately outlined. This uncertainty is particularly large at the lateral edge of MT due both to the sharp angle of the floor of the sulcus to the plane of the histological section and to increased ambiguity when the chemical lesion invades this area. MST includes a densely myelinated area on the anterior bank (DMZ) and is shown enclosed by the dotted lines on all drawings. Area FST is indicated more laterally.

cently been observed in humans with ocular muscle weakness (36). This would produce the higher pursuit speed where no deficit was present in the ipsilateral field. This increase is also evident in the field ipsilateral to the retinotopic deficits in some of the other experiments (see graphs in Fig. 3, *D* and *F*), and might reflect the strategy of the individual monkey in dealing with its pursuit deficit.

Figure 2*C* shows the directional pursuit deficit for target motion toward the side of the lesion, also for the same experiment shown in Fig. 1. Here the mean speed taken is 200–300 ms after the initial saccade to the target so that, where present, the contribution of the retinotopic deficit is minimized. The striking feature of Fig. 2*C* is the reduced pursuit speed following steps throughout the visual field, with all points but one showing a significant difference between pre- and postlesion records. The time course of the pursuit after the initial saccade to the target (Fig. 2*D*) shows that for pursuit toward the lesion the deficit persisted throughout the period studied (up to 500 ms after the saccade). The magnitude of the deficit does, however, decline over time (compare, e.g., 0–100 with 400–500 for the right, ipsilateral, field where no retinotopic deficit is evident). Note also in Fig. 2*D* that the deficit is largest in the left, contralateral, visual field in the first several hundred ms after the initial saccade (compare 0–100 with 200–300); we interpret this as a combination of the directional and retinotopic deficits in this part of the visual field.

PURSUIT DEFICIT AND LESION LOCATION. Figure 3 compares the magnitude of the retinotopic and directional deficits to the area within the STS damaged by the ibotenic acid lesions. The *left side* of Fig. 3 shows the magnitude of the pursuit deficits by using graphs for each lesion identical to those described in Fig. 2, *A* and *C*. The retinotopic deficit is summarized by showing the reduction in pursuit speed after the monkey acquired a target moving away from the side of the lesion (see the drawing in the upper right corner of the graph). The relevant side of the graph for the retinotopic deficit is that showing the contralateral visual field. The directional deficit is indicated by showing target motion toward the side of the lesion. The relevant side of the graph for the directional deficit is that related to the ipsilateral visual field.

The *right side* of Fig. 3 shows maps that unfold the superior temporal sulcus (STS) and aid in localizing the lesions. *Black areas* indicate damage by the injections to all layers of cortex; *striped areas* indicate only partial damage. MT is identified on all sections of Fig. 3 by outlining the densely myelinated area on the posterior bank of the STS. MST includes the densely myelinated zone (DMZ) on the anterior bank and extends posteriorly to MT on the posterior bank of the STS. MST is labeled on Fig. 3*A* as the area including the DMZ on the anterior bank, bordering on MT on the posterior bank, and falling between the dotted lines connecting these two areas. This definition of MST is generally comparable to that introduced by Desimone and Ungerleider (8) and refers to that fraction of the projection zone of MT within the STS having a high proportion of directionally selective cells (see Ref. 20 for discussion). This definition is more restrictive than the original definition of MST as the entire projection zone of MT within the STS (30), and because this definition depends on both anatomic and functional information, which we have not obtained in these experiments, the dotted line outline of MST shown in each section of Fig. 3 can only be regarded as an estimate based on previous experiments in our laboratory (20). The remaining area of the MT projection zone within the STS has a high proportion of cells that are visually responsive but not such a high proportion that are directionally selective. This area is lateral to MT on the floor of the STS, has been named FTS (8), and is so labeled on Fig. 3*A*.

In Fig. 3 the experiments are arranged so that the most striking directional deficits are placed first (Fig. 3*A*), the least, last (Fig. 3*G*). The first major point that is evident from a comparison of pursuit deficits with lesion locations is that the directional deficits follow lesions associated with subregions of MST. The most striking directional deficits are associated with damage to the floor of the STS, including extension onto the lateral posterior bank. Figure 3, *A–D* (*W2, N1, B1, W1*), shows this relationship. The lesions mapped in Fig. 3, *A* and *B*, damaged the floor and posterior bank of the STS lateral to MT with the lesion in Fig. 3*B* being limited to this area. The lesions shown in Fig. 3, *C* and *D*, damaged the floor of the STS, but instead of extending onto the lateral posterior bank, they extended onto the anterior bank. The lesion

in Fig. 3*E* shows an injection that damaged primarily the border between MT and MST, which is on the floor of the STS. While little directional deficit is evident at this speed, a directional deficit becomes evident at higher target speeds (considered later in RESULTS and in Table 1). The next illustrated lesion (Fig. 3*F*) is associated with a small but highly significant directional deficit and, while the lesion invades the fundus of the STS, it should be noted that the damage is much more medial than that of the other lesions. The last case shown in Fig. 3*G* has only minimal damage to the fundus and floor of the STS and only produces a minimal directional deficit. Damage to the densely myelinated area and the more dorsal anterior bank of the STS does not in itself produce the directional deficit as shown by the last injection (Fig. 3*G*).

The second major point is that the directional deficit does not *require* damage to MT. Two injections that led to directional deficits spared MT (Fig. 3, *A* and *B*) and one other barely touched MT (Fig. 3*C*). Damage to MT (particularly the foveal region in lateral MT) is not a necessary condition for the directional deficit. On the other hand, damage to MT fovea might also contribute to the deficit since two of the lesions that invade MT (Fig. 3, *D* and *E*), show directional deficits.

The third major point that is evident from Fig. 3 is that a retinotopic deficit follows all of the injections that include damage to MST. This deficit is clear even when the lesion does not invade area MT as is the case in Fig. 3, *A* and *B*. Thus, like the directional deficit, the retinotopic deficit can result also from lesions of MST that spare MT. However, in contrast to the localized retinotopic deficits following damage to MT (34), most lesions of MST produce a retinotopic deficit throughout the contralateral visual field. This widespread effect is consistent with the larger visual receptive fields found for many MST cells, particularly for cells on the anterior bank of the STS (20, 46). The exception to the widespread retinotopic deficit is the lesion of Fig. 3*A*. This is also the only lesion to spare totally the anterior bank region of MST except for some spread to a point high on the anterior bank of the sulcus.

PURSUIT DEFICITS AND TARGET SPEED. We tested pursuit at higher speeds both to determine whether the deficits became more severe at higher speeds and to measure the gain of pursuit (eye speed/target speed). Figure 4 shows examples from two experiments illustrated earlier, one that showed a clear directional deficit (*W2*), and another that showed primarily a retinotopic deficit (*P*).

Figure 4, *A* and *B*, *top*, emphasizes the retinotopic deficit by showing pursuit for target motion away from the side of the lesion into the contralateral visual field. For an injection that showed a limited retinotopic deficit (Fig. 4*A*, *top*), the deficit over the speeds tested was small and roughly constant. In contrast, for an injection that showed a clear retinotopic deficit (Fig. 4*B*, *top*), the deficit at higher speeds was substantially greater than at lower speeds. The slope of these lines is the gain of the pursuit (eye speed/target speed), and Fig. 4*B*, *top*, shows that for the retinotopic deficit the gain of pursuit was reduced; the reduction of pursuit speed was proportional to target speed.

Table 1 shows the changes in pursuit gain for all experiments (calculated as in Fig. 4) and allows a more quantitative comparison of the deficits. The experiments in Table 1 are in the same order from the largest to the least directional deficit as in Fig. 3. The upper section of Table 1 shows the pursuit gains for target motion into the contralateral visual field (away from lesion). The gain for the pursuit shown in Fig. 4*A*, which was the most restricted retinotopic deficit (*W2* in Fig. 3*A*) changes little, from 0.76 to 0.82. This might reflect mainly the small area of the visual field affected and the minimal time spent by a high-speed target moving across the affected area. The gain for the case shown in Fig. 4*B*, which had a retinotopic deficit extending throughout the contralateral visual field (*P* in Fig. 3*E*), declined substantially from 0.64 to 0.21. Injections that produced the smallest retinotopic deficits as shown in Fig. 3 for *W2*, *B1*, and *W1*, also showed the smallest (if any) reductions in gain. The other experiments showed reductions in gain ranging from ~67%–30%. Thus for the retinotopic deficit the change in gain gave about the same estimate of severity of the lesion as did the deficit in pursuit at 16°/s.

Pursuit of targets moving toward the side of the lesion into the ipsilateral visual field provided a measure of gain for the directional deficit (Fig. 4, *bottom*). The gain reduction in Fig. 4*A* was 48% and in Fig. 4*B* was 38%, and

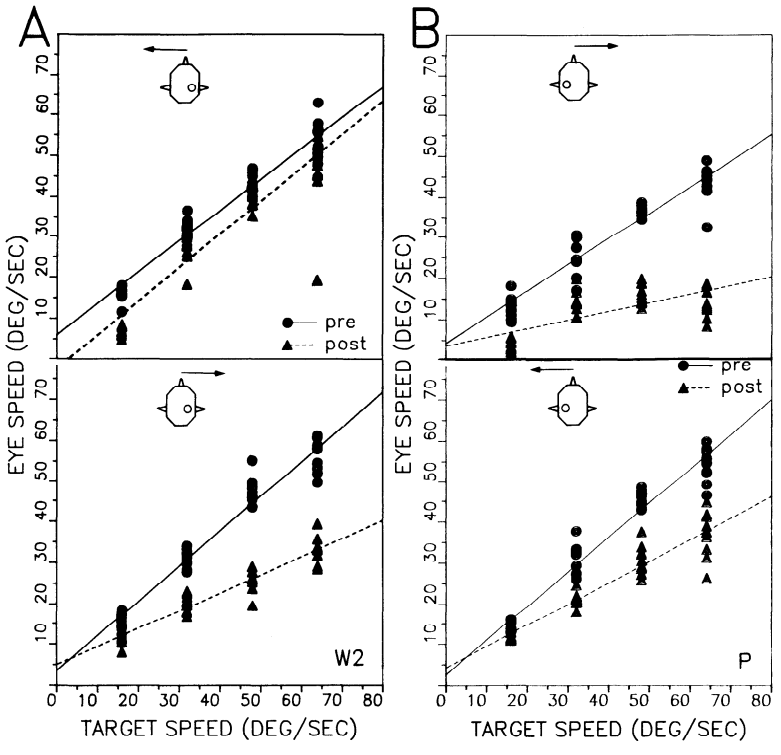


FIG. 4. Pursuit deficit for different speeds of target motion, in *A* for an experiment showing primarily a directional deficit (*W2*) and in *B* for an experiment showing both retinotopic and directional deficits (*P*). Target motion was in total darkness, and speeds tested were 16, 32, 48, and 64°/s. There was either no target step or a step of only 1°, and target motion was always away from the fixation point. *Upper graphs*: pursuit for target motion away from the side of the lesion; the means of ten trials pre- and postlesion are the mean speeds in the first 100 ms after the saccade. *Lower graphs*: pursuit for target motion toward the side of the lesion. The speed here was also measured in the first 100 ms after the saccade; this kept the pursuit measured close to the center of the visual field, and since the motion was in the ipsilateral visual field the pursuit speed was minimally affected by any retinotopic deficit. The regression lines are a least-squares fit, and their slopes indicate the gain of pursuit: eye speed/target speed. *A*: difference in slopes of the *bottom graph* (directional deficit) was significant at the 0.005 level. *B*: differences in slopes both in the *top graph* (retinotopic deficit) and in the *bottom graph* (directional deficit) were significant at the 0.005 level.

the gains for all injections are listed in the lower section of Table 1. Gains were reduced in two cases in which there were clear changes in pursuit at 16°/s (*W2* and *N1*) but only slightly reduced in two cases (*B1, W1*) in which directional deficits were clear at low speeds. A clearer directional deficit is indicated by the change in gain in one case (*P*) that showed little directional deficit at 16°/s. This lesion also included damage to the floor of the STS as did the other cases showing a directional deficit.

TABLE 1. *Pursuit gains*

Pursuit gain:	*W2*	*N1*	*B1*	*W1*	*P*	*B2*	*Y*
Away from lesion							
Pre	0.76	0.85	0.81	0.73	0.64	0.76	0.78
Post	0.82	0.47	0.67	0.88	0.21	0.36	0.55
% Change	+8	−45	−17	+20	−67	−53	−30
Toward lesion							
Pre	0.85	0.99	0.84	0.74	0.84	0.89	0.70
Post	0.44	0.69	0.77	0.62	0.52	0.86	0.75
% Change	−48	−30	−13	−16	−38	−3	+7

DIRECTIONAL DEFICIT AND RETINAL SLIP. One possible mechanism that might underlie the directional deficit is a reduced sensitivity to visual motion, a failure to respond to slip of the target on the retina. If this were the case, any slip superimposed during pursuit should produce less change in pursuit speed when pursuit is toward the side of the lesion than when it is away from the side of the lesion. We tested this possibility by first stabilizing the image on the retina during pursuit,

as described in a previous paper (10), and then superimposing a constant slip of the visual target—a velocity step. Figure 5 shows the result of one of two such experiments (on *B1* and *B2*) that gave the same results. After the monkey was pursuing the target moving at 16°/s, we stabilized the image and then superimposed a velocity step of 4°/s. This 4°/s-step was close to the slip that occurred during pursuit toward the ipsilateral side after a lesion. For example, in the postlesion pursuit of this monkey shown in Fig. 2*C*, the target was moving at 16°/s, the eye at ~10°/s. In Fig. 5*A*, the monkey pursued a target moving to the right toward the side of the lesion, and the superimposed increase in velocity was also toward the right. The schematic drawing above Fig. 5*A* indicates the step of the target to the left, rightward pursuit, and then a superimposed increase in velocity (*dashed line*); the shading indicates the contralateral visual field. An increase in pursuit speed occurred under these conditions before the lesion (*solid lines*—mean and standard error) but much less after (*dashed lines*). Note that the reduced response to the velocity step occurs with target motion in the ipsilateral visual field. When we added this velocity step during pursuit away from the side of the lesion (Fig. 5*B*), little difference between pre- and postlesion pursuit was evident.

This deficit in the response to retinal slip could be due to reduced sensitivity to visual motion toward the side of the lesion, or it

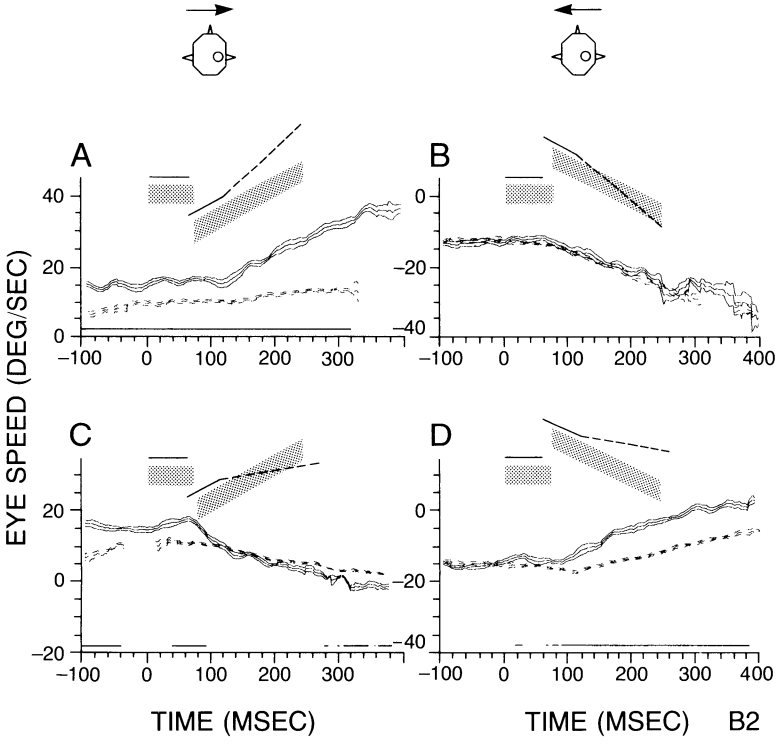


FIG. 5. Sensitivity to retinal slip during pursuit toward the side of the lesion (*left column*) and away from the side of the lesion (*right column*). During pursuit the image was stabilized on the retina (0 on the *abscissa*) and a constant target velocity was superimposed on the pursuit eye velocity. The schematic drawings at the *top* of each section (*A–D*) illustrate the direction of the 15° step and the 16°/s ramp, the direction of the superimposed velocity (*dashed line*), and the side of the visual field contralateral to the lesion (*shading*). The traces in each section show mean and standard error before (*solid lines*) and after (*dashed lines*) the lesion for a superimposed velocity of 4°/s. Differences in the traces at the 0.01 level using a Student's *t* test are indicated by the *horizontal lines above the abscissa*. *A*: pursuit toward the side of the lesion with superimposed stimulus motion in the same direction. *B*: pursuit away from the side of the lesion. Note that the initial pursuit speed before the addition of the velocity step in *A* is less than that in *B* since the step in *A* is into the contralateral visual field where the retinotopic deficit for both directions of movement is evident. *C*: pursuit toward the lesion but with superimposed stimulus motion away from the side of the lesion. *D*: pursuit away but stimulus motion toward the side of the lesion.

could be due to a gating of the visual sensitivity by pursuit movements toward the side of the lesion. In the experiments of Fig. 5, *C* and *D*, we tried to dissociate these two factors by adding a velocity slip in the direction *opposite* to the direction of pursuit. In Fig. 5*C*, even though pursuit was toward the side of the lesion, there was little reduction in sensitivity to motion away from the side of the lesion; the added velocity slowed the pursuit both before and after the lesion. With a higher superimposed velocity (16°/s), the deficit was clearer, but a deficit was not evident at either velocity in the other experiment (*B1*). In contrast in Fig. 5*D*, where pursuit was away from the side of the lesion but slip was toward the side of the lesion, there was a larger deficit. While this deficit was still small, it was also clear in the other experiment and clearer in both cases with a higher superimposed velocity (16°/s). These results indicate that there is a reduced sensitivity for visual slip after the lesion, and that it seems to be greater for visual motion toward the side of the lesion.

PURSUIT OVER PATTERNED BACKGROUND. All pursuit described so far has been on a homogeneous background, at least within the central 80° of the visual field. We thought that pursuit over a background might be a more difficult task and might amplify the deficit related to the lesion. Furthermore, some of the cells in the areas we have damaged are more sensitive to stimulation by large field stimuli than they are to small spots (21, 46), and the discharge of many of these cells is facilitated during pursuit over a patterned background (21, 41). We therefore compared the effects of the lesions on pursuit in the dark with pursuit across a pattern of stationary random dots. Figure 6 shows the effects of such a patterned background on pursuit for the experiment we have considered previously (*W2*). For simplicity just the means and the regression lines are shown for the dark and patterned conditions. In the normal monkey, we found pursuit across a patterned background to be only slightly less efficient than pursuit in the dark. This is consistent with the observation in both humans and monkeys that the effect of a patterned background on pursuit maintenance is slight (7, 18). For pursuit initiation, however, we would have expected a larger reduction in pursuit speed on a patterned background judging from the recent experiments of Keller and Khan (18). We do not know

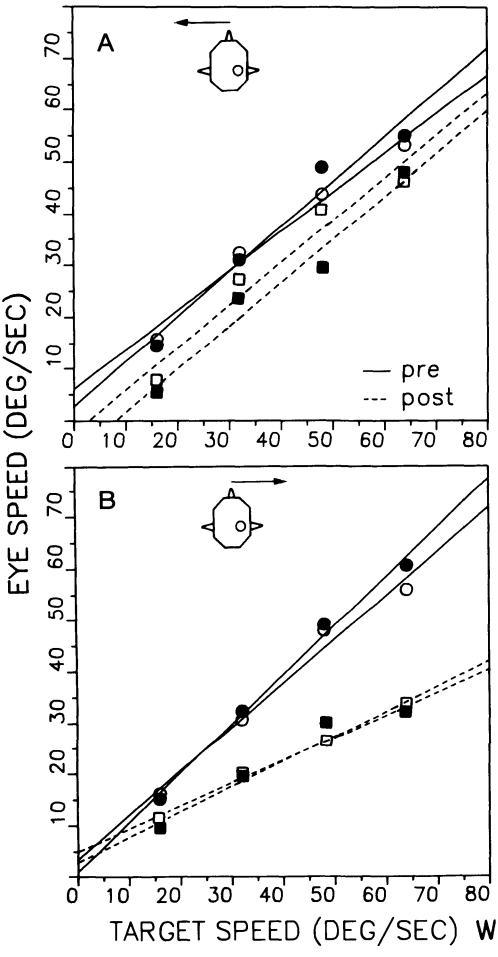


FIG. 6. Effect of a patterned background on pursuit and the deficits in pursuit following ibotenic acid lesions. The points and the regression lines were derived as described in Fig. 4. *Solid lines* and *circles*: prelesion, *dashed lines* and *squares*: postlesion. *Closed symbols*: pursuit of the target against the patterned background; *open symbols*: pursuit of the same target in the dark. The patterned background made little difference in pursuit either before or after the lesion for target motion either away from (*A, top graph*) or toward (*B, bottom graph*) the side of the lesion. The gains (slope) prelesion for pursuit over a patterned background were 0.87 and 0.96 for the two directions, and for pursuit in the dark they were 0.76 and 0.85. The differences in slopes pre- and postlesion are significant at the 0.005 level (using a *t* test) in the lower but not the upper graph.

what accounts for the difference, but use of the target dimming task or extent of overtraining may be factors. The ibotenic acid lesion reduced pursuit to the same extent regardless of background or direction of pursuit (Fig. 6, *A* and *B*). The only exception to this lack of effect of a patterned background in

three monkeys tested (*W2, W1*, and *P*) was a slight increase in the deficit in one monkey (*P*) for pursuit at higher speeds, and this monkey was also less consistent in its pursuit before the lesion.

AMPLITUDE OF SACCADES DURING PURSUIT. As mentioned earlier, the amplitudes of saccades were affected under some conditions, and Fig. 7 quantifies these effects for the experiment (*W2*) used to illustrate the deficits in pursuit speed (Figs. 1 and 2). The *ordinates* in Fig. 7 show the difference in eye and target position at the end of the saccade (saccadic error) for the same target step sizes shown on previous graphs. For the initial saccade to the target (Fig. 7*A*), there was clearly a deficit when the target moved in the contralateral visual field. For target motion away from the side of the lesion (Fig. 7*A, top*) or toward it (Fig. 7*A, middle*), the saccades to the moving targets were either too short or too long, respectively. No such deficit was seen in the ipsilateral visual field (right half of each graph). The deficit in saccadic amplitude therefore appears to be limited to target motion in the contralateral visual field where we observed

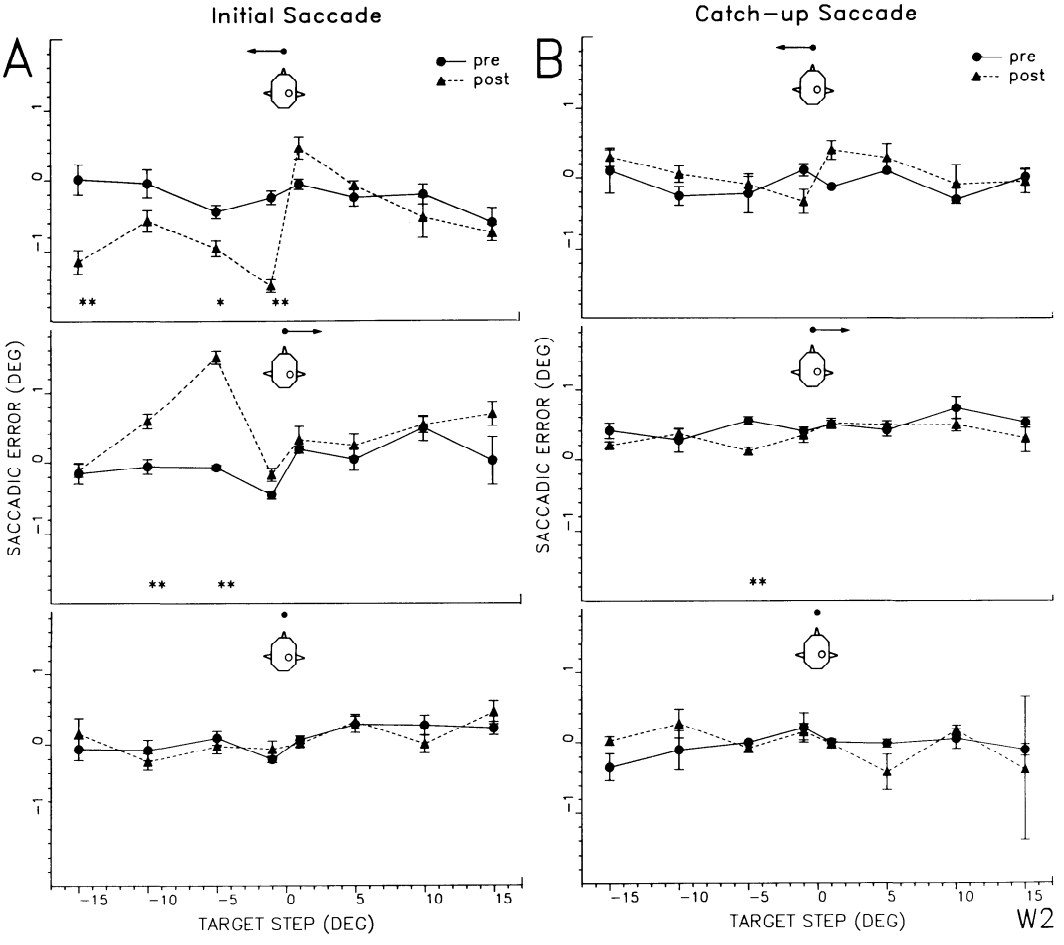


FIG. 7. The effect of an ibotenic acid lesion on the amplitude of saccades. *A*: amplitude of the initial saccades made to acquire targets are shown for motion away from the side of the lesion in the *top graph*, for motion toward the side of the lesion in the *middle graph*, and for stationary targets in the *lower graph*. The same steps into the left and right visual fields are shown on the *abscissa* as were shown previously (Fig. 2). *Ordinate*: difference in eye and target position at the end of the initial saccade (eye position error). An overshoot of the target by the saccade is positive, undershoot is negative. After the lesion, saccades into the contralateral (left) visual field were short for targets moving away from the side of the lesion, and long for target motion towards the side of the lesion. *B*: saccadic errors for catch-up saccades, those made after the initial saccade to the target. Catch-up saccades are grouped according to where the target started within the visual field. No consistent deficit was evident in any case.

the retinotopic deficit. Furthermore, the deficit was limited to moving targets since saccades to stationary targets in either visual field remained accurate after the lesion (Fig. 7*A, bottom*). This is similar to the deficit in amplitude of saccades following MT lesions: it is limited to those saccades made to moving targets (34).

Figure 7*B* shows saccadic errors for the catch-up or corrective saccades made after the initial saccade to the target. The differences pre- and postlesion in these cases were slight for target motion toward or away from the side of the lesion, (Fig. 7*B, top* and *middle*) or for stationary targets (Fig. 7*B, bottom*). The saccadic error was limited to the retinotopic deficit; it was evident in neither the initial saccade to establish the pursuit that showed a directional deficit nor was it evident with small catch-up saccades.

Figure 8 shows the change in saccadic error with increasing target speed. For target steps into the contralateral visual field and target motion away from the side of the lesion (Fig. 8, *top*), the saccadic errors showed the same trend as the speed errors in Fig. 4: a constant error in *W2* (Fig. 8*A*) and an increasing one in *P* (Fig. 8*B*). For the target motion that produced a directional deficit (Fig. 8, *A* and *B, bottom*), however, we saw little eye position error at the end of the saccade regardless of the speed of target motion. Thus the amplitudes were altered for saccades made to targets in the contralateral visual field (where we observed the retinotopic deficit) but not for targets moving toward the side of the lesion in the ipsilateral visual field (where we observed the directional deficit). Higher speeds of target motion, therefore, do not reveal deficits in saccadic amplitude related to the directional deficit.

OKN deficits

We obtained quantitative measures of the optokinetic response before and after the injection of ibotenic acid in five of the seven experiments already described. In normal monkeys, the slow phase of OKN consists of two components: an immediate rapid rise

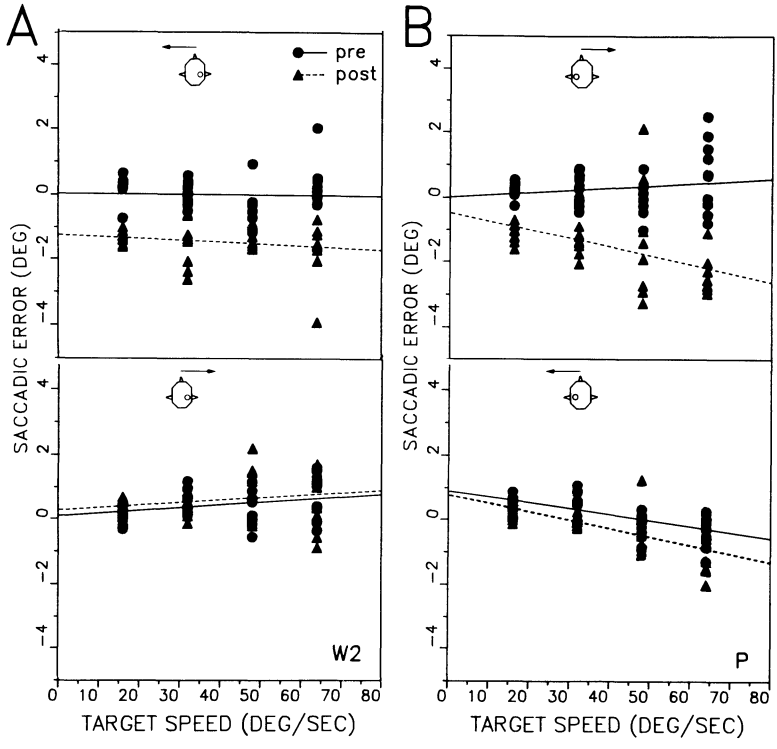


FIG. 8. Saccadic errors for different speeds of target motion. Target steps are 1° or less with motion away from the fixation point. Same target step sizes, speeds, and conventions as described in Fig. 4. The example in *A* had primarily a directional deficit in pursuit (*case W2*) and in *B* both a retinotopic and pursuit deficit (*case P*). The difference in slopes pre- and postlesion is significant at the 0.01 level (using a *t* test) only for the *top graph* in *B*.

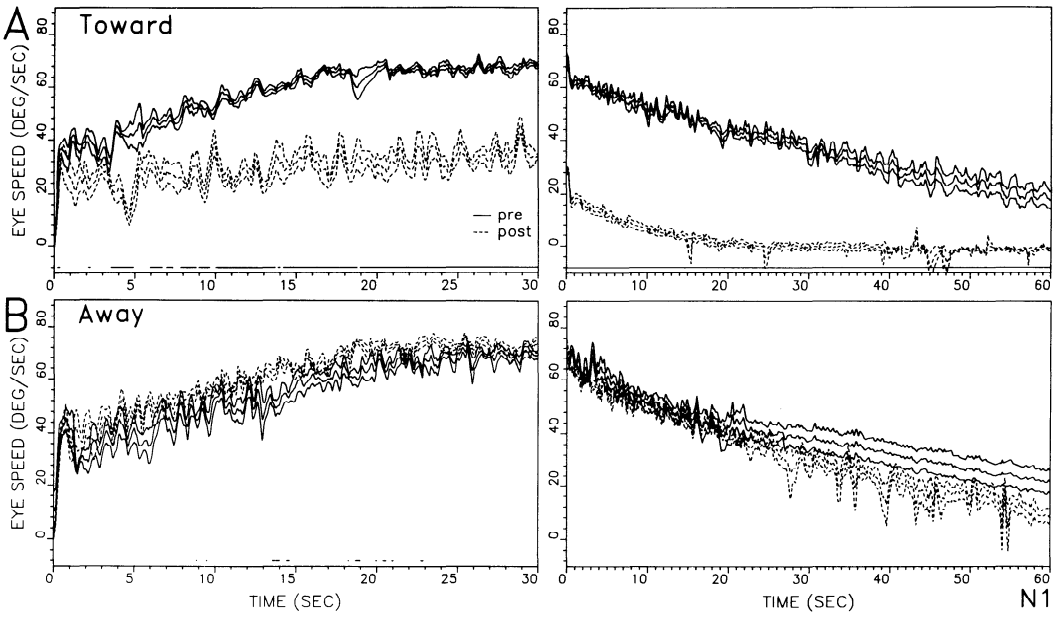


FIG. 9. Deficits in OKN and OKAN for stimulus motion toward the side of the lesion in an experiment (*N1*) that showed a prominent direction pursuit deficit. *A*: motion is toward the side of the lesion; *B*: it is away from the side of the lesion (traces are inverted in this case—left is up). OKN is shown on the *left*; 0 on the *abscissa* is time of drum illumination. OKAN is on the *right*; 0 is the time drum illumination was turned off. The *solid* and *dashed lines* represent the mean and standard error of the slow phase eye speed (see METHODS for details of derivation) for 5 superimposed prelesion trials and five superimposed postlesion trials. *Solid lines on the abscissa*: significant difference at the $P \leq 0.01$ level between the mean pre- and postlesion means (Student's *t* test). Such differences were clear in the slower buildup of OKN with stimulus motion toward the side of the lesion (*A, left*) and in the lower OKAN level (*A, right*), but not with stimulus motion away from the side of the lesion (*B*). The experiment was done binocularly with drum rotation of 72°/s and a period of stripes of 15.6°/cycle.

that might share common neural mechanisms with the pursuit system, and a slow buildup of response that is dependent on a velocity storage mechanism (6, 38). We found deficits in both responses.

DEFICITS IN FINAL OKN. Figure 9 shows the results of a lesion that produced a decrease primarily in the slow buildup of OKN. The records indicate the mean and standard error of 100-ms periods of the slow phase of nystagmus averaged over 5 trials. The *solid lines* show prelesion values, and the *dashed lines* show postlesion values. The records on the *left* show the slow rise in OKN over a 30-s period after illumination of the drum rotating at 72°/s toward the side of the lesion (Fig. 9*A*) or away from the side of the lesion (Fig. 9*B*). The records on the *right* show the succeeding 60-s period of optokinetic after-nystagmus (OKAN) in the dark. Fig. 9*A* shows a decrease in the buildup of OKN in the 30-s period of stimulation; the difference was consistently significant after the first 8 s of OKN stimulation. Similarly, the initial OKAN was reduced, and the decrease was statistically significant throughout the entire period. In contrast, for drum motion away from the side of the lesion (Fig. 9*B*), we saw no difference between pre- and postlesion records.

To compare results across monkeys we quantified the observations from the OKN tests in three experiments where we recorded OKN for several speeds of drum rotation and where a deficit in the slow buildup was evident. We took the maximum slow-phase eye speed in the first 2 s of OKN as an indication of the initial rapid rise in OKN, the mean of the final 10 s of the 30-s OKN period as an indication of the maximum slow phase speed achieved, and the maximum speed in the second 2-s period in the dark as a measure of initial OKAN. We did not measure the time constant of the slow rise of OKN since the shape of this curve varied markedly before and after the lesion. Also, we did not measure the duration of OKAN because we found that

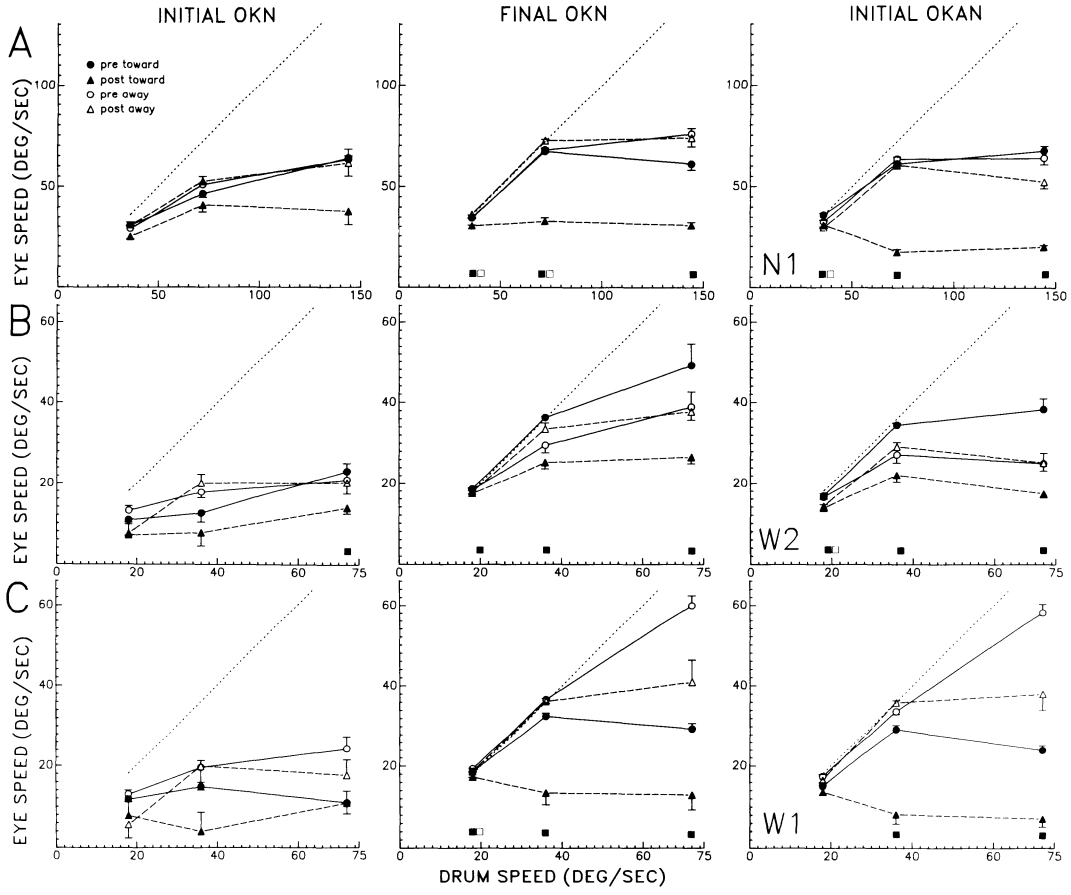


FIG. 10. Deficit in final OKN and initial OKAN for three experiments that showed a directional pursuit deficit. The initial OKN, final OKN and initial OKAN are shown for experiment *N1* (*A*), *W2* (*B*) and *W1* (*C*). *Dashed line on each graph*: where eye speed equals drum speed. In all cases, the initial OKN is little affected, but final OKN and initial OKAN are reduced for motion toward the side of the lesion (*dark circles* and *triangles*). *Filled squares on the abscissa*: significance level of $P \leq 0.01$ for motion toward the lesion; *open squares*: motion away from the lesion. Initial OKN is the maximum slow phase speed in the first 2 s after the OKN drum was illuminated. This period is inappropriately long for the initial phase but because the onset of drum illumination was not synchronized with the presence of eye position in the center of the field, we lengthened this interval to include all of the initial pursuit response. Final OKN is the mean of the last 10 s of drum illumination. Initial OKAN is the maximum speed in the *second* 2-s period after the light was turned off. These values represent the means for the entire slow phase segments, not for the 100-ms periods shown in Fig. 9 so that the tests of statistical significance in Fig. 9 and this figure are based upon different sample periods.

this changed with repeated testing (as we describe later in Fig. 11).

Figure 10*A* shows OKN measures for the same drum speed shown in Fig. 9 (72°/s) and one speed above and one below. *Filled symbols* are for drum motion toward the side of the lesion, *open symbols* for motion away from the side of the lesion; *circles* are for prelesion, *triangles* are for postlesion. Symbols along the *abscissa* indicate the statistical significance between the pre- and postlesion responses: *filled* for motion toward, *open* for motion away from the side of the lesion. Final OKN (*middle column* in Fig. 10) showed a striking reduction at higher speeds for motion toward the side of the lesion (although the difference was statistically significant at all speeds). Initial after nystagmus (OKAN—*right column* in Fig. 10) was similarly reduced. Motion away from the side of the lesion (*open symbols*) was not similarly reduced: the two symbols indicating significance at 36 and 72°/s were for small *increases* in final OKN speed. Initial OKN (*left column*

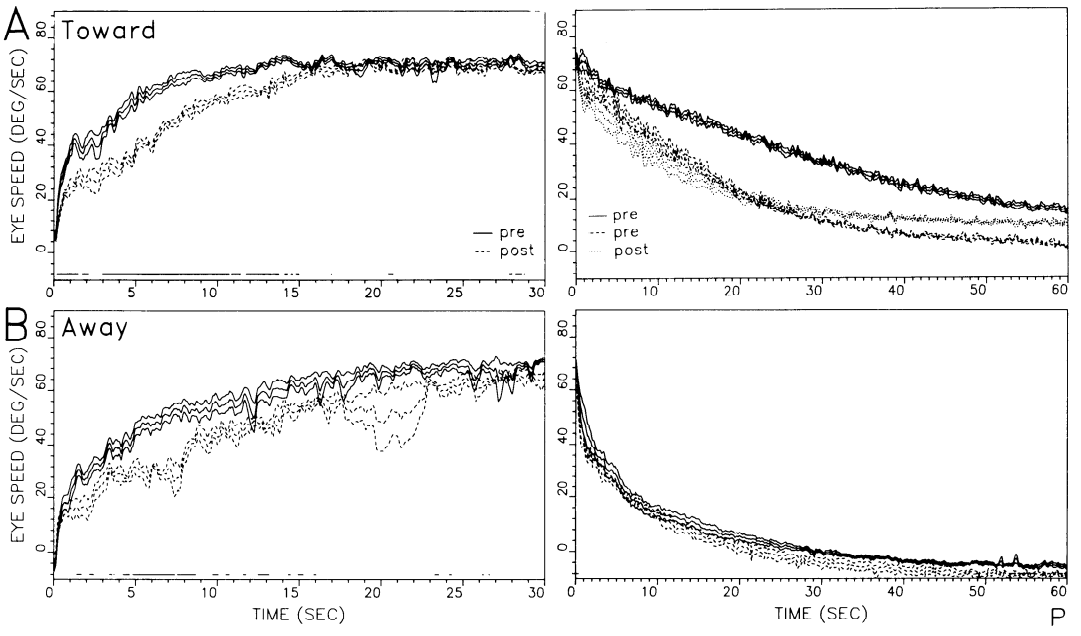


FIG. 11. Reduction in initial OKN for stimulus motion toward (*A*) and away (*B*) from the side of the lesion in an experiment (*P*) showing both retinotopic and directional pursuit deficits. Same conventions as Fig. 9 except that the means are for 10 prelesion trials and 5 postlesion trials. Slower initial rise in the OKN is evident from the traces and the statistical significance indicated by the *solid line above the abscissa*. Final OKN and OKAN show no differences in pre- and postlesion records. Variation in prelesion OKAN time course is indicated in *A, right*, where two groups of prelesion trials (five each) are shown, indicating that with repeated exposure the time course of OKAN decreases even in the absence of any lesion. Drum rotation was 72°/s and the period of the stripes was 1.6°/cycle.

in Fig. 10) was not reduced except for the highest speed.

Figure 10, *B* and *C*, shows the effects of the lesion in two other experiments, *W2* and *W1* for drum motion ranging from 18 to 72°/s (note change in *abscissa* between Fig. 10*A* and Fig. 10, *B* and *C*). Again, final OKN and initial OKAN showed a reduction for motion toward the side of the lesion, and most of these differences were statistically significant. *Case W1* in Fig. 10*C* also shows a substantial reduction in final OKN and initial OKAN for motion away from the side of the lesion, but these differences are usually not statistically significant.

In net, these three experiments (*N1, W2,* and *W1*) show a similar pattern of deficits, a decrease in final OKN and initial OKAN for motion toward the side of the lesion, although other deficits may also be present. These three monkeys also showed the most striking directional pursuit deficits (see Fig. 3, *A, B,* and *D*) and the lesion in each case included the floor of the STS. The remaining monkey that had a prominent directional pursuit deficit (Fig. 3*C*—*case B1*) showed qualitatively the same effect in final OKN and initial OKAN but we did not collect a sufficient number of trials for quantification. These limited cases suggest that the slow OKN buildup, which is indicative of the charging of a velocity storage mechanism, is related to the directional pursuit mechanism and that both are related to damage to the floor of the STS.

DEFICITS IN INITIAL OKN. Figure 11 shows the clearest example of a second type of deficit: a decrease in the initial rapid rise of eye speed seen in the first few seconds of OKN. This phase of OKN was reduced for drum motion in both directions, although the decrease for motion away from the side of the lesion was not as clear possibly because the prelesion eye speed in this direction was low. The final OKN and initial OKAN values were unaffected, in contrast to the example shown in Fig. 9.

In Fig. 11*A* we show the prelesion OKAN for drum rotation toward the lesion for two

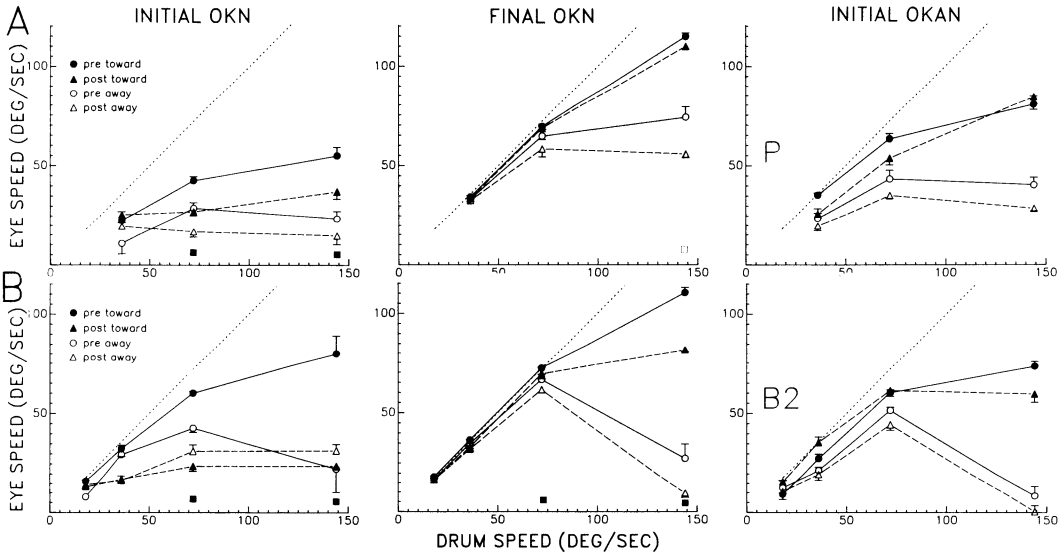


FIG. 12. Deficit in initial OKN in two experiments (*P, B2*) that show a clear retinotopic pursuit deficit. Same conventions as in Fig. 10. Effect of these lesions is primarily on initial OKN in both directions. Note that in these two experiments, OKN continued at higher drum speeds (144°/s) than was the case for 2 of the 3 experiments shown in Fig. 10.

separate days to indicate the changes in time course we encountered on successive days of testing. If we had compared postlesion OKAN (*dotted line, middle trace*) to just the first prelesion day (*solid line, upper record*), the more rapid postlesion decay of OKAN would have led us to conclude that the lesion had produced a change in time course. When we made the comparison to the second prelesion day (*dashed line, lower trace*), no deficit was evident. Because of this variation in OKAN time course in the absence of any lesion we did not analyze it in relation to the lesion. We saw no such variation in the slow rise in OKN.

Figure 12*A* shows quantitative data for the lesion illustrated in Fig. 11. For initial OKN only two points (at 72 and 144°/s) for motion toward the side of the lesion are statistically significant—possibly due to the prelesion asymmetry in this monkey. Final OKN and initial OKAN are not significantly altered. Figure 12*B* shows the results of the other experiment that also showed a decline in initial OKN (*B2*), but again the decrease was statistically significant only for motion toward the lesion. While this monkey also had some asymmetry in initial OKN before the lesion, it seems unlikely that this slight asymmetry accounts for the much greater deficit for motion toward the side of the lesion. The deficit in initial OKN seems to occur for both directions of stimulus motion but to be more pronounced for motion toward the side of the lesion. A deficit in final OKN was also evident in some cases in this latter experiment.

In net, while these lesions produced a mixture of deficits, there was a tendency for these latter monkeys (Fig. 12—*cases P* and *B2*) to show a decrease in the initial rapid rise of OKN primarily for motion toward the side of the lesion. These monkeys also had prominent retinotopic pursuit deficits and minimal directional pursuit deficits, at least at slow pursuit speeds (see Fig. 3, *E* and *F*). In contrast, the monkeys that tended to show a directional deficit in final OKN and initial OKAN (Fig. 10–*cases N1, W2*, and *W1*) also showed clear directional pursuit deficits (Fig. 3, *A, B*, and *D*). Both pursuit and OKN deficits in these latter cases were for stimulus motion toward the side of the lesion.

MONOCULAR STIMULATION. A striking feature of the optokinetic system in afoveate mammals, such as the rabbit, is that with monocular stimulation, temporal to nasal motion is effective for producing the slow buildup of OKN, while nasal to temporal is not. Furthermore, cats that show more symmetrical OKN to monocular stimulation are able to do so because of the contribution of

visual motion processing in the cerebral cortex (15, 44, 55). To investigate this aspect of OKN in the monkey, we tested OKN monocularly, using the second highest speed to which the particular monkey responded. We did not see consistent results for initial OKN. For final monocular OKN with motion toward the lesion, the decrease was striking in three experiments (*N1, W2*, and *W1*), but for *both* temporal to nasal and nasal to temporal motion. For motion away from the side of the lesion, a clear pattern of deficits was not evident. Our experiments did not reveal any preference for temporal to nasal motion under monocular stimulation.

DISCUSSION

Chemical lesions of MST produce deficits in both pursuit and optokinetic eye movements. We identified two deficits in pursuit: a retinotopic deficit for initiation of pursuit to targets moving in the contralateral visual field and a directional deficit for maintenance of pursuit for targets moving towards the side of the brain with the lesion. We also observed two deficits in OKN: a decrease in the slow buildup phase of OKN and initial OKAN and a decrease in the initial rapid rise in OKN. The same lesions that produced the clearest directional deficits in pursuit also tended to produce the clearest deficits in the slow buildup of OKN. We will discuss the possible neuronal basis of these deficits and their relationship to each other.

Retinotopic pursuit deficits

All of our injections of ibotenic acid were centered on the area we have referred to as MST. Within this MST area, all of our injections produced a retinotopic deficit in pursuit initiation for targets moving in the visual field contralateral to the side of the injection. The deficit was a failure to match initial eye speed to target speed and to adjust the amplitude of the saccade made to acquire a target. This deficit was present for any direction of motion, was more severe for higher speeds, and recovered almost completely within 2 wk. In these respects, the retinotopic deficits following injections in MST were identical to those following injections in extrafoveal MT. We therefore consider these MST retinotopic deficits to result from a disruption of visual motion processing for the same reasons we enumerated for the MT lesions (34).

The major difference in the retinotopic deficits following MT and MST injections was the extent of the visual field that was affected. Injections of extrafoveal regions of MT led to pursuit deficits restricted to one area of the contralateral visual field that could be predicted from the location of the visual receptive fields of cells at the site of the injection. Injections of MST frequently led to a deficit spreading throughout the extent of the extrafoveal visual field that we tested, usually out to 15–20°. The extent of the retinotopic deficit for a given injection bore some relationship to the distribution of receptive field sizes that we found in different subregions of MST (20). Receptive field sizes in lateral MST (MSTl—on the posterior bank and floor of the STS) were only slightly larger than in MT while those in more dorsal medial areas of MST (MSTd—on the anterior bank of the STS) were substantially larger, frequently covering a quadrant of the visual field. Our lesions that invaded MSTd produced deficits throughout the contralateral visual field tested (Fig. 3). In contrast, the lesion that most clearly spared the entire anterior bank of the STS related to MSTd (*W2* in Fig. 3*A*) also produced the most restricted retinotopic pursuit deficit.

The similarity of the retinotopic deficits between MT and MST is in itself significant. An efferent pathway from the cortex, which is almost certainly relevant for the pursuit system, is the projection to the dorsolateral pontine nuclear area (3, 13, 31). This pontine area has cells that discharge during pursuit eye movements (33, 45) and probably in turn projects to the contralateral flocculus of the cerebellum (4, 22). The flocculus has been demonstrated to have cells that discharge in relation to pursuit eye movements (26, 32). The projection from the STS to the dorsolateral pontine nuclei is from a wide area of extrastriate cortex (13) including regions that would include both MT and MST (M. Glickstein, personal communication). Therefore, the consistent retinotopic deficit following MST injections suggests that a functionally significant proportion of the output related to visual motion processing in the STS reaches these pontine areas, not only directly from area MT, but in parallel via MST or beyond. If the functionally significant projection to the pons were only directly from MT, then damage to the next sequential area,

MST, should have minimal effect since the information should already have been transmitted to the pons directly from MT. The prominent retinotopic deficit following damage to MST indicates that this is not the case. An alternative interpretation might be that damage to MST interrupts the projection back from MST to MT and that this in turn disrupts visual activity in MT. In either case, the retinotopic deficit following damage to MST emphasizes the role of MST in the visual motion processing necessary for pursuit initiation.

We draw two conclusions related to the retinotopic deficit following lesions of MST. First, damage to visual motion processing in MST underlies the retinotopic deficit just as it does in MT. The major difference between the areas is the size of receptive fields and size of the area showing the retinotopic deficit. Second, MST is probably a sequential step in visual motion processing that is critical for pursuit, and this step might well occur *before* this motion information is conveyed to the pontine areas of the brain stem.

Directional pursuit deficits

The directional deficit in the maintenance of pursuit was characterized by a failure to match eye speed to target speed after the monkey had made a saccade to place the target on or near the fovea. This deficit was evident whenever pursuit was toward the side of the brain with the lesion, regardless of the visual field in which the target began to move, and was similar to the directional deficit we observed previously following lesions directed at the foveal representation within MT (10). Similar deficits follow large cortical lesions in monkeys (29, 57) and hemispherectomy or lesions of the parietal-occipital lobe in the human (2, 5, 11, 24, 25, 43, 48). In humans a distinction between directional and retinotopic deficits following unilateral cortical lesions has recently been made (47). Reversible lesions of the dorsolateral pontine nuclei (45) also produce such a directional pursuit deficit.

In contrast to the retinotopic deficit, the directional deficit in pursuit maintenance tended to follow damage to restricted areas within MST. Lesions that invaded the floor and posterior bank of the STS adjacent to MT produced the greatest deficits. Part of this subregion of MST seemed to correspond to an area where cells have a directionally selective visual response and discharge during pursuit. This lateral-anterior area was designated as MSTl by Komatsu and Wurtz (20). Some pursuit cells in this area continue to discharge during pursuit even in the absence of a visual pursuit target, suggesting that these cells have an extraretinal input (35). In addition, damage to regions lateral to MT and on the floor and posterior bank of the STS also produced directional deficits. This damaged area must certainly have included an area designated as FST by Desimone and Ungerleider (8). That damage to FST is associated with a directional deficit is surprising since FST does not have a large proportion of directionally selective cells and these cells do not discharge during pursuit. The directional deficit might also be the result of damage to areas beyond FST where the visual response of neurons and their discharge during pursuit have not been adequately investigated.

Damage limited to areas on the anterior bank of the STS, which would include in large part the dorsal-medial area of MST—MSTd (20), produced minimal directional deficits. Extension of the damage onto the dorsal lip of the anterior bank of the STS, which would include area PP of Desimone and Ungerleider (8), also produced only slight directional pursuit deficits.

Given this relationship of a directional deficit and damage to a restricted region of the STS (MSTl and FST), the next question is whether this deficit results from an invasion of the adjacent foveal MT. Our previous study (10) showed that injections in foveal MT produce directional pursuit deficits, and in that study we hypothesized that the directional deficit resulted from an invasion of MST. In the present experiments there were two cases with small lesions in MST that produced a directional deficit (Fig. 3, *A* and *B, cases W2* and *N1*) but did not invade MT. In a third case, the directional deficit was also clear, and the invasion of MT was minimal (Fig. 3*C, case B1*). In contrast, a lesion *centered* on MT, which includes MT fovea (Fig. 3*E, case P*), produced a directional deficit only at higher pursuit speeds. Our working hypothesis, therefore, is that the directional deficit is due to damage to MST although the possible halo effects of chemical lesions, which we have outlined previously (10), do

not allow a definitive differentiation between such contiguous areas as MT fovea and MSTl.

Regardless of the exact substrate within the STS that underlies the directional deficit, the nonretinotopic nature of the deficit poses interesting questions about the cortical mechanisms controlling this movement. Lesions separated by only a few millimeters within the STS produce deficits that have shifted from being understandable as scotoma for stimulus motion in a retinotopic map to being related only to a direction of eye movement.

The characteristics of neurons within MT and MST suggest that reduction of either of two possible signals might underlie the directional deficit. One is the visual signal carried by cells in both MT and MST. The other is a signal related to eye movement that probably results from the addition of an extraretinal input (probably a corollary discharge) to the visual signal and that is largely limited to cells in MST (35). We have argued previously (10) that the directional deficit might result from reduction of the movement signal rather than the visual signal. The logic of this argument is that because the directional deficit is independent of the contralateral visual field, it should be related to a neuronal correlate that is also independent of that visual field. The extraretinal input is dependent only on the direction of the pursuit movement, and would therefore serve as an ideal candidate for the neural substrate which, if damaged, would lead to the pursuit deficit.

The other signal whose reduction could lead to a directional pursuit deficit would be a directionally selective visual one. Because cells in MST have large receptive fields that extend well across the vertical meridian, a loss of these visual cells would also be consistent with a directional deficit that is related to both the ipsilateral and the contralateral visual fields. The experiments that have tested the sensitivity to visual slip during pursuit before and after the lesions (Fig. 5) provide some evidence for this visual mechanism. Sensitivity to retinal slip was reduced more for visual motion toward the side of the lesion than for motion away from the side of the lesion. This decreased sensitivity was independent of the direction of pursuit, showing that the direction of visual slip is important, not the direction of pursuit per se. Further evidence on this point comes from experiments in which these areas of the STS were electrically stimulated during pursuit (19). Stimulation produced an acceleration of pursuit toward the side of the brain being stimulated. For pursuit toward the side of stimulation, this was an increase in pursuit speed; for pursuit away from the side of stimulation, this was a decrease in pursuit speed. Furthermore, because the stimulation was equally effective when retinal slip was present and when it was not (when the image was stabilized), the stimulation acted as if it replaced the normal visual input. Thus both these observations on sensitivity to slip and those on the effect of stimulation are consistent with the hypothesis that the lesions remove a visual signal and that it is the removal of this visual signal that leads to the directional deficit.

We have not come close to answering the most interesting question, namely, why the deficit is *directional.* The simplest explanation for the directional effect would be that neurons on one side of the brain are related to pursuit with a horizontal component in one direction while those on the other side are related to the opposite direction. This possibility can be rejected by our single cell recording experiments (20) because cells related to all directions of visual motion or pursuit were found on both sides of the brain. A more subtle explanation for the directional pursuit deficit would be a bias in the population of cortical cells that would not be apparent if a small sample of cells were studied, as was the case in our experiments, but would become apparent if the population of cells were damaged. For example, groups of cells in primate motor cortex have been shown to convey more precise information about the trajectory of an arm movement than is evident from the discharge of individual cells (12). A slight bias has been observed in small samples of cells within the STS (20, 41), but we do not know if the bias in fact accounts for the directional deficit. An alternative to such a bias is a selective projection of a subset of cortical cells with a uniform direction preference to an area of the brain related to only one direction of movement. In this case, we would not expect to find any bias in preferred direction in the population of cortical cells. This latter possibility emphasizes the importance of understanding the functional connections of particular cells.

Optokinetic deficits

Raphan and Cohen (39) have recently summarized the evidence that optokinetic nystagmus results from the activation of two processes, one direct and one indirect. The initial rapid rise in OKN is regarded as reflecting the direct process; the eye movement output is related to the velocity of stimulus motion input. The slow buildup phase is taken to reflect an indirect process; it exhibits a memory or velocity storage that is related not only to the velocity of visual motion but to its duration as well. We find deficits in both of these components of OKN following the chemical lesions of MST.

The most consistent optokinetic deficit was related to the indirect process, a reduction in the slow buildup of OKN, a deficit that can be interpreted as a failure to charge the velocity storage mechanism. This suggests that in the normal monkey, cortical area MST contributes to the charging process. We think that it would be surprising if MST acted directly on the velocity storage mechanism that probably is located in the vestibular nuclei (39). What seems more likely is that the cortex acts on the visual input that is usually thought to drive the velocity storage, the accessory optic system. In the cat, visual cortex has identified projections to the pretectal region of the brain stem, but in the monkey such projections from MST have not been investigated and from MT have not been found (30, 50). While any such input from cortex can probably best be regarded as a modulation of brain stem visual activity, the extent of the deficit emphasizes the importance of the visual input from cortex to the velocity storage phase of OKN in the monkey.

The deficit in buildup of OKN was more severe for motion toward the side of the brain with the lesion than for motion away from that side. This suggests that velocity storage is separate for each horizontal direction and that the cortex acts only on the mechanism related to one direction of motion. This is entirely consistent with the argument for two horizontal velocity storage mechanisms that was based on the asymmetry of OKN buildup in normal monkeys (6), which we have confirmed (e.g., see Fig. 9).

Lesions that produced this directional OKN deficit also tended to produce a directional pursuit deficit. Like the directional pursuit deficit, eye speed did not approach stimulus (target or drum) speed, even after an extended period of visual stimulation. This overlap of the two deficits in the same area of the cortex raises the possibility that the same mechanism underlies both deficits, but this overlap might be fortuitous and requires verification using more restricted lesions within the STS.

Ablation experiments on the visual cortex of the cat have led to the conclusion that the contribution of cerebral cortex to the slow buildup of OKN is at least twofold: *1*) facilitation of OKN at higher speeds of stimulus motion and *2*) production of symmetric responses for monocular visual stimulation in the nasal-to-temporal and temporal-to-nasal directions (15, 44, 55). The first expectation of the role of cortex in OKN is fulfilled in our experiments on the monkey since the deficits in buildup of OKN were always more severe at higher speeds of drum rotation. The difference in response at higher and lower speeds might have been even more striking had we used drum speeds lower than our usual 18°/s minimum. Furthermore, the buildup of OKN after the lesion seemed to reach an asymptote, with further increase in drum speed producing little increase in final OKN eye speed (as in Fig. 10). This apparent limiting of the final OKN at higher speeds is in contrast to the proportional reduction in pursuit speed with increased target speed as indicated by the reduced pursuit gain. The second expectation, a directional asymmetry with monocular viewing following cortical lesions, has not been fulfilled, possibly because our small and unilateral lesions within the STS affected too small a fraction of the cortical input to the optokinetic system.

The other deficit in OKN, the decrease in the initial rapid rise, can be regarded as acting on the direct process. This deficit was for both directions of drum motion but was somewhat stronger for motion toward the side of the lesion. It tended to be associated with those injections that also showed a marked retinotopic pursuit deficit. One possible reason for this association of deficits might be that the clearest retinotopic deficits were associated with lesions that affected large areas of the visual field, and damage to large areas of the visual field might be necessary to produce the clearest OKN deficits. Another more interesting possibility is that the same mechanism underlies the initiation of pursuit and the ini-

tial rapid rise of OKN. That this early phase of OKN and the pursuit system share the same underlying neural mechanisms has been suggested previously (6, 27, 40, 54, 56). The present experiments support this conclusion since lesions within the STS in some cases altered the initial rapid rise of OKN without affecting the slow buildup. These lesions appear to act on the visual motion processing stage on which both pursuit initiation and the rapid rise of OKN are dependent. It would be interesting to see the deficit following lesions within the STS in those *Macaca fascicularis* recently reported to have normal pursuit but no rapid rise in OKN (17).

We think that the salient point from the present experiments is that restricted damage to cortex reveals a possible differential relationship of subregions within the STS to the two phases of OKN. Our hypothesis is that the initial rapid rise, reflecting the direct process, shares cortical visual processing and probably the same output pathway with the pursuit system. In contrast, the subsequent slow buildup, reflecting the indirect process, acts on the velocity storage of OKN through the accessory optic system. This specificity suggests a substantial degree of functional localization within the STS. However, since the deficits in OKN (and pursuit) recover within several weeks, the information conveyed by these areas within the STS can be provided by other areas as well, as we have discussed previously (10, 34). Because following larger unilateral lesions of monkey cerebral cortex recovery of pursuit or OKN proceeds much more slowly (42, 49), other areas of cortex would seem to be the source of the information. Whether this information is derived from areas of MT and MST that were spared by our restricted lesions, or from cortical areas outside of the STS, remains to be determined.

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