

Effects of Superior Temporal Polysensory Area Lesions on Eye Movements in the Macaque Monkey

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SUMMARY AND CONCLUSIONS

1. On the basis of its anatomic connections and single-unit properties, the superior temporal polysensory area (STP) would seem to be primarily involved in visuospatial functions. We have examined the effects of lesions of STP on saccadic eye movements, visual fixation, and smooth pursuit eye movements to directly test the hypothesis that STP is involved in visuospatial and visuomotor behavior.

2. Seven monkeys were trained to make saccades to targets 8, 15, and 22° from a central fixation point along the horizontal meridian and 8° from the central fixation point along the vertical meridian. One monkey was also trained to make saccades to auditory targets. The same monkeys were trained to foveate a stationary central fixation point and to follow it with a smooth pursuit eye movement when it began moving 5, 13, or 20°/s. Four monkeys received unilateral STP lesions, one received a bilateral STP lesion, and as a control, two received unilateral inferior temporal cortex (IT) lesions. After testing, three of the animals with unilateral STP lesions received an additional STP lesion in the hemisphere contralateral to the first lesion. Similarly, one animal with a unilateral IT lesion received an additional IT lesion in the hemisphere contralateral to the first lesion.

3. All monkeys with complete removal of STP showed a significant increase in saccade latency to the most peripheral contralateral target, and most also had increased saccade latencies to the other contralateral targets. Saccades directed to targets along the vertical meridian or toward targets in the hemifield ipsilateral to the lesion were not impaired by removal of STP. By contrast, IT lesions did not impair the monkeys' ability to make saccadic eye movements to visual stimuli at any location, showing that saccades to visually guided targets are not impaired nonspecifically by damage to visual cortex.

4. The deficit in making eye movements after STP lesions was specific to saccade latency, with little effect on the accuracy of saccades to visual targets.

5. In the one monkey trained to make saccades to auditory targets, removal of STP did not impair saccades to auditory targets contralateral to its lesion, despite this monkey showing the largest increase in saccade latencies to visual targets. This result suggests that the impairment in the monkey's ability to make saccades to visual targets was not a purely motor deficit.

6. There was complete recovery of saccade latency to the baseline level of performance on the saccade task after all STP lesions. The median deficit took 2 wk of postoperative testing to recover.

7. Neither STP lesions nor IT lesions impaired the monkeys' ability to foveate the central fixation point.

8. Monkeys with removal of STP showed a decrease in smooth pursuit speed to downward moving targets. This impairment was more severe after bilateral lesions than after unilateral lesions. STP lesions also caused a less consistent decrease in smooth pursuit velocity to targets moving ipsilaterally to the lesion. Control lesions of IT did not impair smooth pursuit eye movements, demonstrating that, like the impairment in making saccades, the impairment in

smooth pursuit is a specific result of damage to STP, rather than a general effect of damage to visual cortex.

9. The impairment in making both saccadic eye movements and smooth pursuit eye movements after STP lesions is similar to that seen after lesions of posterior parietal cortex and suggests that, like posterior parietal cortex, STP is involved in visuospatial perception and visuomotor behavior.

INTRODUCTION

The superior temporal polysensory area (STP) of the macaque lies in the upper bank and the fundus of the anterior portion of the superior temporal sulcus (Bruce et al. 1981; Desimone and Gross 1979). Cytoarchitecturally, STP corresponds to the portion of area T3 of Jones and Burton (1976) lying within this sulcus and to the areas TAa, PGa, TPO and the dorsal half of IPa (Seltzer and Pandya 1978). Virtually all STP neurons are visually responsive, and almost one-half also respond to auditory or somesthetic stimuli or both (Bruce et al. 1981). STP is not visuotopically organized; its receptive fields are very large, often encompassing most of the visual field. STP cells are particularly sensitive to stimulus movement, especially in the far periphery. Most STP cells are directionally selective; some are highly selective for complex forms of movement including specific biological motions (Bruce et al. 1981; Perrett et al. 1985a). Some STP neurons show increased firing during saccades or smooth pursuit eye movements (Colby and Miller 1986). In contrast to the prevalence of motion sensitivity, only a minority of STP cells are sensitive to stimulus form. Some of this group are selective for aspects of face stimuli, such as head or eye orientation (Baylis et al. 1987; Bruce et al. 1981; Perrett et al. 1985b).

Consistent with the response properties of its cells, STP is connected to both the frontal eye fields (FEF; Barbas and Mesulam 1981; Jacobson and Trojanowski 1977; Kawamura and Naito 1984; Seltzer and Pandya 1989a; Stanton et al. 1989) and posterior parietal cortex (Baizer et al. 1991; Cavada and Goldman-Rakic 1989; Jones and Powell 1970; Mesulam et al. 1977; Morel and Bullier 1990; Neal et al. 1988; Seltzer and Pandya 1984), two cortical areas involved in the control of eye movements and spatial vision. There is also a projection to STP from areas medial superior temporal visual area (MST) and fundus of the superior temporal sulcus visual area (FST) (Boussaoud et al. 1990), both of which are specialized for the analysis of complex stimulus movement; and a weaker projection from inferior temporal cortex (IT) (Baizer et al. 1991; Morel and Bullier 1990), which is specialized for the analysis of shape. Finally, STP receives afferents from the medial pulvinar (Burton and Jones 1976),

which, in turn, is connected to the FEF (Bos and Benevento 1975; Huerta et al. 1986; Shook et al. 1991; Stanton et al. 1988; Trojanowski and Jacobson 1976), the posterior parietal cortex (Divac et al. 1977; Kasdon and Jacobson 1978; Mesulam et al. 1977), the deep layers of the superior colliculus (Benevento and Fallon 1975; Harting et al. 1980), and a variety of other areas (see Hardy and Lynch 1992 for a review).

Before the present study there were no data on the behavioral effects of lesions confined entirely to STP. Large lesions of the superior temporal sulcus that included STP had been found to impair the ability to carry out a manual visuomotor task (Petrides and Iversen 1979), to recognize angle of gaze (Campbell et al. 1990), and to orient the head and eye to visual stimuli (Luh et al. 1986). By contrast, these lesions had little or no effect on visual discrimination learning of faces or other visual stimuli or on cross-modal recognition (Ettlinger and Garcha 1980; Streicher and Ettlinger 1987). In another study in which lesions that included STP did slightly impair visual discrimination learning (Eacott et al. 1993), the deficit resembled that following posterior parietal lesions (Eacott and Gaffan 1991) in that discriminations between patterns that differed only in orientation were impaired, unlike IT lesions, which do not impair the discrimination of pattern orientation (Gross 1978; Holmes and Gross 1984).

Thus, in terms of neuronal response properties, anatomic connections, and the behavioral effects of lesions, STP appears to be predominantly but not exclusively part of the dorsal cortical stream specialized for the analysis of movement and for visuospatial and visuomotor function (Goodale and Milner 1992; Ungerleider and Mishkin 1982). In further support of this notion, some visual responsiveness of STP survives removal of striate cortex (Bruce et al. 1986) as is also the case for the dorsal stream area MT (middle temporal visual area) (Rodman et al. 1989) but not for the ventral stream area IT (Rocha-Miranda et al. 1975).

In the present study we have further examined the contribution of area STP to visuospatial and visuomotor behavior by studying the effects of STP lesions on the production of saccadic and pursuit eye movements. Our results suggest that STP is indeed involved in the control of eye movements to visual targets, and thus in visuomotor and visuospatial functions.

A preliminary report of this study in abstract form has been presented previously (Skelly et al. 1989).

METHODS

Subjects

The subjects were seven macaque monkeys: three *Macaca mulatta* and four *Macaca fascicularis*, ranging in weight from 4.0 to 6.5 kg. All animal handling, surgical procedures, training, and testing were done in accordance with protocols following National Institutes of Health guidelines and approved by the Princeton University Institutional Animal Care and Use Committee and the consulting veterinarian. Table 1 lists the animals and lesions, with u denoting the first (unilateral) lesion and b denoting the second (2-stage bilateral) lesion. *Monkey 4* received only a unilateral STP lesion, and *monkey 5* had STP removed from both sides in one operation (1-stage bilateral). *Monkeys 1–3* had STP removed on one side, recovered to preoperative performance on the saccade task, and then had STP removed in the opposite hemisphere. Simi-

TABLE 1. *Monkeys and lesions*

First STP Lesion	Second STP Lesion	Bilateral STP Lesion	First IT Lesion	Second IT Lesion
1u	1b			
2u*	2b			
3u	3b			
4u		5b		
			6u	6b
			7u	

STP, superior temporal polysensory area; IT, inferior temporal cortex.
* Partial STP lesion.

larly, IT cortex was removed from one hemisphere of *monkeys 6* and *7* (unilateral lesions), and, after postoperative testing was complete, IT was removed from the opposite hemisphere of *monkey 6* (bilateral lesion).

Implantation

Before any surgery or training, the monkeys were adapted to being handled and to sitting in a primate chair. Then the monkeys were implanted with a head bolt and a scleral eye coil for measuring eye position. Surgery was performed under pentobarbital sodium anesthesia with the use of standard sterile procedures. The eye coil surgery was similar to the procedure described in detail by Judge et al. (1980). To stabilize the animal's head during behavioral procedures, a head bolt was implanted onto the skull with skull screws covered by dental cement. This procedure yields a strong, stable, and painless means of head restraint. After recovery from the eye coil surgery, the animals were put on a controlled drinking schedule and began training.

Apparatus

During training the monkeys sat in a primate chair with their head held in a fixed position by the implanted head bolt. In a dark room the animals faced a $98 \times 98^\circ$ translucent screen. An optical bench behind the tangent screen was used to project the fixation and target stimuli. The fixation stimulus was a 0.3° diam spot of light that was projected onto the tangent screen by a high-intensity light emitting diode (LED) and a focusing lens. The saccade target stimulus was another 0.3° diam spot of light that was positioned by scanner mounted mirrors (General Scanning model G325D). The use of a separate fixation point and saccade target allowed us to present saccade targets without a streak of light appearing on the tangent screen. For auditory saccade testing there were three speakers located at 90° to the right of the animal, straight ahead, and 90° to the left of the animal. On the auditory saccade task the monkey responded to one of three LEDs positioned 22° to the right, straight ahead, or 22° to the left. The stimulus used in the pursuit task was the same as the saccade target stimulus. For all tasks eye position was measured to within 0.5° accuracy by a magnetic search coil apparatus (C-N-C Engineering). A Digital Equipment Corporation PDP-11/73 computer monitored eye position, presented stimuli, controlled reinforcement contingencies, and collected performance and eye movement data.

Fixation training

The monkeys were trained by successive approximation to fixate within 1.5° of the fixation stimulus for 1,000 ms to receive a fluid reward (apple juice or water). If the animal's eye position left the fixation window, the fixation light extinguished, no reward was delivered, and a 1-s time out was imposed in addition to the 1-s intertrial interval. Throughout all behavioral training and testing,

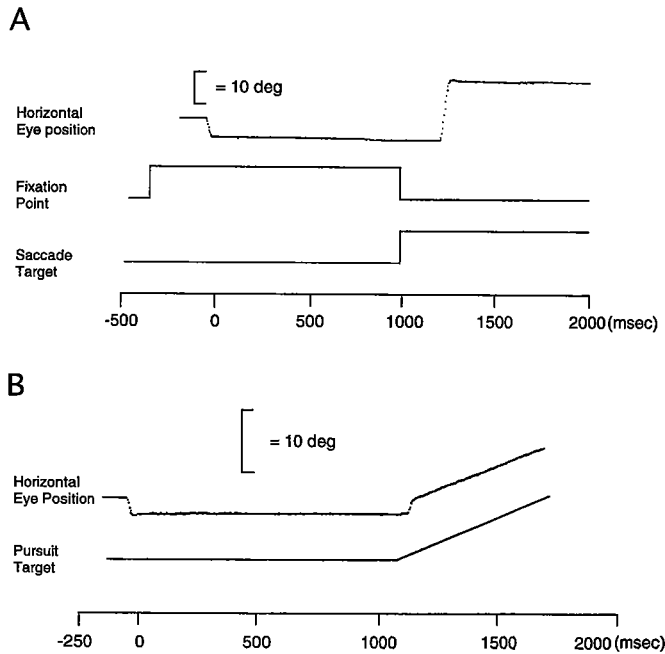


FIG. 1. *A*: typical saccade: 1st the fixation point appears, then the monkey foveates the fixation point; after 1,000 ms the fixation point disappears, and the saccade target appears; the monkey then makes a saccade to the target and foveates it for the remainder of the trial. *B*: smooth pursuit eye movement: 1st the stimulus appears, then the monkey foveates it; after 1,000 ms the stimulus begins moving, and the animal makes a catch-up saccade to it followed by a smooth pursuit eye movement.

the monkeys were run for either 5 or 6 days a week, and daily training sessions lasted until the animal completed ~1,000 correct trials (1–3 h). After fixation training to 90% correct in a block of trials including 500 correct trials for 2 consecutive days, the animals' training was begun on the saccade task.

Visual saccade training

After the monkey maintained fixation on the fixation point for 1,000 ms, a target stimulus in one of eight randomly chosen locations came on immediately after the offset of the fixation point. Target stimuli appeared at 8° above or below the fixation point and at 8, 15, or 22° to the right or left of the fixation point. After the simultaneous offset of the fixation point and onset of the target, the monkey had 1,000 ms to fixate within either 2° of the target (for those targets 8 and 15° from the fixation point) or 3° of the target (for those targets 22° from the target). The animal then had to maintain fixation for the remainder of the 1,000-ms interval to receive reward (see Fig. 1*A*). Breaking fixation or failing to make a saccade to the target and fixate it resulted in termination of the trial and imposition of a 1-s time out as in the fixation training sessions. A noncorrecting procedure was used (i.e., the stimulus after an incorrect trial was randomly chosen). When the monkey achieved 85% correct in a block of trials including 500 correct trials on 2 consecutive days, training on the saccade task was terminated, and training on either the pursuit task (monkeys 1–3 and 5–7) or the auditory saccade task (monkey 4) was initiated.

Auditory saccade training

One monkey (4) was trained to make saccades toward auditory stimuli. The animal had to fixate a central fixation point for 1 s as in the visual saccade task. Then a 3,000-Hz tone straight ahead or at 90° to the left or right of the animal came on. Simultaneously, LEDs lit just beneath the fixation point and at 22° to the left and

right of the fixation point. A 90° speaker separation was used to facilitate training on the task, and a 22° visual target separation was used so that the monkey's saccades would be similar to the size of saccades that monkeys normally make and so that the saccades would be in the range of measurement of our eye coil apparatus.

Pursuit training

The monkeys were trained to fixate and pursue a 0.3° diam spot of light that was positioned by scanner mounted mirrors. After a 1-s fixation period, the fixation point began moving at 5°/s, and the animal subsequently was required to maintain eye position within 4° of the stimulus. Either breaking fixation or failing to pursue the target stimulus within these limits resulted in termination of the trial and imposition of a time out. As in the saccade task, a noncorrecting procedure was used. The targets moved to an eccentricity of 8° left, right, up, or down. When the stimulus reached its final eccentricity, it shut off, and the animal received a reward (see Fig. 1*B*). After attaining 85% correct in a block of trials including 500 correct trials, the animals were trained to track within 5° of a stimulus moving at 13°/s to an eccentricity of 10° either up or down along the vertical meridian or left or right along the horizontal meridian. When 85% correct was reached on this task, they were trained to track within 5° of a stimulus moving at 20°/s to an eccentricity of 10° as in the 13°/s pursuit task. On the 20°/s trials, horizontal stimuli also were presented that moved to an eccentricity of 20°. Data for the latter trials were similar to the results for targets traveling to an eccentricity of 10° and will not be considered further in this report. One monkey (4) was only trained to pursue targets moving 5°/s. Immediately after completion of pursuit training baseline testing was begun.

Baseline testing

Each day during the baseline period, the monkeys were tested until they achieved 300 trials correct on the visual saccade task. Monkey 4 was tested until it got 300 trials correct on the visual saccade task and 300 trials correct on the auditory saccade task. The monkeys were also tested until they achieved 150 trials correct on the pursuit task at each of the three speeds. Monkey 4 was tested until it obtained 300 trials correct on the 5°/s pursuit task. After a criterion of 85% correct was achieved for 2 consecutive days on a task, the animal was run on the task for 2 more days, and the data for these 4 days were used for postlesion comparisons. During this period eye movement records were saved for all correct and incorrect trials by the same computer that controlled stimulus presentation and reinforcement contingencies. Fixation latency (the time between fixation stimulus onset and achievement of fixation) and saccade latency (the time between saccade target onset and fixation of the target) and percent correct to each saccade target for all tasks were also saved during this period as was done during the training phase of the experiment. After completion of baseline testing, the animal was given free access to water for a period of not less than 6 wk before cortical surgery.

Postoperative testing and data analysis

Seven to 10 days after surgery, the animals were tested on all tasks as in the baseline testing period for either 5 or 6 days/wk. The animals were tested until the mean postoperative saccade latencies and percent correct over a week of testing either were no longer significantly increased ($P < 0.05$, 2-tailed t -test for unpaired observations) for all contralateral targets over preoperative performance or until all of the mean postoperative contralateral saccade latencies were within 7.5% of the mean preoperative level. In the one animal (monkey 4) that occasionally did not make saccades to peripheral contralateral targets on the first few days of test-

ing, trials without saccades were counted as having latencies of 1,000 ms.

For STP lesions the saccade task data were analyzed both as each individual lesion and as the entire group of lesions. This allowed us to determine in the former analysis how an individual lesion affected performance and in the latter analysis how STP lesions *in general* affect performance. For individual lesions, preoperative and postoperative saccade latency, percent correct, and fixation latency over each week of postoperative testing were compared with baseline performance, and differences that reached a level of $P < 0.05$ (2-tailed t -test for unpaired observations) were considered statistically significant. Therefore the mean saccade latency and percent correct of all preoperative trials to a given target [(300 trials per day/8 randomly selected targets) \times 4 days = approximately 150 trials per target] was compared with the mean value of all postoperative trials to the same target [(300 trials per day/8 randomly selected targets) \times 5 or 6 days = approximately 188 or 225 trials per target]. For each lesion the mean preoperative fixation latency (300 trials per day \times 4 days = 1,200 trials) was compared with the postoperative fixation latency (300 trials per day \times 5 or 6 days = 1,500 or 1,800 trials). To analyze the STP lesions as a group, changes in both mean saccade latency and percent correct were tested for using an 8×2 analysis of variance (ANOVA) for repeated measures (preoperative data and 1st postoperative week) with the use of target location and lesion type (unilateral vs. bilateral) as factors. From each lesion there was one preoperative and postoperative mean for each location. Additionally, the mean fixation latency, percent correct, and saccade latency over all STP lesions was compared with baseline with the use of a two-tailed t -test for paired observations (preoperative and postoperative for each monkey) and an alpha level of 0.05. Saccade latency, percent correct, and fixation latency was analyzed for the group of animals with IT lesions in the same way except that lesion type was not used as a factor in the ANOVA.

To analyze the pursuit data from the STP lesions, the digitally recorded eye position signal on the pursuit task was differenced (the mean slope was determined by the use of a method similar to differentiating a continuous function) to yield eye speed, any saccades made were excluded from each record, and the mean eye speed was calculated for each trial. Similar to the individual lesion analysis of the saccade data, for each individual lesion the mean preoperative and postoperative eye speeds for each week of testing were compared with the use of a two-tailed t -test for unpaired observations and differences that reached a level of $P < 0.05$ were considered statistically significant. So, for each lesion and target speed, the mean eye speed of all preoperative trials to a given target [(150 trials per day/4 randomly selected directions) \times 4 days = approximately 150 trials per direction] was compared with the mean eye speed of all postoperative trials to the same target [(150 trials per day/4 randomly selected directions) \times 5 or 6 days = approximately 188 or 225 trials per direction]. To analyze the effects of STP lesions as a group on eye speed a $4 \times 3 \times 2$ ANOVA for repeated measures (preoperative data and 1st postoperative week) with target direction (ipsilateral, contralateral, up, and down), target speed (5, 13, and 20°/s) and lesion type (unilateral vs. bilateral) as factors was used. From each lesion there was one preoperative and postoperative mean for each target direction and speed combination. Additionally, both one-way ANOVAs with Tukey's Honest Significance Difference (HSD) comparisons and two-tailed t -tests for paired observations ($P < 0.05$) were used to help determine the source of significant effects and interactions. Eye speed was analyzed in the same way for the group of animals with IT lesions except that lesion type was not used as a factor in the ANOVA.

Only changes in postoperative performance that are statistically significant ($P < 0.05$) are referred to as increases or decreases throughout this report.

Cortical surgery

For surgery the monkey was first given an intramuscular injection of atropine sulfate (0.4 mg) followed by a restraining dose of ketamine (11 mg/kg). Animals were then either 1) anesthetized with 1.5% halothane, respired with 68.5% nitrous oxide and 30% oxygen, and immobilized with pancuronium bromide (0.24 mg bolus followed by an infusion of 0.02 mg/kg per hour), or 2) anesthetized with intravenous pentobarbital sodium (15 mg/kg, supplemented as necessary). Pentobarbital sodium anesthesia was found to yield shorter recovery times than halothane in combination with muscle relaxation. Body temperature and heart rate were monitored throughout surgery and recovery in all animals, and expired carbon dioxide level was also monitored in the animals anesthetized with halothane. After the skin and muscle were retracted, the bone overlying the temporal lobe was removed. Then the dura mater was opened, and either STP or IT was removed by aspiration with the aid of a Zeiss operating microscope. When the lesion was completed, the overlying tissue was sutured closed, and the animal was allowed to recover from anesthesia. After surgery the animal was given a prophylactic course of penicillin (Bicillin).

Histology

After all postoperative testing, the monkeys were overdosed with pentobarbital sodium and perfused through the heart with saline followed by either 1) 10% buffered Formalin or 2) first, 10% buffered Formalin; second, 10% buffered Formalin with 10% sucrose; third, 10% buffered Formalin with 20% sucrose; and finally, 10% buffered Formalin with 30% sucrose. Before removal of the brain from the skull, two insect pins were driven through each hemisphere from back to front to facilitate the alignment of drawn brain sections (see below). The brain was then placed in 30% sucrose Formalin until it was sectioned. The temporal lobe was sectioned in 50- μ m-thick slices in the coronal plane. Every 10th section was mounted on a glass slide and stained with cresyl violet. As the posterior boundary of STP was approached, sections were also mounted at intervals of 10 for myelin staining.

The intended STP and IT lesions were drawn on standardized sections from a monkey brain atlas (Szabo and Cowan 1984). For each lesion cresyl violet and myelin stained sections were examined and drawn at 5 times actual size. To get a more easily visualized representation of the lesion's size and location, a flattened map of the superior temporal sulcus was then constructed for the intended lesions and for each STP and IT lesion with the use of a procedure similar to that of Van Essen and Maunsell (1980).

STP lesions

The superior temporal polysensory area was described by Bruce and colleagues as "the dorsal bank and fundus of the anterior portion of the superior temporal sulcus" (Bruce et al. 1981, p. 369) on the basis of the similarity of single-unit response properties throughout the region. Baylis and colleagues (1987) found that cells in an area roughly corresponding to STP had similar visual response properties with the use of a cluster analysis. Seltzer and Pandya (1989b) found that the cortex corresponding to STP (areas TAa, PGa, TPO, and part of IPa) form a tightly interconnected region, as does the cortex in the lower bank of the superior temporal sulcus. STP borders high-order auditory cortex on the superior temporal gyrus, IT cortex in the lower bank of the superior temporal sulcus, and area MST, area FST, and posterior parietal cortex at its posterior extent (see Fig. 2).

Desimone and Ungerleider (1986) have mapped out the locations of MT, MST, FST, and posterior parietal cortex bordering STP. In concordance with previous work showing that MT does not extend beyond "... an imaginary line connecting the dorsal tip of the inferior occipital sulcus and the anterior tip of the intra-

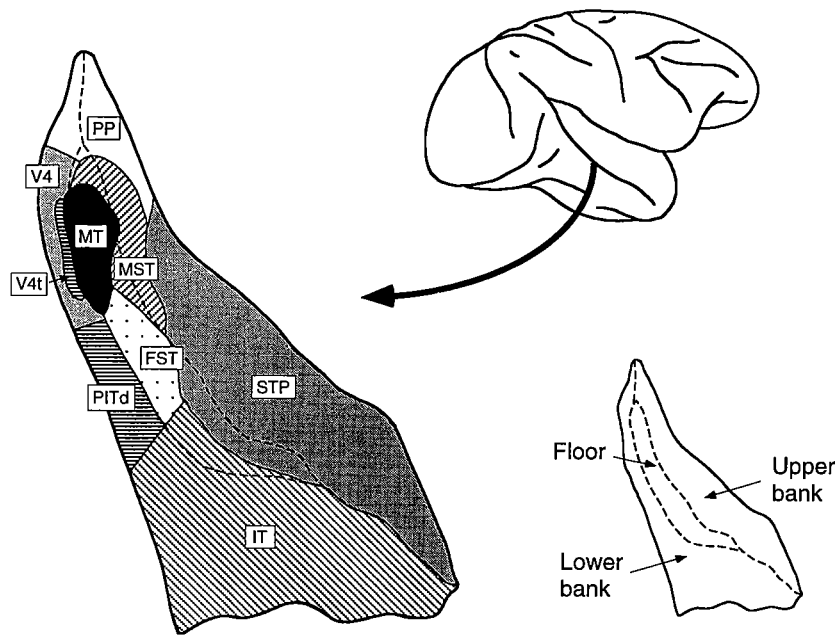


FIG. 2. Opened, "flattened" representation of the superior temporal sulcus illustrating the location of visual areas contained within it, based on Bruce et al. (1981), Felleman and Van Essen (1991), and Distler et al. (1993). FST, fundus of the superior temporal sulcus visual area; IT, inferior temporal cortex; MT, middle temporal visual area; MST, medial superior temporal visual area; PITd, dorsal portion of the posterior inferior temporal cortex; PP, posterior parietal cortex; STP, superior temporal polysensory area; V4, visual area V4; V4t, transitional zone of V4.

rietal sulcus" (Gattass and Gross 1981) or >20 mm anterior to the posterior tip of the superior temporal sulcus (Fig. 6, Van Essen et al. 1981), they found (Fig. 1) that MT did not extend >22 mm anterior to the posterior tip of the superior temporal sulcus. The anterior extent of area FST, however, is not clear neither in myelin or cresyl stained sections nor from its visual response properties. Given this, along with the variability of the location of MST and FST based on gross anatomic landmarks, our intended lesion was based on a conservative estimate of the size of STP (see Figs. 2 and 3) so as not to impinge on these areas. The intended lesion also included a small amount of cortex on the surface of the superior temporal gyrus. This predominantly auditory cortex (Merzenich and Brugge 1973) was removed to gain access to STP.

One can see from the coronal sections through the lesions and from the flattened reconstructions (Fig. 3) that none of the lesions encroached on any of the caudal visual areas in the superior temporal sulcus; in fact, the lesions were somewhat conservative, as intended, and only came within 1–4 mm of where areas FST, MST, and posterior parietal cortex usually lie in the superior temporal sulcus based on sulcal landmarks. The unilateral lesion in animal 1 (*lesion 1u*) involved STP almost exactly as was intended, but IT cortex in the bottom bank of the superior temporal sulcus, and more posteriorly on the inferior temporal gyrus, was also lost. As is the case with all of the STP lesions in which there was loss of IT cortex, this cortex was not surgically removed and was presumably lost because of impaired blood circulation after the STP lesions. The second lesion in animal 1 (*lesion 1b*) was similar to *lesion 1u*; it involved all of STP and also much of IT on the bottom bank of the superior temporal sulcus and more posteriorly on the inferior temporal gyrus. One can see that *lesion 2u* was far from a complete STP lesion; it does not extend to the caudal and dorsal extent of STP and, much more strikingly, does not include the anterior portion of the superior temporal sulcus. Because only approximately one-half of STP was ablated in *lesion 2u*, the data from this lesion are not further considered in this report. *Lesion 2b* included all but approximately the most posterior and dorsal few millimeters of STP. There was also a very small amount of damage to IT cortex on the bottom bank of the superior temporal sulcus. *Lesion 3u* was as intended except for sparing of both the small amount of STP that lies in the floor of the superior temporal sulcus and of the posterior and dorsal few millimeters of STP. *Lesion 3b* also spared STP on the floor of the superior temporal

sulcus but extended quite close to the posterior and dorsal borders of STP. *Lesion 4u* included all of STP except approximately the posterior and dorsal most millimeter. There was also extensive damage to IT cortex in the bottom bank of the superior temporal sulcus and some damage to IT cortex on the inferior temporal gyrus. The one-stage bilateral lesion (*5b*) included all of STP to within approximately a millimeter of its posterior and dorsal extent on both the right and left side. The anterior most few millimeters of STP were spared on the left side. On both sides there was extensive loss of IT cortex in the superior temporal sulcus and a small amount of tissue lost from the inferior temporal gyrus. Overall, with the exception of *lesion 2u*, the ablations removed all, or almost all, of STP. The main source of variation in lesion placement was the amount of damage done to IT in the bottom bank of the superior temporal sulcus. Partly because *lesions 1u*, *1b*, *4u*, and *5b* involved a considerable amount of damage to IT cortex in the bottom bank of the superior temporal sulcus, we added a control group of monkeys with lesions of IT cortex alone.

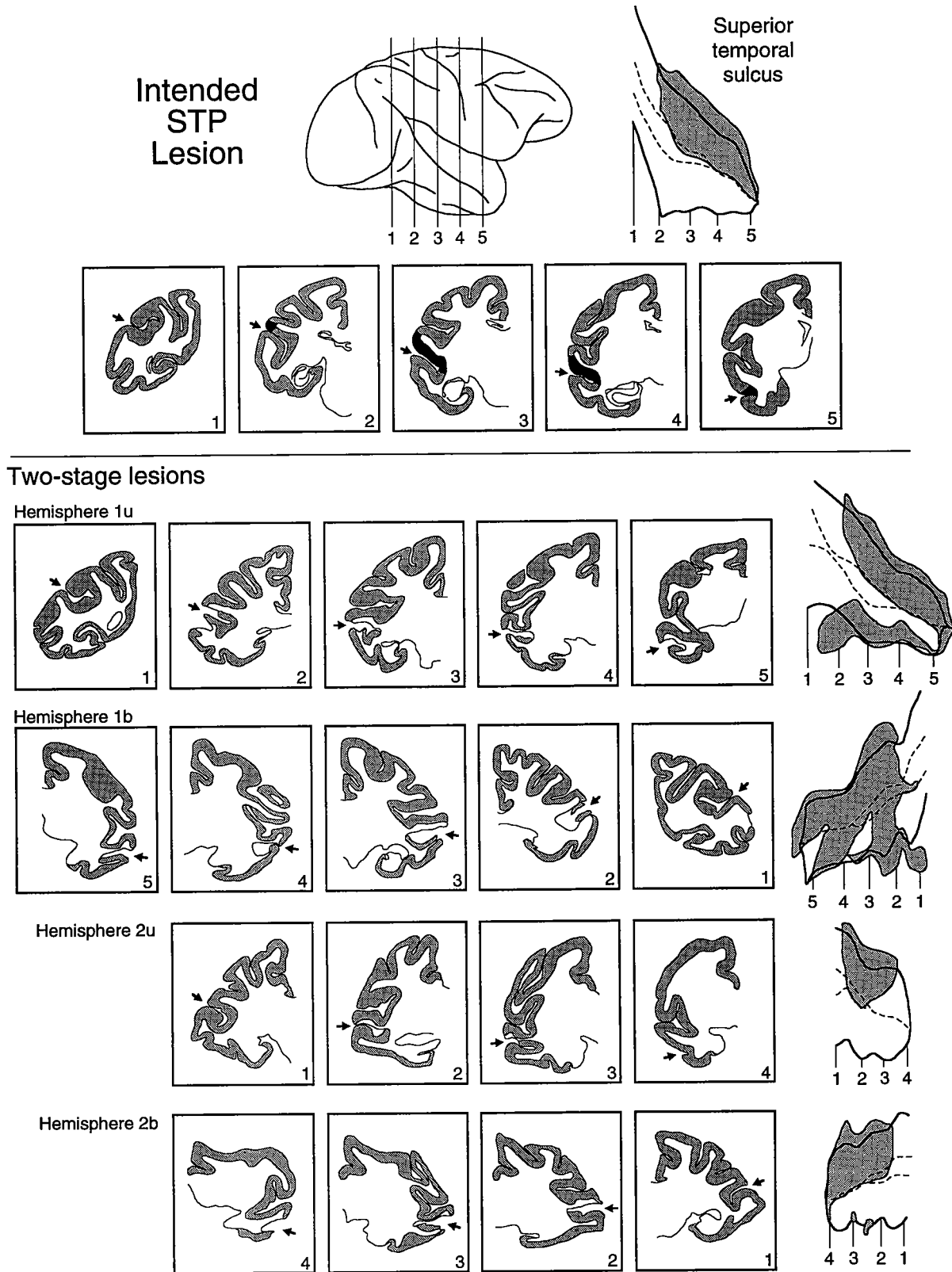
IT lesions

The intended IT lesion was chosen on the basis of the damage to IT incurred by the STP lesions and as such does not include all of IT on the inferior temporal gyrus. *Lesions 6u* and *6b* mimicked the damage to IT after *lesions 1u*, *1b*, *4u*, and *5b*, whereas *lesion 7u* was for the most part restricted to the surface of the inferior temporal gyrus and more closely mimicked the amount of IT cortex damaged after *lesion 2b* (see Fig. 4).

RESULTS

Visual saccade latency

Lesions of STP produced an increase in saccade latency to targets contralateral to the lesion. Figure 5 shows 10 superimposed eye movement traces to a target that was 22° in the visual field contralateral to a STP lesion (*lesion 3b*) before and 8 days after the lesion. Postoperatively, the saccade latency is increased by ~ 70 ms to this target location. That the saccade latency also becomes slightly more variable can be seen by noticing the increased range over which saccades

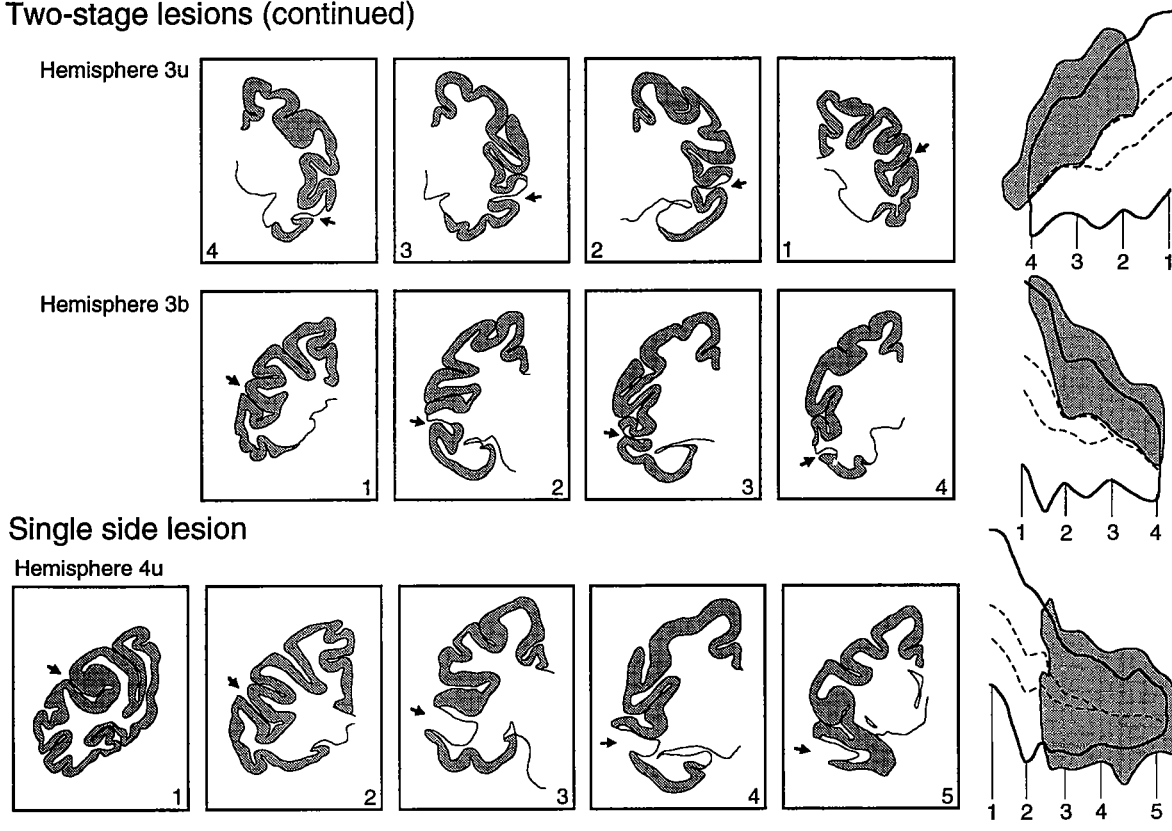


occur postoperatively. Figure 6 shows the day-by-day saccade latency to contralateral targets after an STP lesion (*lesion 1b*). It can be seen that, in addition to day-to-day variation in saccade latency, the deficit decreases during postoperative testing until the saccade latency to contralateral targets

is at the preoperative level. The data presented in these figures are representative of the saccade results to peripheral contralateral targets in general.

Figure 7 shows the individual monkey data for the preoperative testing period and the 1st wk of postoperative testing. All

Two-stage lesions (continued)



One-stage lesion

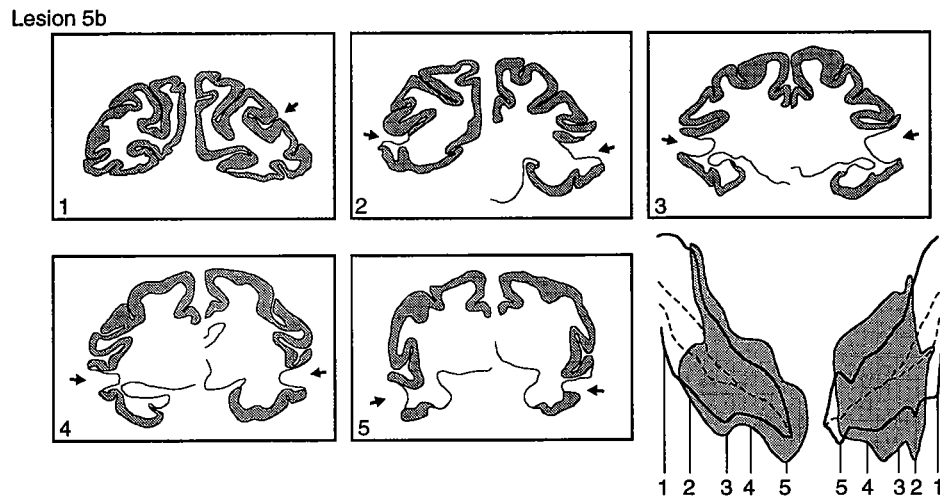


FIG. 3. STP lesions. Lateral view of the right hemisphere shows the location of the coronal sections below it. The intended lesion is the blackened region of the cortex in the sections immediately below the lateral view (arrows indicate the superior temporal sulcus). To the right of the hemisphere is a flattened representation of the superior temporal sulcus with the intended lesion shown by shading. Dark lines are the top and bottom lips of the superior temporal sulcus, and dashed lines are the fundus and the floor of the superior temporal sulcus. Numbers below the flattened representation correspond to the location of the sections. Similarly, the actual lesions are shown in both sections and flattenings below the intended lesion.

six unilateral and two-stage bilateral STP lesions (*1u*, *3u*, *4u*, *1b*, *2b*, and *3b*) caused an increase in saccade latency to the most peripheral contralateral target. Four of six lesions (*4u*, *1b*, *2b*, and *3b*) caused an increase in saccade latency to the 15° contralateral target, and the same four lesions produced an increase in saccade latency to the 8° contralateral target. The largest increases in saccade latency to contralateral targets (after

lesions *1b* and *4u*) were accompanied by a small increase in saccade latency to the most peripheral ipsilateral target. In contrast, when the saccade latency to the peripheral contralateral targets was smaller, there was either no change or a slight decrease in saccade latency to the most peripheral ipsilateral target (lesions *1u*, *2b*, *3u*, and *3b*). There was an increase in saccade latency to the upward target after two lesions (*4u* and

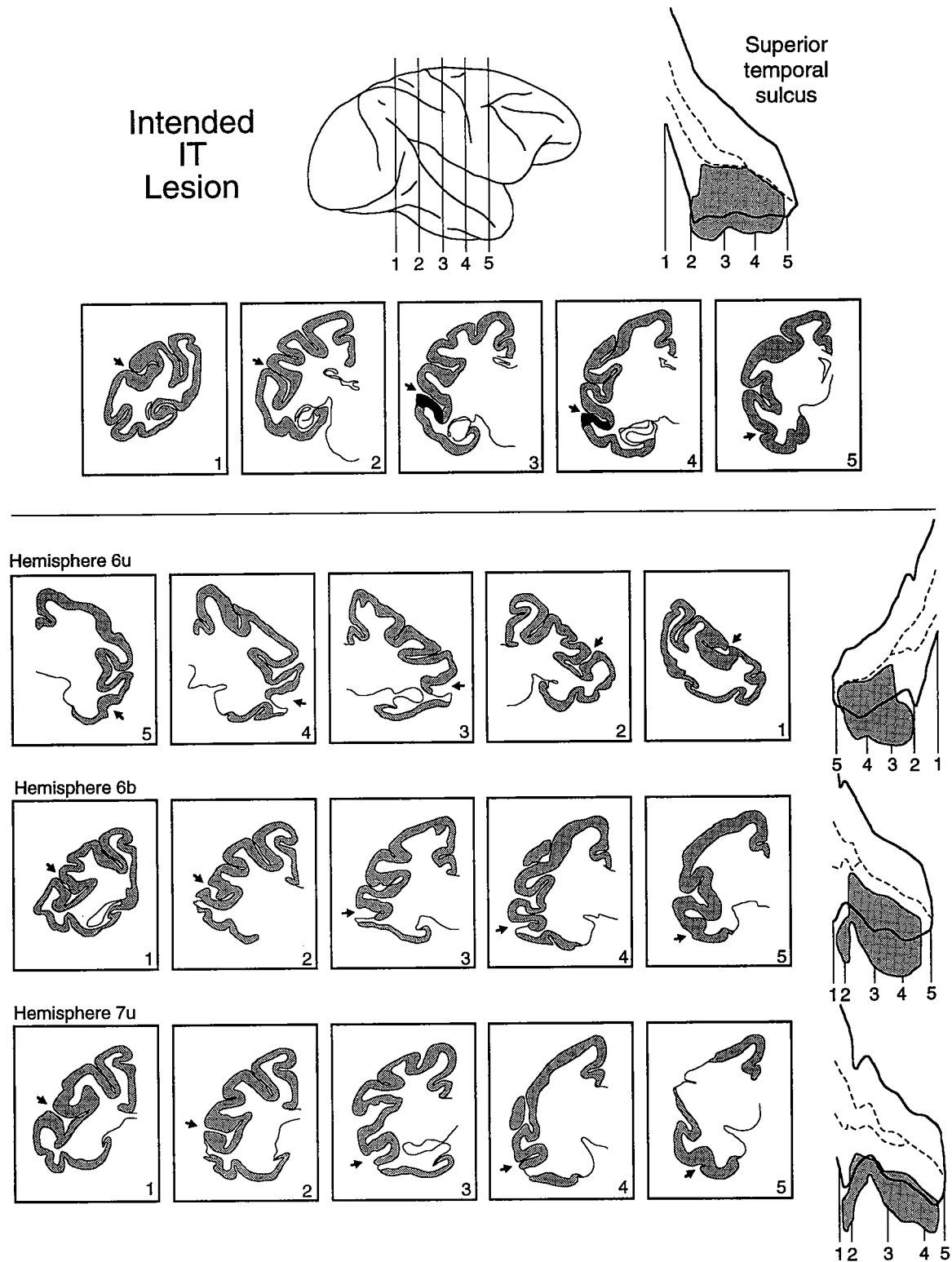


FIG. 4. IT lesions. Lateral view of the right hemisphere shows the location of the coronal sections below it. See also legend to Fig. 3.

3b) and to the downward target after two lesions (1u and 4u). There was a decrease in saccade latency to the upward target after one lesion (2b) and to the downward target after one lesion (3u). There was no discernible difference between left hemisphere (1b, 2b, and 3u) and right hemisphere (1u, 3b,

and 4u) lesions. In one animal (5) we ablated STP bilaterally in one operation. This lesion produced a bilateral saccade latency increase similar to that seen for targets contralateral to unilateral lesions. The mean saccade latencies to vertical targets were also increased in this animal.

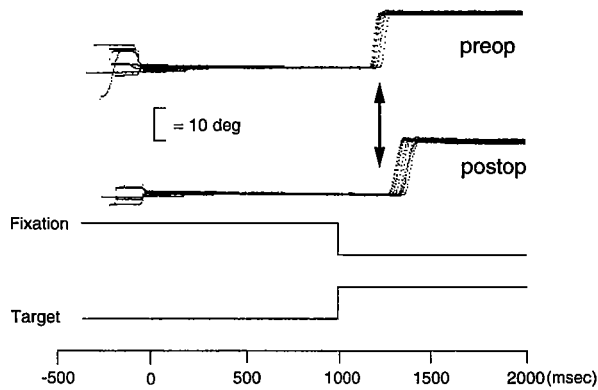


FIG. 5. Preoperative and postoperative eye movement records to a target that appears 22° from the fixation point in the visual field contralateral to a STP lesion (*lesion 3b*). The vertical line is drawn to facilitate comparison of the saccade latencies. Eye movement records are lined up on when the monkey achieved fixation.

A summary of the saccade latency deficits for all STP lesions is shown in Fig. 8. The saccade latencies to the horizontal targets for the one stage bilateral lesion (*lesion 5b*) are included as contralateral data points (there was no difference between unilateral and bilateral lesions; see below). The mean saccade latency collapsed over all targets is increased after STP lesions (ANOVA, $P = 0.001$). The saccade latency increase is due to an increase in saccade latency to contralateral targets (t -test for paired observations, $P < 0.05$) and becomes larger as the targets become more peripheral. The saccade latency was increased by 67.8 ms (25%) to the 22° contralateral target, by 35.4 ms (14%) to the 15° contralateral target, and by 15.8 ms (8%) to the 8° contralateral target. Consistent with this, there was an effect of target location on the magnitude of change in saccade latency (ANOVA, $P = 0.001$). Because of the saccade latency to ipsilateral targets and to vertical targets sometimes being increased, sometimes being decreased, and sometimes being unchanged (see Fig. 7), there was no significant change in saccade latency to these targets when analyzed as a group (t -test for paired observations). There was no effect of lesion order (unilateral vs. bilateral) on the magnitude of the increase in saccade latency (ANOVA, $P = 0.615$) or interaction between target location and lesion order (ANOVA, $P = 0.894$). Accordingly, the summary figure (Fig. 8) accurately reflects the central tendency of STP lesions to increase saccade latency to contralateral targets and leave saccade latency to ipsilateral and vertical targets unchanged.

Recovery

After removal of STP, all animals recovered to preoperative levels of performance within 4 wk of testing (see Fig. 9). Our criterion for recovery was that saccade latency to no contralateral target was significantly increased by $>7.5\%$ of the preoperative level. The saccade latency recovered after *lesion 4u* in 4 wk, after *lesion 1b* in 3 wk, after *lesions 2b, 3b, and 5b* in 2 wk, and after *lesions 1u and 3u* in 1 wk of postoperative testing. The saccade latencies to the most peripheral contralateral target were usually the last to recover. The average (median) monkey took 2 wk of testing to recover.

Visual saccade accuracy

In contrast with the monkeys' saccade latency, there was no consistent effect on the accuracy of the saccadic eye movements as assessed by the percent of correct trials (see Fig. 10). Postoperatively, as preoperatively, errors occurred almost exclusively on trials when animals left the fixation window before the saccade target appeared and only involved $\sim 5\%$ of the trials. Although there appears to be a small decrease in accuracy in the group data, this is entirely due to *lesion 4u* and is not statistically significant. After surgery, this animal occasionally failed to make an eye movement to the peripheral saccade targets, and therefore the deficit was in saccade latency rather than in accuracy per se. This occasional failure to make a saccade recovered in 1 wk of postoperative testing, which was before the saccade latency impairment recovered in the same animal. Although the animals occasionally showed a significant increase or decrease in performance to individual stimuli, when analyzed as a group there was no effect across all targets and lesions (ANOVA, $P = 0.170$). Furthermore, there was no effect of target location (ANOVA, $P = 0.953$) or of lesion order (ANOVA, $P = 0.439$) or interaction between target location and lesion order (ANOVA, $P = 0.456$).

IT lesions

In contrast to STP lesions, IT lesions did not impair saccadic eye movements. It can be seen from the individual lesion data that saccade latency was not increased to any target after either the first (*6u* and *7u*) or the second (*6b*) IT lesion (see Fig. 11). Saccade latency was decreased to vertical targets after *lesion 6u*; to 15° and 8° ipsilateral, 8° contralateral, and 8° upward targets after *lesion 6b*; and to vertical targets, 15° ipsilateral and 22° ipsilateral targets after *lesion 7u*. When the data were analyzed as a group of lesions, saccade latency collapsed across all target locations decreased slightly after IT damage (ANOVA, $P = 0.001$; see Fig. 12), although this is rarely significant in the group analysis with t -tests of individual target differences because of the small number of lesions ($n = 3$). There was no effect of target location (ANOVA, $P = 0.517$) on the change in saccade latency.

As after STP lesions, there was no loss of saccade accuracy after IT lesions (ANOVA, $P = 0.693$), as measured

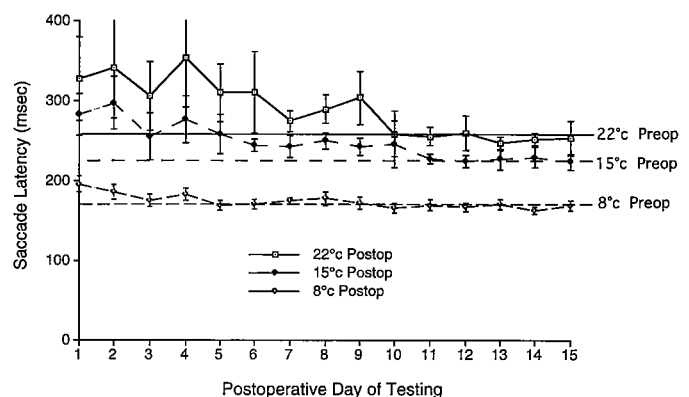
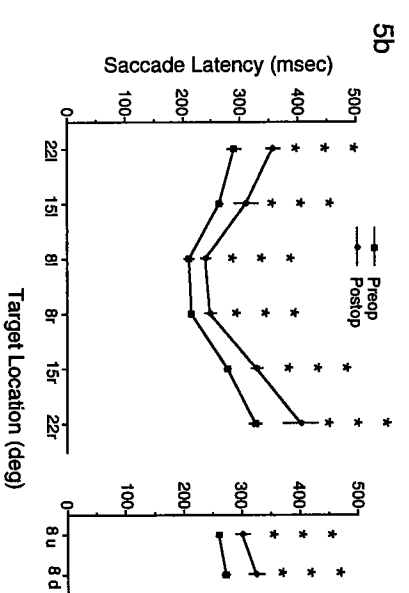
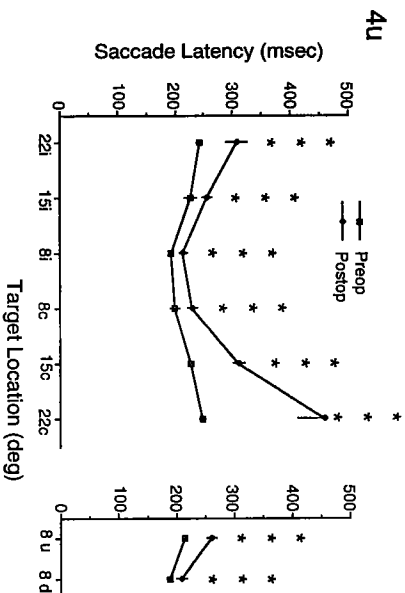
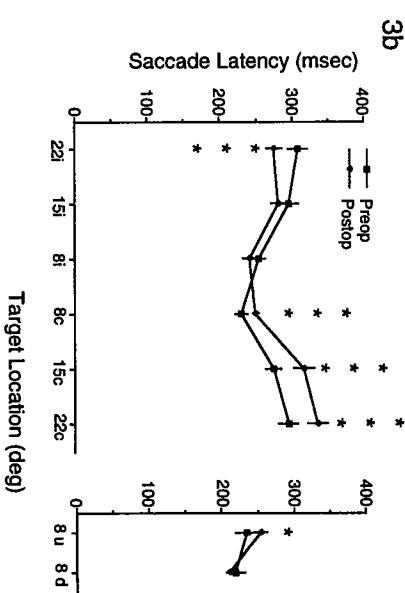
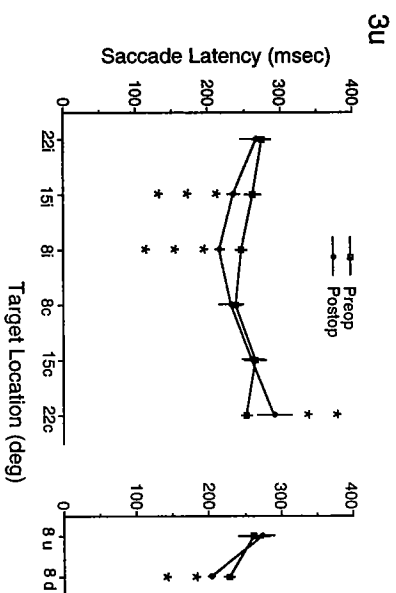
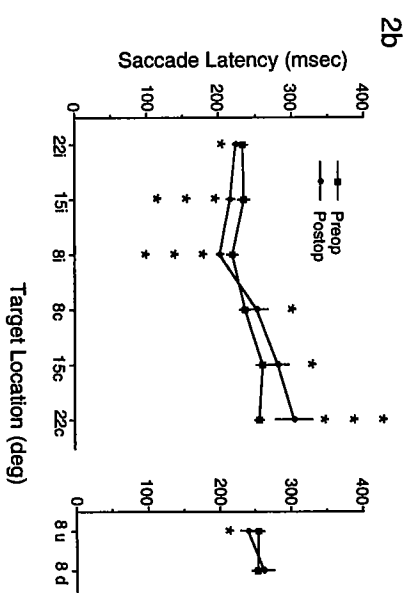
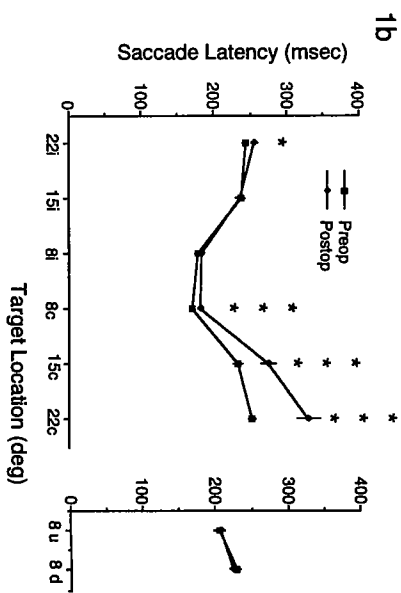
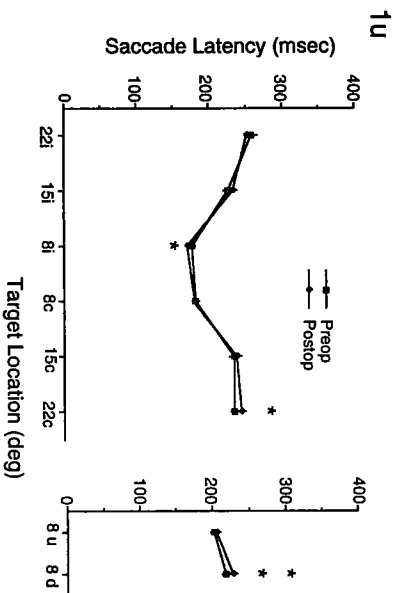


FIG. 6. Daily saccade latencies to targets 8° , 15° , and 22° from the fixation point in the visual field contralateral to *lesion 1b*. Horizontal lines indicate preoperative performance, and error bars are 95% confidence intervals.



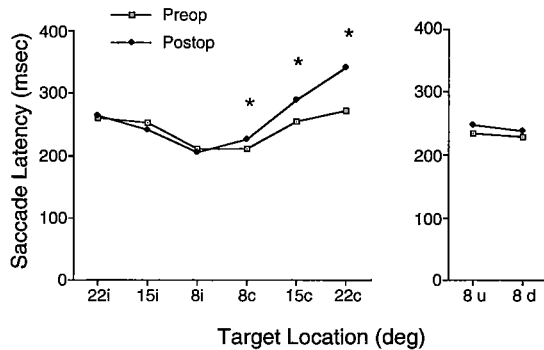


FIG. 8. Mean preoperative and postoperative (1st wk of testing) saccade latencies after all STP lesions (i, ipsilateral; c, contralateral; u, up; d, down) with significance levels (* $P < 0.05$; 2-tailed t -test for paired observations).

by the percent of trials correct (see Fig. 13). There was also no effect of target location (ANOVA, $P = 0.977$) on accuracy after IT lesions.

Auditory saccade task

The STP lesion (4u) that produced the largest increase in saccade latency to visual targets did not impair the monkey's ability to make saccades to contralateral auditory stimuli. In fact, as can be seen from Fig. 14, the postoperative performance was slightly improved to contralateral targets.

Visual fixation

After three STP lesions there was a decrease in fixation latency, after one STP lesion there was an increase in fixation latency, and after three STP lesions there was no change in the fixation latency; so, overall, there was no effect of STP lesions on fixation latency. There was also no significant change in fixation latency after any of the three IT lesions. Figure 15 shows that STP and IT lesions did not impair the monkeys' ability to foveate quickly the central fixation spot. This figure shows the mean preoperative and postoperative fixation latency for all STP lesions. The mean fixation latency for IT animals appears greater than that of STP animals because one monkey (6) had relatively high preoperative and postoperative fixation latencies; however, this apparent difference was not significant (ANOVA, $P = 0.208$). By observing the monkeys' eye movements with our on-line eye position display, we saw that their eye position was usually within 15° of the fixation point when the fixation point came on, consistent with the monkey anticipating the upcoming trial. Typically, the animals foveated the fixation point by making one or two saccades to it immediately after its onset, both preoperatively and postoperatively.

Smooth pursuit task

Figure 16 shows 10 superimposed eye movement traces to a target moving $13^\circ/\text{s}$ away from the side of one animal's STP lesion (2b). The preoperative and postoperative traces

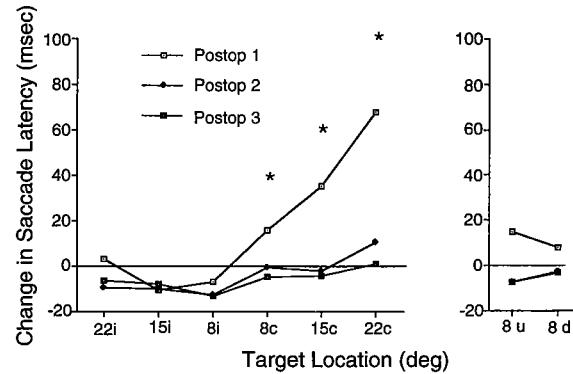


FIG. 9. Mean change in saccade latency after all STP lesions for the 1st, 2nd, and 3rd wk of postoperative testing. Conventions are as in Fig. 8.

are indistinguishable. This is representative of the fact that, for all but downward moving targets, changes in eye speed were subtle after STP lesions.

The individual subject data show that STP lesions decrease eye speed to downward moving targets and that after STP lesions there is both a variable decrease in eye speed to ipsilateral moving targets and an increase in eye speed to contralateral moving targets (see Fig. 17). Eye speed was significantly decreased after STP lesions to 14 of 19 downward moving targets. In contrast, eye speed to upward moving targets was decreased to 3 of 19 targets, unchanged to 13 of 19 targets, and increased to 3 of 19 targets. Either one- or two-stage bilateral lesions were more likely to cause a decrease in eye speed to downward moving targets (11 of 12 targets) than unilateral lesions were (3 of 7 targets). The eye speed to ipsilateral moving targets decreased to 6 of 16 targets, increased to 0 of 16 targets, and was unchanged to 10 of 16 targets; the eye speed to contralateral moving targets increased to 6 of 16 targets, decreased to 1 of 16 targets, and was unchanged to 9 of 16 targets (not counting the 6 horizontally moving targets from lesion 5b as either ipsilateral or contralateral; see below). The size of these effects increased with increasing target speed (note the scales are different in Fig. 17 for different speeds). In summary, the individual lesion analysis suggests that STP lesions cause a decrease in eye speed to downward moving targets, especially after bilateral lesions, and less consistently are followed by a decrease in eye speed to targets moving ipsilateral to the lesion and an increase in eye speed to targets moving contralateral to the lesion.

The comparison between preoperative and postoperative eye speed collapsed over all targets, and speed did not reach statistical significance (ANOVA, $P = 0.112$); however, the lack of an effect with the data collapsed over all targets, directions, and lesions was due to the deficit being specific to certain targets with *increases* in eye speed to other targets. Consistent with the deficit being specific to certain target directions, there was a highly significant effect of target direction on change in eye speed (ANOVA, $P = 0.0002$).

FIG. 7. Mean preoperative and postoperative (1st wk of testing) saccade latencies for each STP lesion (i, ipsilateral; c, contralateral; u, up; d, down). Error bars represent the mean 95% confidence intervals (sometimes the error bars are smaller than the data points for the means), and asterisks indicate statistical significance (* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$; 2-tailed t -test for unpaired observations). Asterisks are above the data points for increases in saccade latency and below the data points for decreases in saccade latency.

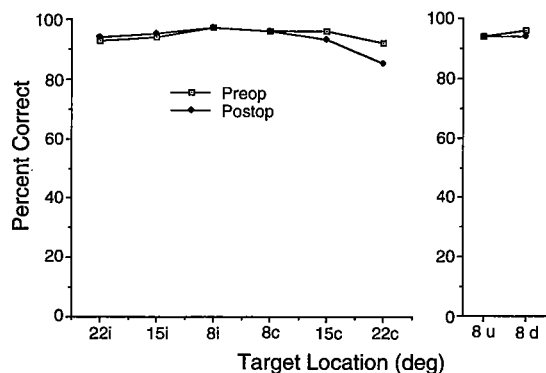


FIG. 10. Accuracy as measured by mean preoperative and postoperative (1st wk of testing) percent correct after all STP lesions. The appearance of a slight decrease in postoperative performance is due to *monkey 4*'s pronounced saccade latency increase to the point where it occasionally did not make a saccade within the time allowed. There were no statistically significant differences between preoperative and postoperative performance ($P < 0.05$, 2-tailed t -test for paired observations).

As can be seen from Fig. 18, which shows the change in mean eye speed for all STP lesions, there was a decrease in eye speed to targets moving into the visual hemifield ipsilateral to the STP lesion (2-tailed t -test for paired observations, $P = 0.018$) and to targets moving downward (t -test, $P = 0.005$). Eye speed was increased to targets moving contralateral to the lesion (t -test, $P = 0.011$) and unchanged for

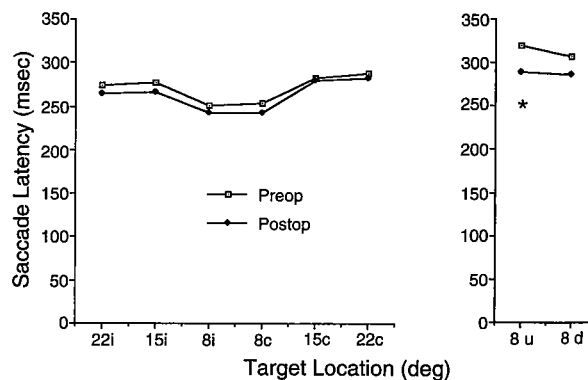


FIG. 12. Mean preoperative and postoperative (1st wk of testing) saccade latency after all 1st- and 2nd-stage IT lesions. Conventions are as in Fig. 8.

targets moving upward (t -test, $P = 0.358$). The horizontal smooth pursuit eye speeds for the one-stage bilateral lesion (*monkey 5*) are not included in the summary figure; given that cortical lesions can impair pursuit to either ipsilateral moving (MacAvoy et al. 1991) or ipsilateral and contralateral moving targets (Keating 1991), it was not clear a priori whether and how the single-stage bilateral lesion should be included with the others. In any case, including the results for horizontal pursuit from this lesion as ipsilateral data does not change the statistical significance for the ipsilateral mov-

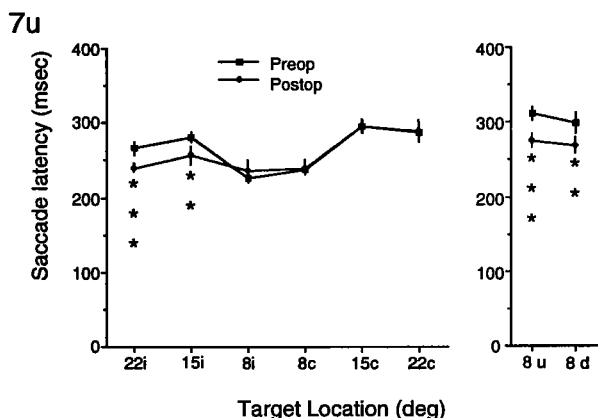
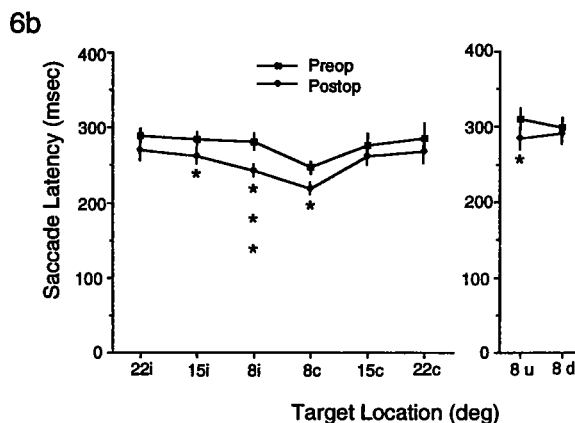
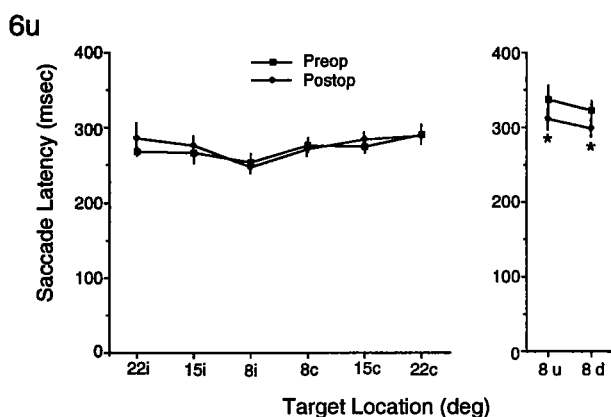


FIG. 11. Mean preoperative and postoperative (1st wk of testing) saccade latencies for individual IT lesions. Conventions are as in Fig. 7.

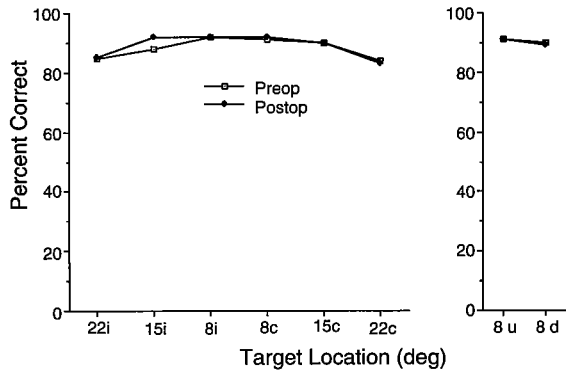


FIG. 13. Accuracy as measured by mean preoperative and postoperative (1st wk of testing) percent correct after all 1st- and 2nd-stage IT lesions. Conventions are as in Fig. 10.

ing target group data and changes the means only slightly. There was no significant effect of target speed on change in eye speed collapsed across target direction and lesion order (ANOVA, $P = 0.431$). There was, however, an interaction between target speed and direction ($P = 0.048$); this reflected the effects of target direction on eye speed becoming more pronounced at increased target speeds. There was also an effect of the type of lesion (unilateral vs. bilateral) on the size of the deficit collapsed across target direction and target speed (ANOVA, $P = 0.044$), consistent with unilateral lesions producing smaller deficits on the pursuit task than bilateral lesions (see Fig. 17). There was an even stronger interaction between lesion order and target direction ($P = 0.005$), because of the effect of target direction being larger after bilateral lesions for both targets with increased and decreased eye speed to them. Finally, there was no three-way interaction between target direction, target speed, and lesion order ($P = 0.218$). Thus, consistent with the individual subject data, the group ANOVAs show that there is an impairment in making downward and ipsilateral smooth pursuit after STP lesions and an increase in eye speed to contralateral targets. These effects are larger to faster target speeds and after bilateral lesions.

Lesions of IT cortex did not impair smooth pursuit eye movements (see Fig. 19). The individual lesion data show that there was a significant increase in eye speed to 19 out of 36 target speed and direction combinations. When the IT

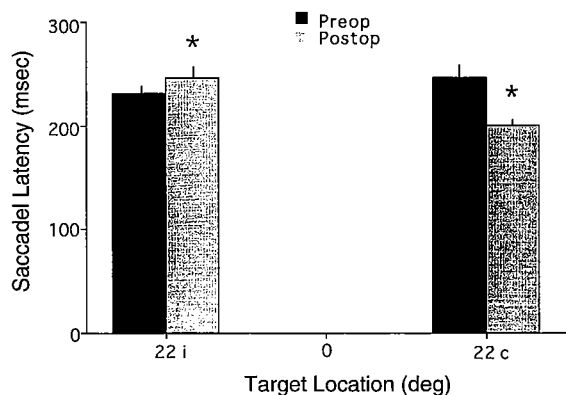


FIG. 14. Mean saccade latency to auditory targets before and after (1st postoperative wk) a STP lesion in 1 monkey (4). Error bars represent mean 95% confidence intervals, and symbols denote the level of statistical significance (* $P < 0.05$, 2-tailed t -test for unpaired observations).

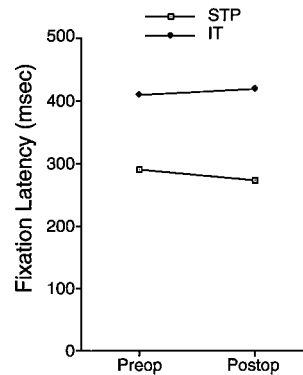


FIG. 15. Fixation latency before and after (1st postoperative wk of testing) lesions of STP and IT. There were no statistically significant differences between preoperative and postoperative performance for either group ($P < 0.05$, 2-tailed t -test for paired observations). The fixation latencies for the IT group appear to be longer because of one monkey having long preoperative and postoperative fixation latencies, but this difference is not significant.

lesions were analyzed as a group (see Fig. 20), there was a slight increase in eye speed after IT lesions over all target directions and speeds (ANOVA, $P = 0.001$). There was no effect of target direction on the increase in eye speed (ANOVA, $P = 0.492$), but there was an effect of target speed (ANOVA, $P = 0.031$) with faster targets showing larger improvements. There was no interaction between target speed and target direction (ANOVA, $P = 0.948$). The postoperative mean eye speed is always greater than the preoperative eye speed, although this is rarely significant in the group analysis with t -tests of individual target differences because of the small number of lesions ($n = 3$).

DISCUSSION

Lesions of STP produced an impairment in making saccadic eye movements that was similar to those seen after lesions of posterior parietal cortex (Lynch and McLaren 1989) and the FEF (Lynch 1992). After removal of STP, there was also an impairment in making smooth pursuit eye movements similar to that seen after posterior parietal lesions and smaller than that seen after FEF lesions. Control lesions of IT cortex did not impair eye movements, implying that the deficits seen after STP lesions are not merely a nonspe-

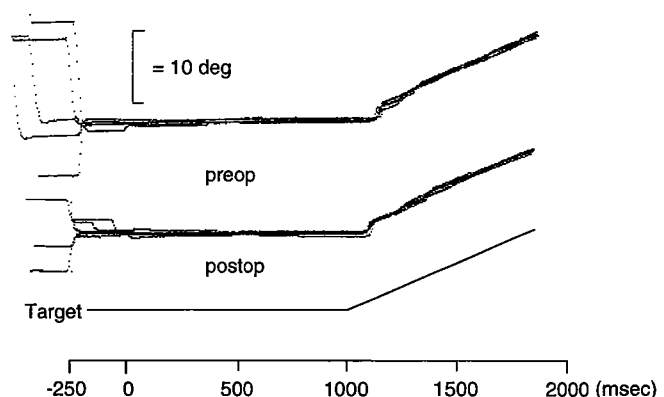
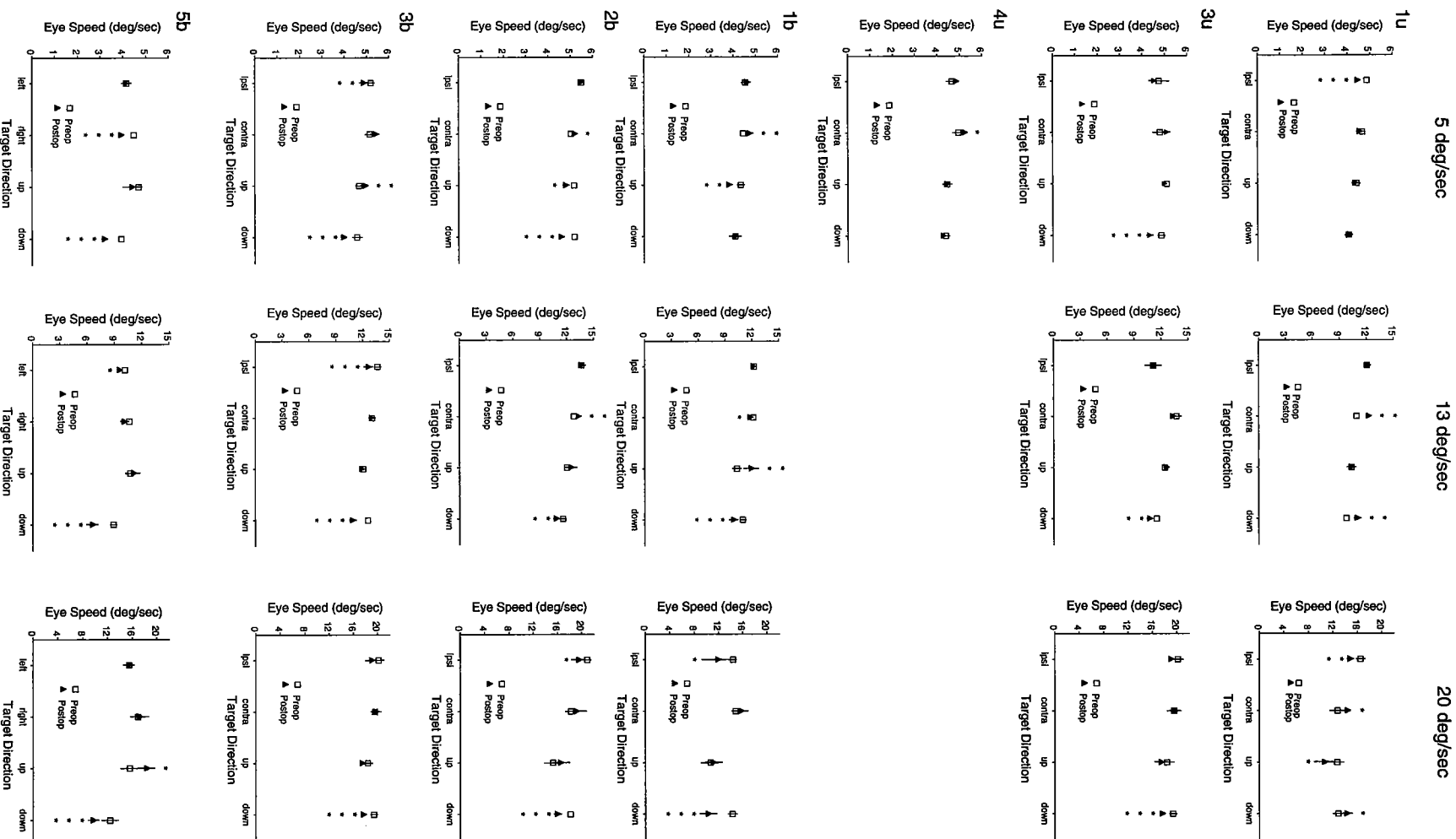


FIG. 16. Five superimposed eye movement records from eye movements to a target moving $13^\circ/\text{s}$ toward the side of one monkey's STP lesion (lesion 2b). Smooth pursuit was not impaired to this target.



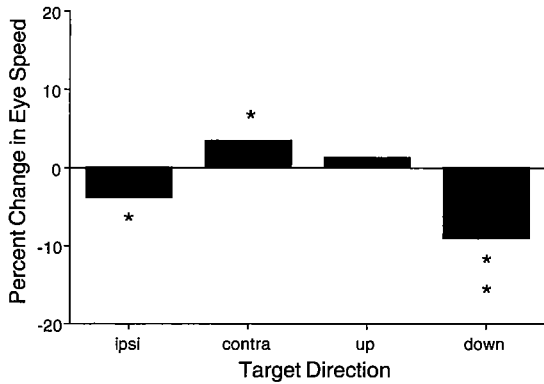


FIG. 18. Mean percent change in eye speed after all STP lesions to all target speeds. Asterisks denote statistically significant differences (* $P < 0.05$, ** $P < 0.01$; 2-tailed t -test for paired observations).

cific result of visual cortex damage. Our results suggest that STP plays a role in visual behavior more akin to that of posterior parietal cortex than to that of IT cortex.

Visually guided saccadic eye movements

Lesions of STP produced an increase in saccade latency to contralateral targets. Although this was consistently significant across animals, there was some interanimal variability in the magnitude of the deficit. The most obvious potential explanation for this is that the size of the deficit is simply related to the size of the lesion. The lesions that produced the largest deficits (*1b* and *4u*) also involved damage to cortex in the lower bank of the superior temporal sulcus. Nevertheless, the lesion that produced the smallest deficit (*1u*) also involved similar damage. Rather than a simple effect of lesion size, we suggest that there are a number of processes that influence the exact magnitude of saccade latency seen after the STP lesions. First is the effect of STP lesions themselves, which is to increase saccade latencies to contralateral targets. Second is a practice effect that decreases saccade latencies to all targets. This perhaps is best illustrated by the animals with IT lesions who have *decreased* saccade latencies to all targets. Although improvement due to practice can explain the decrease in saccade latency to ipsilateral targets after some of the STP lesions, it does not explain the increase in saccade latency to the most peripheral ipsilateral target seen after other STP lesions (*4u* and *1b*). A third factor in the size of the monkeys' impairments appears to be a nonlateralized effect of damage to STP that increases saccade latency to all targets and may be related to lesion size.

Damage to STP produced saccade latency increases that became larger with increasing target eccentricity. That damage to STP resulted in deficits in programming eye movements to peripheral space is in a sense not surprising because receptive fields in STP, like those in posterior parietal cortex, tend often to be large, often as large as an entire quadrant or even the whole visual field (Bruce et al. 1981; Yin and Mountcastle 1977). This contrasts with most retinotopically organized vi-

sual areas, which contain an exaggerated representation of the fovea and with IT, where responses are typically strongest at the center of gaze. Some cells in STP, in fact, respond preferentially to large stimuli appearing suddenly in peripheral space (Bruce et al. 1981; Rodman et al. 1993). The increased impairment to peripheral targets is consistent with both STP and posterior parietal cortex playing a role in visuomotor and visuospatial behavior, because, although the visual acuity and the ability to identify objects rapidly drops off as a stimulus is presented farther away from the fovea, the ability to detect and respond to the appearance of a novel object and its location in the periphery remains quite acute.

Comparison with the effects of other lesions

Lynch and McLaren (1989) have shown that posterior parietal cortex lesions produce saccade latency increases that are quite similar to those seen after lesions of STP. The lesions most effective at producing saccade latency increases included cortex in the intraparietal sulcus as well as area 7a on the surface of the inferior parietal lobule. Lynch and McLaren observed a mean increase in saccade latency of 37.5 ms (117% of preoperative saccade latency) to targets 24° contralateral to the central fixation point. This is similar to the effects of STP lesions, which produce a mean increase in saccade latency of 67.8 ms (125% of preoperative saccade latency) to targets 22° contralateral to the fixation point. Any difference between the effects of posterior parietal cortex lesions and STP lesions on saccades to visual targets is so small as to be indistinguishable with the use of our paradigm.

Like posterior parietal cortex lesions and STP lesions, lesions of the FEF produce a small deficit in making saccades. Monkeys with unilateral and bilateral FEF lesions tended not to make saccades to peripheral contralateral targets in a task that required the animals to remove pieces of apple from a slotted board (Schiller et al. 1980). Using a behavioral procedure similar to ours, Lynch (1992) found saccade latency increases of ~30% after bilateral FEF lesions to targets 24° from the fixation point. This is similar to the deficits seen after both posterior parietal cortex lesions and STP lesions (but see Deng et al. 1986).

Lesions closer to either the retinal input or the motor output tend to produce larger impairments in making saccades than lesions of extra striate cortical areas. For example, lesions of the superior colliculus produce deficits that are slightly more severe than those seen after STP lesions. Wurtz and Goldberg (1972) found that superior colliculus lesions increased saccade latencies by 150–300 ms immediately after the lesions. The effects of striate cortex lesions on saccadic eye movements are more pronounced yet, in that the animals with striate cortex damage initially appear blind and are unable to make saccades to objects of interest that are presented in the contralateral visual field (see Segraves et al. 1987; Zee et al. 1987). Destruction of the paramedian pontine reticular formation causes a complete paralysis of saccadic eye movements (Goebel et al. 1971). Presumably,

FIG. 17. Mean preoperative and postoperative (1st wk of testing) eye speed for each STP lesion to all target speeds. Error bars represent the mean 95% confidence intervals (sometimes the error bars are smaller than the data points for the means), and asterisks denote statistically significant differences (* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$; 2-tailed t -test for unpaired observations). Asterisks are above the data points for increases in saccade latency and below the data points for decreases in saccade latency.

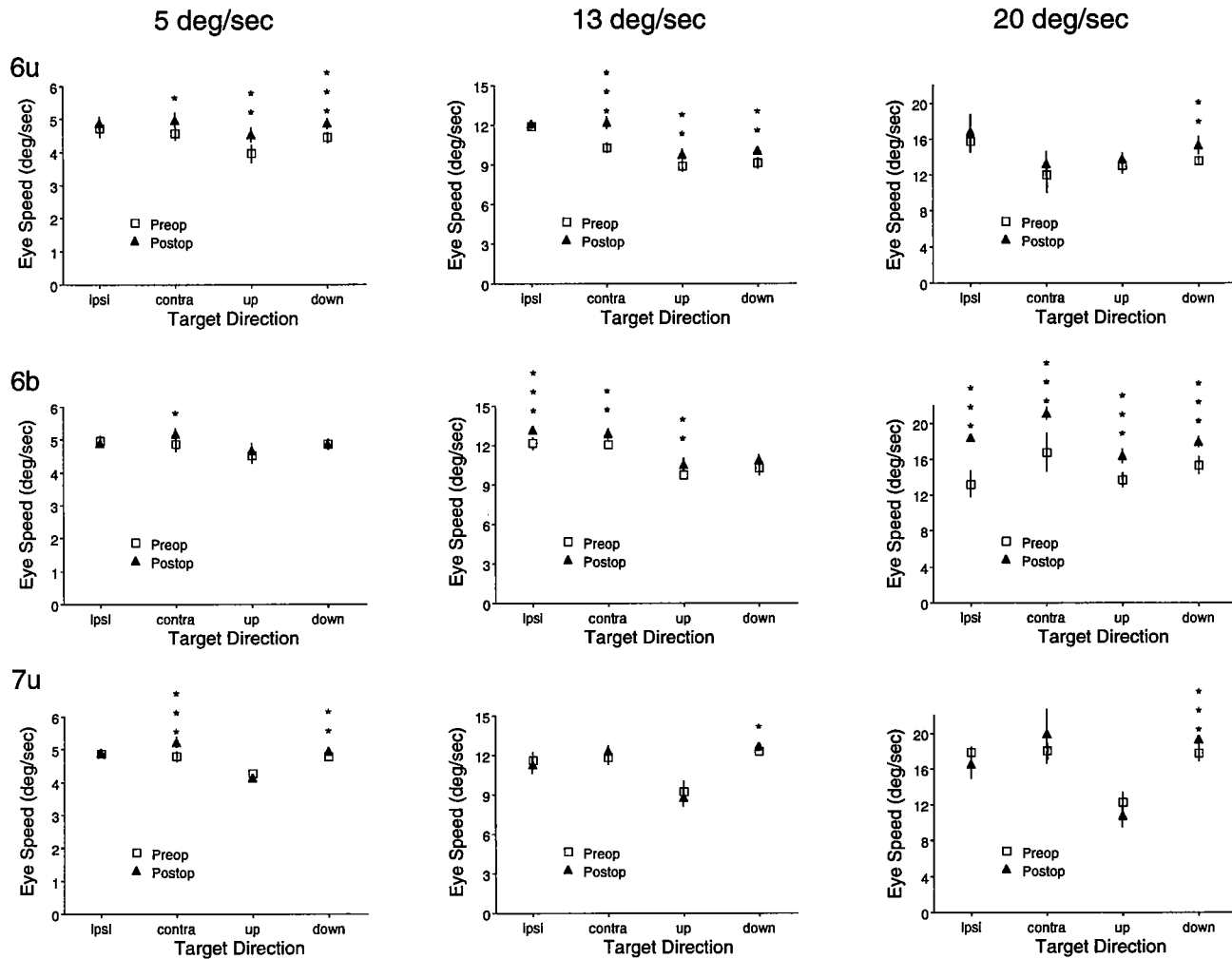


FIG. 19. Mean preoperative and postoperative (1st wk of testing) eye speed for each IT lesion to all target speeds. Conventions are as in Fig. 17.

the difference between the effects of damage to high-order cortical areas such as STP and posterior parietal cortex and areas close to either retinal input or motor neuron output reflects the former areas' involvement in higher cognitive functions rather than purely sensory or motor functions.

Recovery of saccadic eye movements after STP lesions

Consistent with the similarity of effects on saccadic eye movements after STP and posterior parietal cortex lesions, there was a similar course of recovery. Most of the monkeys in Lynch and McLaren's study recovered to preoperative performance in the 3–4 wk that they were tested (J. C. Lynch, personal communication). In our study the animals recovered to preoperative levels within 5 wk of surgery.

Saccades to auditory targets

The one monkey (4) tested with an auditory saccade paradigm did not show any deficit in making saccades that were cued by contralateral auditory stimuli. There was in fact a slight decrease in saccade latency to contralateral targets and a slight increase in saccade latency to ipsilateral targets, perhaps reflecting a shift in the monkey's response bias. Any such shift

is unlikely to have been due to a speed/accuracy tradeoff masking a deficit, because the total percent correct was unchanged. Although single subject data must always be interpreted with caution, the fact that this animal had the largest impairment of any animal in making visual saccades after a STP lesion suggests that STP lesions do not, in fact, impair the ability to make saccades to auditory targets. This finding implies that the saccade latency increase cannot be described as a basic motor deficit (i.e., resembling the effects of oculomotor muscle loss). It would be inaccurate to conclude conversely that the visual saccade latency deficits can usefully be described as sensory, that is, resembling the effects of retinal damage. In fact, it seems that neither STP nor any other cortical area that is multiple synapses away from either the sensory epithelium or the ocular muscles is likely to function in a strictly sensory or motor fashion. As is the case with posterior parietal cortex, it remains an open question whether the deficits following STP lesions are best characterized as an attentional impairment or as a loss of command of movement (for reviews of this issue, see Lynch 1980; Robinson et al. 1978).

Visual fixation

The lack of change in fixation latency after STP lesions provides behavioral evidence that the animals were the same

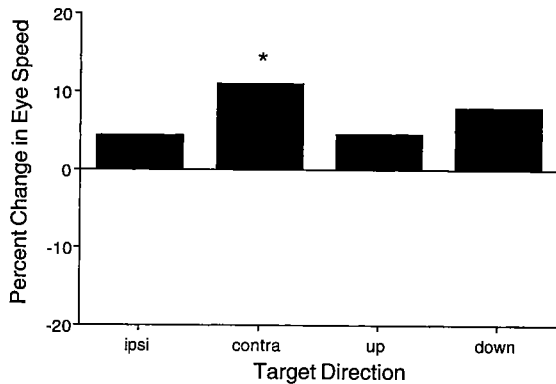


FIG. 20. Mean percent change in eye speed after all IT lesions to all target speeds. Conventions are as in Fig. 18.

level of motivation as they were during preoperative testing. It also implies that the effects of STP lesions on eye movements cannot be attributed to a generalized deficit of attention or visual perception.

It may seem incongruous that there was an effect on saccade latency and not on fixation latency; however, there are two reasons that these findings are not contradictory. First, there was no experimental control of the monkeys' eye position before the fixation point's onset. Although the animals did not adopt a strategy such as diverting their eyes so that the fixation point appeared in the visual field ipsilateral to the lesion, their eye position was usually within 15° of the fixation point at the start of the trial. Because the saccade latency increase was most pronounced to the most peripheral targets, changes in fixation latency might not be apparent if fixation involved, on average, relatively short saccades. Second, as we have shown (Skelly et al. 1991), lesions of the FEF can produce the opposite effect: a fixation latency increase without a saccade latency increase. These findings suggest that visual fixation and the saccade task rely at least in part on different brain areas.

Smooth pursuit eye movements

Damage to posterior parietal cortex produces variable and transient smooth pursuit eye movement deficits (Lynch 1982). Unilateral lesions of posterior parietal cortex have no effect on smooth pursuit. Bilateral (2 stage) posterior parietal cortex lesions produced deficits in making smooth pursuit eye movements to targets moving vertically (4/4 monkeys) and sometimes caused deficits to horizontally moving targets (2/4 monkeys). These effects were similar to those seen after STP lesions in that there was a consistent effect on vertical eye movements (downward only after STP lesions) after bilateral lesions and a less consistent effect after unilateral lesions. Our finding that smooth pursuit speed is decreased to downward moving targets more reliably after one- and two-stage bilateral STP lesions than after unilateral STP lesions appears to be due to the second lesion producing a larger effect than the first rather than an effect of lesion size. There is no consistent relationship between lesion size and whether it was the first or second lesion. The variable effect on horizontal smooth pursuit eye movements seen after STP lesions is also consistent with the effects of posterior parietal lesions.

There is ambiguity over the effects of lesions of the FEF on smooth pursuit eye movements. Following lesions of the FEF, there is sometimes a large reduction in smooth pursuit eye movement velocity (Lynch 1987; Keating 1991). In contrast, Schiller and Logothetis (1987) found no effect of FEF lesions on smooth pursuit. MacAvoy et al. (1991) found that the variable effects of unilateral FEF lesions on smooth pursuit can be explained by the existence of a specific sub-area in the depths of the arcuate sulcus being important for smooth pursuit, as suggested by the effects of lesions, electrical stimulation, and the responses of FEF cells while a monkey does a smooth pursuit task. If it is assumed that the larger impairments seen after some FEF lesions reflect removal of a critical subarea, the effects of FEF lesions on pursuit seem to be larger than those of STP lesions.

Lesions of areas closer to the retinal input such as MT (Newsome et al. 1985), MST (Dursteler et al. 1987), and especially striate cortex (Segraves et al. 1987; Zee et al. 1987) produce larger impairments on smooth pursuit tasks than do lesions of STP.

In summary, STP and posterior parietal cortex lesions produce small deficits in making smooth pursuit eye movements. Frontal eye field lesions and MT/MST lesions appear to produce larger deficits, whereas striate cortex lesions produce even larger impairments.

Role of STP in smooth pursuit

Lesions of STP most reliably caused a decrease in eye speed to targets moving down from the center of gaze. This deficit may be one of downward pursuit per se, or, alternatively, of pursuit of objects moving within the bottom portion of extrapersonal space, because our paradigm does not allow us to differentiate between these possibilities. Indeed, STP and other cortical visual areas involved in the control of pursuit might be expected to devote more processing to either or both downward-directed pursuit or lower field pursuit in ground-dwelling animals. For example, a macaque moving forward toward an object of interest located on the ground a short distance in front of him effectively pursues a downward moving object. In addition, monkeys and other animals that manipulate objects manually, forage on the ground, and groom from a sitting position have a preponderance of visual stimulation within the lower portion of extra personal space, which presumably elicits both upward and downward pursuit movements. It is worth noting that area MT, which provides visual input to STP via a multisynaptic pathway within dorsal extrastriate cortex, contains both a larger representation of the lower visual hemifield and a centrifugal bias in neuronal direction selectivity for moving peripheral stimuli (Albright 1989). It is not yet known whether similar representational biases exist in STP.

FUNCTION OF STP. The anatomic connections, typical single neuron properties, and effects of removal of STP all indicate a closer similarity of STP to the visual areas of the dorsal cortical visual processing stream than to those of the ventral one. Indeed, many of the properties of STP are strikingly similar to those of the terminus of the dorsal stream, posterior parietal cortex. For example, most of the neurons in both areas have large receptive fields that are not visuotopically organized and are often selective for specific types of visual movement (Bruce et al. 1981; Motter and Mountcastle 1981;

Robinson et al. 1978; Yin and Mountcastle 1977). Both areas have cells whose firing is related to saccadic eye movements (Colby and Miller 1986; Lynch et al. 1977; Robinson et al. 1978). The two areas are reciprocally connected to each other and to other dorsal areas such as MST and FST as well as to the frontal eye fields (Boussaoud et al. 1990; Cavada and Goldman-Rakic 1989; Jacobson and Trojanowski 1977; Jones and Powell 1970). Removal of either STP or posterior parietal cortex produces visuomotor deficits while failing to impair object or pattern discrimination learning (Luh et al. 1986; Petrides and Iversen 1979). Finally, as shown for the first time by the present study, lesions of STP have effects on both visually elicited saccades and smooth pursuit eye movements very similar to those reported after lesions of posterior parietal cortex (Lynch 1982; Lynch and McLaren 1989). These findings strongly imply that STP, like posterior parietal cortex, is involved in visuospatial and visuomotor functions. The difference in the roles of these two areas remains a subject for further inquiry.

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