Role of Striate Cortex and Superior Colliculus in Visual Guidance of Saccadic Eye Movements in Monkeys

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SUMMARY AND CONCLUSIONS

1. We studied the effect of lesions placed in striate cortex or superior colliculus on the detection of visual stimuli and the accuracy of saccadic eye movements. The monkeys (Macaca mulatta) first learned to respond to a 0.25° spot of light flashed for 150–200 ms in one part of the visual field while they were fixating in order to determine if they could detect the light. The monkeys also learned in a different task to make a saccade to the spot of light when the fixation point went out, and the accuracy of the saccades was measured.

2. Following a unilateral partial ablation of the striate cortex in two monkeys they could not detect the spot of light in the resulting scotoma or saccade to it. The deficit was only relative; if we increased the brightness of the stimulus from the usual 11 cd/m² to 1,700 cd/m² against a background of 1 cd/m² the monkeys were able to detect and to make a saccade to the spot of light.

3. Following about 1 mo of practice on the detection and saccade tasks, the monkeys recovered the ability to detect the spots of light and to make saccades to them without gross errors (saccades made beyond an area of ±3 average standard deviations). Lowering the stimulus intensity reinstated both the detection and saccadic errors. There was no increase in latency to initiate the saccades, no increase in the number of double saccades, but a slight increase in scatter of the initial saccades around the target as reflected in the standard deviation of the amplitude of the initial saccade.

4. The recovery of performance in an area of the visual field was related to practice since the areas of the visual field that were practiced recovered more rapidly than those areas that were not.

5. Following partial lesions of the superior colliculus there was no detection deficit and no large deficit in saccadic accuracy. There was, however, an increase in the frequency of small corrective saccades and also an increase in the latency for initiation of the saccades as reported previously. The initial saccade to each point was consistently short so that no increase in the standard deviation of the amplitude of the initial saccade was observed.

6. Following joint lesions of both striate cortex and superior colliculus, the monkey appeared to be blind in the part of the visual field related to the jointly lesioned area. No recovery was observed on either the detection or the saccade task after 15 wk of testing.

7. Single units in the superior colliculus that were related to the same area of the visual field as the ablated striate cortex were found to show a higher frequency of enhanced visual responses when the monkey used the receptive-field stimulus as the target for a saccade.

8. From these experiments we conclude that either the striate cortex or the superior colliculus is sufficient to produce visual detection and visually guided saccades. One or the combination of these structures is necessary, since lesion of both structures leads to permanent deficits of visual detection and saccadic accuracy. While the striate cortex and superior colliculus could form parallel and redundant visual inputs to the oculomotor system, our evidence suggests that these structures are complementary and act in concert to accurately guided saccades.

INTRODUCTION

Primates have both visual acuity concentrated in the foveal area of the retina and a saccadic eye movement system to move the fovea from one part of the visual field to another. In the process of initiating such saccades, the nervous system must both select the visual targets and transform the spatial coordinates of these targets into the pulse and step code necessary for production of accurate eye movements by the oculomotor sys-

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tem (22). Information about the location of the target could be derived from either branch of the visual system: the midbrain pathway terminating primarily in the superior colliculus or the geniculocortical pathway terminating in the striate cortex.

In the superior colliculus of the monkey, cells in the superficial layers discharge in response to visual stimulation (8, 10, 25, 36) and frequently respond better when the visual stimulus is the target for a saccadic eye movement (9, 39). Cells in the intermediate layers discharge before eye movements (15, 25, 26, 28, 36, 37). In addition, electrical stimulation of the superior colliculus elicits an all-or-none saccade of a specific amplitude and direction (21, 26), which is consistent with the retinotopic map of the visual and eye movement-related cells. Taken together, this evidence suggests that the superior colliculus plays a central role in the initiation of visually guided saccades. However, there is disagreement in the literature regarding the effect of collicular lesions on the guidance of saccadic eye movements. Several studies demonstrate that monkeys with collicular lesions can apparently make accurate saccades (17, 38) while other authors suggest that monkeys with collicular lesions are not able to make accurate eye movements (5, 24). In addition, Wurtz and Goldberg (38), using monkeys trained to make saccades to a visual target, found that there was a longer latency to initiate visually guided saccades in monkeys with collicular lesions.

In the striate cortex of monkeys no cells discharging before saccadic eye movements have been reported (35) although some possible eye movement-related input might be present (see ref 40 for discussion and references). The retinotopic map and precise localization of the visual receptive fields of striate cortex cells make this area a logical one for providing precise visual target location for guiding eye movements. In addition, electrical stimulation of the striate cortex of monkey can elicit saccadic eye movements (23). While perceptual and detection deficits have been studied in detail following ablation of striate cortex (6, 32), no measure of the saccadic accuracy following lesions restricted to the striate cortex of monkeys has been reported.

In this study we attempted to study the role of the superior colliculus and striate cortex in the visual guidance of saccadic eye movements. The logic of our experiments rested on two key strategies. First we determined whether the monkey could "see" a given point of light before measuring his ability to saccade to the point. To do this we used a detection task comparable to static perimetry tests used to detect visual def-

icits in humans and then measured the latency and accuracy of the saccadic eye movement to the same point. Second, we made no attempt to ablate the entire striate cortex and superior colliculus with the attendant problems of overlapping other areas or leaving parts intact. Instead we used the known retinotopic organization of each structure (confirmed with direct microelectrode recording and visual receptive-field mapping) to select a part of the visual field to be damaged. Evaluation then concentrated on comparing the accuracy of saccades to targets in the visual field related to the damaged brain to accuracy to targets in undamaged areas and to prelesion controls.

Our results suggest that either striate cortex or superior colliculus, but no other nuclei, are sufficient for the visual guidance of saccadic eye movements. This paper also demonstrates that the superior colliculus mediates the recovery from the scotoma produced by a striate cortex lesion and reports on changes in the response of collicular cells that are correlated with this recovery.

A report of part of this work has been presented earlier (14).

METHODS

General behavioral and recording procedures

All monkeys were initially trained on a behavioral fixation task which has been described previously (34). During the experimental sessions the monkeys sat in a primate chair facing a tangent screen 57 cm in front of them. By depressing a bar the monkey turned on a fixation light which stayed on for a variable length of time between 1 and 3 s. The fixation light then dimmed for about 0.5 s, and if the monkey released the bar during this time he was rewarded with a drop of water. The fixation light was small enough and the dim subtle enough so that the monkey fixated on the point during the fixation period. The bar was disabled for several seconds following each release of the bar; early or late release of the bar was neither rewarded nor punished. The fixation point was either a 0.1° or 0.25° circular spot of light with a brightness of 11 cd/m² against a usual background illumination of 1.0 cd/m², as measured by a Salford Electrical Instruments, Lancashire, England photometer.

The monkeys were generally allowed as much water as they were willing to work for in a test session. At the end of the session the monkeys were removed from the primate chair and returned to their home cages. The monkey's weight and water consumption were recorded daily.

During all behavioral testing the monkey's

head was held rigidly. Four bolts were implanted into the skull (7). A horseshoe-shaped ring was attached to the bolts with the open end pointed toward the back of the head and a socket attached to the forward end. The socket was attached to a rod fastened to the primate chair by a single taper pin connection so that the head was held in exactly the same position on successive recording days. Electrooculograms (EOG) were recorded via nonpolarizeable electrodes (1) implanted at the outer canthi of each eye and above and below one eye. Band pass of the amplifier used was usually DC to 25 Hz.

An on-line PDP-12 digital computer was used (as previously described (15)), to program the monkey's behavioral tasks, record his responses, present a moving display of his eye movements, trigger a Grass camera to photograph eye movements on selected trials, and measure parameters of eye movements and behavioral responses.

Detection task

Following training on the fixation task and implantation of EOG electrodes, monkeys were trained in a visual detection task. The monkey again looked at the fixation light which dimmed on 25% of the fixation trials. On about 70% of the trials the fixation point did not dim, but instead another spot of light on the screen was flashed on for 150 or 200 ms. On these trials the monkey was rewarded if he released the bar within 500 or 600 ms of the onset of the flash. These spots of light were 0.25° in diameter with a brightness of 11 cd/m² and were generated by fixed bulbs behind the screen. The duration of the flash was set so that it was shorter than the reaction time for a saccadic eye movement to the stimulus, but no effort was made to prevent any attempted eye movement to the area of the flashed stimulus. The light could come on in three areas. On approximately 45% of the trials (two-thirds of the flashed trials) the light was located at one point in a grid of points centered on the right visual hemifield (illustrated in Fig. 1A) which covered the area of the visual field where vision would be impaired following later cortical or collicular ablations. In Fig. 1A the fixation point is indicated by FP and the numbers (0 or 1) are placed at the position of the various target lights; data during experimental sessions were obtained entirely from detection of points on this test grid. On approximately 25% of the trials (one-third of the flashed trials), the fixation point remained on and one of two other "control" lights in other quadrants of the visual field were flashed on; this forced the monkey to use all quadrants rather than concentrate on just the hemifield with the test grid. The
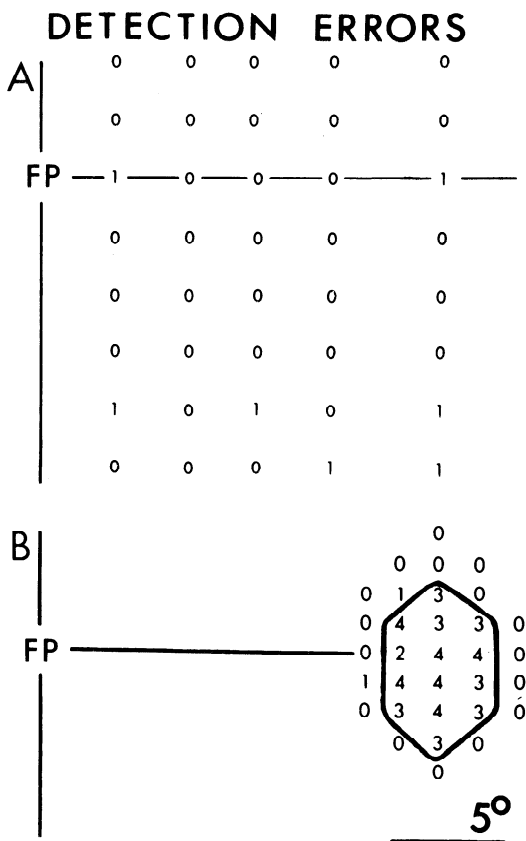

FIG. 1. Detection task. Position of numbers on test grid in *A* showing spacing of 0.25° lights which were flashed on while the monkey fixated at point FP. The grid lights were separated by 3.2° vertically and 2.4° horizontally between the first four columns and 3.6° between the last two columns. In this and all subsequent figures the intensity of these lights was 11 cd/m² against a background of 1 cd/m² unless specifically noted. Numbers indicate the errors in four trials at each point of the grid for monkey *553* and is typical of prelesion performance by the monkeys on the detection task. In *B* the location of the visual blind spot of the right eye (with the left eye covered) is shown using the same detection task but with lights more closely spaced in one part of the test grid. The solid lines surround the area where more than one error was made in four trials. Monkey *L414*.

remaining 5% of the trials were blank trials with no light flashed on; this permitted evaluation of the monkey's response when no detection of the light flash was possible. The monkeys were not able to guess when a flash should occur and therefore never received reward for a correctly timed response on one of these blank trials. The order of occurrence of fixation trials with no detection flashes, of flashes on the test grid, and

of flashes at the two control points was randomized by the computer. Therefore, on each trial the monkey did not know whether or not a flash detection might be required, when in the fixation period the flash might occur, or where on the tangent screen the flash might occur.

In a testing session the grid points, control points, and blank trials were given in a sequence that was selected so that the target did not appear in the same part of the grid on successive trials, i.e., in geometrically random order. After one response to a grid point had been recorded, the next grid point was selected and another series of fixations and flash detections was run until the flash occurred on the next grid point. Four runs through the entire grid were usually done in each experimental session; the session usually required about 350 fixation trials. The computer collated the errors (failure to release the bar within 500 or 600 ms after the flash on the test grid) made for each point on each trial, and the latency of the response if a correct response was made, and it then printed these results out for each grid point at the end of the four runs through the grid list. The computer also automatically photographed the display of the EOG record for each trial on selected days to make certain the monkey was not making eye movements before the flashed point of light was turned off.

Monkeys were trained on this task until the monkey made no more than one error in four trials of each point on the test grid from day to day. An example of the behavior of one fully trained monkey is shown in Fig. 1A for four runs through the list of grid points on one day; numbers of errors are indicated on each grid point. As was typically the case for normal monkeys, this monkey was able to perform the detection task with only a few scattered errors. The ranges of latency for bar release on this task for two monkeys tested extensively prior to any lesion were 353–457 ms (monkey 553, 500-ms error criteria used) and 420–539 ms (monkey L414, 600-ms error criteria used). There was a tendency for the longer latencies to be associated with more distant flashes.

Evidence of the sensitivity of this task was obtained by testing for detection within the blind spot. In this experiment the monkey had the vision in his left eye blocked by a patch. Detection errors in four runs through a part of the grid in the area of the presumed blind spot is shown in Fig. 1B. The area with more than one error at each point is outlined and is 4.5° across, 7.0° up and down, and centered 17.5° lateral to the fixation point. The blind spot in two other monkeys was found to be about the same size and 16° and 17° lateral from the fixation point. These values are comparable to those reported earlier by Cowey ((2), approximately 6° in diameter and centered 16.5° lateral to the fovea).

Saccade task

Each monkey also learned a task which required a saccadic eye movement from the fixation point to another point in the visual field. Each time the monkey depressed the bar, the fixation point came on and then dimmed, as described above on about 25% of the trials. On 70% of the trials the fixation point was turned off and simultaneously a target light (one of the same lights used in the detection task) was turned on at a different point on the screen. The monkey made a saccadic eye movement from the fixation point to the target light so that he could now fixate on the target light and release the bar when the target light dimmed. The signal for the monkey to make a saccade was that the fixation point went out; in the detection task the flash occurred while the fixation light remained on. Like the detection task, on about 45% of the trials (two-thirds of the saccade trials) these saccade targets were located in the same grid of points in the right hemifield. On the other 25% of trials (one-third of the saccade trials) the target point was in one of two other quadrants. In order to measure the monkey's strategy when no target was available, on approximately 5% of the trials the fixation point was turned off, but no target light was turned on. The occurrence of fixations, blank trials, and saccades to the various visual quadrants was again randomized so that on any given trial the monkey did not know whether or not he would be required to make a saccade nor did he know where the saccade target would be. An experimental session again generally consisted of presenting each point on the test grid four times.

The amplitude of the monkey's eye movements during the saccade task was determined from digitized horizontal and vertical electrooculograms. This digitized record was generally adjusted to have a resolution of one bit per 0.5° of eye movement amplitude in either horizontal or vertical channel; eye movements were sampled every 4 ms. From this digitized data the computer identified the start of eye movements as a deflection in one oculogram channel that was greater than 1.5° within two consecutive samples (8 ms). The termination was identified as a failure to detect a difference greater than 1.5° within two consecutive samples. The amplitude of an eye movement was then calculated as the change in level between start and end of the eye movement and was independent of the absolute poten-

tial of the oculogram at the start of the eye movement.

During the experimental sessions using the saccade task, the computer used this eye movement-recognition procedure to identify the first saccade occurring after the signal to saccade and stored the amplitude of the horizontal and vertical components and the latency of the saccade (measured from trigger to saccade to the start of the eye movement). These values were collated for each point during the four runs through the test grid and then printed out by the computer along with the means and standard deviations for each of the values. For each saccade to a point on the test grid the computer also activated a camera which photographed and labeled the digitized display of an eye movement on that trial. This permitted visual inspection of the eye movements to identify double saccades as well as occasional elimination of trials in which the EOG was off one edge of the recording channel or was contaminated by an artifact.

Monkeys were trained on the saccade task until the standard deviation of the initial saccade was about ± 1° for each target point on the test grid and consistent from day to day. This usually required about 10 days of practice on the task. At this time an "average standard deviation" for horizontal and vertical saccade components was computed by taking the mean value for all of the horizontal and vertical standard deviations, respectively. For the three monkeys on which these data were obtained, the average horizontal standard deviation was ± 0.6–0.75° and the average vertical standard deviation was ± 0.9–1.0° for the points within 15° of the fixation point. When evaluation of the lesion required testing beyond 15° horizontally, separate average standard deviations were calculated for those points beyond 15° on the horizontal; for this more distant grid of points the average standard deviations were approximately 50% larger than for the near grid. The range of latencies for the saccades was: 188–229 ms (553), 195–350 ms (H072), 165-223 ms (L414), and 189-240 ms (M648). Latency tended to increase with length of saccade.

The accuracy of the monkey's initial eye movement to each target light was then evaluated in three ways, from least to most sensitive: number of gross errors, frequency of small corrective saccades, and size of standard deviations of the initial saccades. Any initial saccade which missed the saccade target by more than 3 average standard deviations was arbitrarily called a gross error of saccadic accuracy. In Fig. 2, the solid line rectangle encloses this 3 standard deviation region about the target point (located at the number 1). Note that one of the four end points of the saccades (filled circles) was outside

the rectangle and, therefore, was an error. While the magnitude of this error criteria depended on the average standard deviation found for each monkey, roughly speaking, any saccade which missed the target by more than 2° horizontally or 3° vertically was called a gross error. Figure 2 illustrates the frequency of saccadic errors for a normal monkey at the end of training; this monkey made one saccadic error in four trials at a few points and no saccadic errors at the other points.

The occurrence of small corrective saccades was determined by visual inspection of the film record or the on-line display of the digitized electrooculograms of each trial. If a second saccade occurred within 100–400 ms after the end of the initial saccade and was greater than 0.5° (the limit of resolution of our display), it was counted as a corrective saccade.

Smaller changes in the accuracy of the visually guided saccades were evaluated by calculating the standard deviation as a measure of scatter of the initial eye movements. In order that the standard deviation would be sensitive to slight inaccuracies in the guidance of saccades, all trials on which the monkey made gross errors of saccade accuracy were eliminated prior to calculation of the standard deviation.

Lesion procedures

When monkeys had been trained on both the detection and the saccade tasks, their perfor-
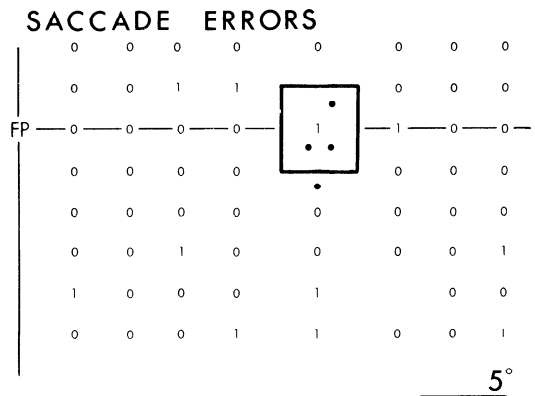
SACCADE ERRORS

FIG. 2. Saccade task. Position of numbers show location of saccade target lights which came on as fixation point (FP) went off. Same fixation point and grid of lights as used in Fig. 1. Numbers indicate the number of gross saccade errors made (see METHODS for explanation) at each point in four trials through the grid. The rectangle around one point shows schematically the area of one standard deviation around the target point. Dots near the rectangle indicate end points of the actual saccades made to that point. Prelesion data for monkey 553.

mance was recorded over several days to obtain a prelesion baseline. The amount of testing each animal did per day varied from a complete run of four trials on half of the grid for one task to a complete run of the full grid for both tasks. Monkeys were usually tested on 5–6 days/wk just prior to the lesion and for several weeks after the lesion and then on 2 to 3 days/wk after that. The monkeys and the sequence of lesions in each monkey are indicated in Table 1.

Focal lesions of the striate area of visual cortex were made by subpial suction under aseptic conditions. The monkeys were immobilized with 6 mg intramuscular Sernylan (phencyclidine hydrochloride) and anesthetized with 30 mg/kg intrahepatic Nembutal (sodium pentobarbitol). A bone flap over the right occipital lobe was removed for replacement following surgery and the dura retracted. An area of striate cortical gray matter was then removed so as to affect the central 10–30° of the contralateral lower visual field but spare the fovea (4, 29). The monkeys were allowed to recover 5–7 days before behavioral testing.

One monkey had a complete unilateral ablation of the striate cortex. The gray matter was removed from the dorsolateral striate cortex to within 2 mm of the inferior occipital and lunate sulci. In addition, we aspirated the gray matter in the medial and deep calcarine gyri corresponding to von Bonin and Bailey's area OC (30).

Electrolytic lesions of the colliculus were made by passing current through a microelectrode. The lesion was placed so as to affect the same portion of the visual field as was or would be affected by the striate cortex lesion. This localization was made possible by recording the position of the visual receptive field of the first superficial layer cells encountered as the microelectrode reached the superior colliculus. When this testing indicated that the electrode was in the proper portion of the superior colliculus, the electrode was advanced 1–2 mm further, the visual receptive field of the cells was verified, and a lesion of approximately 2 mm diameter was made by passing an electrode-positive DC current of 400 μA for 20 min. This was done in

awake monkeys who showed no signs of discomfort. The monkeys were tested starting on the following day.

Electrophysiology

Single cells were recorded from the superficial layers of the superior colliculi of two monkeys (L414 and M648) after they had recovered from a striate cortex lesion but before any lesion had been made in the superior colliculus. The monkeys sat in the primate chair with head restrained and faced a tangent screen as in the behavioral testing. Visual receptive fields were mapped with stationary stimuli while the monkey fixated (34). Poststimulus rasters and histograms were obtained while the monkey fixated and were compared to the visual response when the monkey used the stimulus as a target for a saccadic eye movement (9, 39). Details of the single cell-recording procedures have been described in detail previously (14).

Histology

Under deep nembutal anesthesia all animals were perfused with normal saline followed by 10% formalin. Dura was removed, the brain photographed, and an outline of the dorsal cortical lesion area drawn onto a standard brain drawing. Sagittal or coronal 50-μm frozen sections were then made of the occipital lobe containing the striate cortex lesion and used to verify the extent of the lesion into the underlying cortex. Coronal 50-μm frozen sections were made through the superior colliculi and stained with cresyl violet. The extent of the collicular lesions was indicated on tracings made from enlargements of these sections.

RESULTS

Deficits following striate cortex lesions

Unilateral lesions were made in part of the striate cortex of two monkeys and all of the striate cortex in one monkey. Figure 3 illustrates the trapezoid-shaped area of removal of cortical gray matter on the dorsal surface of one monkey. The base of the trapezoid-shaped lesion

TABLE 1. *Experimental monkeys and sequence of lesions*

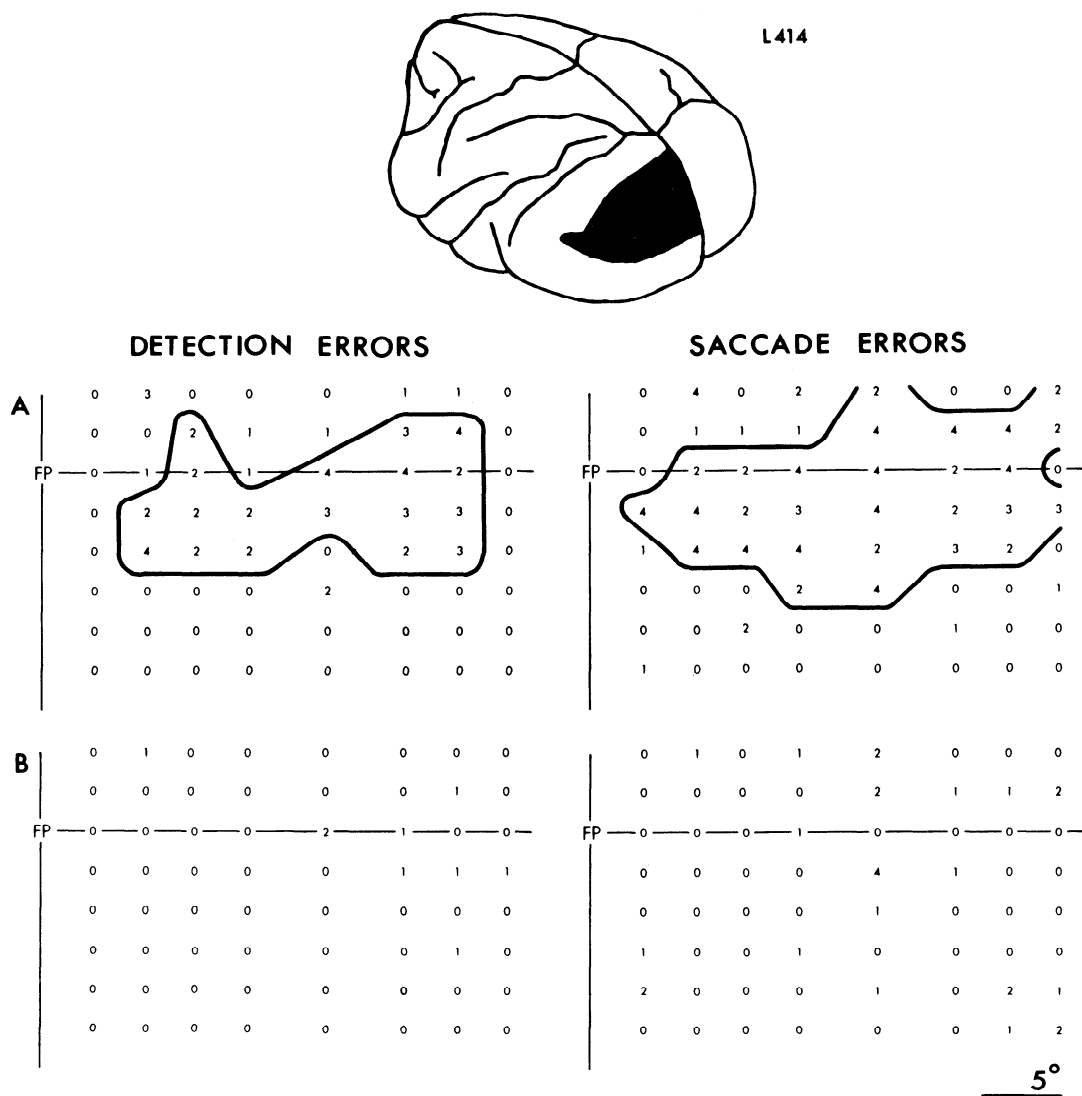| Monkey No.	Weight, kg	Sex	1st Lesion	2nd Lesion
L 414	4.5	F	Striate cortex	Superior colliculus
M 648	5.5	F	Striate cortex	Superior colliculus
P 496	5.3	F	Striate cortex (complete)	
553	4.0	M	Superior colliculus	Striate cortex
H 072	9.0	M	Superior colliculus	

FIG. 3. Detection errors and gross saccade errors following partial lesion of the left striate cortex. The drawing shows the extent of the lesion on the dorsal surface but it also included the lateral bank of the ascending limb of the medial calcarine gyrus from midline to about 15 mm lateral. Histological sections showed that the lesion was restricted to striate cortex. In *A* the number of detection errors on the 8th prelesion day and number of gross saccade errors on the 6th and 7th post lesion days are indicated for four runs through the test grid. The solid lines enclose points where the monkey made two or more errors; note that the areas of detection and gross saccadic errors lie in the same region of the visual field. In *B* the numbers indicate the number of errors in four runs taken over several days about 4 wk postlesion.

was 15 mm long on the midline positioned so that the anterior border (side of the trapezoid) was 6 mm posterior to the lunate sulcus. The height of the trapezoid-shaped lesion was 15 mm extending ventrolaterally from midline and the width of the lesion at the top of the trapezoid was about 7 mm. In addition to that illustrated in Fig. 3, the lesion included a triangular-shaped segment of

adjacent cortical gray matter in the lateral bank of the ascending limb of the medial calcarine sulcus. The base of the triangle was the same as the base of the trapezoid and the height of the triangle was about 15 mm.

The effect of the illustrated lesion on visual detection after one week of recovery is shown on the left of Fig. 3A (see Fig. 9 for the extent

and effect of a cortical lesion on monkey M648). The solid lines enclose those points where the monkey made two or more detection errors in four trials, that is, he failed to respond within 500 ms after the test flash. In this and following figures, we enclosed the areas with two or more errors in such a way as to include a minimum number of points for which there was no apparent deficit. The errors are in contrast to nearly error-free performance prior to the lesion. The deficit could not be accounted for simply by an increase in reaction time: extending the flash to 1 s (5 times normal duration at 11 cd/m²) and time to respond to 1 s (2 times the normal reaction time) in one monkey (M648) did not reduce the number of detection errors.

The effect of the lesion on performance of the saccade task is illustrated on the right in Fig. 3A. The solid lines outline the area where 2 or more gross saccadic errors (initial saccade was more than 3 average standard deviations away from target point, see METHODS) were made on the 1st day of testing following the lesion. Both the deficits in visual detection and saccadic accuracy are confined to roughly the same portion of the visual field, an area extending approximately 25° laterally from the fixation point and from 2.5° above to 5° below the level of the fixation point. This corresponds closely to the portion of the visual field that one would predict from the study of Daniel and Whitteridge (4).

Because of the combined detection deficit and visually guided saccade deficit, the most parsimonious conclusion is that the saccadic deficit is simply secondary to the visual deficit. Further confirmation for this conclusion came from the results from blank trials when no target light was presented. On these blank trials the monkey adopted a strategy of making saccades to the middle of the deficit area; this initial erroneous saccade was followed by a second saccade to the target. Most of the saccades to targets in the deficit area fell within the range of saccades made when no target appeared. We assume that the monkey could not distinguish betweeen no-saccade targets and targets which appeared in the deficit area. This strategy was useful to the monkey in that it brought his eyes closer to the target for targets in the "scotoma," but it confounds the results in that some of these saccades would be near the target by chance. We rewarded another monkey for making a saccade to the left of the fixation point on the blank trials in order to have a clearer response indicating that the monkey had not seen the target. This monkey then made saccadic eye movements to the left of the fixation point when the saccade target came on in the deficit area. In other words, the monkeys with cortical lesion made the same saccades as if no

target was present when the target was in the deficit area. We conclude that the monkeys with striate cortex lesions were making grossly inaccurate saccades because they could not see the saccade target.

These deficits in visual detection and saccade accuracy were not absolute. If the intensity of the target light was raised from the usual 11 cd/m² to 1,700 cd/m² at about a week after the lesion, the monkey was able to detect the flashes and saccade with no gross error to the target points. This "recovery" was not due to light scatter since the same intensity lights on the blind spot (as in Fig. 1) were not detected.

Recovery following striate cortex lesion

The deficits following cortical lesions gradually recovered so that after 3 wk of testing (4 wk postlesion) the deficit shown in Fig. 3A had recovered to the point shown in Fig 3B. In the visual detection task only one point showed as many as two errors, and the deficit at this point was not stable from day to day. There was also no increase in latency of response in the visual detection task; response latency decreased somewhat through the test grid in comparison to prelesion trials. On the saccade task there were only four points in the initial deficit area that showed two or more gross saccade errors, and these three points did not show a consistent deficit from day to day.

The time before recovery was longer for the other two monkeys with striate cortical lesions. The monkey with the smallest cortical lesion (monkey M648 in Fig. 9) required 6 wk for recovery comparable to that shown in Fig. 3B. The monkey with the total striate cortex lesion (P496) completely recovered for 1,000 cd/m² spots in 2 wk of testing and had recovered for test spots of 11 cd/m² at most points in the central 20° of the tested field after 3 wk of testing. This animal died of an acute gastric infection before testing was completed, but the progression of recovery was similar, but slower than monkeys with partial cortical lesions. Each monkey required at least 30 trials to each point before recovery was noted.

Following recovery from striate cortex lesions, when the monkeys made few gross errors on the saccade task, it was possible to evaluate the accuracy of the monkey's saccadic eye movements using the more sensitive measures of saccadic accuracy. There was no consistent change in the frequency of small corrective saccades in monkeys L414 and M648. For monkey L414 (see Fig. 3), the monkey made more small corrective saccades to targets more than 15° away, but this increase in frequency of corrective saccades was seen for points both inside and

outside the original deficit area; this probably reflects a change in strategy rather than a specific deficit. For the second monkey, *M648*, with a small cortical lesion (see Fig. 9), the monkey made no small corrective saccades prior to the lesion and no corrective saccades after recovery from the lesion.

The standard deviation is one measure of the scatter of several saccades around their end point and should reflect slight changes in the accuracy of the saccadic guidance system. Figure 4 shows samples of the standard deviation of seven saccades to targets both inside (shaded) and outside (not shaded) the defective field for the monkey whose lesion is illustrated in Fig. 3. Each rectangle is the graphic representation of ± 1 standard deviation from the average end point of the initial saccade to that point. The clearest change occurs in the right-hand column of Fig. 4; the standard deviations are increased slightly in the cortically deficient field after the lesion (lower part of Fig. 4) as compared to before the lesion (upper part of the figure). The shift in the position of the two points near the bottom of the column is partly ascribable to the occurrence of corrective saccades. While this increase in standard deviation was not large, it was consistent from day to day for the one monkey tested repetitively. For this monkey (*L414*) we compared the areas of the standard deviation rectangles (based on eight saccades to each target point) prior to the cortical lesion to the corresponding areas on 3 days after recovery from the lesion. On the postlesion days 75, 79, and 81% of the points in the initial cortical deficit area (inside solid lines of Fig. 3*A*) had an increased area of the standard deviation rectangle. In contrast, 47, 59, and 66% of the points outside the cortically deficient area showed an increased standard deviation area. The percentage of points with an increased area was greater than 50% for the points in the deficit area and was no different than 50% for points outside the deficit area (90% confidence interval). Quantitatively this effect was very small. Prior to the cortical lesion the average area of the standard deviation rectangles was 0.6 deg^2 for this monkey and the average increase was 0.2, 0.8, and 1.0 deg^2 for the points in the cortically deficient field. This is analogous to a change from a 0.8° by 0.8° square to no more than 1.3° by 1.3° square. Monkey *M648*, with a small number of lesion points, showed no increase in the area of the standard deviation rectangles. There was no increase in the latency for initiation of saccades over prelesion values. Therefore, after recovery from striate cortex lesion, when the monkey was able to detect visual stimuli, he could saccade to the visual stimuli with the same reaction time and very nearly the same degree of accuracy as before the lesion.
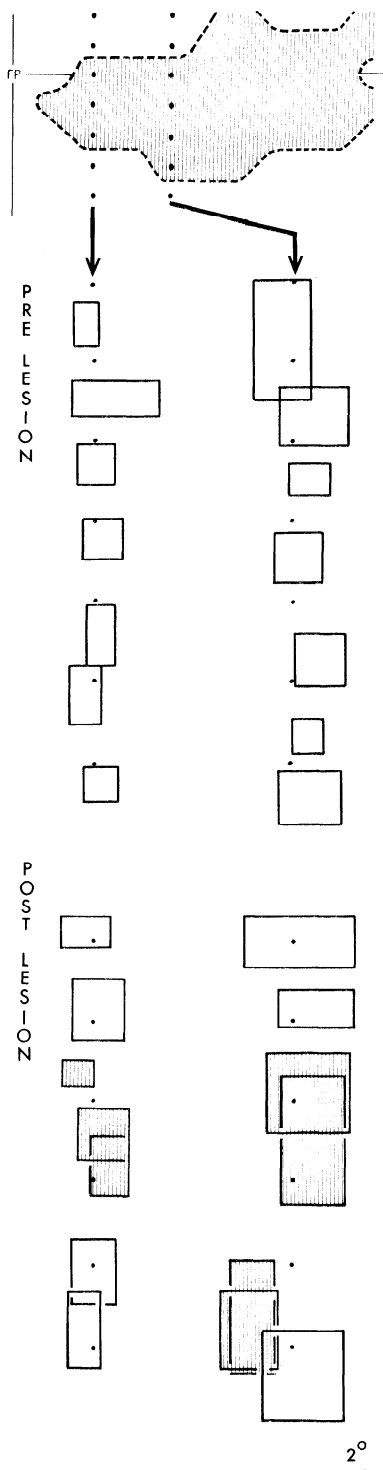

FIG. 4. Slight increase in standard deviation of saccadic eye movements following recovery from partial lesion of striate cortex. The shaded area on the drawing at the top outlines the area of gross saccade

As mentioned previously, the deficit immediately following a striate cortex lesion was dependent on stimulus intensity; the monkey made gross saccadic accuracy errors to targets of 11 cd/m² but did not make such errors if the intensity was raised to 1,700 cd/m². Similarly, when the monkey was tested at 3 cd/m² after it had recovered from the effects of the striate cortex lesion as measured with 11 cd/m² spots, the monkey again made saccadic accuracy and visual detection errors in the cortically deficient field. The recovery process was not complete but represented a decrease in the visual threshold elevation that was produced by the cortical lesion.

Deficits following superior colliculus lesions

Two monkeys received unilateral lesions of the left superior colliculus. The drawing of coronal sections through the superior colliculus in Fig. 5 shows the extent of the collicular lesion in one of these monkeys (553). Note that the lesion primarily involves the superficial layers with a small extension into the intermediate layers. This lesion would be expected to affect vision in the lower visual field contralateral to the lesion and to spare the fovea (3, 8).

The errors in the visual detection task following this lesion are shown on the left in Fig. 5. The monkey made only one error in four trials to several points and no errors to the other points. In the visual detection task, the monkey responds to the occurrence of the test flash with a hand movement and in this task there was no increase in response latency after the collicular lesion: 4 of 19 points in the presumed area affected by the superior colliculus lesion (see saccade latency-deficit area in Fig. 5, right) showed an increase in latency of 35 ms or less while the latency of the remaining 15 points was reduced by as much as 60 ms. To try to bring about a deficit in visual detection we reduced the inten-

sity of the target lights from 11 to 2 cd/m² against a background of 1 cd/m² and the monkey was retested in the visual detection task on the 8th day following the lesion. The monkey made no more than one error in four trials at any point in the visual field affected by the collicular lesion. We conclude that in our tests there was no visual detection deficit following a lesion of the superior colliculus. These target lights might still have been brighter than those of Latto and Cowey (12) who found a slight detection deficit following lesion of the colliculus.

The right grid in Fig. 5 shows the frequency of gross eye movement-accuracy errors following the same lesion of the superior colliculus. The monkey made two grossly inaccurate saccades in four trials to one point in the visual field and no more than one error to all other points; this was similar to this monkey's prelesion performance which was illustrated in Fig. 2. There was, however, a consistent increase in the latency for initiation of the eye movement as demonstrated previously by Wurtz and Goldberg (38). The dashed line encloses all points where saccade latency increased by more than 50 ms over prelesion latency to the same point. The maximum latency increase was 240 ms.

Figure 6 illustrates that a superior colliculus lesion produces an increase in the occurrence of small corrective saccades. Monkey 553 (left column of Fig. 6) received the lesion illustrated in Fig. 5 and monkey H072 (right column of Fig. 6) received a similar lesion in the right superior colliculus. Monkey H072 was tested on a larger grid using a slightly different sequence of stimulus presentations. Prior to the collicular lesion, the monkeys made no more than one corrective saccade in four trials if the target was within 25° of the fixation point. Monkey H072 made corrective saccades on nearly all trials if the target was beyond 30° from the fixation point and the initial saccade was nearly always short of the target. Immediately after the collicular lesion, there was a marked increase in the number of corrective saccades which was most pronounced in the portion of the visual field affected by the collicular lesion (the dashed lines surround points which showed an increase in saccade latency). The solid line encloses points in the visual field where the monkeys made three or four corrective saccades in four trials. The amplitude of the second saccade of the double saccade ranged from 0.5° to 2.5° (limit of resolution for the electrooculogram) for monkey 553 and from 0.5° to 5° for monkey H072. The corrective saccades were nearly always in the direction of the initial saccade indicating that the initial saccade was short of the target. On monkey H072, we determined that the small second saccade was a visually guided corrective saccade; turn-

errors found immediately postlesion for the same monkey (L414) as in Fig. 3. Dots on the drawing show the locations of two columns of grid points through the lesion area for which the standard deviations are drawn below for a prelesion day (upper set of rectangles) and a day 6 wk postlesion (lower set of rectangles). Rectangles in these columns are centered at the average end point of the initial saccade and represent the standard deviations of the horizontal and vertical EOG for the target points indicated by the adjacent dots. Mean and standard deviations are based on seven or eight saccades to each grid point. Shaded rectangles indicate those points falling in the lesion area. All values on the postlesion day were multiplied by 1.2 to compensate for a decrease in the gain of the EOG electrodes; this value was determined from the changes observed at grid points unaffected by the lesion.
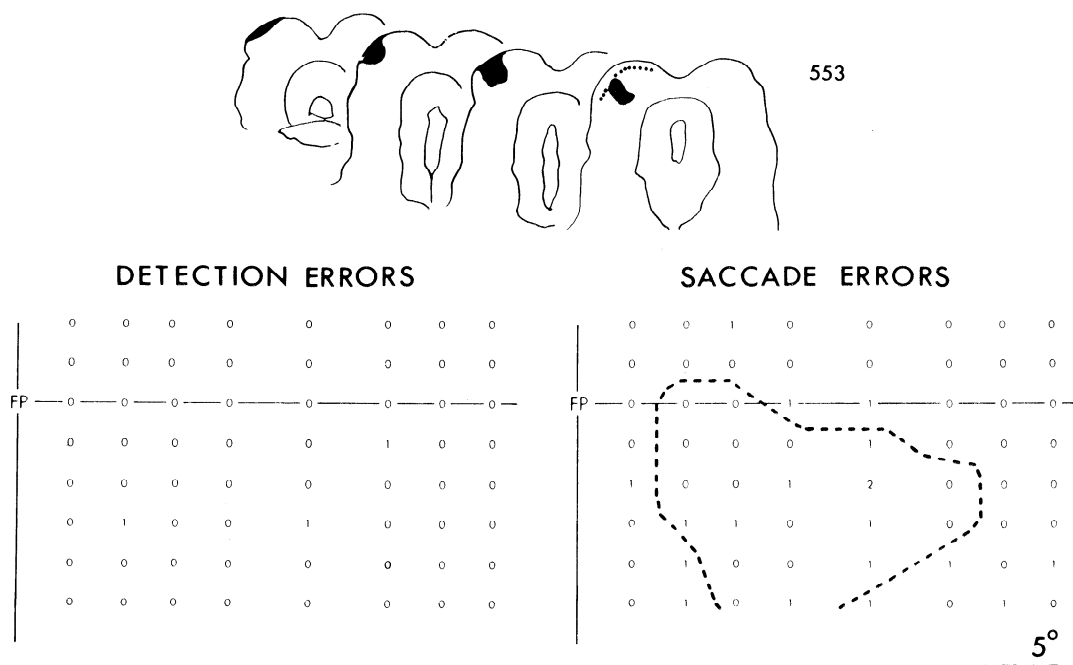
FIG. 5. Lack of detection errors and gross saccade errors following partial lesion of the left superior colliculus. The drawing shows coronal sections through the superior colliculus; from posterior (on the left) to anterior (on the right) at 500-μm intervals; the lesion extended no more than 250 μm anterior to the section on the right. The dotted line on the right section indicates the approximate upper boundary of the stratum opticum. Numbers on the test grids indicate the number of detection errors (on the left, taken on the 7th and 8th days postlesion) or gross saccade errors (on the right, taken on the 5th and 6th day postlesion) made to each point in four trials. No significant deficit was observed on either the detection or the saccade task when these results were compared to prelesion responses (Fig. 1A and Fig. 2). Dashed lines on the saccade error grid outline the area where the latency of the saccade was increased by more than 50 ms on the 3rd and 4th day following the lesion (saccade task).
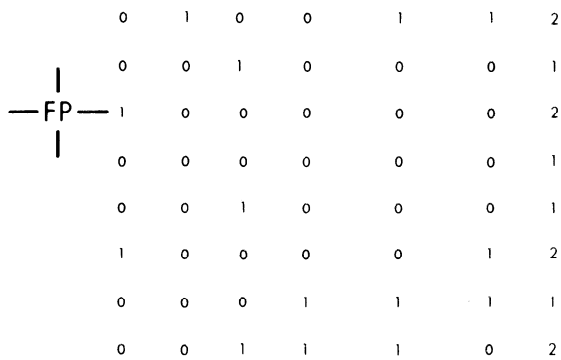
ing off the target light after 150 ms, that is, before the monkey had started to make a saccade to the target, did not change the initial saccade but eliminated the small corrective saccade. In addition to the increase in corrective saccades in the portion of the visual field affected by the collicular lesion, monkey 553 also showed an increased number of corrective saccades for other points in the field. Presumably, this nonspecific increase in corrective saccades reflects a change in behavioral strategy by this animal.

Figure 7 shows the effect of the collicular lesion in Fig. 5 on the standard deviation of the initial saccade. The dots on the grid at the top of the figure illustrate those points whose standard deviations were compared pre- and postlesion. The shading covers the portion of the visual field affected by the superior colliculus lesion, i.e., the points where the saccade latency was increased. In the portions of the figure labeled prelesion and postlesion, the rectangles are graphic representations of ± 1 standard devia-
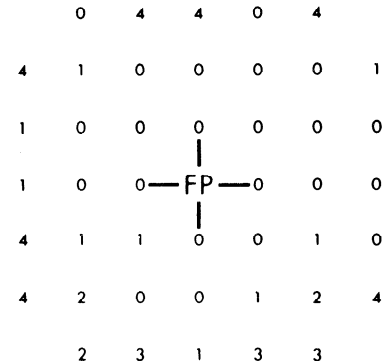
tion from the average end point of the initial saccade to that point. Each standard deviation was calculated from the horizontal and vertical components of seven saccades after eliminating those few trials on which the monkey made gross errors in saccadic accuracy. In the postlesion portion of Fig. 7, the standard deviation rectangles for saccades made into the collicluar lesion area are shaded. Although the standard deviation rectangles in the area of the visual field affected by the collicular lesion are slightly larger postlesion than the corresponding rectangles prelesion, the standard deviation also increases slightly on the postlesion grid for points outside the visual field affected by the collicular lesion, possibly due to a strategy change by the monkey or a degradation in the electrooculogram. The analysis of standard deviation of the saccade following a collicular lesion indicates that there is no more "scatter" around the end point of the initial saccade. This result was confirmed on monkey H072. However, the initial saccade tended to be consistently short as indi-

PRE-LESION

MONKEY 553

0	1	0	0	1	1	2
0	0	1	0	0	0	1
1	0	0	0	0	0	2
0	0	0	0	0	0	1
0	0	1	0	0	0	1
1	0	0	0	0	1	2
0	0	0	1	1	1	1
0	0	1	1	1	0	2

—FP—

MONKEY H072

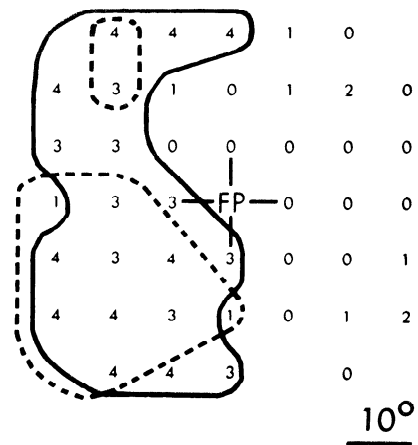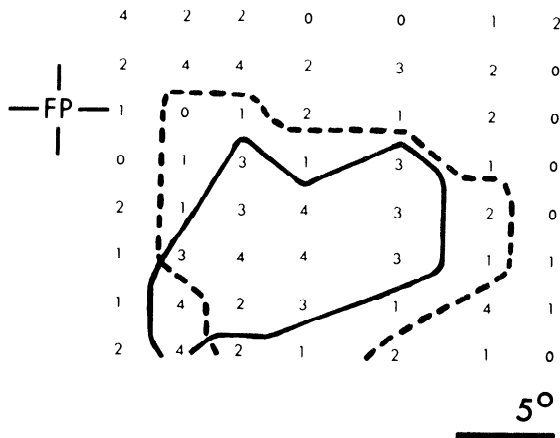|				
0	4	4	0	4
4 1	0	0	0	0 1
1 0	0	0	0	0 0
1 0	0	0	0	0 0
4 1	1	0	0	1 0
4 2	0	0	1	2 4
2	3	1	3	3

0—FP—0

POST-LESION

FIG. 6. Increased frequency of small corrective saccades following lesion of superior colliculus. Monkey 553 has lesion and grid points illustrated in Fig. 5; monkey H072 has a similar lesion in the right colliculus and was tested on a different set of grid points. Numbers indicate the number of corrective saccades (0.5°–2.5° in amplitude for monkey 533 and 0.5°–5° for monkey H072) in four trials. The postlesion trials (lower grids) were made on the 5th and 6th postlesion days for monkey 533 and on the first postlesion day for monkey H072. Solid line on postlesion grid encloses those points with three or four corrective saccades lying in the lesion area; dashed line indicates the area affected by the lesion as indicated by an increase in the latency to make a saccade.

cated by the increased frequency of small corrective saccades which are always in the direction of the initial eye movement.

The saccade latency and corrective saccade deficits showed some slight recovery with time. In the one monkey (553) that was followed for 3.5 wk, the prolonged saccade latency decreased slightly (comparable to ref 38), and the number of corrective saccades decreased by one or two at all test points.

In summary, following the superior colliculus lesions, the monkeys are able to make saccades to visual targets. But there is an increase in the latency to initiate the saccade and a tendency for the initial saccade to be short of the target and to be followed by a second small corrective saccade. Since there is no visual detection, i.e., sensory deficit following collicular lesion, this slight deficit in the accuracy of saccades is probably a visuomotor rather than a visual deficit.
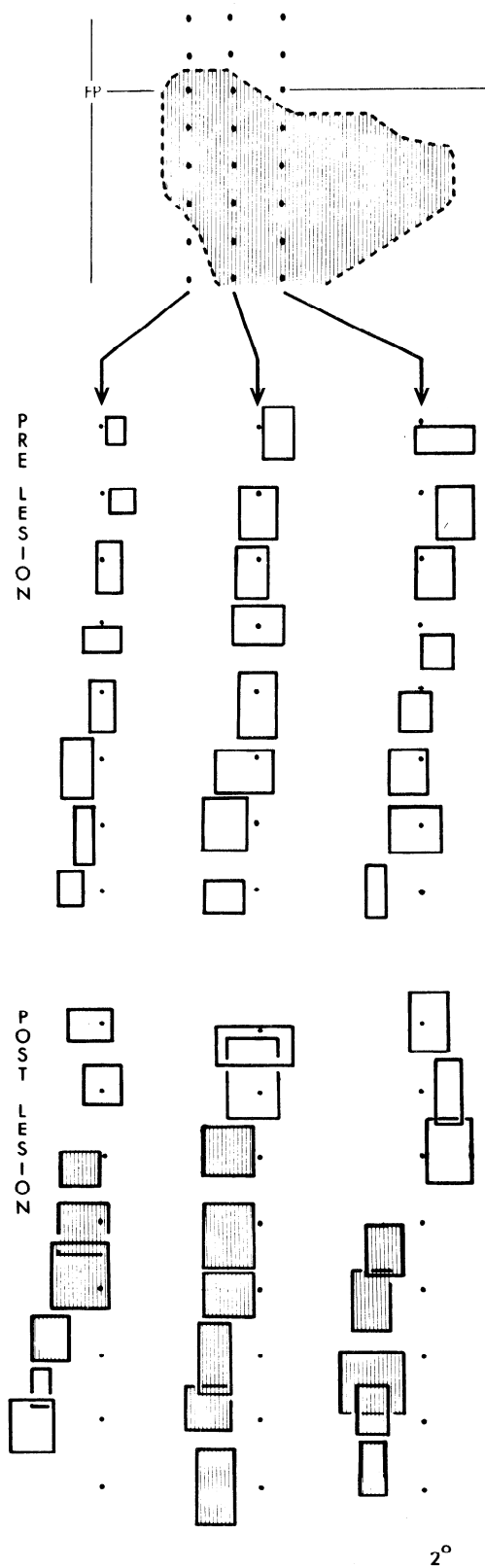
Joint lesions of striate cortex and superior colliculus

Three monkeys had focal, homolateral lesions of both striate cortex and superior colliculus placed so as to affect the same portion of the visual field. Two of the monkeys initially received and recovered from the cortical lesion prior to the collicular lesion while the third monkey received a collicular lesion one month prior to the cortical lesion (see Table 1).

Figure 8 illustrates the effect of a subsequent collicular lesion on one of the monkeys which had recovered from the cortical lesion illustrated in Fig. 3. The colliculuar lesion almost completely removed the lateral posterior part of the superficial collicular layers, should affect the left visual field on and below the horizontal meridian with the exception of the central 2 or 3° (3, 8), and should overlap the portion of the visual field affected by the previous cortical lesion. The solid line in Fig. 8 surrounds those points where the monkey made two or more visual detection or saccadic accuracy errors in four trials after this subsequent collicular lesion; the shaded areas represent the deficit area immediately following the initial striate cortex lesion as shown in Fig. 3A. As a result of the subsequent collicular lesion, both the detection and saccadic accuracy errors, which had decreased with recovery after the striate cortex lesion (see Fig. 3B), reappeared in about the same areas as the original cortical deficit. Since there is no deficit in detection or gross saccadic accuracy associated with a colliculus lesion alone, this suggests that the superior colliculus was involved with the recovery from the cortical lesion illustrated in Fig. 3B.

The combined corticollicular deficit was different from the deficit produced by the cortical

FIG. 7. Similarity of standard deviation of saccadic eye movements before and after lesion of the superior colliculus. The shaded area on the drawing at the top outlines the lesion area (as determined by an increased latency for the saccade) for the same monkey (553) as in Figs. 5 and 6. Dots on the drawing show the locations of three columns of grid points through the lesion area for which the standard deviations are shown below. Rectangles in these columns are located with their centers at the average end point of the initial saccade and show graphically the standard deviation of the horizontal and vertical EOG for saccades made to the adjacent target points (dots). Scale in the lower right corner is for distance within the column but columns are spread apart for ease of display. Standard deviations are based on seven saccades to each grid point; half of the points were run on the 5th postlesion day and half on the 6th postlesion day. Shaded rectangles on the postlesion days indicate that the target point was in the lesion area.
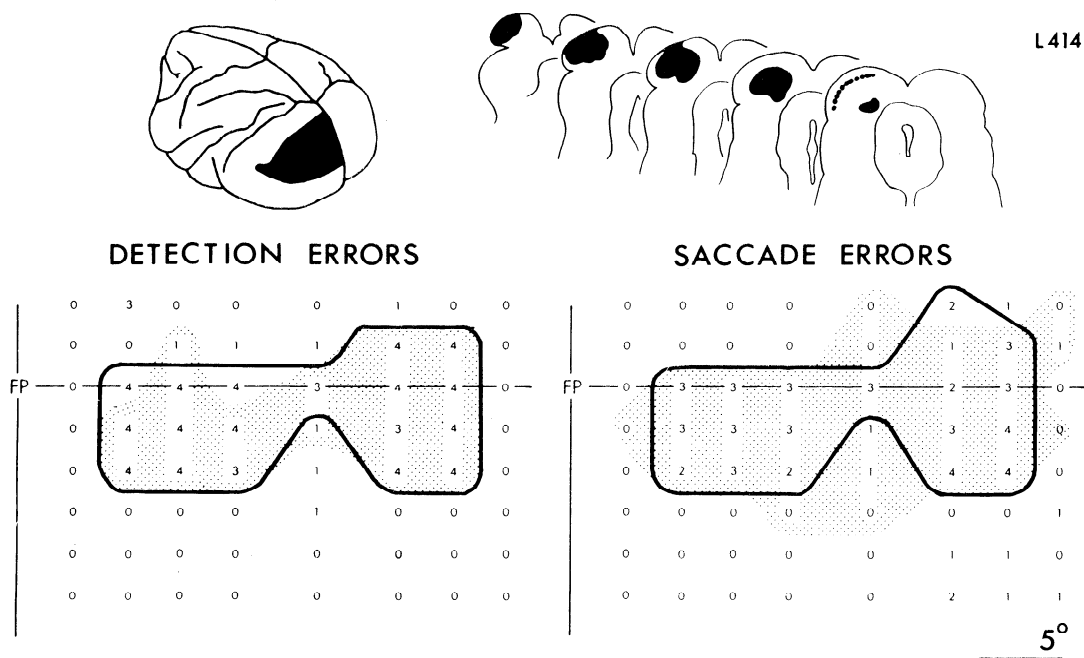
L 414

DETECTION ERRORS

SACCADE ERRORS

5°

FIG. 8. Detection errors and gross saccade errors following a combined partial lesion of the left striate cortex and left superior colliculus. The striate cortex lesion is the same as that shown in Fig. 3 (*L414*). Tracings of coronal sections through the brain stem are separated by roughly 600 μm with the section on the left being most posterior. A dotted line on the most anterior section indicates the upper boundary of the stratum opticum. Numbers on both grids indicate errors in four trials and at each point on the 10th day following the superior colliculus lesion which, in turn, was 9 wk after the striate cortex lesion. Solid lines surround the area with two or more errors. Shaded area is the area associated with the deficit following the initial lesion of striate cortex.

lesion in that increasing the target intensity from 11 to 1,700 cd/m² did not reduce the detection or saccade errors. In this respect the defective field produced by the combined cortical-collicular lesion acted like the retinal blind spot caused by the optic disc; the monkey could not make accurate saccades or detect small spots of light even at very high intensities. The combined cortico-collicular deficit was also different from the cortical deficit in that there did not seem to be any recovery from this combined lesion. The monkey whose lesions and deficits are illustrated in Fig. 8 showed no recovery in the 6 wk it was studied following the lesions. Two other monkeys showed no recovery in the 15 wk (*M648*) and 4 wk (*553*) over which they were studied. This lack of recovery suggests that the monkeys had a permanent loss of ability to detect small spots of light and to make accurate visual saccades following a combined striate cortex-superior colliculus lesion.

Figure 9 illustrates the effect of cortical and then collicular lesion (left) compared to collicular and then cortical lesion (right). The solid lines enclose the grid points where the monkey made two or more visual detection errors in four

trials after the joint lesion; the dotted lines enclose the grid points where there was an initial visual detection deficit following the cortical lesion and, the dashed lines enclose points thought to be affected by the collicular lesion using the criterion of a saccadic latency increase of at least 50 ms. Presumably points enclosed by the dotted line and/or the dashed line but not enclosed by the other line were grid points where only cortex or colliculus had been removed. Figure 9 illustrates two points. First, the combined lesion deficit is the same regardless of the order in which the striate cortex and superior colliculus lesions are made. Second, as in Fig. 8, the combined lesion effects are seen only in the portion of the visual field where the striate cortex and superior colliculus lesions overlap.

We want to emphasize that the deficits described above are likely to be the minimal ones since in order to perform consistently on the tests the monkeys must be highly trained and well motivated. Any number of factors could contribute to poorer performance and thereby produce larger deficits following the lesion. Figure 10 illustrates one such motivational effect on the size of the deficit following a combined
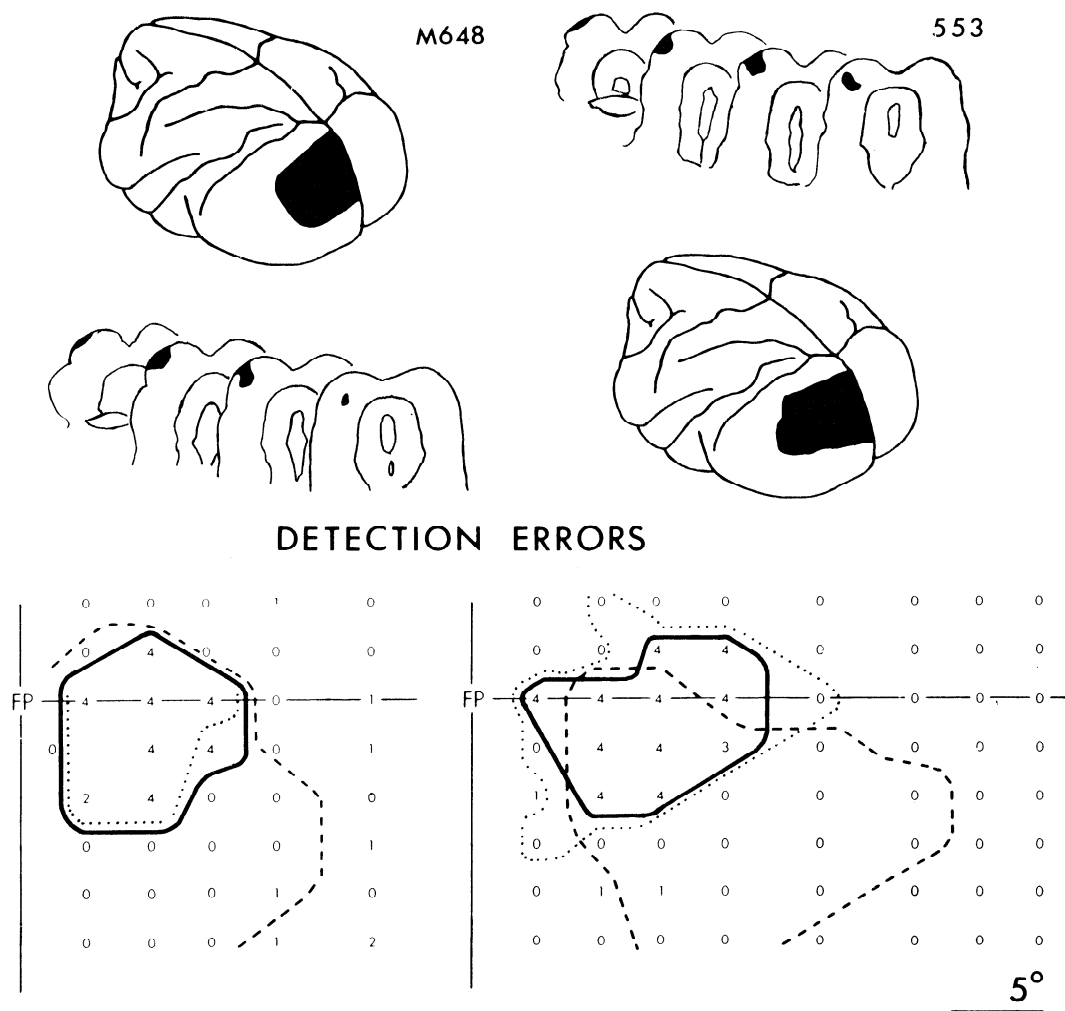
DETECTION ERRORS

FIG. 9. Detection deficits following lesion of striate cortex first and superior colliculus second (drawing and test grid on left, *M648*) and following lesion of superior colliculus first and striate cortex second (drawing and test grid on right, *553*). Numbers on grids are detection errors in four trials after the combined lesion; solid lines surround areas of two or more errors. Dotted lines enclose the area of detection errors following striate cortex lesion and dashed lines enclose the area with saccade latency increase of more than 50 ms following the superior colliculus lesion. Saccade errors are not shown since, like the errors illustrated in Fig.8, the detection errors and saccade errors were generally concentrated on the same grid points. For monkey *M648*, the cortical lesion was limited to the striate cortex and included the dorsal surface as shown in black and the lateral bank of the ascending limb of the medial calcarine gyrus from midline to about 5 mm lateral; the colliculus lesion was made 6 wk later, the coronal sections through the superior colliculus are 500 μm apart. For monkey *553* the superior colliculus lesion was made first and the sections are explained in Fig. 5; the cortical lesion was made a month later and was limited to striate cortex including the dorsal surface shown in black and the lateral bank of the ascending limb of the medial calcarine gyrus 8 mm lateral from midline.

cortex-colliculus lesion (shown in Fig. 9, right). Detection errors shown in Fig. 10*A*, *B*, and *D* show a consistent pattern and were obtained under the usual conditions at 5, 7, and 13 wk, respectively, after the addition of the striate cortex lesion. However, in Fig. 10*C* (11 wk postlesion) the monkey was tested after receiving un- limited water in the home cage on the preceding day; the deficit area was increased by several points, and the number of errors within this area was maximal. While this increase is slight, the motivational decrease must also have been slight or the monkey would not have performed the task at all. Other extraneous factors, not tested
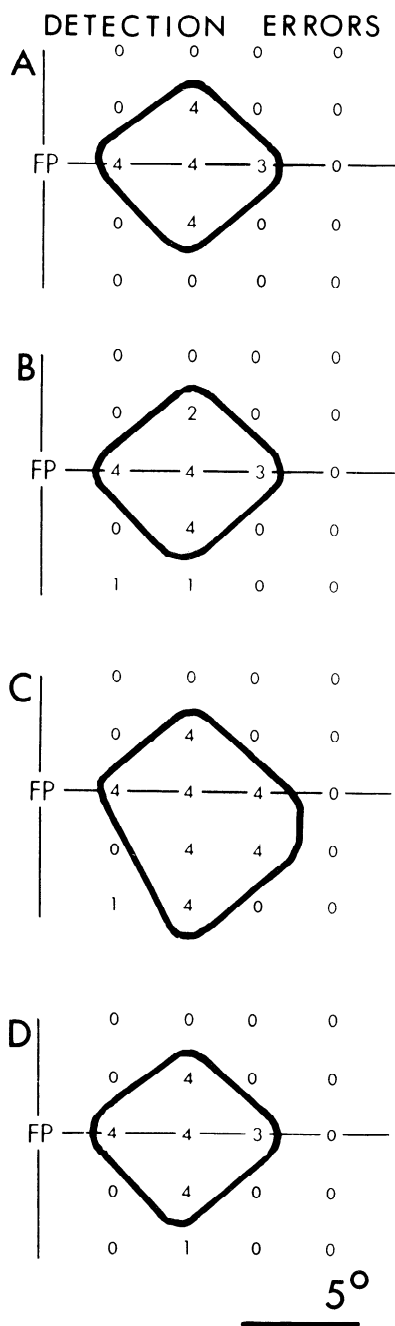
DETECTION ERRORS

FIG. 10. Modification of lesion deficit with change in motivation. Detection errors in four trials following both striate cortex and superior colliculus lesion is shown for the same monkey as in the left of Fig. 9. *A* is about 5 wk postlesion, *B* is 7 wk, *C* is 11 wk, and *D* is 15 wk. All except *C* were tested following water deprivation; expansion of lesion size in *C* occurred on a day when the monkey had had free access to water on the preceding day.

in these experiments, could presumably also act to make the deficits larger but probably not smaller.

Effect of field specific practice on recovery from striate cortex lesions

The data from the three monkeys with cortical lesions suggested that the recovery process was at least in part the result of the testing and not due just to the passage of time. In one monkey the initial cortical deficit was mapped in 3 days of testing (as indicated by the stippled area in Fig. 11). Then the half of the deficit to the left of the arrows was tested each day until recovery was noted. The entire grid was then tested, and the results are illustrated by the number of saccadic accuracy and detection errors in Fig. 11. The solid line encloses the points in the visual field where the monkey made two or more errors in four trials. For both the visual detection and saccadic accuracy tasks, the deficit area was still present in the portion of the visual field which had not been tested. There was some recovery noted to the right of the arrows on the untested portion of the grid, but this recovery was much less than the recovery at the tested points. For the second monkey all of the cortical deficit except for one point was on or below the horizontal meridian. The points on or below the horizontal meridian were tested for 6 wk, and recovery was noted in all but one point. In contrast, the single point above the horizontal meridian was tested for approximately 1 of the 6 wk and this point did not show any recovery. These observations indicate that the monkey shows some spontaneous recovery from a striate cortex lesion, but this recovery was greatly accelerated by forcing the monkey to use visual stimuli in the deficit area as part of the saccadic accuracy and visual detection tasks.

Single-cell changes in superior colliculus following striate cortex lesions

Since the gradual recovery from striate cortex lesions appeared to depend on the superior colliculus, we recorded the activity of colliculus neurons after such recovery occurred. We studied cells with visual receptive fields found in the superficial gray and stratum opticum layers. While we did not analyze receptive-field properties in detail, we found cells related to the same part of the visual field as the cortical scotoma to be normally active and visually responsive (in agreement with Schiller et al. (27)).

We tested the cells in a paradigm which measured changes in the cell's visual response when the monkey was required to use the stimulus as a
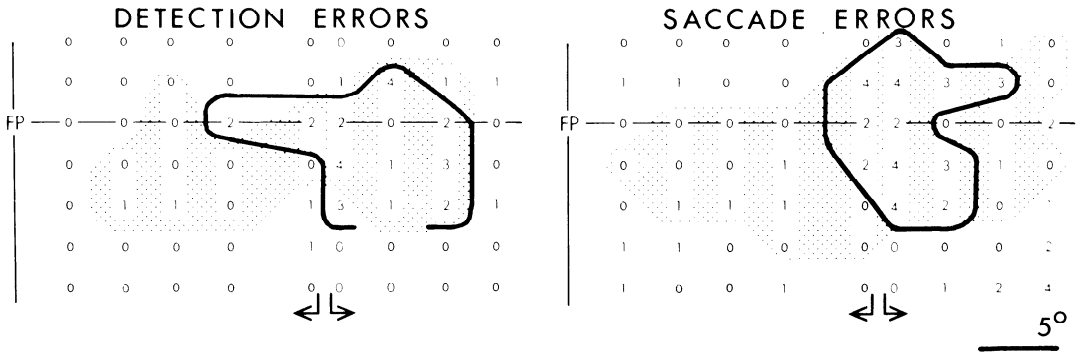
DETECTION ERRORS

SACCADE ERRORS

FIG. 11.   Effect of practice on recovery from striate cortex lesion. Numbers on the grid are for errors on four trials at each point on the 14th and 15th days postlesion. Shading outlines area of original detection errors and gross saccade errors. Each point on the grid area to the left of arrows has been practiced a total of 6 days on flash and saccade trials, while points to the right of the errors had been practiced 2 days. Remaining area of deficit (with two or more errors) is outlined by solid lines and is predominantly in the unpracticed area.

target for a saccadic eye movement (9, 39). In an intact monkey approximately 40% of the cells show an increased or enhanced response when the monkey uses the stimulus as a saccade target (9, 39), and this percentage did not vary markedly at different retinal eccentricities (39). The results of recording from collicular cells after recovery from striate lesions is summarized in Table 2. The column labeld "Ipsilateral to ablation, inside scotoma" shows that 50 and 89% of the cells that have no striate cortex input and presumably were involved in the recovery process increased their visual response when the two monkeys used the stimulus as a saccade target. These percentages are slightly higher than the 40% seen in normal monkeys, and the significance of this increase is strengthened by comparing the percent of cells showing enhancement in the scotoma produced by the cortical lesion to the percent in the same colliculus but outside the scotoma (50% inside versus 22% outside for monkey L414 and 89% inside versus 33% outside for the monkey M648 in Table 2). In the superior colliculus contralateral to the ablation, there is the mirror image of this effect (Table 2). There is a decreased percentage of cells showing the enhancement effect for cells

with receptive fields in the mirror image of the cortical scotoma as compared to cells in the contralateral colliculus with receptive fields outside the scotoma (22% inside versus 57% outside for monkey L414 and 57% inside versus 70% outside for monkey M648 in Table 2).

The recovery from striate cortex lesions is therefore correlated with an increased frequency of cells which can increase their visual response when the monkey is required to use the visual stimulus as a target for an eye movement. Increase in the enhancement in one colliculus may be associated with a decrease in the other colliculus.

DISCUSSION

Parallel input for visually guided saccades

The major results of these experiments are summarized in Table 3. Following partial removal of the striate cortex (Table 3, col 1), the monkeys were unable either to detect or to make saccades to the visual targets. After a month or more of practice (Table 3, col 2), these monkeys recovered the ability both to detect and to make saccades to visual targets without gross

TABLE 2.   *Percent enhanced responses in superior colliculus following partial striate cortex ablation*

| Monkey | Ipsilateral to Ablation, % | | Contralateral to Ablation, % | |
	Inside scotoma	Outside scotoma	Inside scotoma*	Outside scotoma
L414	50 (16)	22 (54)	22 (9)	57 (28)
M648	89 (9)	33 (21)	57 (7)	70 (16)

Numbers in parentheses are total number of units.   * Refers to the area of the visual field symmetrical and contralateral to the scotoma.

TABLE 3. *Summary of lesion deficits*

	Striate Cortex Lesion	Recovery From Striate Cortex Lesion	Superior Colliculus Lesion	Joint Striate Cortex and Superior Colliculus Lesion
Detection deficit	Yes	No	No	Yes
Saccade deficit				
Gross error	Yes	No	No	Yes
Increase in frequency of double saccades	*	No	Yes	*
Increase in SD of EOG amplitudes	*	Slight	No	*
Latencies for saccade initiation	*	No	Yes	*

* When gross errors in saccadic accuracy were frequent, no attempt was made to measure increase in double saccade frequency, increase in standard deviation of amplitudes, or change in latency.

errors. Following an initial lesion of the superior colliculus (Table 3, col 3), the monkey could both detect the visual targets and make saccades to them without gross errors. The first point that emerges from these results is that either striate cortex or superior colliculus alone is sufficient to provide the visual input necessary for the guidance of saccades, although the accuracy, latency, and time for recovery of the saccades may vary with the structure ablated.

Perhaps the major finding of these experiments is that following a combined lesion of striate cortex and superior colliculus (Table 3, col 4), the monkey could neither detect nor saccade to a visual target. Unlike the results following a lesion in either cortex or colliculus, the deficits following the joint lesion did not recover (at least not within 15 wk) and were not reduced by an increase in light intensity. We conclude that either striate cortex or superior colliculus or the combination of these two structures is necessary for information on location of the visual target for the guidance of saccadic eye movements. The presence of other structures which receive retinal afferents, such as pretectum, pregeniculate, and accessory optic nucleus are not sufficient for the guidance of saccadic eye movements.

The finding of a very severe visual detection deficit after the combined lesion might at first appear surprising in light of recent evidence that the discrimination of differences in gross luminous flux can be carried out in the absence of both striate cortex and superior colliculus (16). These flux discriminations, however, require no localization in space as does our saccade task and probably utilizes a larger stimulus area than does our detection task.

After recovery following striate cortex lesions, the finer measures of accuracy could be tested. We found no consistent increase in dou-

ble saccades, but a slight increase in the scatter of the initial saccades in one monkey. This suggests that even after recovery the saccades were not quite as accurate as in the intact monkey; the deficit might be larger than it appeared since the EOG itself introduced variance into the prelesion base-line data. It suggests furthermore that since this recovery is in part mediated by the superior colliculus, the colliculus, even with the changes accompanying recovery, cannot provide guidance as accurate as can the intact striate cortex and normal superior colliculus acting together.

After lesions of the superior colliculus, there was an increase in the frequency of double saccades. The initial saccades were short of the target, and the second saccade was presumably used to bring the fovea onto the target. In addition, the standard deviation of the initial saccade did not increase, indicating that there was no more scatter around the end point of the initial saccade than before the lesion. This is clearly a deficit in saccadic guidance but a rather specific one in that it entails a highly consistent error rather than a random pattern of missing the target. This deficit may have led to an earlier report that collicular lesions produced large saccadic guidance errors (24). The data obtained by Wurtz and Goldberg (38) was not sufficiently quantitative to detect the corrective saccade deficit reported here. We have, however, looked at their data and seen what is probably an increased frequency of double saccades following collicular lesions in several monkeys. The striking exception was the monkey with the largest lesion and clearest latency deficit (Fig. 4 of ref 38), which showed no indication of an increase in double saccades. This raises the possibility that the occurrence of double saccades and increased latency are reflections of different compensatory mechanisms. We confirm the earlier

finding (38) that a colliculus lesion produces an increase in the latency for initiating a saccade; this effect is specific to the colliculus since we see no such latency increase following striate cortex lesions.

The different deficits following collicular and cortical lesions suggest that these two structures may be more than redundant visual inputs to the oculomotor system (Table 3). There are at least two speculative views which would fit our data and attempt to explain the unique nature of the collicular and cortical inputs. A very simple view would hypothesize that in order to make an accurate saccade to a visual target in our task, the monkey must visually recognize that the test spot is a saccade target and determine the direction and distance of the target point from the fovea. Striate cortex lesions left the monkey functionally blind to all but intense stimuli immediately after the lesion, and since he could not see the stimulus he could not saccade to it. The deficit was a visual one and the lack of saccadic guidance presumably resulted from the monkey's inability to see the target of his saccade. In contrast, the colliculus lesion led to no detection deficit and must have been primarily an oculomotor deficit, not a visual one. In this view the target recognition would be mediated by striate cortex and the metrics of the saccade would be calculated largely by the colliculus. This hypothesis does not, however, explain why the guidance of saccades is as good as it is after collicular lesions nor does it allow for the possibility that the detection deficit following cortical lesions might simply mask an additional saccade deficit.

An alternative, but not exclusive view of the nature of collicular and cortical inputs to the oculomotor system, is suggested by the different visual receptive-field properties of cells in these two structures. For the superior colliculus, McIlwain (13) suggested that the relatively large visual receptive fields of collicular cells might serve to amplify neural power, i.e., a single spot excites many collicular cells with each one having only rough guidance information. In the striate cortex, in contrast, a single spot excites a smaller proportion of cortical cells and each cell has precise location information. In the later oculomotor processing, it is possible that in response to a small target the cortex would indirectly excite only a few cells and that colliculus would produce an excitatory effect on many cells. In the absence of cortical input, the many collicularly activated cells, each having rough guidance information, could act in concert to produce a grossly accurate saccade. But because the precise information from the cortical cells is not present, each saccade is slightly inaccurate,

as indicated by the increased standard deviation seen after recovery from cortical lesions. Following collicular lesions, only the smaller number of cells with the precise cortical localization might be activated. If fewer oculomotor neurons were activated or if each oculomotor neuron was activated less intensely, it is possible that the resultant force would move the eyes a shorter distance, a result found in this study following collicular lesions. We have discussed elsewhere (39) the relation of colliculus to anticipation and readiness to respond which might, in turn, relate to the latency deficit. We conclude that while damage to either striate cortex or superior colliculus produces a slight guidance deficit, the effects are somewhat different. After recovery in cortex there is an increase in scatter, while in the colliculus there is a consistently short initial saccade with no increase in scatter, followed by a second saccade. These differences following the two lesions suggest that, while either structure can in the absence of the other provide the visual guidance information for saccadic eye movements, in the intact animal their organization and contribution to guidance are unique and complementary rather than equal and redundant.

The interpretation of the finer measures of saccadic accuracy becomes more difficult, however, when it is realized that the behavior of the monkey following a lesion reflects not only the lack of the structure ablated (with the consequent disruption of the normal afferent and efferent connections of other intact brain structures), but also the compensation occurring in other brain areas. For example, it is clear from the changes found in the behavior of single cells in the superior colliculus following recovery from a striate cortex lesion, that cells in this brain area do alter their function. Thus the behavior seen after the recovery from a striate cortex lesion reflects not only the deficit resulting from the lack of striate cortex, but also the compensation that is in large part responsible for the recovery.

Recovery from striate cortex lesions

The recovery of visual detection following cortical lesions in our monkeys was very similar to the recovery from similar lesions seen by Cowey (2), and probably parallels the ability to make visually guided arm movements (L. Weiskrantz and C. Passingham, personal communication), and recovery of more complex visual behavior (11). Cowey (2) found that monkeys recovered in 2 mo of behavioral testing, but did not recover in 2 mo if left in the home cage. Our findings support this observation and indi-

cate that recovery is dependent on the monkey being tested specifically in the part of the field related to the lesion. Our results suggest that there might be field-specific changes associated with recovery rather than a nonspecific change in the way the monkey solves the task.

As noted above, recovery from striate cortex lesions is completely reversed by subsequent damage to the ipsilateral colliculus. Therefore, it is reasonable to assume that the colliculus is mediating this recovery. We have, in fact, noted that there are changes in the visual response of collicular cells following recovery from striate cortex lesions: a larger proportion of the collicular cells show an enhanced response to a visual stimulus after the lesion than has been found in normal monkeys (39). The increased frequency of this enhancement effect was specifically related to that portion of the colliculus that would have been used for recovery from the cortical lesion. We have discussed elsewhere (39) the relation of this enhancement effect to a readiness to make an eye movement. This increased frequency of the enhancement effect is presumably part of the monkey's compensation for the cortical deficit. We do not know the reason for this change or what other modifications might also have occurred in the colliculus. In net, both the lesion and the single unit results are consistent with the idea that the superior colliculus is necessary for the recovery process from cortical lesions.

Human subjects with postgeniculate or visual cortical lesions are classically reported to be blind if the lesion is bilateral and hemianopic if the lesion is unilateral (31). The initial visual detection deficit after cortical lesions in monkeys is measured in much the same way and is analogous to the partial hemianopic scotoma measured in humans. Humans with occipital cortical lesions have recently been shown to have some residual function within the scotoma (18–20, 33). Although the patient is not aware of any visual images he is able to localize visual stimuli with an arm or an eye movement or detect moving visual stimuli when placed in a forced choice paradigm. We have demonstrated that the recovery of the monkey's ability to make accurate eye movements is dependent on the superior colliculus. It is possible that the residual vision in humans is also mediated by the superior colliculus.

This study and the study of Cowey (2) both suggest that behavioral testing situations in which the monkey is forced to use the lesioned part of the visual field result in some recovery. Since the effect of repeated testing of the lesion related part of the field does result in improvement of performance in the monkey, it might be useful to try these methods on patients with similar lesions.

REFERENCES

1. BOND, H. W. AND HO, P. Solid miniature silver-silver chloride electrodes for chronic implantation. Electroencephalog. Clin. Neurophysiol. 28: 206–208, 1970.
2. COWEY, A. Perimetric study of field defects in monkeys after cortical and retinal ablations. Quart. J. Exptl. Psychol., 19: 232–245, 1967.
3. CYANDER, M. AND BERMAN, N. Receptive-field organization of monkey superior colliculus. J. Neurophysiol. 35: 187–201, 1972.
4. DANIEL, P. M. AND WHITTERIDGE, D. The representation of the visual field on the cerebral cortex in monkeys. J. Physiol., London 159: 203–221, 1961.
5. DENNY-BROWN, D. The midbrain and motor integration. Proc. Roy. Soc. Med. 55: 527–538, 1962.
6. DOTY, R. W. Ablation of visual areas in the central nervous system. In: Handbook of Sensory Physiology, edited by R. Jung. Berlin: Springer, 1973, vol. VII/3 B, p. 483–541.
7. EVARTS, E. V. A technique for recording activity of subcortical neurons in moving animals. Electrocencephalog. Clin. Neurophysiol. 24: 83–86, 1968.
8. GOLDBERG, M. E. AND WURTZ, R. H. Activity of superior colliculus in behaving monkey. I. Visual receptive fields of single neurons. J. Neurophysiol. 35: 542–559, 1972.
9. GOLDBERG, M. E. AND WURTZ, R. H. Activity of superior colliculus in behaving monkey. II. Effect of attention on neuronal responses. J. Neurophysiol. 35: 560–574, 1972.
10. HUMPHREY, N. K. Responses to visual stimuli of units in the superior colliculus of rats and monkeys. Exptl. Neurol. 20: 312–340, 1968.
11. HUMPHREY, N. K. What the frog's eye tells the monkey's brain. Brain Behav. Evol. 3: 324–337, 1970.
12. LATTO, R. AND COWEY, A. Visual field defects after frontal eye field lesions in monkeys. Brain Res. 30: 1–24, 1971.
13. MCILWAIN, J. T. Large receptive fields and spatial transformations in the visual system. In: International Review of Physiology, edited by R. Porter. Baltimore: Univ. Park Press, 1976.

14. MOHLER, C. W. AND WURTZ, R. H. Role of striate cortex and superior colliculus in visual guidance of saccadic eye movements in monkey. *Soc. Neurosci. Abstr.* 342, 1974.

15. MOHLER, C. W. AND WURTZ, R. H. Organization of monkey superior colliculus: intermediate layer cells discharging before eye movements. *J. Neurophysiol.* 39: 722–744, 1976.

16. PASIK, T. AND PASIK, P. Extrageniculostriate vision in the monkey. IV. Critical structures for light vs. no-light discrimination. *Brain Res.* 56: 165–182, 1973.

17. PASIK, T. PASIK, P., AND BENDER, M. B. The superior colliculi and eye movements. *Arch. Neurol.* 15: 420–436, 1966.

18. PERENIN, M. T. AND JEANNEROD, M. Residual vision in cortically blind hemishields. *Neuropsychologia* 13: 1–7, 1975.

19. PÖPPEL, E., HELD, R., AND FROST, D. Residual visual function after brain wounds involving the central visual pathways in man. *Nature* 243: 295–296, 1973.

20. RICHARDS, W. Visual processing in scotomata. *Exptl. Brain Res.* 17: 333–347, 1973.

21. ROBINSON, D. A. Eye movements evoked by collicular stimulation in the alert monkey. *Vision Res.* 12: 1795–1808, 1972.

22. ROBINSON, D. A. On the nature of visualoculomotor connections. *Invest. Ophthalmol.* 11: 497–503, 1972.

23. SCHILLER, P. H. The role of the monkey superior colliculus in eye movement and vision. *Invest. Opthalmol.* 11: 451–460, 1972.

24. SCHILLER, P. H. Some functional characteristics of the superior colliculus of monkey. In: *Cerebral Control of Eye Movements and Motion Perceptions*, edited by J. Dichgans and E. Bizzi. Basel: Karger, 1972, p. 122–129.

25. SCHILLER, P. H. AND KOERNER, F. Discharge characteristics of single units in superior colliculus of the alert rhesus monkey. *J. Neurophysiol.* 34: 920–936, 1971.

26. SCHILLER, P. H. AND STRYKER, M. Single-unit recording and stimulation in superior colliculus of the alert rhesus monkey. *J. Neurophysiol.* 35: 915–924, 1972.

27. SCHILLER, P. H., STRYKER, M., CYANDER, M., AND BERMAN, N. Response characteristics of single cells in the monkey superior colliculus fol-

lowing ablation or cooling of visual cortex. *J. Neurophysiol.* 37: 181–194, 1974.

28. SPARKS, D. L. Response properties of eye movement-related neurons in monkey superior colliculus. *Brain Res.* 90: 147–152, 1975.

29. TALBOT, S. A. AND MARSHALL, W. H. Physiological studies on neural mechanisms of visual localization and discrimination. *Am. J. Ophthalmol.* 24: 1255–1263, 1941.

30. VON BONIN, G. AND BAILEY, P. *The Neocortex of Macacca mulatta.* Urbana: Univ. of Illinois Press, 1947, p. 44–49.

31. WALSH, F. B. AND HOYT, W. F. *Clinical Neuro-ophthalmology.* Baltimore: William & Wilkins, 1969, vol. I, p. 77–84.

32. WEISKRANTZ, L. Behavioral analysis of the monkey's visual system. *Proc. Roy. Soc. London, Ser. B* 182: 427–455, 1972.

33. WEISKRANTZ, L., WARRINGTON, E. K., SANDERS, M. D., AND MARSHALL, J. Visual capacity in the hemianopic field following a restricted occipital ablation. *Brain* 97: 709–728, 1974.

34. WURTZ, R. H. Visual receptive fields of striate cortex neurons in awake monkeys. *J. Neurophysiol.* 32: 727–742, 1969.

35. WURTZ, R. H. Response of striate cortex neurons to stimuli during rapid eye movements in the monkey. *J. Neurophysiol.* 32: 975–986, 1969.

36. WURTZ, R. H. AND GOLDBERG, M. E. Superior colliculus cell responses related to eye movements in awake monkeys. *Science* 171: 82–84, 1971.

37. WURTZ, R. H. AND GOLDBERG, M. E. Activity of superior colliculus in behaving monkey. III. Cells discharging before eye movements. *J. Neurophysiol.* 35: 575–586, 1972.

38. WURTZ, R. H. AND GOLDBERG, M. E. Activity of superior colliculus in behaving monkey. IV. Effects of lesions on eye movements. *J. Neurophysiol.* 35: 587–596, 1972.

39. WURTZ, R. H. AND MOHLER, C. W. Organization of monkey superior colliculus: enhanced visual response of superficial layer cells. *J. Neurophysiol.* 39: 745–765, 1976.

40. WURTZ, R. H. AND MOHLER, C. W. Enhancement of visual responses in monkey striate cortex and frontal eye fields. *J. Neurophysiol.* 39: 766–772, 1976.