Visuomotor Deficits Following Ablation of Monkey Superior Colliculus

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SUMMARY AND CONCLUSIONS

1. Previous experiments have shown that the initiation and accuracy of saccades made to visual stimuli in order to obtain a reward are only minimally affected by ablation of the primate superior colliculus. The goal of the present experiments was to examine whether ablation of the colliculus affected the generation of unrewarded, spontaneous saccades, and to what extent any such effects might depend on a sensory loss.

2. Monkeys were trained on a stimulus-detection task that required response to a peripheral stimulus while fixation of a central target was continued. Unilateral surgical ablations produced a deficit in stimulus detection in the visual field contralateral to the ablation. This failure to respond to stimuli recovered during the second postoperative week. Stimuli more eccentric than 20–30° elicited the largest and most persistent deficit. There was no indication that the time course of recovery was modified by visual experience, since the amount of postoperative visual experience did not appear to speed recovery. These experiments show that a transient sensory loss follows collicular ablation.

3. Monkeys were tested for distractibility by presentation of a brief irrelevant peripheral stimulus while they were being rewarded for fixation of a central spot of light. After the lesion, monkeys made fewer saccades than before toward distracting stimuli appearing in the visual field contralateral to the ablation.

4. A similar reduction in the frequency of saccades was observed during the stimulus-detection task. After the ablation, two monkeys that consistently made saccades toward stimuli in the field ipsilateral to the ablation showed a reduction in the number of saccades into the contralateral field. This decrease occurred even when the monkeys detected the stimuli.

5. The pattern of spontaneous eye movements recorded while the monkey was looking around the experimental room was also modified even after recovery from the detection deficit. The greatest postoperative effect was that the monkeys tended to look toward the side ipsilateral to the ablation; the proportion of ipsilateral fixations and the proportion of time spent in ipsilateral eye positions increased. In contrast, only a slight change was found in the proportion of contralaterally directed and ipsilaterally directed saccades. Also, slight decreases in the frequency of eye movements in the light were evident.

6. The visuomotor deficit that persists after recovery from the transient sensory loss can be characterized as a decrease in the frequency of saccades to visual targets contralateral to the lesion when the visually guided saccades are not required by the behavioral paradigm. The present experiments indicate that the superior colliculus plays an important role in the selection of a visual target for a visually guided saccade. This hypothesis is consistent with previous observations suggesting that one effect of collicular lesions is a visual neglect that may be related to but not necessarily limited to the generation of saccadic eye movements.

INTRODUCTION

The preceding study (4) and several others (25, 29, 34) have investigated the effect of ablation of the superior colliculus on the ini-
tiation and accuracy of saccadic eye movements. These studies found that when the lesions are restricted to the colliculus, the deficits in a monkey's ability to make saccades to specified visual targets in order to obtain a reward are minimal; primarily an increase in the latency to initiate the saccade. The saccades do fall somewhat short of their intended target, but these errors are readily corrected (4, 25). In contrast to these minimal effects on eye movements in monkeys trained to perform them to specified targets, a marked reduction in eye movements had been reported earlier by numerous investigators who had simply observed their operated animals in free situations (5, 10, 28). Taken together, these observations suggest that although a monkey with damage to its superior colliculus is able to make visually guided saccades if required to do so, it nevertheless tends to make fewer of them if they are not required.

Our goal in the present experiments was to investigate the role of the superior colliculus in the initiation of saccades under conditions in which the saccades were not required by the behavioral paradigm. We did this by recording eye movements following unilateral ablation of the superior colliculus in three different situations: 1) in a distraction paradigm in which the monkey was presented with a peripheral stimulus irrelevant to the behavioral task, 2) in a detection paradigm in which the monkey was presented with a peripheral stimulus relevant to the task but in which eye movements were irrelevant, and 3) during spontaneous eye movements. First, however, we used the stimulus-detection paradigm to determine whether a purely sensory deficit might be responsible for any alteration in saccadic activity.

We found that ablation of the superior colliculus did produce a deficit in the detection of visual stimuli, but this sensory deficit faded during the second postoperative week. A visuomotor deficit, which was expressed in these experiments as a marked reduction in saccades to distracting peripheral stimuli and significant changes in the pattern of spontaneous eye movements, persisted beyond recovery from the sensory deficit.

Brief reports of these results have appeared previously (1-3).

METHODS

The experiments were performed on four adult rhesus monkeys (Macaca mulatta) that were subjects in the preceding paper (4), and the experimental procedures followed the same sequence as described there. In addition, the spontaneous eye movements of one monkey with a bilateral collicular lesion were examined pre- and postoperatively.

Behavioral training and testing

DETECTION TEST. The animals were trained and tested on a detection task previously described by Mohler and Wurtz (25). Depression of a bar initiated a trial and turned on a fixation light in the center of a tangent screen. On 25% of the trials (fixation trials), the fixation point dimmed after a variable period of time (1-3 s) and the monkey was rewarded if it released the bar within 500 ms of the dimming. On 73% of the trials (detection trials), the fixation point did not dim, but instead another spot of light (detection stimulus) was briefly presented at a more eccentric and unpredictable position on the screen. On these trials the monkey was rewarded if it released the bar within 500 ms of the onset of the detection stimulus. On the remaining 2% of the trials (control trials), no detection stimulus was presented so that the strategy of the monkey when it could not detect a stimulus could be evaluated.

In an experimental session, the monkey was tested 4 times on each of over 80 stimulus positions located across an area subtending ±45° vertically and horizontally from the fixation point. A detection error was scored for any given point when the animal failed to release the bar within 500 ms of the onset of the detection stimulus. Monkeys were easily trained on this task and soon made no more than 10% errors during a test session.

Detection stimuli were the same as those described for the saccadic task in the preceding paper (4). The duration of the stimulus was limited to either 150 or 200 ms so that it would be shorter than the reaction time for a saccadic eye movement, but no effort was made to prevent eye movements toward the stimulus. A digital computer collected the data and controlled the behavioral tasks (25). Eye movement signals, stimulus conditions, and reaction times and errors were digitized, displayed, photographed, and stored under computer control. In addition, the computer pseudorandomly varied the timing and occurrence of fixation trials, detection trials, and control trials. Therefore, on a given trial the monkey could not predict whether a detection stimulus would be presented, when its onset might occur, or where it would be located on the tangent screen.
DISTRACTION TEST. During a series of fixation trials, when the monkey was therefore "set" to fixate the central light, a visual stimulus was occasionally presented briefly in the periphery. On these trials we determined whether the monkey broke fixation and made a saccade toward the stimulus. Three different visual stimuli were used: flashed, moving, and displaced. A flashed stimulus was a 1° spot of light that was presented for 200 ms on the horizontal axis 15 or 30° from the fixation point. The moving stimulus was 1° in diameter with a velocity of 600°/s and appeared while moving on a trajectory from 10° above to 10° below the horizontal axis at 15 or 30° from the fixation point. The displaced stimulus was a 1° spot that remained in the field throughout the fixation trials but was displaced downward 20° at 600°/s during the fixation period. All stimuli were 100 c/°/2° on a background of 1 c/°/2°. Eye movements that occurred during the trials were monitored but not rewarded or punished, except that the monkey risked missing the dimming of the fixation period if it broke fixation. In order to minimize learning or habituation on this task, each stimulus type was presented no more than 8 times in each visual field during a series of several hundred fixation trials in a single test session. The time at which the distraction stimulus was presented within a trial was varied.

SPONTANEOUS EYE MOVEMENTS. We recorded on magnetic tape (Hewlett-Packard, model 1530) continuous 5-min periods of eye movements while the monkeys were looking about the lighted experimental room or while they were in total darkness. These records were later analyzed off-line for saccade frequency, saccade amplitude, and eye position at the start of a saccade. Recorded eye signals were digitized and sampled by a PDP-11/34 computer to detect the occurrence of saccades identified on the basis of movement duration (less than 300 ms but greater than 50 ms) and velocity (initial velocity greater than 49°/s, final velocity less than 49°/s). Saccades were detected and measured by a computer program that displayed each saccade and permitted the user to verify and adjust the computer-detected initial and final eye positions. Computer sampling errors representing undetected saccades or artifacts detected as saccades were estimated to be less than 10%, based on direct examination of sampled records.

RESULTS

The unilateral collicular ablations were virtually complete in all four monkeys as indicated in the preceding paper (4).

Detection deficit

The polar plots in Fig. 1 show the frequency and distribution of preoperative and postoperative errors made in the detection task by three monkeys on the first day of testing. The monkeys were deprived of patterned visual experience prior to testing by the insertion of translucent contact occluders immediately after surgery, using the precautions described in the preceding paper (4). The translucent contact occluders were removed the evening before testing and the animal was placed in darkness until the following morning. Each polar plot in Fig. 1 shows the number of errors that occurred in four trials at each location. A dot indicates that the position was tested but no errors.
DEFICITS AFTER COLLICULAR ABLATION

occurred. Before the lesion (Fig. 1, left column), the monkeys made few errors in a daily test session, and errors that did occur were more frequent to far peripheral targets than to more central targets.

Following the lesion (Fig. 1, right column), a detection deficit was present in the visual field contralateral to the lesion in all three monkeys. The severity of the deficit was related to the length of the postsurgical recovery period. The most severe deficit was seen in monkey Ner (Fig. 1, postoperative day 8). This animal made numerous errors throughout the visual field contralateral to the lesion (shaded half of the field), even to targets presented in the central 10°-20° of the visual field. There was also a slight increase in the frequency of errors to peripheral targets ipsilaterally. Monkey Pec, first tested on day 10, was less severely affected. This animal’s detection errors were graded in number across the contralateral visual field, with the greatest frequency occurring for visual stimuli beyond 20°-30° eccentricity. Monkey Haw, first tested on day 13, made few detection errors to targets in either the central or peripheral visual field.

The frequency of detection errors soon declined in Ner, Pec, and the fourth monkey, Mou, whose pattern of recovery is shown in Fig. 2. Mou was first tested on day 10, 2 days after the occluders were removed, and the errors were restricted to targets beyond 20° into the visual field contralateral to the lesion. By day 11 most errors were restricted to targets 35° into the field, and by day 20 even these peripheral errors were less frequent.

The time course of recovery was similar across monkeys despite differences in postsurgical visual experience. Figure 3 shows the percentage of detection errors in the visual field contralateral to the lesion on successive test days. As indicated earlier, Ner, tested on day 8, was most severely affected, but the frequency of errors decreased over the next two sessions to reach preoperative levels by day 12. Pec, first tested on day 10 immediately after removal of the occluders, made fewer initial errors and showed recovery over the next 2 days by reaching preoperative performance values. Mou, Ner, and Pec all had the same frequency of errors on day 10 despite their different visual experience: Pec had none; Mou had 2 days of prior visual experience in his cage and in the experimental room but no testing on the detection task; and Ner had 2 days of prior testing on the detection task. Haw did not have occluders removed until the 13th day and showed no significant increase in detection errors from preoperative values.

Throughout postoperative testing the animals showed an elevation in the average reaction time to release the bar when detection stimuli appeared in the visual field contralateral to the lesion. Ner had the greatest

FIG. 2. Spatial pattern of recovery of detection errors. Polar maps and symbols same as in Fig. 1. Detection errors occurred for stimuli presented beyond 20° into the visual field contralateral to the lesion on day 10, persist only for stimuli beyond 30° on day 11, and are further reduced on day 20.
elevation in reaction time, averaging more than 100 ms above base line for the contralateral field but also 50 ms above for the ipsilateral field. This increase in reaction time in Ner diminished slightly during the first 2 postoperative weeks but was still greater than the increases seen in the other monkeys. Reaction-time increases in Mou and Haw averaged about 30 ms, and in Pec they averaged only 15 ms for stimuli in the contralateral visual field and were equal to preoperative values in the ipsilateral visual field. To determine whether this increased reaction time produced errors in stimulus detection, we allowed a longer period for the monkey to respond in two test sessions. While an allowance of 750 or 1,000 ms instead of the usual 500 ms reduced the total number of errors slightly, the number of false positives (appropriately timed bar releases when no target was flashed) rose sharply. The distribution of errors in the visual field did not appear to change. The elevation in reaction times persisted beyond recovery from the detection deficit.

The results of the detection experiments show that a visual detection deficit follows complete unilateral collicular ablation. In our experimental paradigm, however, the number of detection errors decreased during the second postsurgical week until the monkeys could again detect the lights at nearly preoperative performance levels.

**Visuo motor deficits**

The monkeys tended to ignore visual stimuli on the side contralateral to their lesion whether they were unrestrained in their cages, in a primate chair with their heads free, or in a chair with their heads held rig-

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**TRIAL #**

37

38

39

40

41

42

43

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NER 200 msec
idly. The tendency to look toward the side ipsilateral to the lesion was particularly obvious during the first 2 postoperative weeks and became more subtle over time. We examined this tendency more carefully by recording eye movements in three situations: during two behavioral tasks in which saccades to visual targets were not required and during spontaneous eye movements.

DISTRACTION SACCADIES. During a series of trials in which the monkeys were rewarded for looking at a central fixation point, an occasional unpredictable stimulus (1° spot of light) was either moved or flashed at 15 or 30° in the periphery. The eye movement records from a series of such trials are shown in Fig. 4. Trials 37–39 and trials 41–43 were fixation trials, i.e., no distraction stimulus appeared. Trial 40 was a distraction trial; a peripheral stimulus was presented at the time of the arrow and the monkey made an eye movement toward the stimulus. On subsequent trials (41 and 42) the monkey again tended to break fixation.

The results of the distraction experiments are shown in Fig. 5. Each bar graph shows the percentage of preoperative and postoperative trials on which a distracting stimulus elicited an eye movement. Results for all distraction trials were combined, since there were no differences associated with the different types of stimuli (flashed, moving, or displaced) or with the position of the stimuli in the field (15 or 30° from the fixation point).

Before the lesion, there was considerable variability among animals in the relative frequency of saccades of both fixation and distraction trials (Fig. 5, left side). Two monkeys, Haw and Ner, were induced to break fixation and make an eye movement toward the irrelevant stimulus only infrequently (less than 50%), while the other two animals did this frequently (greater than 50%).

In both pairs of monkeys, the effect of unilateral collicular ablation was to reduce the frequency of saccades to the distracting stimuli presented in the visual field contralateral to the side of the ablation (Fig. 5, right side). This effect could not be due to a general reduction in distractibility, since they continued to be distracted by stimuli flashed or moved in the ipsilateral visual field. Furthermore, the effect could not be due to an inability to detect the peripherally presented stimuli, since animals were tested in portions of the visual field that had already recovered from the detection deficit to even smaller stimuli (20° as compared to 1°).

This effect could also be observed on ordinary fixation trials. We observed that preoperatively one monkey, Mou, broke fixation frequently, making leftward saccades on 24% of the trials and rightward saccades on

![Graph showing reduction in frequency of saccades elicited by distracting stimuli in the visual field contralateral to the ablation.](image)

**FIG. 5.** Reduction in frequency of saccades elicited by distracting stimuli in the visual field contralateral to the ablation. Graphs show the percentage of distraction trials in which a saccade was made toward a stimulus on one side of the visual field or the other. The number of test trials are shown beside each bar of the graph. Preoperatively (left graph), two monkeys made relatively few saccades to these distracting stimuli (Haw and Ner, less than 50% of trials), while two others made frequent saccades (Mou and Pec, more than 50% of trials). After the ablation (right graph) both pairs of monkeys showed a decrease in the frequency of these eye movements, but particularly toward the field contralateral to the lesion. Data from postoperative days 5 in Mou, 15–18 in Ner, 8–13 in Mou, and 14 in Pec.
54%. Postoperatively, breaks in fixation decreased (to about 15%) but these breaks were always toward the ipsilateral side (left). DETECTION SACCADVES. Although the detection task did not require the animals to move their eyes to the peripheral stimulus, two monkeys nevertheless developed the habit of attempting to fixate it. They thus provided us with an opportunity to examine the probability of saccade initiation when a saccade was not required. The brief presentation of the stimulus (150-200 ms) did not allow the animals to achieve fixation of it since the stimulus was turned off at about the time the eyes began the saccade.

Figure 6 shows that the frequency of these saccades was reduced postoperatively, but only to detection stimuli in the visual field contralateral to the lesion. Comparison of these grids with the plots of detection errors for the same day (postoperative day 10 in Fig. 1 and Fig. 6) indicates that the loss of saccades occurred not only to stimuli that revealed a detection deficit but also to stimuli in the central visual field that did not. In view of the known deficits in saccade latency arising from collicular lesions, it should be emphasized that these saccades were not simply delayed but did not occur at all during the 1-s poststimulus interval.

The reduction in saccades to the detection stimuli did not recover along with the detection deficit. Figure 6 shows that on day 12 Pec was still making eye movements only infrequently toward stimuli in the visual field contralateral to the lesion, yet the number of detection errors to these same targets had already fallen to preoperative values, as shown in Fig. 4.

SPONTANEOUS EYE MOVEMENTS. Representative samples of spontaneous eye movements taken both before collicular ablation and during the second postoperative week are illustrated in Fig. 7A. These EEG records show that while the monkeys (Mou and Nex) continued to make rightward and leftward saccades, the eyes tended to remain turned to the side of the lesion. The magnitude of this ipsilateral bias in eye position is illustrated in Fig. 7B. Each graph represents the proportion of time spent in central eye positions (±10°) and in more eccentric rightward or leftward eye positions (greater than 10°). In contrast to preoperative samples, postoperative samples are not laterally symmetrical but show eye positions biased toward the ipsilateral side. Both monkeys

FIG. 6. Decrease in the frequency of saccades toward detection stimuli in the visual field contralateral to the ablation. Traces at the top of the figure show an example of a saccade made during a detection trial. Top two traces show horizontal and vertical eye positions, respectively. Third trace shows the sequence of events during the behavioral trial: the end of the fixation period, the detection flash (200 ms), remaining reward interval (during which time the bar release was rewarded, 500 ms total), and part of the intertrial interval. Time between dots is 100 ms. In this particular trial, the monkey broke fixation to look toward the detection target and missed the reward. Polar plots show the frequency of saccades made by Pec to each stimulus position during four trials (polar maps are the same as in Fig. 2). On postoperative day 10, saccades to detection stimuli presented contralateral to the ablation elicited fewer saccades than in preoperative tests. The decrease in the frequency of saccades persisted at least to postoperative day 12, well beyond recovery of the detection deficit seen in this monkey.
spent about 25% more time postoperatively than preoperatively looking to the side ipsilateral to the lesion and about 15% less time looking to the contralateral side. Such striking changes were seen only during the first 2 wk, and thereafter became more subtle.

The bias in eye position was examined more fully in the two monkeys with eye coils (see Table 1 of Ref. 4). Figure 8 shows eye position and saccade characteristics based on samples of spontaneous eye movements from Pec and Haw taken in the preoperative period and 2 wk into the postoperative period, after recovery from the detection deficit. Eye movement samples were analyzed for the relative frequency of fixations contralateral to the lesion (Fig 8A), the proportion of time spent in such contralateral fixations (Fig. 8B), and the relative frequency of contralaterally directed saccades (Fig. 8C).

The most marked change in the pattern of spontaneous eye movements following collicular ablation was in the relative frequency of contralateral fixations (Fig. 8A). The proportion of contralateral fixations decreased by 15% in Pec and 25% in Haw, changes
A B C

PEC HAW

CONTRALATERAL FIXATIONS (%) 60 40 20 0

TIME CONTRALATERAL SACCADIES (%) 60 40 20 0

CONTRALATERAL SACCADIES (%) 60 40 20 0

FIG. 8. Proportion of fixations on the side contralat-
eral to the lesion (A), proportion of time spent in con-
tralateral fixations (B), and proportion of contralaterally
directed saccades (C). Decreases from preoperative lev-
els (open bars) to postoperative levels (filled bars) that
were statistically significant at the 0.001 confidence level
on the basis of a χ² test are indicated with asterisks.
These observations were based on a 5-min sample period
during which eye movements were recorded with the
magnetic search-coil technique while the monkeys sat
in the lighted experimental room on postoperative day
16 for Pec and day 30 for Haw. The same patterns were
seen on day 19 for Pec and day 15 for Haw.

The proportion of time spent in contralateral eye
positions (Fig. 8B) also decreased, although not as
dramatically, reaching significance in
Pec (P < 0.001) but not in Haw. The direc-
tion of saccades (Fig. 8C), on the other hand,
was only mildly affected by the lesions. The
frequency of contralaterally directed sac-
cades decreased by less than 10% and this
change was not statistically significant.

In an attempt to determine whether the
changes in fixation positions depended on
visual stimulation, we repeated the same
analysis for spontaneous eye movements re-
corded in total darkness in these two mon-
keys with eye coils, but the results were in-
conclusive. When placed in total darkness,
both Pec and Haw showed an initial nystag-
mus with a slow phase of 1–2°/s toward the
side contralateral to the lesion. After the ini-
tial nystagmus subsided, eye position seemed
to depend on state of arousal; alert periods
with crisp saccades were interspersed with
intervals where the eyes rolled toward the
contralateral side as the animals became
drowsy. On day 15, during the nystagmus,
the eye remained predominately in ipsilat-
eral eye positions in Pec and in contralateral
eye positions in Haw. By day 30, however,
the eye position preference reversed in Haw.

The total frequency of spontaneous saccades
in the light was not affected consis-
tently in the four monkeys with unilateral
ablations (Fig. 9). Although the frequency
decreased postoperatively in Mou, Ner, and
Pec, this decrease reached statistical signifi-
cance in Mou only (Mann-Whitney U test,
P < 0.01).

In the one monkey tested with bilateral
collicular lesions, however, spontaneous eye
movements in the second postoperative week
showed a marked change from the preop-
erative measure. The most striking change
was a reduction in the frequency of spons
taneous saccades by about 60% (from a pre-
operative mean of 195 ± 27 to a postoper-
ative mean of 54 ± 5 per minute). The
frequency of smaller eye movements, those
under 10° in amplitude, was particularly
affected. Histological reconstruction of the
ablation in this case revealed that both col-
lliculi were nearly entirely removed and an
infarct extended anteriorly on both sides.
Thus, unlike monkeys with unilateral abla-
tions, which show little decrease in the fre-
quency of saccades, the monkey with a bi-
lateral ablation did show a marked reduction.
The reason for the difference could be that,
in unilateral cases, a reduction in the pro-
portion of fixations and saccades on the side
contralateral to the ablation is partially com-
pensated by an increase on the ipsilateral
from that following ablation of striate cortex in that the recovery is both more rapid and apparently less dependent on visual experience or task-related practice (9, 25, 35).

The transient visual deficit seen in our monkeys is consistent with the observations of Latto and Cowey (22). Using perimetry methods, these investigators found a mild visual-field defect following subtotal electrolytic lesions of the colliculus in one monkey that had previous ablations of the frontal eye fields. The deficit they found was most severe for peripheral stimuli beyond 30° eccentricity, and this deficit also recovered during the second postoperative week. Since they used a hemisphere to present their stimuli, peripheral targets did not suffer from the error in stimulus size introduced by our use of a tangent screen, and therefore, their findings provide strong support for the conclusion that the severity of the deficit increases with the eccentricity of the visual stimulus. Furthermore, their results suggest that lesion size is not crucial, since their lesions were incomplete, having involved primarily the superficial layers. Finally, their observation indicates that detection deficits after collicular lesions are not a result of added callosal damage (see Table 1 of Ref. 4) or surgical trauma since their lesions were made electrolytically.

The visual loss reported here is also consistent with other observations of losses in reactivity to visual stimuli following collicular damage. In the immediate postoperative period following unilateral collicular removal in man (17) and monkey (10), the visual deficit has been described as a complete hemianopia. In the monkey, large bilateral lesions were reported to produce peripheral visual-field defects (28) or deficits in detecting or localizing flashed stimuli, particularly in the periphery (7, 18–20). In addition, Latto (21) showed that collicular lesions produced a fourfold increase in brightness threshold for foveal stimuli. This loss in visual sensitivity persisted for as long as 6 wk.

Mohler and Wartz (25), on the other hand, did not find any detection deficit after ablations of the colliculus. They used nearly the same behavioral paradigm and testing situations as those used in the present experiment, with these exceptions: their mon-
keys had only partial collicular lesions, they were tested primarily in the central part of the visual field (where, according to the present results, the detection deficits are least dramatic and persistent), and they were not deprived of visual experience between surgery and testing. In view of the findings by Latto and Cowey (22) and Latto (21), none of these differences alone would seem to be sufficient to account for the discrepancy, but perhaps the combination is.

The decreased sensitivity for detection found in the present experiments, as well as comparable observations in other experiments, support a suggestion (31) that the superior colliculus in the primate makes a contribution to the sensitivity of the visual system. This visual function would appear to be independent of collicular functions involving eye movements, since the detection task did not require saccades. Ascending pathways from the superficial layers of the superior colliculus through the pulvinar to prefrontal visual cortex could mediate any such contributions to the sensitivity of the visual system. This pathway has been accorded great importance in nonprimate species (8, 11). The present results are consistent with the notion that the polysynaptic projection from colliculi to cortex contributes to visual function in primates as well, although the rapid recovery in sensitivity suggests that the contribution may be a limited one.

**Visuomotor deficits following collicular lesions**

The visuomotor deficit following collicular lesions was expressed in the detection and distraction tasks as a relative decrease in the frequency of saccades to stimuli in the contralateral visual field and, during spontaneous eye movements, as a relative decrease in the total time spent fixating the to the side contralateral to the lesion. These visuomotor deficits cannot be easily explained either by a simple sensory deficit such as a difficulty in detecting the stimulus or by a simple oculomotor deficit such as a difficulty in generating saccades.

A simple sensory loss does not appear to explain the asymmetry in saccade frequency, since it persisted after the monkeys were able to detect the visual stimuli in the affected field. This was demonstrated in the detection task by their ability to respond to the stimuli by a bar release. In the distraction task, the monkeys were not required to respond to the visual stimuli but these stimuli must have been suprathreshold, since they were as bright as the detection stimuli, and they were larger. The asymmetrical shift in the pattern of spontaneous eye movements that occurred as a result of the ablations are also difficult to explain in terms of a detection impairment. When the head is held, the most adaptive strategy in dealing with a contralateral visual-field defect presumably would be to shift the average position of the eye toward the side of the defect, thereby maintaining stimuli of interest in the unaffected visual hemifield. But this was not the strategy that was adopted. Instead, the average position of the eye was shifted toward the ipsilateral side.

Nor are the persisting visuomotor effects only the result of a simple oculomotor loss. While three of the four animals were unable to make accurate saccades or fixations to eccentric visual targets (4), these difficulties cannot account for their visuomotor deficits within the central portion of the visual field where their eye-position errors were small. Moreover, the oculomotor loss cannot account for any of the visuomotor deficits found in the normal animal, which had only slight saccade inaccuracies even for peripheral targets. Finally, in view of the similarity of the visuomotor deficits in all four animals, it is unlikely that the additional oculomotor deficits in three of them made any contribution to the asymmetry in saccade frequency.

The visuomotor deficits seen in the detection and distraction tasks and in spontaneous eye movements could all reflect the same underlying disorder: a reduction in the efficacy of stimuli in the contralateral visual field to elicit saccades. This hypothesis implies that the visuomotor deficits result from a removal of some input that facilitates either the selection of a visual target for a saccade or the initiation of a visually guided saccade into the contralateral visual field. This loss of facilitation would result in a simple decrease in the frequency of contralaterally directed saccades when the monkeys begin each trial from the fixation point directly ahead, as in the detection and distraction tasks. In the case of spontaneous eye
movements, the monkey is presumably choosing the stimulus for a saccade from a larger stimulus set and initiating the saccade from many eye positions. If targets in the ipsilateral visual field were more effective in eliciting saccades than targets in the contralateral visual field, then this relatively greater effectiveness would produce a shift in average eye position toward the ipsilateral side. Only slight differences in the relative frequency of ipsilaterally and contralaterally directed saccades would be needed to produce such a shift. Due to mechanical limitations of the eye, which limit saccades to a preferred range of eye positions, and due to the generation of nonvisually guided saccades, the average shift in eye position would never become extreme.

This hypothesis of collicular function—facilitation of stimulus selection, or saccade initiation, or both—is also supported by previous observations. The preceding paper (4) and earlier reports (25, 34) demonstrate that collicular ablation results in an increased latency for the initiation of saccades to visual stimuli. This increase in saccade latency may be considered as another example of a stimulus-selection/saccade-initiation deficit. In fact, under some conditions requiring rapid search, or in situations involving responses to briefly presented targets, increased saccade latencies might actually be responsible for decreases in the probability of saccade initiation.

It is important to note that all of the visuomotor deficits that have been described in this study were uncovered in situations in which the animals were not required to perform saccades. This fact may have particular relevance in connection with the phenomenon of neglect.

Mechanisms of neglect

The term neglect is used to describe the symptoms of patients who can be shown to both sense the stimulus and respond to it, but usually fail to do so. The symptoms resulting from collicular lesions in animals have frequently been described as neglect: they tend to look toward the side of a unilateral collicular lesion, tend to ignore stimuli in their contralateral visual field, and are less reactive to sudden or threatening stimuli in this field (10, 22, 32). The present experiments have provided a quantitative demonstration of each of these effects and specify, in addition, that after collicular ablation monkeys often fail to make saccades to contralateral visual targets even when it is known that they can sense the stimulus and make a saccade to it if it is required.

Of course, the neglect that has been described after collicular ablation may also involve a failure in responses other than saccades. That is, saccades may be only one component of a larger constellation of impaired orienting behaviors. This has been suggested by a number of recent experiments in both primate and nonprimate species. For example, Milner, Foreman, and Goodale (23) found that collicular lesions in the monkey lessen the disruption in visual performance caused by the sudden appearance of a brief peripheral stimulus. Similar decreases in distractibility have been observed in rats and hamsters (15, 14, 26). While eye movements were not monitored in any of these studies, eye movements were probably affected as one component of a more general orienting response. When compared to their effects in primates, collicular lesions in nonprimates appear to produce a more generalized deficit in the orienting response, presumably because the range of eye movement in nonprimates is more restricted, and head and body movement, therefore, become the principal means of directing the eyes toward novel or biologically significant stimuli. For example, rats (6, 12, 24), hamsters (30), tree shrews (8), and cats (32) all fail to orient to stimuli in the visual field opposite the ablated colliculus.

At least two hypotheses have previously been proposed concerning the physiological mechanisms underlying neglect (see Refs. 15, 16, 33 for review). One hypothesis is that neglect is the passive result of reduced sensitivity; thus, damage to a structure may decrease sensory awareness directly by reducing the effectiveness of incoming signals. A second hypothesis considers neglect as a deficit in the intention to move such that disturbances in the initiation of movement in response to sensory stimulation tend to occur. Behaviors that serve to direct the sensory apparatus, such as orienting responses, may be especially affected in such movement-initiation deficits.

These two hypotheses are not mutually exclusive, of course, and indeed the evidence
from the present study suggests that the neglect that has been described after collicular lesions involves both mechanisms. First, a visual deficit may contribute to the effect, particularly in the initial postoperative period when the monkeys have deficits in visual detection. In addition, persisting subtle decreases in visual sensitivity might contribute to longer lasting differences in saliency of stimuli in the two visual fields. Second, a visuomotor deficit also may contribute to the neglect. As we have already argued, this visuomotor deficit, which is expressed as an increase in the time required to generate the saccade to a visual target and a decrease in the frequency of a saccade to a visual target, may be due to a deficit in target selection, or saccade initiation, or both. Such deficits could indicate a condition in which the monkey is capable of sensing the stimulus and responding to it if required, but otherwise tends not to.

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