A VISUAL DEFICIT AFTER SUPERIOR COLLICULUS LESIONS IN MONKEYS

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Abstract. The superior colliculus has been implicated in visual orientation and localization in several mammalian species. To examine this possibility in rhesus monkeys, we trained monkeys to respond to one of six locations signaled by a small light of 5 s duration that was turned off immediately before they were allowed to respond. The locations were directly behind the light. After tectal lesions they continued to perform this task normally, but showed a severe deficit when the duration of the signal was reduced to 1 s. However, when the stimulus remained on throughout the trial and the site of the stimulus and response were further separated, performance of the animals with tectal lesions was normal. We suggest that this deficit in the accuracy of reaching guided by a transient visual stimulus may be related to the role of the superior colliculus in the control of eye movements.

INTRODUCTION

The superior colliculus has been implicated in visuomotor functions in several mammalian species. In the hamster, tree shrew, and cat, lesions of the colliculus have been reported to impair visually-guided orientation and localization (e.g. 3, 12, 13). Many of the properties of single colliculus neurons in the cat also support the idea that this structure is involved in visuomotor orientation functions (e.g. 5). The elegant recording and stimulation studies of Schiller and Koerner (11), Robinson (9), and Wurtz and Goldberg (14) have suggested a highly specific orientation role for
the superior colliculus of the rhesus monkey: targeting the eyes, or "foveating", on a visual stimulus.

However, when we began the present study there was little specific evidence that destruction of the simian colliculus would interfere with visual orientation and localization. A transient reduction in spontaneous eye movements and a small and transient increase in latency and decrease in accuracy of saccades had been reported to follow tectal lesions in rhesus monkeys (15), but eye movement reflexes were normal unless the lesions included the adjacent posterior commissure or pretectal area (8). Performance of visual discrimination tasks involving cues of pattern, color, flux, and flicker were also reported to be unimpaired by tectal lesions, although a deficit in the discrimination of rates of movement had been noted (1, 10).

Therefore, we set out to test directly the possibility that superior colliculus lesions in monkeys would impair localization of a visual stimulus. Monkeys were trained to reach to a specific site indicated by a small light; after tectal or control lesions they were tested for retention of the task and then trained on two further versions of it.

EXPERIMENT I

The monkeys were trained to reach for a hidden raisin, the location of which had been signaled by a small light presented for 5 s; after termination of the signal, the animal was allowed to respond. Following bilateral tectal or control lesions the animals were retested on this localization task.

Methods

Subjects. Seven experimentally naive rhesus monkeys (Macaca mulatta), weighing between 4.6 and 6 kg at surgery, served as subjects. Three received lesions of the superior colliculus (No. 1-3) and formed the Experimental Group. Two animals received subcortical lesions sparing the superior colliculus (No. 4 and 5) and together with two unoperated animals (No. 6 and 7) formed the Control Group.

Apparatus. Behavioral testing was carried out in a Wisconsin General Test Apparatus. The monkey's cage was separated from the illuminated stimulus compartment by two sliding screens, one opaque and the other transparent. Raising the transparent screen activated a timer which measured response latency to about 0.1 s accuracy. The experimenter was separated from the stimulus compartment by a sliding one-way vision screen.
When the opaque screen was raised, the animal faced the raised platform shown in Fig. 1. Six 6 V (GE No. 44) bulbs were mounted along the front edge of the platform. A 3 cm high vertical barrier, extending the width of the stimulus compartment, was located 1 cm behind the bulbs. Raisins were used as rewards and were placed beyond this barrier in one of six food compartments. The monkeys could see neither the food compartments nor the raisin, which was always placed in the center of one of the compartments. To obtain a raisin the monkey had to reach over the barrier into one of the compartments. The floor of each food compartment was marked with a grid to measure the site of the initial response on each trial.

Fig. 1. Apparatus used for all experiments. The upper row of lights was used for Experiments I and II, the lower for Experiment III.

White noise at a 70 db level was present throughout each session. Behavioral procedures. The monkeys were taught to reach over the barrier to obtain a raisin and then trained on two preliminary tasks. In the first task, only two light bulbs were used (No. 2 and 5). At the start of each trial, the opaque screen was raised, one of the bulbs illuminated, and after 5 s the transparent screen was raised. A raisin had been placed on the other side of the vertical barrier behind the illuminated bulb. When the animal reached over the barrier and picked up the raisin, the light was turned off, and both screens were lowered, thereby terminating the trial. If the animal's initial response was to the half of the platform on which the bulb was illuminated, the trial was counted as a correct one. Initial responses to the other side were counted as errors. In both cases the animal was given the opportunity to retrieve the raisin. The animals were trained until they made 27 correct responses on 30 consecutive trials. On this and all subsequent tasks, 30 trials per day were run 5 days each week, and the intertrial interval was about 10 s.

In the second preliminary task, the procedure was identical except that (a) one of the entire set of six bulbs was illuminated and the corresponding food compartment was baited, and (b) the animal was allowed to search for the raisin in only one food compartment on each trial. Responses to the compartment in which the raisin had been placed were counted as correct. Responses to other compartments were counted as
errors and terminated the trial; unlike the previous stage, the animal never received a raisin on an incorrect trial. The stimulus light was turned off after the animal made its response. Training was continued until a criterion of 27 correct out of 30 trials was again attained.

The same procedures were used in the final preoperative task except that when the transparent screen was raised, the stimulus light was turned off. That is, unlike the situation in the preliminary tasks, the animal could respond only in the absence of the cue. If the monkey failed to respond within 10 s the screens were lowered, and the trial was counted as an error. The monkeys were again trained to a criterion of 27 correct in 30 trials. Two types of errors were distinguished: "no-response errors", in which the animal failed to respond in the allotted 10 s, and "misreaching errors", in which the animal reached into an incorrect compartment. By this point all the animals were responding with a high degree of accuracy. That is, if they reached into the correct compartment they almost invariably contacted the raisin at once, showing little of the "tactile searching" or fumbling. Moreover, if they reached into the incorrect compartment, they immediately withdrew their hand.

Within 5 days of attaining criterion on the final task the animals underwent surgery. After surgery, the operated animals were tested on the second preliminary task until they reattained criterion in order to determine whether or not any overt motor dysfunction had resulted from the surgery. They were then retested on the final task until the original criterion was attained. Animal 1 was retested on the final task learned prior to operation on postoperative days 15 and 16, Animal 2 on days 8–20, Animal 3 on days 10–14, Animal 4 on day 24, and Animal 5 on days 5–10. The two unoperated monkeys, No. 6 and 7, were not retested.

Surgical procedures. The animals were anesthetized with halothane or pentobarbital sodium and placed in a Kopf Stereotaxic machine. After a skin incision, a section of skull was removed, the dura cut, and the electrode lowered. The exposed tip of the electrode was 1.5 mm long, and its diameter was 0.7 mm. The lesions were made with a Kopf radio-frequency lesion generator set at a tip temperature of 70°C for a duration of 60 s. The intent was to make six burns in each superior colliculus. Following the lesions the dura was sutured and the bone-flap replaced. Wounds were closed in anatomical layers, and 2 cm³ penicillin G (Bicillin) were administered intramuscularly.

As described below, three animals actually sustained superior colliculus damage and formed the Experimental Group, whereas the colliculus was entirely spared in two other animals. These latter two animals served as operated control animals and with the addition of two unoperated animals formed the Control Group.
**Histological procedures and results.** After completion of the study, each animal was deeply anesthetized with pentobarbital sodium (36 mg/kg) and perfused through the aorta with isotonic saline followed by 10% formalin. A few days later the brain was blocked in the coronal stereotaxic plane, placed in 30% sucrose formalin until it sank, embedded in albumin-gelatin, and cut in 25 μm frozen sections. Determination of the extent of the lesions was based on examination of serial sections (0.5 mm apart) stained with cresyl violet.

Representative cross-sections of the lesions are shown in Fig. 2.

![Diagram of brain sections](image)

**Fig. 2.** Representative cross-sections through the lesions of two experimental animals (No. 1 and 2) and one control animal (No. 5). Areas of cell loss and intense gliosis are shown in black. Caudate nucleus; CC, corpus callosum; CG, central grey; H, habenular area; IC, inferior colliculus; MD, medial dorsal nucleus; MG, medial geniculate nucleus; PC, posterior commissure; PL, inferior pulvinar; PL, lateral pulvinar; PM, medial pulvinar; SN, substantia nigra; VL, ventral lateral nucleus; VPI, ventral posterolateral nucleus.

Animal 1 sustained almost complete destruction of the superior colliculus except for the rostral superficial layers on both sides and the anterolateral portion on the left. In addition there was some damage to the central grey and midbrain reticular formation on both sides and to
the pretectum on the right side. In Animal 2 the lesion was deep, sparing
the superficial layers and the medial portion on both sides. There was
also damage to the midbrain reticular formation and the brachium of
the inferior colliculus. In Animal 3 the middle third of the left superior
colliculus was destroyed. Unfortunately, adequate histology was not ob-
tained for the right side, but there probably was little or no damage.
In both operated control animals, midbrain structures were entirely
spared. Animal 4 sustained very slight damage to occipital cortex on the
medial tentorial surface of the right hemisphere. In Animal 5 there was
bilateral involvement of the lateral dorsal, anterodorsal and anteroventral
nuclei of the thalamus, the corpus callosum at the level of the lateral
geniculate bodies, and the stria terminalis.

Results

Prior to operation, there were no significant differences between the
Experimental and Control groups in the number of trials or errors to
criterion on either the preliminary or final tasks (U = 2, Mann–Whitney
U test). On the preliminary tasks the animals required 474 to 1094 trials
and made 132 to 399 errors. The performance of each animal on the final
preoperative task is shown in Fig. 3. Almost all the errors for both groups
were misreaching rather than no-response errors.

![Error and Trial Graph](Fig. 3. Experiment I: number of errors (bars) and trials (dots) made by each ani-
mal in reaching criterion before (PRE-OP) and after surgery (POST-OP). The pre-
sence of a horizontal line within the bar indicates the number of misreaching er-
rors (below line) and of no-response errors (above line). The absence of this line
indicates that all the errors were misreaching ones. The stimulus duration was 5 s.

After surgery all the animals quickly reattained criterion on the
second preliminary task. None showed any sign of motor dysfunction.
There was no difference between the groups in postoperative retention
of the final preoperative task as shown in Fig. 3. As before operation
most of the errors were misreaching errors rather than no-response errors. The groups did not differ in their mean response latency; furthermore, latency and accuracy of response were not correlated for any animal.

EXPERIMENT II

The duration of the visual stimulus was reduced from 5 s to 1 s, and the task was otherwise identical to that in Experiment I.

Methods

Subjects. All seven animals from Experiment I served as subjects. This experiment was conducted on postoperative days 17–40 for Animal 1, days 22–60 for Animal 2, days 15–26 for Animal 3, days 28–44 for Animal 4, and days 11–12 for Animal 5.

Apparatus and procedures. The apparatus and procedures were the same as those of Experiment I except that the duration of the stimulus light was 1 s.

Results

Each Experimental animal required at least three times as many trials and errors to reach criterion on this task as every Control animal (Fig. 4); the groups were significantly different on both measures ($U = 0$, $P < 0.03$).

Fig. 4. Experiment II: errors and trials to criterion. The stimulus duration was 1 s. See also legend to Fig. 3.

The animals with tectal lesions made both more misreaching errors and more no-response errors than the Control animals ($U = 0$, $P < 0.03$). As in the previous experiment, there were no differences between the
groups in the mean latency of response, nor was there any correlation between response latency (for trials on which responses were made) and accuracy of performance for any animal.

![Graph](https://via.placeholder.com/150)

**Fig. 5.** Experiment III: errors and trials to criterion. In this task there was an increased separation between stimulus and response, and the stimulus remained on throughout the trial. See also legend to Fig. 3.

**EXPERIMENT III**

The animals with tectal damage were not impaired when the light signaling the location of the reward was on for 5 s prior to the response (Experiment I), but when the duration of the light was shortened to 1 s (Experiment II), they were impaired. In this experiment the spatial requirements of the task were made more stringent and the temporal requirements less so; the spatial separation of the signal from the response site was increased, but now the visual signal remained on throughout the trial.

**Methods**

*Subjects.* The same animals used in Experiments I and II served as subjects. Animal 1 was trained on postoperative days 41–42, Animal 2 on days 61–79, Animal 3 on days 37–47, Animal 4 on days 49–94, and Animal 5 on days 13–53.

*Apparatus and procedures.* Except for the location of the stimulus lights, the apparatus was identical to that used in previous experiments. Now the lights were located on the front of the panel 3 cm below the horizontal platform rather than on the horizontal platform (see Fig. 1).

On each trial the opaque screen was raised, one light turned on, and then the transparent screen raised. The light was left on until the animal’s response was completed. The procedures, methods of scoring, and criterion were otherwise the same as before.
As shown in Fig. 5, there were no significant differences between the groups in either trials or errors to criterion ($U = 3$). Whereas all the Experimental animals found this task less difficult than the previous one (Experiment II), three of the four Control animals found it more difficult. In fact, the Experimental animals actually required fewer trials and errors to reach criterion than these three Control animals. As in the previous experiments, the response latency of the groups did not differ, nor did response latency and accuracy correlate for any animal.

DISCUSSION

Tectal lesions impaired the ability to respond accurately to a specific location on the basis of a visual cue when the cue was 1 s in duration (Exp. II), but not when the cue was 5 s long (Exp. I). In Experiment III where the stimulus and response sites were further separated but the stimulus light remained on throughout the trial, the monkeys with tectal lesions performed at least as well as the Controls. Thus, the variable that made the task in Experiment II sensitive to tectal lesions was probably the short (1 s) duration of the visual signal.

The three experiments also differed in whether they tested acquisition or retention, in the spatial separation of stimulus and response site, and in their relative difficulty, but none of these differences account for the impairment of the Experimental Group in Exp. II. The difference in results between Experiments I and II was unlikely to have been due solely to the acquisition-retention difference, because Experiment III also involved an acquisition task on which the animals with tectal lesions performed normally. The impairment in Experiment II was also probably not due to the spatial separation of stimulus and response, because the same separation was present in Experiment I, and an even greater one was present in Experiment III. Finally, the deficit after tectal lesions in Experiment II cannot be attributed to the difficulty of the task, since this task was actually the easiest of the three tasks as measured by the performance of the normal monkeys. Thus, in our testing situations, tectal lesions impair visuomotor localization only when the visual cue is sufficiently brief.

Previous work on the primate colliculus suggests several dysfunctions that could have produced the deficit in Experiment II. Monkeys with tectal damage show a lower frequency of spontaneous eye movements and a longer latency for a saccade (e.g. 15) either of which could have resulted in a lower probability of detecting a brief stimulus (as in Experiment II), as compared with a longer stimulus (as in Experiment I). Similarly, single-unit recording and stimulation studies have implicated
the superior colliculus in foveation (e.g. 11), and rapid foveation would seem more important in Experiment II than Experiments I and III. These speculations impute the deficit shown in Experiment II to a change in the frequency, latency, or accuracy of eye movements. However, a more "cognitive" possibility has also been suggested by studies of tectal unit activity. Goldberg and Wurtz (4) have proposed that attention toward a stimulus may be a parameter of tectal unit activity; an attentional disability, particularly a failure to attend to peripheral stimuli, might be expected to produce the deficit as seen in Experiment II.

Recently, two other demonstrations of positive effects of tectal lesions have been published. In the first, Butter (2) showed that although monkeys with tectal lesions learn color discriminations as well as control animals when the stimulus and response sites are identical, they are impaired when the stimuli are separated from the response site. A separation of stimulus and response site was also present in all three parts of this study, but our animals with tectal lesions were impaired only in Experiment II, although the stimulus-response separation was identical to that in Experiment I. However, (a) our animals, unlike Butter's had received very extensive preoperative training with stimulus-response separation, (b) our discrimination task (light vs. no light) was presumably a simpler one than Butter's color discriminations, and (c) the stimulus-response separation in our experiments was less than the minimum one used by Butter. Any or all of these factors could explain the normal performance in Experiments I and III, although both involved a separation of stimulus and response site.

In the second experiment, Keating (7) trained monkeys to respond to the location of the brighter of two lights. The lights were presented for only 0.2 sec, and their spatial location varied from trial to trial. Following tectal damage there was a severe deficit in performance. Thus Keating's experiment was very similar to our Experiment II; both demonstrated a deficit in localizing the site of a brief visual stimulus after tectal lesions.

In both Keating's and our studies the visual stimulus was a brief one. Could the use of a brief stimulus have been a crucial element in the impaired performance after the tectal lesions? Two unpublished studies suggest that use of a brief visual cue is not, by itself, sufficient to demonstrate a deficit in monkeys with superior colliculus damage. In the first, C. W. Mohler and R. H. Wurtz (in preparation) showed normal detection of a small spot presented for 150 ms to the portion of the visual field whose representation on the tectum had been destroyed. In the second, after tectal lesions, Handelsman (6) showed accurate discrimination of the color and of the position of a small stimulus presented for 260 ms.
In neither experiment was the animal required to respond to the site of the brief stimulus. Thus, the use of brief discriminanda does not itself seem to be sufficient for demonstrating a dysfunction after tectal lesions in monkeys.

In summary, as in previous studies, tectal lesions in monkeys did not produce the severe and generalized visual orientation and localization impairments that they do in other mammals. However, they did produce a clear deficit in the accuracy of reaching guided by a brief visual stimulus. This specific visuomotor deficit may be related to the role of the superior colliculus in the control of eye movements that has been suggested by other lines of evidence.

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