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LOCALIZATION AND DETECTION OF VISUAL STIMULI FOLLOWING SUPERIOR COLLICULUS LESIONS IN RHESUS MONKEYS

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SUMMARY

Rhesus monkeys were trained to fixate a central stimulus and to detect and localize a 50 msec light flash presented 6–80° on either side of the central stimulus. Following large lesions of the superior colliculus, they showed persistent deficits in localizing flashes presented 43–80° from the fixation stimulus. However, they were not consistently impaired when the flashes were presented more centrally, and their performance with peripheral stimuli improved when the stimulus duration was 1 sec. Thus, the superior colliculus appears to be necessary for the localization of brief visual stimuli in the far periphery.

INTRODUCTION

Electrical stimulation, recording and ablation experiments have demonstrated a crucial role for the superior colliculus in visual orientation and localization in a variety of subprimate species[15,16]. The situation is less clear in primates. On the one hand, the elegant recording and stimulation studies of Schiller, Wurtz and Robinson and their colleagues have provided a detailed account of how the colliculus may serve to direct the eyes and perhaps the animal's attention to peripheral stimuli[5,6,12–14,17]. On the other hand, there is little direct evidence that removal of the colliculus in primates markedly impairs visual localization and orientation toward peripheral stimuli.

Reflexly induced eye movements are unaffected by colliculus removal[11], and the deficits in voluntary saccades reported after colliculus lesions in monkeys are usually slight and transient[10,18]. While visual learning impairments have been reported in monkeys with collicular lesions[1], orientation was not observed in that study. A tectal role in orientation and localization is more directly suggested by the findings that accuracy of reaching to the site of a brief visual stimulus is impaired after colliculus lesions[8,9]. However, it is not clear whether and to what extent these deficits were due to

impairments in detecting or in localizing visual stimuli. Furthermore, in neither of these two studies was fixation controlled, and therefore accuracy of detection and localization in different parts of the visual field could not be determined. In the present experiment we studied the effects of colliculus lesions on performance of a task in which detection and localization of brief light flashes presented in various parts of the visual field could be independently assessed.

METHODS

Subjects

The subjects were 5 rhesus monkeys (*Macaca mulatta*), 7.3–11.5 kg in weight. All had undergone visual discrimination testing prior to the present experiment. They were housed in individual cages and fed Purina Monkey Chow supplemented with fresh fruit. Isoniazid in tablet form (25 mg) was administered daily as a prophylaxis against tuberculosis. During testing, the monkeys were water deprived so that their fluid intake was restricted to approximately 200 ml daily, including the water they received as reinforcement during testing.

Apparatus

All training and testing were conducted while the monkeys were seated in a plastic chair which limited body movement, but allowed free use of their arms, hands and head. The testing apparatus consisted of a sound-attenuating chamber containing a metal screen painted flat black. The screen contained a series of 14 circular holes, each 1.4 cm in diameter, located at eye level and arranged in a horizontal array symmetrically around the screen center (see Fig. 2). Mounted behind each of these holes was a translucent plastic strip, which served both as a stimulus and a response panel. These plastic panels, labelled 'side panels' in Fig. 2, were hinged at the top so that a microswitch, mounted behind each panel, closed when the panel was depressed. The metal screen was curved so that all the side panels were equidistant from the animal's eyes when its head was centered, as shown in Fig. 2. At the viewing distance employed, these panels were 3.3° in diameter and 12.4° apart, center to center. The centers of the side panels were located 6.4°, 18.7°, 31.0°, 43.5°, 55.7°, 68.1° and 80.4° from the center of the metal screen. Each panel was illuminated by a rearmounted 28 V incandescent lamp (Sylvania 28ESB5). A low current was passed through the lamps, thus allowing a brief surge in current to produce a light flash with rise and fall times of 9.5 msec. When lit, the luminance of the different panels varied from 89.3 to 148.5 cd/sq.m; when unlit by the lamps, their luminance varied from 0.1 to 0.2 cd/sq.m, which was provided by a lamp mounted in the ceiling of the chamber.

In addition to the side panels, two other panels were located on the metal screen. One of these, the 'fixation' panel, a square plastic button, 13 sq.mm, was located in the center of the metal screen. The other, the 'no-light' panel, was a round plastic panel, 2.5 cm in diameter, located above the fixation panel, as shown in Fig. 2.

Behavioral procedures

Prior to surgery the monkeys were trained to fixate directly ahead and then

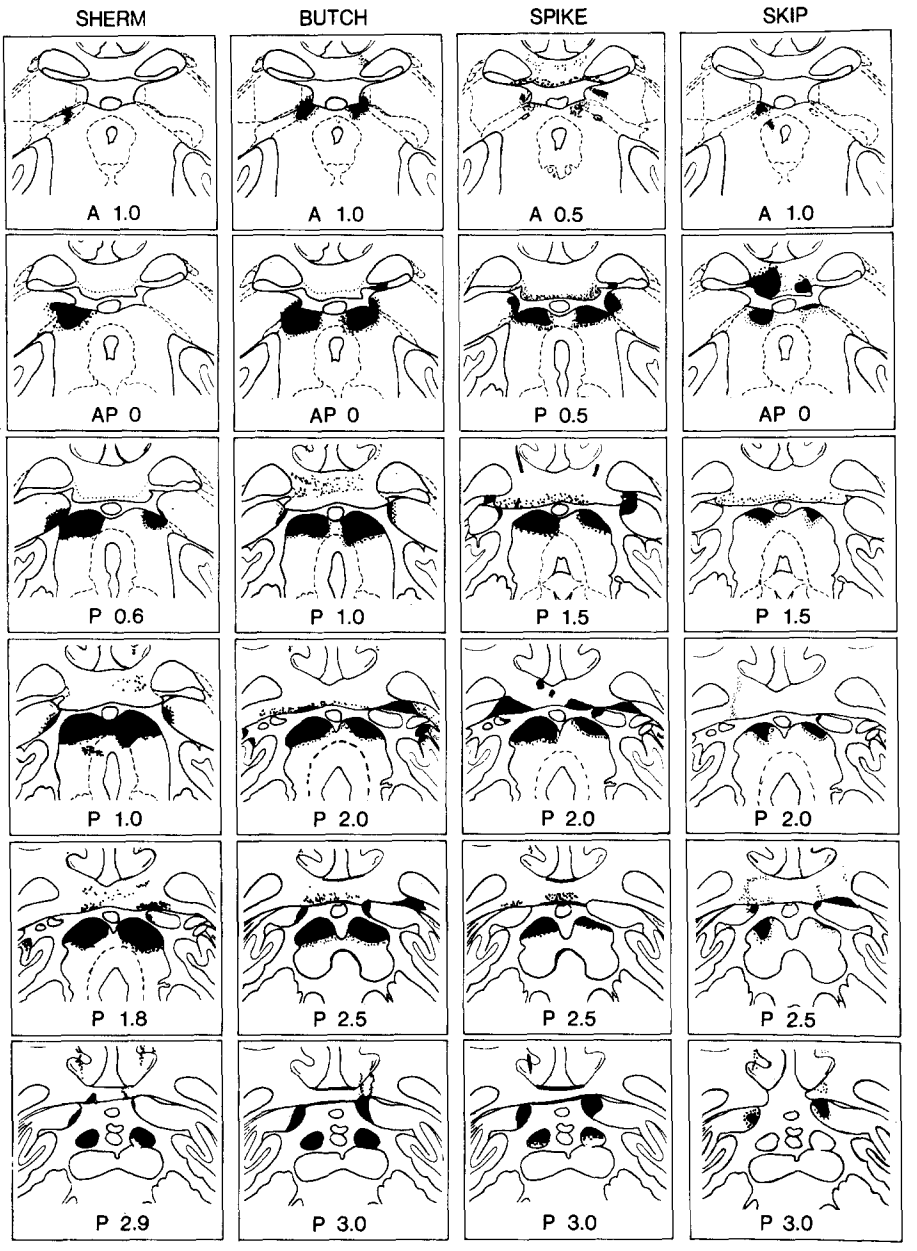

Fig. 1. Cross-sections through lesions of the superior colliculus. Areas in black indicate complete cell loss; dots indicate areas of severe cell loss or gliosis, or both.

respond to the side panel on which a light flash appeared, or to press the no-light panel if no light flash was presented. The training procedures and the parameters of the final task are described in detail in the following sections.

Preliminary training. The monkeys were first trained to press the side panels to obtain water reinforcement. Initially, all the side panels were continuously illuminated; subsequently, the number of side panels illuminated on any trial was gradually reduced until only one was lit on each trial. After the monkeys learned to respond differentially to a single, continuously illuminated side panel, the panel illumination was changed to a series of 0.5 sec light flashes. Responses to unlit panels were punished by time-outs, during which the entire chamber was darkened and no responses were reinforced for 7 sec. In the next stage, the monkeys were required to press the center fixation panel when it changed color (as described below) in order to initiate presentation of light flashes on a side panel. The size and brightness of the color cues on the fixation panel were then gradually reduced to small, dim spots. After this fixation task was mastered, the number of light flashes presented on a side panel was gradually reduced to one, and subsequently the flash duration was reduced to 50 msec. In the final stage of preliminary training, no-light trials were introduced, and the monkeys were trained to press the no-light panel on these trials.

Final preoperative task. Five seconds after reinforcement for a correct response or 7 sec after an incorrect response, a dim red light appeared on the fixation panel. A response to the fixation panel now changed the light from red to green. The duration of the green light varied randomly from a minimum of 0.25 sec to a maximum of 2.5 sec. Immediately after the green light was extinguished, a dim yellow spot appeared for 0.25 sec on the fixation panel. The monkey was required to press the fixation panel within 0.75 sec after the yellow spot was extinguished in order that the trial proceed. The colored lights were 0.06° in diameter and rapidly flickered on and off so that they appeared to pulsate. An incorrect fixation panel response, i.e. responding while the green spot was present or responding later than 0.75 sec after the yellow spot disappeared, resulted in a time-out. The parameters of the stimuli on the fixation panel were such that fixation was required to detect the change from green to yellow[3,5].

On one-half of the trials, a 50 msec light flash was presented on one of the side panels 0.5 sec after the response to the yellow fixation spot was correctly executed, and, if this side panel was pressed within 10 sec after the flash, water reinforcement was presented. In each session, light flashes were presented on 8 of the 14 side panels, 4 on each side of the screen, in pseudo-random orders. The side panels on which the flashes were presented varied from session to session, but in any session they included two of those located furthest (68° and 80°, both left and right) from center, at least two in intermediate positions (55°, 43° and 31°, both left and right of center) and two located closest (18° and 6°, both left and right) to the center. In every block of 5 consecutive sessions, light flashes were presented equally often on all 14 side panels. On the remaining half of the trials in each session, no light flash was presented after successful completion of the fixation phase. On these trials, water reinforcement was contingent upon pressing the no-light panel within 10 sec of completing the fixation task. If the animal failed to respond either to a side panel or to the no-light panel within 10 sec of

completing the fixation task, the trial was terminated, whether it was a light or a no-light trial. The order of light and no-light trials was randomly varied in each session, which consisted of 250 trials. Throughout testing, the chamber was illuminated by an overhead lamp.

Error analysis. In the final task, 3 kinds of errors could be made on light trials: (a) a 'localization' error — pressing a side panel other than the one illuminated; (b) a 'detection' error — pressing the no-light panel, and (c) failure to press either the no-light panel or any of the side panels. Two kinds of errors could be committed on the no-light trials: (a) a 'false positive' — pressing one of the side panels, and (b) failure to respond.

Design. The monkeys were trained on the final task until they performed at least 90% correct responses in light trials and in no-light trials in each of 8 consecutive sessions. Two weeks after reaching this criterion, the monkeys were tested for retention of the task in 5 sessions, and then were operated.

During the postoperative recovery period and for several weeks thereafter, the animals' visuomotor and ocular behavior was evaluated by two observers. These behaviors included orienting with the eyes and head to objects in the periphery of the visual field, reaching for small objects, visual fixation of stationary and moving objects, the occurrence of spontaneous eye movements, strabismus, retraction of the eyelids, and direct pupillary responses to light.

Approximately 3 weeks following surgery, the monkeys were tested for post-operative retention of the final preoperative task. Testing continued for 2–3 months. All the animals were initially tested by the same methods used in the final preoperative task, except for Sherm. Since Sherm failed to respond to any light flashes in the initial postoperative sessions, it was tested in each session with 1 sec light flashes (to which it frequently and correctly responded) on one-half of the light trials, and with the standard 50 msec flashes on the remaining light trials. Starting with the sixth postoperative session, the other 4 monkeys were also tested with 1 sec flashes in every sixth session. After completion of postoperative testing with brief and long flashes, 3 of the monkeys, Spike, Skip and Lloyd, were tested for two weeks with dimmer light flashes (0.5–0.9 cd/sq.m) than those presented previously. The dim flashes were presented only on the 5 outermost panels on each side of the metal screen.

Surgical procedures

Lesions were aimed at the superior colliculus in 4 monkeys (Butch, Skip, Spike and Sherm) by first plotting the receptive field positions of single units in the colliculus and then using this information, together with retinotopic maps of the colliculus[4], to guide the lesion electrodes.

The animals were first restrained with ketamine hydrochloride (1 mg/kg body weight injected i.m.) following which they received atropine sulphate (0.02 mg/kg body weight) and 450,000 units of penicillin i.m. Following induction of light halothane anesthesia, the animals were intubated with a tracheal cannula and anesthetized with 2.5% halothane together with a mixture of 70% nitrous oxide and 30% oxygen. The halothane was later reduced to 1%. Body temperature, pulse and respiration were

monitored throughout the operation. The animals were placed in a stereotaxic instrument modified so that their visual fields were unobstructed. The right pupil was dilated with 0.25% scopolamine hydrochloride, the lids of the right eye were retracted, and the right eye was refracted with a contact lens so it was focused on a translucent screen 57 cm away. The right fovea and blind spot were projected on the screen with a reversible opthalmoscope. The horizontal meridian was defined as a line passing through the center of the blind spot and the fovea. An orthogonal line passing through the fovea defined the vertical meridian. The eye position was checked (and replotted if necessary) during the period of receptive field plotting.

Under aseptic conditions, the scalp, fascia and temporal muscle were cut and retracted, and a bilateral opening was rongeured in the skull around the AP-0 stereotaxic position. The femoral vein was then cannulated, and the animal was immobilized with 20 mg of flaxedil administered i.v. Supplementary doses of 2 mg flaxedil were administered during the course of the operation. Recording electrodes were next advanced through a dural opening to the superior colliculus with an hydraulic microdrive. The electrodes were made of varnished tungsten wire, 30 μm in diameter, with tips 2 μm in diameter. The electrodes were connected to a cathode follower, which provided input to a preamplifier. Signals were monitored on an oscilloscope and, after further amplification, over a speaker. Visual stimuli used to elicit neuronal discharges consisted of moving spots of light back-projected onto the tangent screen. Receptive fields of several units recorded on at least two electrode penetrations in each superior colliculus were plotted in order to estimate the location of the representations of the horizontal and vertical meridian on the colliculus. This information was used to guide the positioning of the lesion electrode. The electrode used for producing the lesions was made of 0.75 mm thick stainless steel wire insulated with Teflon, with 0.5 mm of the tip exposed. This electrode was lowered to 4 positions in each side of the brain. Radio frequency current intensity passing through the electrode was adjusted so as to heat the tip to 73–80 °C for 1 min.

The dura was then replaced over the cortex. Gelfoam was placed over the bone opening, and the wound was sutured in anatomical layers. For several days following surgery, the animals were kept in a humid room in order to avoid respiratory complications and were given additional injections of penicillin.

Control surgery was performed in one monkey, Lloyd. The procedures were identical to those used for the experimental animals, except that both the recording electrodes and the lesion electrodes were only lowered to a point approximately 2 mm above the superior colliculus, and no lesions were made.

Histological methods and results

Following the completion of testing, the monkeys were deeply anesthetized and perfused intracardially with 0.9% saline followed by 10% formalin. After 3–5 days in formalin, the brain was removed from the head, placed in a stereotaxic instrument and the tissue containing the lesion was blocked in stereotaxic planes. The blocked tissue was placed in a sucrose–formalin solution until it sank. The block was then embedded in albumen gel, and frozen sections 40 μm in thickness were cut. Every fifth section

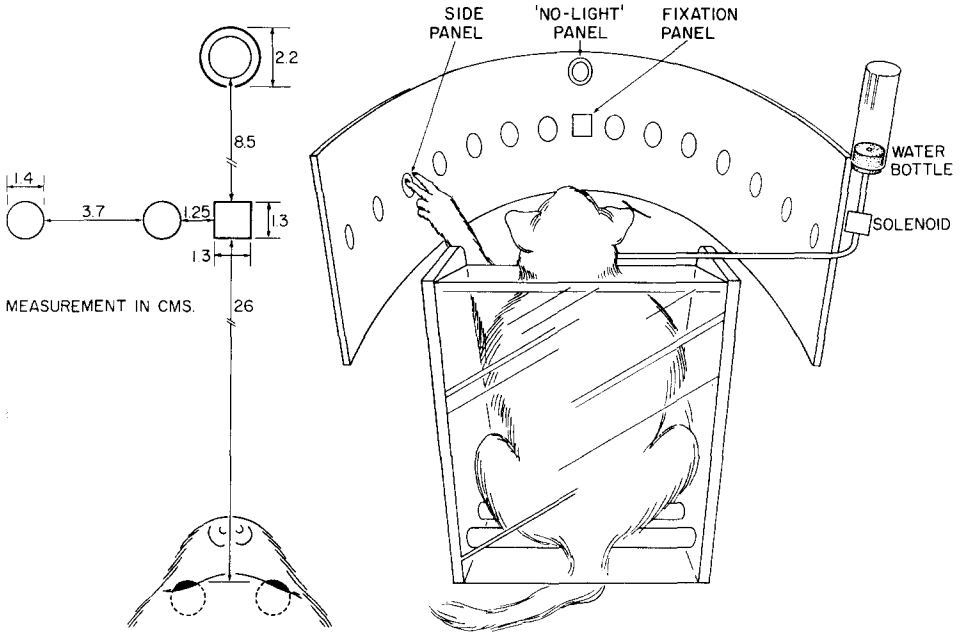

Fig. 2. Schematic view of the testing apparatus (right) and measurements of the panels on the metal screen (left).

was stained with thionin, and adjacent sections stained with the Weil method.

Fig. 1 shows representative cross-sections through the collicular lesions in the 4 monkeys. It will be noted that 3 of the monkeys — Sherm, Butch and Spike — sustained extensive lesions of the superior colliculus, whereas Skip's lesion was small and superficial. As seen in Fig. 1, Sherm's lesion destroyed all but the most anterior portion of the superior colliculus bilaterally, with only slight damage to the central gray and none to the tegmentum. On the left side, the lesion extended anteriorly and dorsally into the brachium of the superior colliculus and n. limitans, and produced slight damage in the pretectum. The medial edge of the pulvinar and prestriate cortex above the anterior end of the calcarine fissure was also slightly damaged. In addition, the passage of the electrodes in Butch, as well as in all the other monkeys, was responsible for slight damage and gliosis in the white matter adjacent to the midline cortex and in the fornix and corpus callosum. Butch's lesion was quite similar in locus and extent to that of Sherm, except that the most medial portion of the superior colliculus was spared. The medial portion of the brachium of the superior colliculus and n. limitans was damaged bilaterally, and gliosis extended to the colliculus–pretectum boundary on the right side. In addition, the posteromedial portion of n. medialis dorsalis was damaged bilaterally, along with the medial edge of the pulvinar and, behind it, a small portion of prestriate cortex. Spike's lesion severely damaged the posterior two-thirds of the colliculus, although it did spare medial and ventral portions of the colliculus on the right side. Spike's lesion also extended anteriorly and dorsally into the brachium of the superior colliculus, which showed some gliosis, n. medialis

dorsalis and the medial edge of the pulvinar; posteriorly, medial prestriate cortex was slightly damaged. In contrast, Skip's lesion was quite small and involved mainly the superficial layers in the intermediate portion of the superior colliculus, although slight damage was also found in the central gray, pretectum and brachium of the superior colliculus on the left side. Examination of the brain of the operated control animal, Lloyd, disclosed only slight damage to white matter, fornix and corpus callosum as a result of electrode penetrations, as in the other animals.

RESULTS

Skip's colliculus lesion was much smaller than intended, and was restricted to the superficial layers of a small portion of this structure, as described in detail under *Histological methods and results*. Furthermore, Skip's behavior in all aspects of both formal and informal testing was indistinguishable from that of the operated control animal, Lloyd. Therefore Skip, together with Lloyd, will be considered control animals.

Visuomotor and ocular deficits

Following surgery, Sherm showed few spontaneous saccades, and its eyelids were moderately retracted. It also oriented sluggishly to peripheral visual stimuli, and, when it did orient, the eyes frequently lagged behind head movements. Two weeks following surgery, the only observable deficit in Sherm was sluggish orientation to peripheral stimuli, which persisted for several more weeks. Throughout the post-operative period, foveation of stationary and moving objects was normal, as was reaching for objects, and light reflexes in both pupils appeared unaltered.

Like Sherm, Butch exhibited a paucity of saccadic eye movements after surgery. In addition, Butch showed poor tracking of moving objects, and misreached for objects with either hand. None of these deficits were present by the time formal testing began. Fixation of stationary objects was unaffected by surgery, as was orienting to peripheral stimuli. Likewise, no lid retraction was observed, and light reflexes in both pupils were normal.

Spike, like Butch, did not properly foveate small moving objects, and showed few spontaneous saccades, especially large amplitude ones, after surgery. Spike also showed a slight strabismus, and its orientation to peripheral stimuli was incomplete. These symptoms were gone before formal testing began. No lid retraction was noticeable, and both pupils showed normal reactions to light. Similarly, reaching for small objects was accurate.

As mentioned previously Skip, the monkey with the small colliculus lesion, and Lloyd, the operated control monkey, exhibited no visuomotor or ocular disturbances after surgery.

Performance deficits in testing

Since all the monkeys performed at criterion level in the preoperative retention sessions, their scores in these sessions were combined in the following analyses with those in the prior sessions in which they had attained criterion.

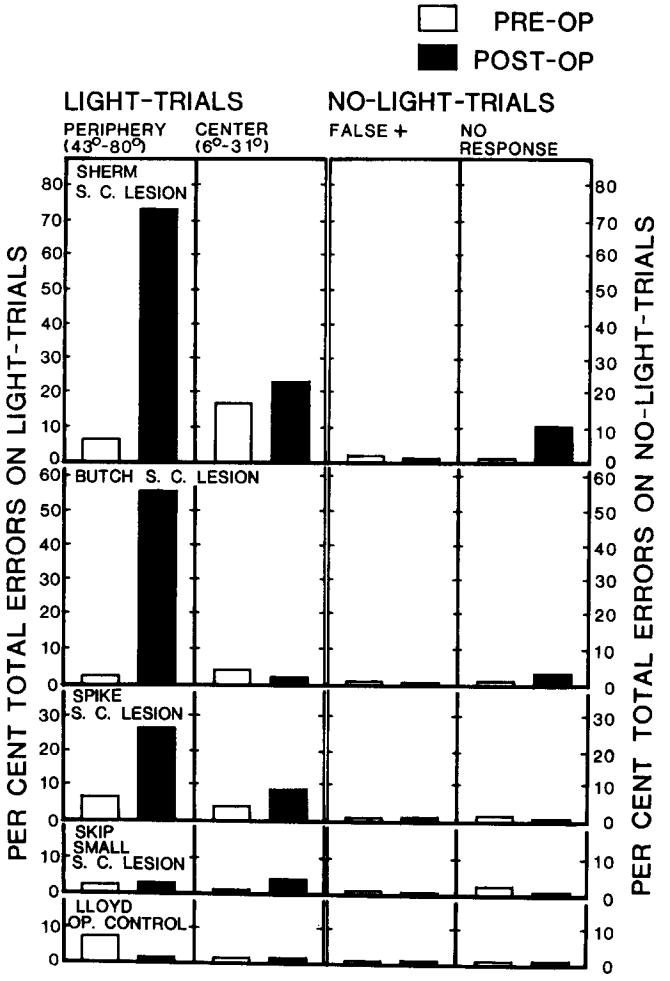

Fig. 3. Per cent total errors made by each monkey on light trials and on no-light trials before and after surgery.

Following surgery, the experimental animals made more errors in the test situation compared either to their own performance prior to the surgery or to that of the control animals after surgery. The performance of the control animals, on the other hand, did not deteriorate postoperatively. As shown in Fig. 3, the experimental animals' impairment was almost entirely restricted to trials on which light flashes were presented. On no-light trials following surgery, the experimental animals, like the controls, performed as well as they had before surgery, with one exception: Sherm omitted responses more frequently than it did preoperatively. Furthermore, the experimental subjects made more errors when light flashes were presented on the peripherally located panels than they did when flashes were presented on the more centrally located panels (see Fig. 3). It will also be noted that the severity of this selective impairment in responding to peripherally presented flashes varied among the experimental animals: Sherm was the most severely impaired and Spike the least.

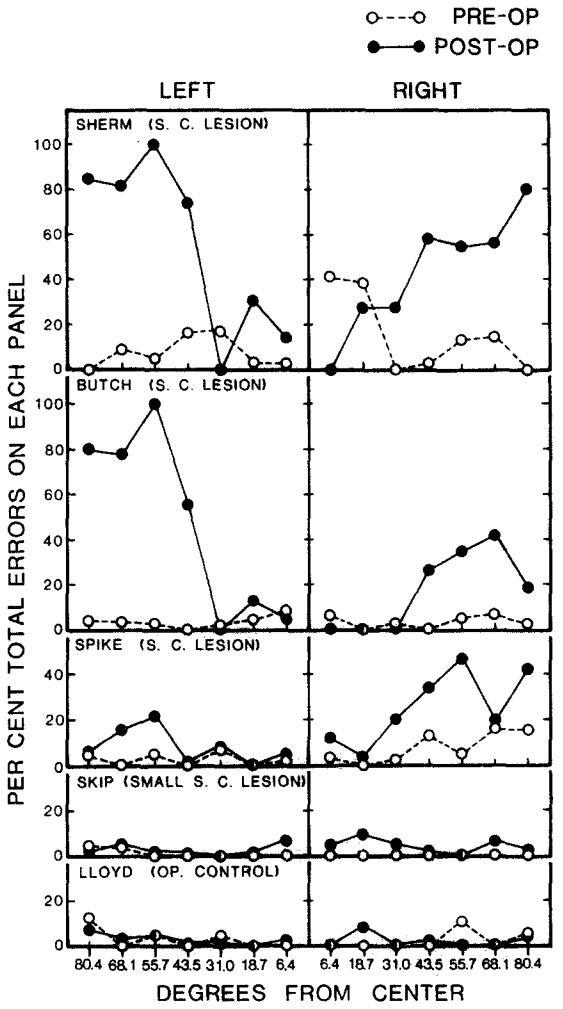

Fig. 4. Per cent total errors made by each monkey when light flashes were presented on each of the 14 panels before and after surgery.

The relationship between performance and spatial position of the light flashes is shown in more detail in Fig. 4. Following surgery, the experimental animals' errors became more frequent as the eccentricity of the light flashes increased. In addition, it may be seen in Fig. 4 that the relative severity of the deficits on the left and right sides differed for two of the experimental animals. Postoperatively, Butch made more errors when lights were flashed on the left side panels than it did when the lights were flashed on the right side panels, whereas Spike showed the opposite tendency.

In Fig. 5, errors on light trials are divided into localization errors (pressing the wrong side panel), detection errors (pressing the no-light panel), and response omissions. The experimental animals showed increases in the frequency of all 3 kinds of errors following surgery, although the relative proportions of the 3 kinds of errors varied among animals. It may also be noted that, following surgery, Spike and Sherm

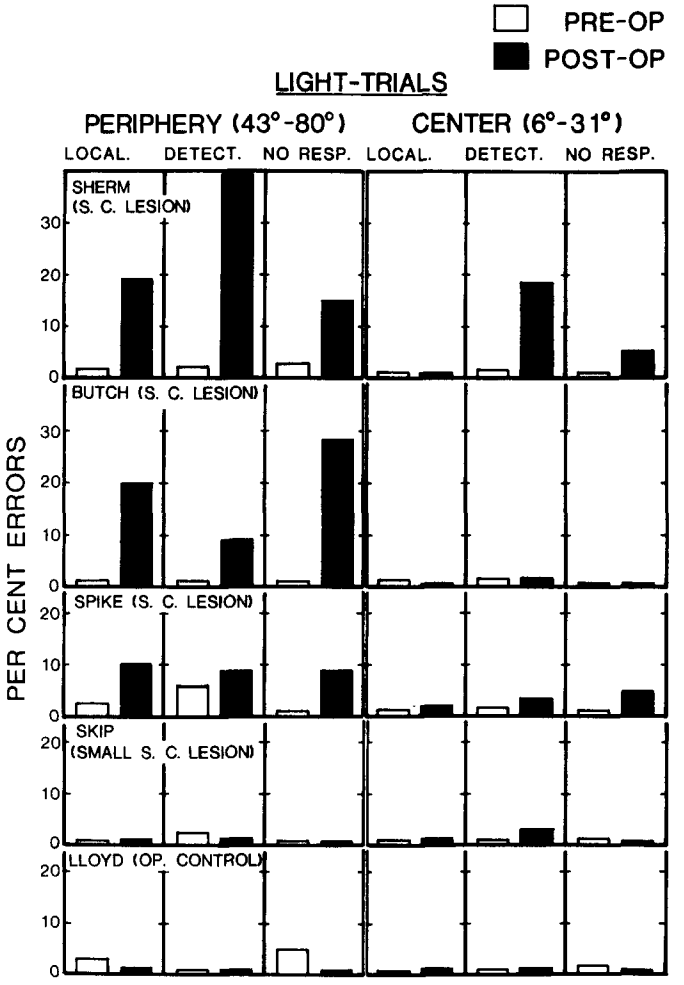

Fig. 5. Per cent localization errors, detection errors and response omissions made by each monkey on light trials before and after surgery.

showed increased detection errors and response omissions when lights were flashed on panels near the center, although these deficits were smaller than the comparable errors to lights flashed on the peripheral panels.

Further, analyses were made of localization errors of the experimental subjects following surgery in order to determine whether they were consistently made in a particular direction in relation to the panel on which light flashes were presented, i.e. to the left vs. right, or medially (toward the center) vs. laterally (away from the center) vs. the opposite side of the panel display. Preoperative scores were not included in this analysis, nor were the postoperative scores of the control animals, for in these cases there were too few localization errors for analysis. As seen in Table I, all the experimental subjects directed more of their localization errors medially than they did laterally or to the opposite side of the screen. Butch also mislocalized more to the right than to the left, while Spike showed the opposite tendency. These error tendencies are

TABLE I

Per cent of total localization errors in different directions from site of light flash presentation following superior colliculus lesions

Subject	*Direction*				
	Left	*Right*	*Medial*	*Lateral*	*Opp. side*
Sherm	48.1	51.9	76.9	3.8	19.2
Butch	22.4	77.6	71.4	4.0	24.5
Spike	98.0	2.0	74.0	26.0	0.0

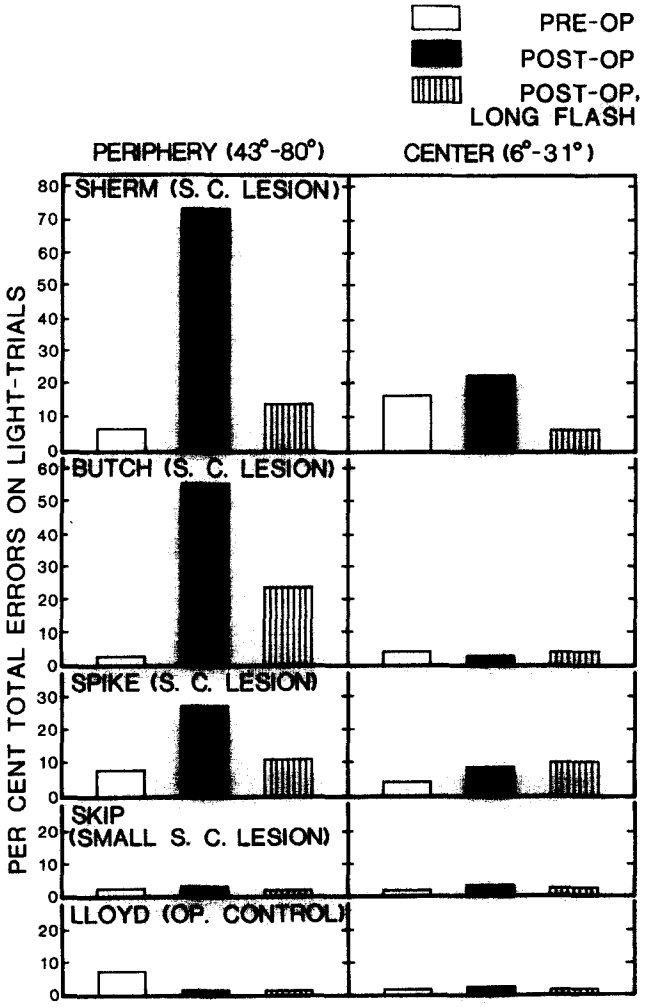

Fig. 6. Per cent total errors made by each monkey when brief light flashes were presented before and after surgery and when 1 sec light flashes were presented after surgery.

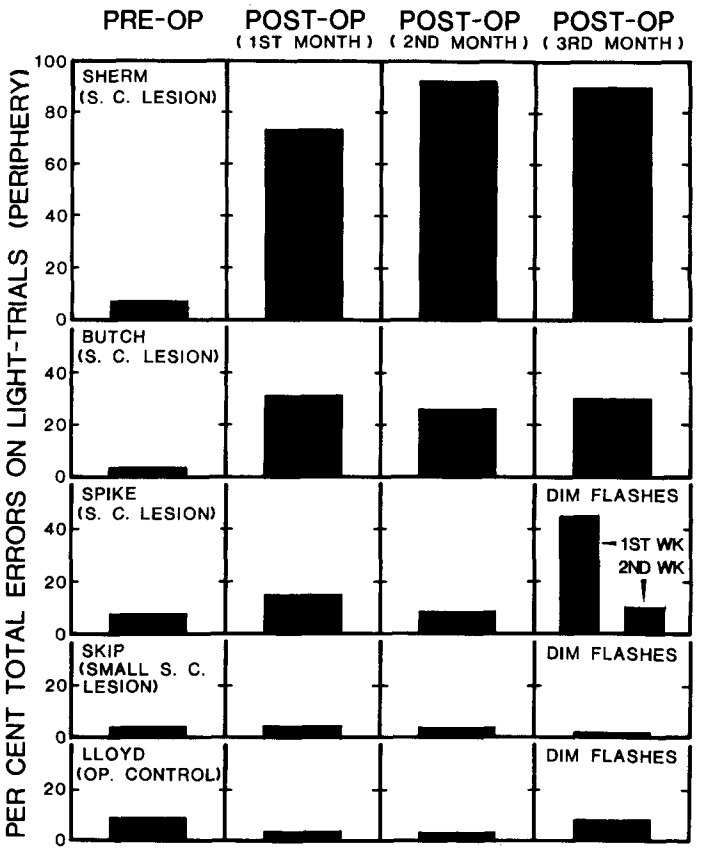

Fig. 7. Per cent total errors made by each monkey when eight flashes were presented on the peripheral side panels before surgery and during each of the 3 months of postoperative testing.

consistent with medially directed errors, for Butch and Spike made more errors (including localization errors) when light flashes were presented on the left and right sides, respectively, than they did when light flashes were presented on the opposite side, as mentioned previously.

Fig. 6 shows total errors committed in sessions in which long (1 sec) flashes were presented postoperatively, together with errors made before and after surgery when the standard 50 msec flashes were presented. It is clear that after surgery the experimental animals' performance improved markedly when long flashes were presented on the more peripherally located panels, although their performance was still slightly inferior to that of the control animals. Furthermore, this improvement was due to decreases in all 3 kinds of errors. The experimental animals' level of performance on no-light trials remained high in sessions when long flashes were presented.

As shown in Fig. 7, both Sherm and Butch continued to demonstrate performance deficits 3 months following surgery, and Sherm's continued to be selective to trials on which lights were flashed on the peripheral panels. On the other hand, Spike's

performance returned to its preoperative level after the first month of postoperative testing. Following the second month of testing after surgery, Spike, along with the two control animals, was tested with dim flashes presented only on the peripheral panels (see Fig. 7). Initially, Spike made many more errors than did either of the control animals, but, after one week of testing with dim flashes, its performance returned to control levels.

None of the monkeys shifted hand preference in the test situation following surgery. As in preoperative training, all the monkeys pressed the panels on one side of the metal screen with the ipsilateral hand.

DISCUSSION

The results of this experiment show that monkeys with large collicular lesions are impaired in localizing and perhaps in detecting brief light flashes in the periphery. Furthermore, the impairments were present for several months following surgery, long after ocular and visuomotor abnormalities were no longer seen. Thus, direct observation of visually guided behavior, which is often used to evaluate the effects of brain lesions on vision, is a relatively insensitive measure of the chronic effects of tectal lesions in monkeys.

The performance deficits of the experimental subjects cannot readily be attributed to a general disturbance in motivational or other factors that could affect responding in the test situation, since these deficits were largely limited to trials in which the peripheral side panels were briefly illuminated. Furthermore, the finding that the experimental animals' performance markedly improved when long flashes were presented, as Mackinnon et al.[9] had found, indicates that the deficits were not due or solely due to a motor impairment in directing the hand to the peripheral panels. When a long flash was presented, the monkeys had sufficient time to (and did) direct their gaze to it. The direction of the eyes could then have been used as a cue to guide correct responding. The 50 msec flashes, however, were too brief to permit their fixation; hence, in this condition, the only cue to the location of the light flash was its position on the retina. Thus, the experimental animals' tendency to press the wrong side-panels when light flashes were presented following surgery may be interpreted as a localization impairment. This interpretation is supported by the finding that these incorrect responses were not randomly directed; a very high proportion were directed to the panel adjacent (and medial) to the illuminated panel.

Postoperatively, the experimental animals also showed an increased frequency of pressing the no-light panel when light flashes were presented peripherally. It is likely that this change reflected their difficulty in detecting peripheral flashes rather than a general loss in control of the no-light panel response, since they did not consistently increase their responses to the no-light panel when light flashes were presented more centrally; nor were they consistently impaired in performing the no-light response on no-light trials. However, it is equally likely that the experimental subjects were uncertain about the flash location, and so pressed the no-light panel simply to terminate the trial.

Mohler and Wurtz[10] failed to find impairments in detecting a brief light flash after colliculus lesions in monkeys. However, their lesions were smaller and more superficial than the ones that produced deficits in our study. Furthermore, they did not present flashes beyond 30° from fixation, and in our study significant impairments only occurred with stimuli 43–80° from fixation. Thus, both the differences in lesion and in stimulus location could account for the differences between our results and those of Mohler and Wurtz.

It has recently been reported that, following collicular lesions, monkeys show a transient elevation in thresholds for detecting light[7]. However, since this threshold elevation apparently involved foveal vision and was no longer present 6 weeks following surgery, it does not seem to be closely related to the detection deficits that our monkeys may have displayed.

The experimental animals in the present study also omitted responses more frequently on light trials following surgery than they did prior to surgery. It is unlikely that this increase in response omissions was due to a failure to detect the light flashes. If this were the case, the experimental animals would have pressed the no-light panel, as they occasionally did on other light trials, rather than fail to respond. Moreover, the experimental animals' tendency to omit responses was not indiscriminate; these errors were largely limited to trials on which light flashes were peripherally presented. It is likely, therefore, that these response omissions were due to the animal's uncertainty about the location of the flashes, or possibly to lack of reinforcement as a result of frequent errors in localizing peripherally presented flashes.

Sherm and Butch, the two animals with the largest lesions, showed the most severe and long-lasting impairments. Spike, whose collicular lesion was more restricted than those of Sherm and Butch, showed less of a deficit in testing, while Skip, the monkey with the smallest collicular lesion, showed no detectable impairment, even with dim flashes. Thus, the severity of the deficits appears to be related to the size of the tectal lesions. Furthermore, Spike, the monkey that was more severely impaired on the right side of space than on the left, had more damage to the left colliculus than to the right. On the other hand, Butch, whose colliculus lesions appeared similar on the two sides, also showed left–right asymmetries in performance. It should also be noted that unilateral colliculus lesions in monkeys produce deficits similar to those reported here, but limited to the contralateral side of space[2]. Finally, there was no relationship between performance deficits and the relatively minor and variable damage to structures other than the superior colliculus in any of the experimental animals.

Our findings that tectal lesions impair accurate reaching toward peripherally presented light flashes suggests that the tectum is not only involved in motor control of foveation, but may also be part of a mechanism that controls reaching toward brief visual targets. Furthermore, it is possible that both the localization and detection deficits that are apparently present after tectal lesions may reflect a disturbance in selectively attending to peripheral stimuli, a function that has been attributed to the superior colliculus on the basis of electrophysiological findings[6]. Recently, Wurtz and Mohler[19] presented evidence that the attentional properties of collicular cells are

specifically related to eye movements and not to a manual response performed when the target appeared. However, the manual response that Wurtz and Mohler employed, unlike the one used in the present experiment, was not directed to the visual targets. The present findings imply that the superior colliculus may control selective attention to visual targets toward which various responses, including manual and ocular responses, are directed.

Although the experimental animals showed consistent deficits only when the stimuli were presented 43° or more from the center of gaze, their lesions destroyed the representation of all but approximately the central 5° of the visual field (according to Cynader and Berman's retinotopic map[4]). Why then was the performance of the experimental subjects usually normal when stimuli were presented between 5° and 43° from the center of gaze? One possibility is that other structures, such as striate cortex, may compensate for loss of the portion of the colliculus representing central vision but not far peripheral vision. A second possibility is that cells in the deeper layers of the intact anterior portion of the colliculus (which sometimes have large receptive fields extending beyond the central 5°) might have mediated the normal performance with stimuli presented between 5° and 43°. More complete superior colliculus lesions might resolve this question.

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