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IMPAIRMENTS IN VISUAL DISCRIMINATION PERFORMANCE AND GAZE SHIFTS IN MONKEYS WITH SUPERIOR COLLICULUS LESIONS

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SUMMARY

Eye movements of monkeys were recorded while they performed a visual discrimination task before and after superior colliculus (SC) lesions or control surgery. The monkeys with SC lesions were impaired in orienting their eyes toward the visual stimuli when they were presented eccentrically 15° to 32° from the center of the display screen, toward which their heads were directed. This impairment in shifting the gaze to eccentric stimuli may account for the concomitant deficit in discriminating between these eccentric stimuli. The eye movement deficit appears to depend on destruction of the deep as well as more superficial layers of the SC and may reflect a disturbance in visual–oculomotor coordination.

INTRODUCTION

Monkeys with superior colliculus (SC) lesions are impaired in discriminating between color cues spatially displaced from the response sites, but are unimpaired when the stimuli are located at the response sites[3,4]. This impairment may be due to a deficiency in shifting orientation from the response sites, which are foci of attention for normal monkeys[14,18,24,27], to the displaced stimuli. The present experiment was undertaken to test this hypothesis. Monkeys were tested for color discrimination performance while their eye position was recorded. The test was designed so that at the beginning of each trial the monkey would look toward the response sites and,

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consequently, the displaced stimuli would fall in the periphery of the visual field. Following SC lesions, according to the hypothesis, deficits in orienting the eyes toward the displaced stimuli would accompany deficits in discriminating between the stimuli. Furthermore, to determine whether the expected performance deficits are due to stimulus eccentricity or to stimulus–response (SR) separation, in some postoperative tests, the response sites were placed so they would fall in the periphery of the visual field at the beginning of each trial.

METHODS

Subjects

The subjects were 5 male and one female rhesus monkeys (*Macaca mulatta*) and one male cynomolgus monkey (*Macaca fascicularis*), weighing 4.0–8.2 kg. Two rhesus monkeys had previously been tested with drugs (experimental narcotic agonists and antagonists), but had received none for at least 6 months prior to this study; they were indistinguishable from the other subjects, which were experimentally naive. The monkeys were fed Purina monkey chow; their daily water intake was approximately 200 ml during training and testing. Isoniazid (25 mg) was given daily in tablet form as a prophylaxis against tuberculosis.

Behavioral apparatus

During training and testing, the monkeys were seated in a restraining chair within a chamber and faced a gray, metal display screen dimly illuminated (0.1 cd/sq. m) by an overhead light (see Fig. 1). The screen included 3 circular openings 1.3 cm in diameter, behind each of which a translucent plexiglas response panel was located, flush with the back surface of the screen. The response panels were hinged so that a microswitch closed when they were pressed. One of the panels, the observing response

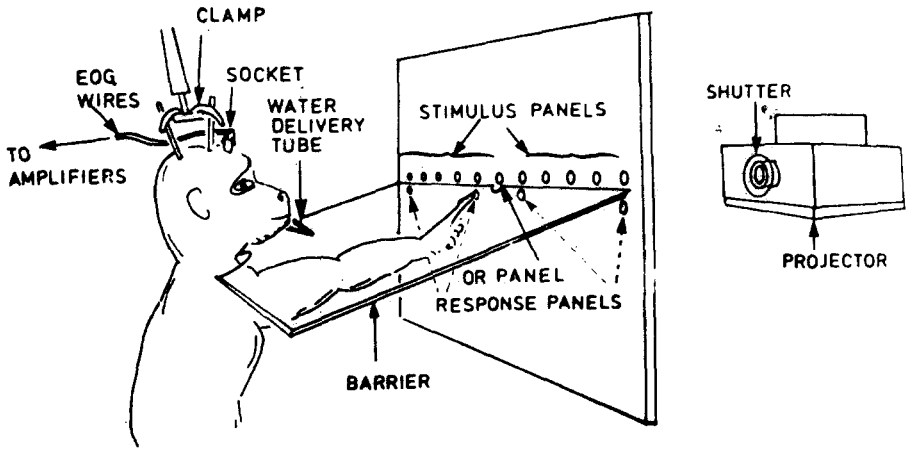

Fig. 1. Diagrammatic representations of the visual discrimination apparatus with the central choice–response panels or the peripheral choice–response panels. Within a single session the choice–response panels were either central or peripheral, but never both (see text for explanation).

(OR) panel, was in the center of the screen at eye level and was back-illuminated by a lamp so that a 0.1° spot appeared on the OR panel. The other two response panels, the choice–response panels, were located centrally 5.7° below and 8° to the left and right of the OR panel, or peripherally 5.7° below the 32° — eccentric stimulus panels (all distances between panels are center to center) (see Fig. 1). Five circular stimulus panels were located on each side of the display screen. These translucent plexiglas panels were located to the left and right of the OR panel and in the same horizontal plane with it (see Fig. 1). At the monkeys' viewing distance, (approximately 21 cm) the central choice–response panels were 8° from the OR panel, and the 5 pairs of stimulus panels were 8°, 15°, 21°, 27° and 32° from the OR panel. The 5 pairs of stimulus panels were separated from the closer of the two choice–response panels by 5°, 8°, 14°, 20° and 25°. Stimuli were back-projected on the panels by a Carousel projector. Water rewards, each 0.4 ml, were delivered through a tube by an electrically-activated valve. An opaque (masonite) or transparent (plexiglas) barrier was placed between the animal and the choice–response panels (see Fig. 1). However, since the two kinds of barriers had no differential effect on performance, they will not be mentioned in the results.

Eye movement recording apparatus

Eye movements and eye position were recorded from miniature Ag–AgCl EOG electrodes[1] implanted in the bones surrounding the orbit. Horizontal and vertical EOG potentials were led to differential DC amplifiers and recorded on a polygraph (Grass model P-7) together with electrical signals synchronized with stimuli and responses. During eye movement recordings, the monkey's head was immobilized and directed straight ahead, toward the OR panel, by securing bolts implanted in the skull to a clamp attached to a universal joint (see Fig. 1).

Discrimination training

The subjects were initially trained to press, for water reward, continuously-illuminated choice–response panels. They were then trained to press the OR panel to initiate a trial and then to press the choice–response panel on which white light was projected. After they achieved 90% correct in 100 consecutive trials in this light, no-light discrimination, skull bolts were implanted as described by others[21,28]. Two weeks later, the monkeys were retrained in the light, no-light discrimination, first with their heads free and then immobilized. They were then trained to discriminate between blue (S+) and pink (S—) light spots, each 3° in diameter, projected on the choice–response panels. The S+ was approximately 15 cd/sq. m in brightness. The S— was 3.3 cd/sq. m on one half of the trials and 33 cd/sq. m on the remainder. After achieving 45 correct responses in 50 consecutive trials, the size of both discriminative stimuli was reduced in steps to 0.2°; training was continued, if necessary, to re-achieve the criterion. Subsequently, the EOG electrodes were implanted, as described by others[21,28].

Discrimination testing with SR separation

Following a two-week recovery period, the monkeys were retrained in the light, no-light discrimination, and then in the color discrimination. Following completion of

the color discrimination, they started color discrimination with stimuli varying in eccentricity, while EOGs were recorded. In each session, the monkey's head was first immobilized and oriented directly toward the OR panel; it was then adapted to the illumination level inside the apparatus for at least 10 min. The experimenter calibrated EOGs by presenting bits of fruit or other small objects through each of a series of 2° holes in a screen in front of the animal and, while looking past the small objects at the monkey's eyes, recording on the EOG record the occasions when the monkey directed its eyes at the hole.

Because EOG voltages occasionally drifted during testing, center fixation was determined by EOG voltages when the monkey pressed the OR panel, on the assumption that the monkey was fixating the spot on the OR panel at this time. This method of determining center fixation is probably accurate to 2–3°. Two other sources of error in estimating eye position were: (a) the calibration procedure, which is accurate to about 2°; and (b) the error of measurement in recording with these electrodes, approximately 2° vertical and 1.5° horizontal[30]. Orientation to a discriminative stimulus was defined as directing the gaze $\pm$ 9° or less from it, a value chosen on the basis of pilot data indicating normal monkeys could accurately discriminate between the stimuli only if they fixated within 9° of the S+. A fixation was defined as a period of at least 100 msec during which eye position, as judged by inspecting EOG records, did not change by more than 0.5°.

At the beginning of each discrimination trial, the monkeys were required to respond to the light spot on the OR panel, in order to present the S+ and S—, which were the same color, size and brightness as they were in the last stage of color discrimination learning. On each trial the discriminative stimuli were projected on a pair of stimulus panels equally eccentric from the screen center. The monkey received reinforcement only when it pressed the choice–response panel closer to the S+ within 10 sec of the onset of the discriminative stimuli. If the monkey pressed the incorrect choice–response panel, or the center panel, or if it failed to respond within 10 sec of the onset of the discriminative stimuli, an error was recorded, the stimuli were turned off, and an intertrial interval (ITI), randomly varied between 5 and 10 sec, ensued. A response during the ITI delayed the onset of the next trial by another 5–10 sec. In each block of 5 trials the stimuli were presented in a random order on each of the 5 pairs of stimulus panels. In each block of 10 trials the S+ appeared equally often on the left and right stimulus panels in a pseudo-random order. A session consisted of 100–133 consecutive trials; if a monkey performed 20–99 trials in a day, its data were combined with data from the following day to comprise a single session. Data from a session were discarded if : (a) fewer than 20 trials were performed; or (b) performance with stimuli of minimal eccentricity was less than 80%. Failure to attain this criterion was followed by retraining with minimal-eccentricity stimuli until the monkey performed 90% correct in 50 consecutive trials.

Preoperative testing was terminated when subjects completed at least 8 sessions, the last 3 of which they performed better than 80% correct with minimally-eccentric stimuli and at least 75% correct with stimuli presented at each of the other eccentricities. Five of the 7 monkeys met this criterion in 8 sessions; the other two required 11 and 17 sessions.

Superior colliculus and control surgery

Surgery was performed 1–2 weeks after completing discrimination testing. Anesthesia was achieved with ketamine HCl (initial dose: 25 mg/kg; supplemental doses: 15 mg/kg), administered i.m.; penicillin (600,000 units) was also injected i.m. just prior to surgery. Dexamethasone (4 mg doses) was injected i.m. 18 h and again 1 h before surgery to reduce brain edema. Prior to surgery, 30% urea (1 g/kg) in invert sugar was injected i.v. to shrink the brain. Using sterile procedures, the scalp and fascia were incised and retracted, a 2 cm square bone flap was removed, and the dura was cut and retracted. The bone flap was centered in the skull over the parietal lobe; thus, the edges of the bone defect were about 1 cm from the skull bolts. The splenium was retracted anteriorly and upward to expose the caudal portion of the SC, which was then aspirated bilaterally in the experimental animals with the aid of a microscope. The SC was only exposed for 30–40 min in the two control animals. The wounds were then closed in anatomical layers with silk sutures.

Postoperative testing

During the 10–20 day recovery period following surgery, the monkey's activity level and reactions to visual stimuli, clicks, and tactile stimulation of the trunk and limbs were observed. The speed and accuracy with which they reached for food were also assessed. Eye movements and pupillary responses were also examined.

Following the recovery period, the monkeys were retrained to discriminate between the large (3°) and finally between the smallest (0.2°) color stimuli by the same procedures used preoperatively. Subsequently, discrimination tests were administered for 8 sessions by the same procedures used before. Following the completion of discrimination testing with the central choice–response panels, the monkeys were trained to press the peripheral choice–response panels, in a light, no-light test, until they performed 90% correct in 100 consecutive trials.

The monkeys were then tested for color discrimination performance with the peripheral panels in two sessions and with the central response panels in two sessions in an ABBA order. If subjects performed below 80% with 8°-eccentric cues in sessions with central response panels, their data were discarded; they were then retrained with only 8°-eccentric cues, as in prior tests. If subjects performed below 80% with *both* 8°- and 32°-eccentric cues in sessions with peripheral response panels, their data were also discarded, and testing was continued with stimuli presented at all eccentricities until they performed better than 80% correct with *either* the 8°- or the 32°-eccentric stimuli. In all other respects, these tests were conducted as prior tests were.

Histological analysis

When testing was completed, the subjects were deeply anesthetized with ketamine HCl and then perfused through the heart with 0.9% NaCl followed by 10% formalin. The brains were blocked in stereotaxic planes, hardened in formalin and then in sucrose formalin. Subsequently, they were embedded in albumin-gel and sectioned at 40 μm while frozen. Every fifth section was stained with thionin.

RESULTS

Preoperative results

The monkeys required 878 trials on the average to acquire the color discrimination. After EOG electrodes were implanted, the monkeys relearned the color discrimination in 195 trials. Preoperative training and testing required 3.5–8 months.

In the first discrimination test session, all the monkeys showed steep gradients of performance as stimulus eccentricity increased (see Fig. 2). Consequently, stimulus eccentricity significantly affected the performance scores, as shown by a Friedman two-way analysis of variance ($X = 17.5$, $n = 7$, $P < 0.01$).

In the last 3 preoperative test sessions, all subjects improved their discrimination performance with stimuli presented at 15°–32° eccentricity (see Fig. 2); nevertheless, they still showed significant performance decrements as stimulus eccentricity increased ($X = 15.2$, $n = 7$, $P < 0.01$).

The monkey's performance in preoperative discrimination tests varied consider-

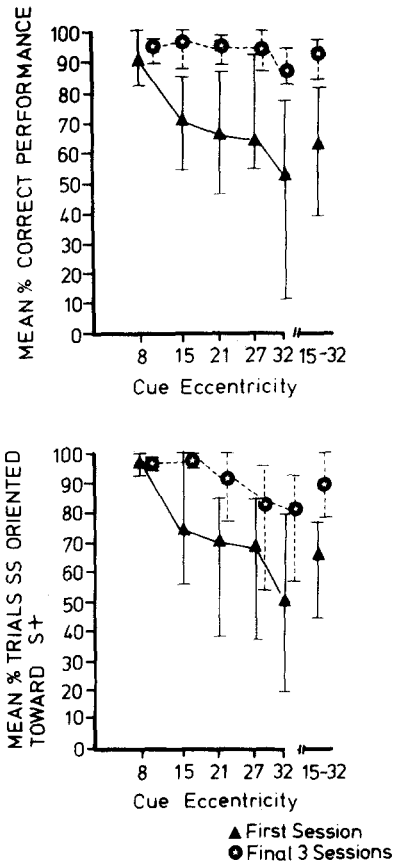

Fig. 2. Mean preoperative discrimination performance and S+ orientation scores as a function of cue eccentricity. Vertical bars represent ranges of individual scores. 15–32 represents the overall mean for cue eccentricities of 15°–32° inclusive.

TABLE I

Per cent correct performance when orienting to S+, S—, or to neither S

Overall means and ranges for trials involving 15°–32° cue eccentricity. Note: orienting to S+: trials in which subject oriented at least once to S+. Orienting to S—: trials in which subject oriented at least once to S—, but not to S+. % correct: numbers in parentheses are ranges of individual scores. No. Ss: proportion of subjects with 10 or more trials in that fixation condition. Proportion Ss above or below chance: $P < 0.05$, binomial distribution.

Session	*Group*	*Stimulus orientation*	*% Correct*	*No. Ss*	*Proport. Ss above chance*	*Proport. Ss below chance*
Preop.: 1st session	all Ss	S+	93.3 (81–100)	7/7	7/7	0/7
		S—	3.5 (0–17)	6/7	0/6	6/6
		neither S	67.3 (45–80)	3/7	2/3	0/3
Preop.: last 3 sessions	all Ss	S+	97.3 (93–99)	7/7	7/7	0/7
		S—	34.6 (6–62)	7/7	0/7	3/7
		neither S	73.7 (60–90)	3/7	2/3	0/3
Postop.: central response panels	SC	S+	93 (88–100)	3/5	3/3	0/3
		S—	54 (47–61)	2/5	0/2	0/2
		neither S	71.4 (52–81)	5/5	4/5	0/5
	Op. C.	S+	94.5 (91–98)	2/2	2/2	0/2
		S—	40 (40–40)	1/2	0/1	0/1
		neither S	74 (74–74)	1/2	1/1	0/1
Postop.: peripheral response panels	SC	S+	94.3 (88–100)	3/5	3/3	0/3
		S—	39 (23–55)	2/5	0/2	1/2
		neither S	69 (62–74)	5/5	5/5	0/5
	Op. C.	S+	98 (98–98)	2/2	2/2	0/2
		S—	30 (30–30)	1/2	0/1	0/1
		neither S	— —	0/2	—	—

ably depending upon their eye position (see Table I). Their performance was consistently high and above chance when they oriented to the S+, but when they oriented only to the S—, their performance was consistently low and significantly below chance. When the subjects oriented to neither stimulus, they performed on the average above 50%, but considerably lower than when they oriented to the S+. Since the monkeys oriented to neither cue infrequently, approximately half of their performance scores in this condition were not significantly above chance. Furthermore, the variations in performance with orienting behavior, were independent of stimulus eccentricity and the amount of testing, except that the monkeys significantly increased their performance scores from the first to last 3 sessions, when they oriented only to the S—, as shown by the results of a Wilcoxon test ($T = 0$, $n = 6$, $P < 0.05$).

Since the monkeys consistently performed the discrimination well only when they oriented to the S+, the percentage of trials in which they oriented to the S+ (referred to as 'S+ orientation scores') was further analyzed. In the first test session (see Fig. 2) the monkeys oriented frequently to the S+ when it was presented centrally, but oriented significantly less to the S+ as its eccentricity increased, according to a Friedman two-way analysis of variance ($X = 19.09$, $n = 7$, $P < 0.001$). S+

orientation scores also decreased significantly with increasing stimulus eccentricity in the last 3 test sessions $X = 18.20$, $n = 7$, $P < 0.01$); however, these decrements were not as pronounced as those found in the first session (see Figure 2).

Postoperative results

Postoperative testing required 1–4.5 months to complete. All of the colliculus-lesioned monkeys, except Spa., and one control monkey (Diz) were sluggish and showed little spontaneous activity for 1–3 weeks following surgery. All the monkeys with SC lesions oriented normally to stimuli in different quadrants of the visual field and followed moving objects normally, but showed reductions in spontaneous saccades. In addition, one of the SC monkeys (Bur.) showed no downward gaze throughout postoperative testing. All the colliculus-lesioned monkeys, when unrestrained, reached accurately for objects and, except for Bur., fixated them while reaching. During EOG calibration tests, (during which the monkeys' heads were immobilized) the SC monkeys fixated objects presented peripherally, especially beyond 20° eccentricity, less often than the control animals did. This deficit persisted throughout postoperative testing (2–4.5 mos.). However, the SC monkeys, like the control monkeys, reached accurately for peripherally-presented objects during EOG calibrations.

The SC monkeys, compared to the controls, performed worse and oriented less to the S+ when it was presented 15°–32° but not when it was presented 8° eccentrically in the first test series. Similarly, when the response panels were centrally located in the second test series, the SC monkeys performed significantly worse with stimuli presented eccentrically and showed significantly lower S+-fixation scores when stimuli were presented 15°–32° eccentrically relative to the control monkeys. In the same test sessions, the SC monkeys' performance and S+-fixations were not impaired when the stimuli were presented at 8° eccentricity (see Fig. 3). The same pattern of results was obtained when the response panels were located peripherally in the second test series. Relative to the control monkeys, those with SC lesions were reliably impaired in orienting to the S+ and in discriminating between the stimuli when they were presented more than 8° eccentrically ($U=0$, $n_1/n_2=2/5$, $P=0.047$, for all comparisons) (see Fig. 3).

To determine where the SC monkeys directed their gaze when the stimuli were eccentrically presented, each subject's distribution of fixations on the display screen during trials was calculated. Only deviations of gaze in the horizontal plane were analyzed, since 95% of the fixations were directed no more than 8° above or below the stimulus panels. The SC monkeys fixated primarily the center of the display screen, regardless of the response-panel location and stimulus eccentricity, whereas the control monkeys looked more to the periphery as stimulus eccentricity increased, especially when the choice–response panels were peripherally located (see Fig. 4).

The interdecile range of each subject's fixation distribution was calculated to measure the degree to which fixations were dispersed across the display screen. When the choice–response panels were central, the control animals' interdecile ranges, unlike those of the S.C. animals, increased as stimulus eccentricity increased, so that the

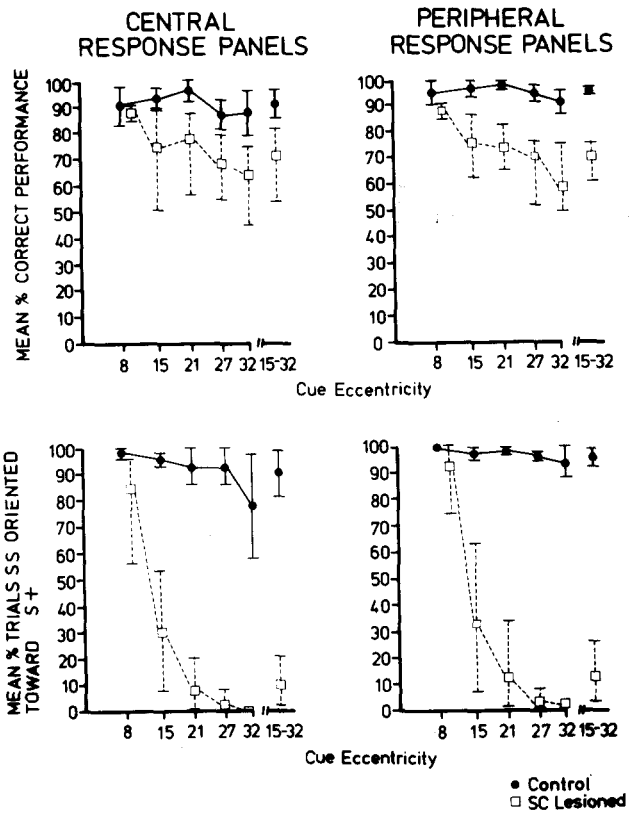

Fig. 3. Mean postoperative discrimination performance and S+ orientation scores as a function of cue eccentricity and response panel location for subjects with superior colliculus (SC) lesions and operated controls. Vertical bars represent ranges of individual scores. 15–32 represents the overall mean for cue eccentricities of 15°–32° inclusive.

interdecile ranges of the two groups did not overlap beyond 8° stimulus eccentricity (see Table II). When the choice–response panels were peripheral, the control monkeys' interdecile ranges were on the average larger than they were when the choice–response panels were central, reflecting their tendency to fixate in the periphery more often in the former condition than in the latter. In contrast, the position of the response panels did not significantly affect the interdecile ranges of the SC monkeys. Consequently, the interdecile ranges of all the SC monkeys were lower than those of the control monkeys when the choice–response panels were located in the periphery (see Table II).

Whereas SC monkeys were deficient in orienting to the S+ when it was eccentrically presented, like the operated controls, they continued to perform well when they did orient to this stimulus (see Table I). As in preoperative tests, all subjects performed less well when they oriented to neither stimulus than they did when they oriented to the S+ and worst when they oriented only to the S—, irrespective of the location of the choice–response panels or of the stimuli.

The SC monkeys' fixation durations, response latencies, and number of fixations

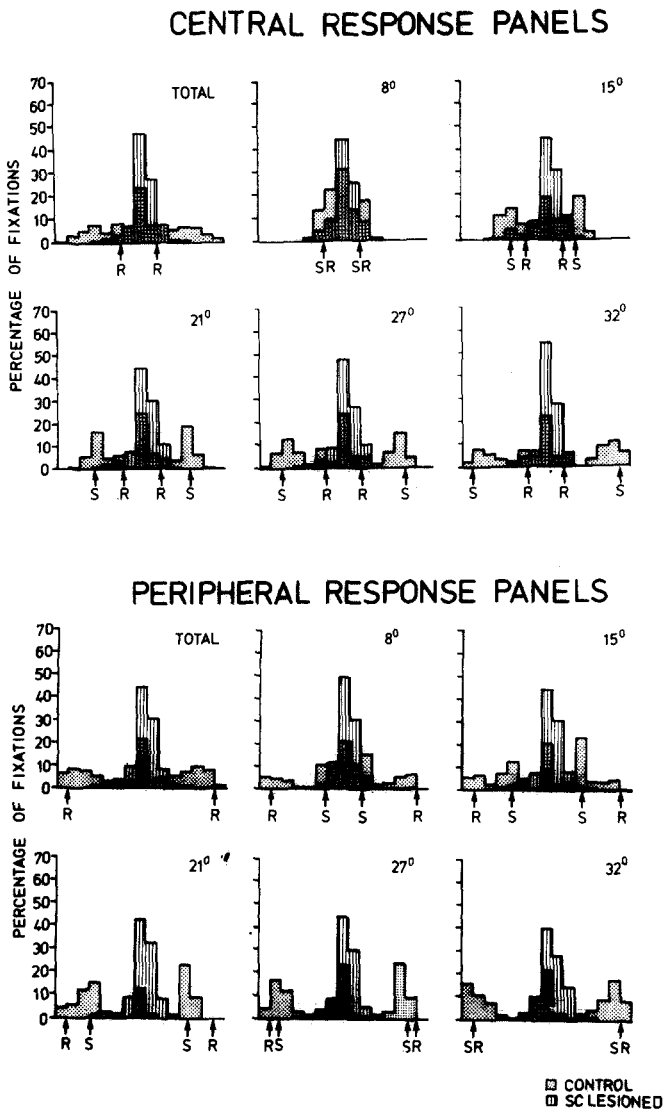

Fig. 4. Mean distributions of fixations on the display screen during the second series of postoperative discrimination tests. Bars: operated controls. Stippled: SC-lesioned. Stippled bars: the overlap of fixations of the monkeys with SC lesions and operated controls. S, locus of stimulus presentation; R, locus of choice-response panel.

per trial in the postoperative discrimination tests were not significantly different from those of the control monkeys, whether they were calculated as absolute measures or relative to preoperative scores.

Histological findings

The brains of all experimental animals showed extensive or total destruction of the caudal one-third to one-half of the SC, representing the visual fields from 10°

TABLE II

Interdecile ranges of fixation distributions

	Stimulus eccentricity	*S.C.*		*Control*	
		Mean	*Range*	*Mean*	*Range*
Central response panels	8	8	5–15	17.5	15–20
	15	7	5–15	35	35–35
	21	9	5–20	45	40–50
	27	9	5–15	47.5	45–50
	32	10	5–15	56.5	50–63
Peripheral response panels	8	10	5–20	45	30–60
	15	13	5–30	47.5	40–55
	21	10	5–20	50	45–55
	27	10	5–20	57.5	55–60
	32	10	5–20	63	63–63

eccentricity to the far periphery. The anterior SC, representing the central visual field, was severely damaged in two experimental monkeys (Bur. and Spa.) and slightly damaged in the others (Lev., All. and Vid.). The pretectal area was variably damaged in 4 of the 5 experimental subjects (All., Lev., Bur. and Spa.). The dorsal portion of the central gray and tegmentum was slightly damaged in all experimental animals. Some of the lesions also slightly damaged the dorsal inferior colliculus, nucleus limitans, medial geniculate nucleus, habenula and nucleus medialis dorsalis. The splenium of

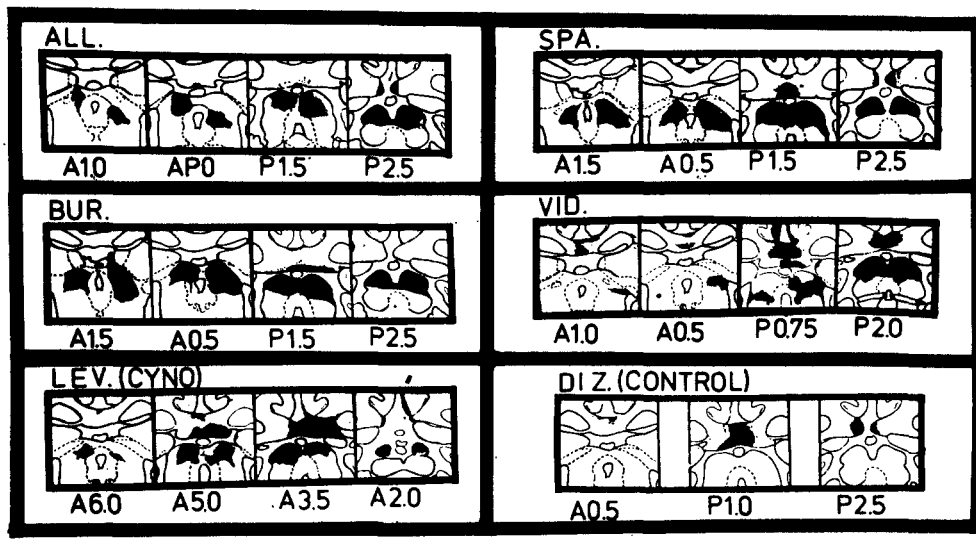

Fig. 5. Representative cross-sections through the SC lesions of the 5 experimental subjects. The numbers under each cross-section refer to stereotaxic coordinates anterior (A) and posterior (P) to ear-bar zero. The coordinates for Lev. are numerically different because this subject was a cynomolgus monkey. Black areas indicate regions of complete cell loss. Stippled areas indicate regions of partial cell loss and gliosis.

the corpus callosum, which was retracted during surgery, was damaged in all experimental animals as well as in one control animal, Diz. No brain damage was found in the other operated control subject, Fur.

The experimental subjects were ranked for extent of damage to the SC and for the severity of performance and orientation deficits; none of the correlation coefficients was statistically significant. No relationships between damage to other structures and performance or S+ orientation were found.

DISCUSSION

It is not clear whether the discrimination performance decrements found prior to surgery were due to stimulus eccentricity or to SR separation, for the control animals performed at high levels in postoperative tests irrespective of the locations of the response panels and stimuli, perhaps because of the extensive discrimination training they had received before and after surgery[13].

However, the performance deficits of the SC monkeys were clearly due to stimulus eccentricity rather than SR separation, suggesting that the SC monkeys were impaired in shifting their gaze from the center of the display screen to eccentrically presented stimuli. This interpretation was amply confirmed by analyses of the SC monkeys' eye movements while they performed the discrimination task. Several additional findings suggest that the SC monkeys' severe deficit in orienting toward the S+ may account for their discrimination deficit when the stimuli were presented eccentrically. The control monkeys performed well only when they oriented to the S+, a finding also obtained in similar investigations of monkeys[17,23]. Likewise, on the few trials in which the SC monkeys did orient to the eccentrically-presented S+, they also performed better than 90% correct. In contrast, when the monkeys oriented only to the S—, they performed consistently below chance, suggesting that they had mistaken it for the S+. Furthermore, S+ orientation scores, like discrimination performance, increased as testing proceeded and decreased as the stimuli were presented more eccentrically. The finding of occasional above-chance performance in the trials when the monkeys oriented to neither stimulus suggests that they may have used a non-visual cue to guide their responses in these trials. However, this interpretation is unlikely, for when some of the subjects were tested for discrimination performance with no visual stimuli, they performed no better than chance or stopped responding. The monkeys may have occasionally performed slightly above chance when they oriented to neither stimulus because they learned to use their near-peripheral vision (within 30° of the fovea) to identify the S+, even though pilot tests conducted prior to the experiment provided no evidence for this.

Why were the monkeys with SC lesions severely impaired in shifting their gaze outside the center of the display screen? A motor impairment or paralysis of gaze is unlikely, for during calibration tests the SC monkeys could be induced to direct their gaze to small objects located more than 20° from center fixation, although these eccentric gaze shifts were rare. Furthermore, total colliculectomy apparently does not impair reflexly-induced saccades in monkeys[19].

Alternatively, the SC monkeys' deficits in shifting their gaze may have been due to an impairment in detecting peripheral stimuli to which saccades are directed. This interpretation is supported by the finding that monkeys with SC lesions are impaired in detecting brief light flashes presented peripherally[5]. However, the finding that most of the monkeys with SC lesions performed significantly above chance when they failed to orient to either stimulus suggests that they could use their peripheral vision to detect and identify the discriminative stimuli.

The deficit in shifting the gaze may have been due to a disorder in shifting attention, which may account for visual deficits in cats[26] and rats[9–11] with SC lesions. Evidence for a shift of attention to an extrafoveal stimulus to which the eyes move is provided by the findings that both detection[25] and identification[2,7] of peripheral visual stimuli are enhanced prior to saccades directed to these stimuli.

A selective impairment in visual–oculomotor coordination might also be responsible for the eye-movement deficits found after SC lesions, as suggested by the finding that the SC monkeys often failed to fixate bits of food when they were presented far from the center of the display screen during EOG calibration, even though all of them quickly and accurately reached for them. Thus, the monkeys with SC lesions apparently detected and attended to these objects without fixating them by saccadic eye movements, as they did before surgery. Thus, after SC lesions the monkeys may have been impaired in stimulus-controlled saccading, which might account for their deficiency in orienting to peripherally-presented stimuli in discrimination testing. However, it should be noted that impaired stimulus-controlled saccading would not account for the finding that SC monkeys are deficient in detecting and localizing peripheral flashes too brief to fixate[5].

Participation of the SC in stimulus-controlled saccading is also suggested by the findings that some neurons in the intermediate layers of the monkey's SC discharge either to a visual stimulus in its receptive field or when the eyes saccade to the receptive field[28]; other intermediate layer neurons discharge only when a stimulus falls in the receptive field *and* the eyes saccade to that stimulus[15].

Our finding that SC lesions alone produce severe deficits in shifting the gaze to the periphery has not been reported in two other investigations involving eye movement recording in monkeys. In those studies, SC lesions resulted only in increased saccade latencies to visual targets[29] and increased frequencies of undersaccading[16]. The discrepancy between these findings and ours is not likely to be due to degree of extracollicular damage. The eye movement deficits we found did not depend on pretectal damage. Moreover, other extracollicular structures were only slightly damaged in the present study. Since our lesions and those made in prior investigations[16,29] included the posterior portion of the SC, which represents the periphery of the visual field, it is unlikely that differences in retinotopic locus of the lesions could account for the discrepancy of results. Furthermore, the retinotopic locus of our lesions was probably not responsible for the *lack* of impairment in gaze shifts 8° from the center of the display screen, for some of the lesions extended into the anterior portion of the SC, representing central vision[8].

However, the discrepancy between the effects of SC lesions in the present and

prior studies may be due to differences in lesion depth. Our lesions, unlike those reported by Wurtz and Goldberg[29] and Mohler and Wurtz[16], consistently included the deepest layers of the SC. The suggestion that the deep SC layers play a critical part in the collicular control of saccades is consistent with the finding that the threshold to elicit a saccade by electrical stimulation decreases abruptly — from 200 μA to 20 μA — when the stimulating electrode advances into the deep fiber layer of the SC[20]. Moreover, a recent study also found reduced saccade amplitude after SC lesions that included the deep layers[22]. Lesions of the deep laminae may be critical, for these layers may receive visual information from dorsal laminae and provide the efferent control of saccades.

This hypothesis has been questioned by the finding that latencies between initiation of unit discharges and saccades shorten, the more dorsally eye movement-related neurons are located[15]. These and other findings prompted Mohler and Wurtz to suggest that the collicular control of saccades may originate from neurons in the upper intermediate layers, which may receive afferents from both deeper and more superficially-located neurons.

Nevertheless, units in the deep layers may play a critical role in the collicular control of saccades. Thus superficial cells, driven by visual inputs, may activate deep neurons, which in turn may facilitate the activity of neurons in the intermediate and superficial layers. These neurons might then facilitate the deep neurons, thus forming a feedback loop in which the activity of cells in the loop or the number of active cells would gradually increase to the point where the SC cells would activate neurons in the brain stem tegmentum. According to this model, collicular cells directly influencing saccades via their brain stem connections[6,12] could be located either in the intermediate, the deep, or in both sets of laminae. The notion that repeated activity in an intracollicular loop precedes saccade generation is consistent with findings that: (a) there is a long latency (about 200 msec) between the increased discharge of some collicular cells and saccade initiation[28]; and (b) the level of activity of many saccade-related SC cells gradually increases and peaks at the time a saccade is generated or during the saccade[15]. The hypothesis that the deep layers of the SC are necessary for eye movements could be tested by observing eye movements after these layers are selectively destroyed.

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REFERENCES

1 Bond, W. H. and Ho, P., Solid miniature silver–silver chloride electrodes for chronic implantation, *Electroenceph. clin. Neurophysiol.*, 28 (1970) 206–208.
2 Bryden, M. P., The role of post-exposural eye movements in tachistoscopic perception, *Canad. J. Psychol.*, 15 (1961) 220–225.
3 Butter, C. M., Effect of superior colliculus, striate or prestriate lesions on visual sampling in rhesus monkeys, *J. comp. physiol. Psychol.*, 87 (1974) 905–917.
4 Butter, C. M., Contrasting effects of lateral striate and superior colliculus lesions on visual discrimination performance in rhesus monkeys, *J. comp. physiol. Psychol.*, 93 (1979) 522–537.
5 Butter, C. M., Weinstein, C., Bender, D. and Gross, C. G., Localization and detection of visual stimuli in monkeys with superior colliculus lesions, *Brain Research*, 156 (1978) 33–49.
6 Buttner-Ennever, J. A. and Henn, V., An autoradiographic study of the pathways from the pontine reticular formation involved in horizontal eye movements, *Brain Research*, 108 (1976) 155–164.
7 Crovits, H. F. and Daves, W., Tendencies to eye movements and perceptual accuracy, *J. exp. Psychol.*, 63 (1962) 495–498.
8 Cynader, M. and Berman, N., Receptive field organization of monkey superior colliculus, *J. Neurophysiol.*, 35 (1972) 187–201.
9 Goodale, M. A. and Lister, T. M., Attention to novel stimuli in rats with lesions of the superior colliculus, *Brain Research*, 66 (1974) 361–362.
10 Goodale, M. A., Milner, A. D. and Rose, J. E. V., Susceptibility to startle during ongoing behavior following collicular lesions in the rat, *Neurosci. Lett.*, 1 (1975) 333–337.
11 Goodale, M. and Murison, R., The effects of lesions of the superior colliculus on locomotor orientation and the orienting reflex in the rat, *Brain Research*, 88 (1975) 243–261.
12 Harting, J. K., Descending pathways from the superior colliculus: an autoradiographic analysis in the rhesus monkey *(Macaca mulatta)*, *J. comp. Neurol.*, 173 (1977) 583–612.
13 Kurtz, D., Leiby, C. C. and Butter, C. M., Effects of S-R separation, cue eccentricity and stimulus duration on discrimination performance in monkeys with superior colliculus lesions, unpublished experiment.
14 Meyer, D. R., Treichler, F. R. and Meyer, P. M., Discrete-trial training techniques and stimulus variables. In A. M. Schrier, H. F. Harlow and F. Stollnitz (Eds.), *Behavior of Non-human Primates, Vol. I.*, Academic Press, New York, 1965.
15 Mohler, C. W. and Wurtz, R. H., Organization of monkey superior colliculus: intermediate layer cells discharging before eye movements, *J. Neurophysiol.*, 39 (1976) 722–744.
16 Mohler, C. W. and Wurtz, R. H., Role of striate cortex and superior colliculus in visual guidance of saccadic eye movements in monkeys, *J. Neurophysiol.*, 40 (1977) 74–94.
17 Oscar-Berman, M., Heywood, S. P. and Gross, C. G., Eye orientation during visual discrimination learning by monkeys, *Neuropsychology*, 9 (1971) 351–358.
18 Ottesen, M. I., Sheridan, C. L. and Meyer, D. R., Effect of stimulus–response isolation on primate pattern discrimination learning, *J. comp. physiol. Psychol.*, 55 (1962) 935–938.
19 Pasik, P. and Pasik, T., Oculomotor functions with lesions of the cerebrum and superior colliculi. In M. Bender (Ed.), *The Oculomotor System*, Harper and Row, New York, 1964.
20 Robinson, D. A., Eye-movements evoked by collicular stimulation in the alert monkey, *Vision Res.*, 12 (1972) 1795–1808.
21 Schiller, P. and Stryker, M., Single-unit recording and stimulation in superior colliculus of alert rhesus monkeys, *J. Neurophysiol.*, 35 (1972) 915–924.
22 Schiller, P. H., True, S. D. and Conway, J. L., Effects of frontal eye field and superior colliculus ablations on eye movements, *Science*, 206 (1979) 590–592.
23 Schrier, A. M. and Wing, T. G., Eye movements of monkeys during brightness discrimination and discrimination reversal, *Anim. Learn. Behav.*, 1 (1973) 145–150.
24 Schuck, J., Pattern discrimination and visual sampling by the monkey, *J. comp. physiol. Psychol.*, 53 (1960) 251–255.
25 Singer, W., Zihl, J. and Poppel, E., Subcortical control of visual thresholds in humans: evidence for modality specific and retinotopically organized mechanisms of selective attention, *Exp. Brain Res.*, 29 (1977) 173–190.
26 Sprague, J. and Meikle, T., The role of the superior colliculus in visually guided behavior, *Exp. Neurol.*, 11 (1965) 115–146.
27 Stollnitz, F., Spatial gradient of performance of monkeys *(Macaca mulatta)* on a single color-pattern discrimination problem, *Amer. Psychol.*, (Abstr.), 18 (1963) 473.

28 Wurtz, R. and Goldberg, M., Activity of superior colliculus in behaving monkey. III. Cells discharging before eye-movements, *J. Neurophysiol.*, 35 (1972) 575–586.
29 Wurtz, R. and Goldberg, M., Activity of superior colliculus in behaving monkey. IV. Effects of lesions on eye-movements, *J. Neurophysiol.*, 35 (1972) 587–596.
30 Young, L. R. and Sheene, D., Survey of Eye Movement Recording Methods, *Behavior Research Methods and Instrumentation*, 7 (1975) 397–429.