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Deficits in Eye Position Following Ablation of Monkey Superior Colliculus, Pretectum, and Posterior-Medial Thalamus

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SUMMARY AND CONCLUSIONS

1. Previous investigations of the effects of ablations of the primate superior colliculus have produced conflicting results: several studies have reported deficits in the accuracy of visually guided saccades while others have reported primarily deficits in the latency of visually guided saccades. The present experiments attempted to resolve this conflict by examining the effects of complete lesions of the superior colliculus on the initiation and accuracy of saccades to central and peripheral visual targets.

2. Four rhesus monkeys were first trained to make saccades to visual stimuli and were then given large unilateral surgical ablations of the superior colliculus. Histological reconstructions of the brain stems of these monkeys showed that colliculus removal in all four monkeys was nearly complete and included the superficial and deep layers, representing the central and peripheral portions of the visual field. However, in three monkeys the lesions also included extensive regions anterior to the colliculus in the pretectum and in the posterior-medial thalamus.

3. After the ablations the three monkeys with more extensive lesions showed a long-lasting deficit in the ability to make accurate saccades from a central fixation point to eccentric visual targets on the side contralateral to the ablation. Saccades to targets greater than about 20° from the central fixation point consistently fell short and were not corrected by subsequent saccades. In the fourth monkey, with the ablation nearly restricted to the colliculus, deficits in the accuracy of initial saccades to visual targets on the side contralateral to the lesion were relatively small and were readily corrected by second saccades.

4. Monkeys were then tested to determine whether the deficits in the accuracy of saccades were related to the amplitude of the saccades or to saccades made to peripheral eye positions. The monkeys were required to make the same-amplitude saccades but from fixation points of varying eccentricity. After the ablations, the error in eye position for saccades to visual targets increased with the eccentricity of the target. Fixations of peripheral targets on the contralateral side were also in error. Errors in fixation and saccades were proportional to the eccentricity of the visual target. Taken together the results of these experiments indicate that the oculomotor deficit was not specifically related to the amplitude of the saccade but rather to the position of the eye in the orbit.

5. While only three of the four monkeys demonstrated deficits in achieving eccentric eye positions, all four monkeys demonstrated increased latencies for saccades to targets throughout the visual field contralateral to the ablation. Latency deficits were not correlated with eye position and largely recovered during the first month of testing.

6. We conclude that the effect of complete unilateral ablation of the superior colliculus alone produces only minimal effects on the initiation and accuracy of saccades. However, when there is additional damage to the pretectum and posterior-medial thalamus, severe and long-lasting deficits in accuracy are produced. The deficit appears to

be an inability to match accurately the position of the eye to visual targets. It is suggested that nuclei in the pretectum and posterior-medial thalamus may be the critical structures in producing this deficit, possibly because of their interconnections with the superior colliculi and frontal eye fields.

7. This deficit is difficult to understand when it is assumed that a retinal error signal is used to guide saccades to visual targets. However, the present observations are consistent with a model of oculomotor control that assumes that a visually guided saccade is generated using a signal specifying the position of the target in space.

INTRODUCTION

The superior colliculus in mammals is involved in visual orienting behaviors. In the primate such orientation is dependent on saccadic eye movements to redirect gaze rapidly and efficiently. Stimulation, recording, and anatomical studies have consistently supported the role of the superior colliculus in the initiation of saccadic eye movements.

On the other hand, ablation studies that have examined deficits in eye movements have produced conflicting results: some find striking deficits while others report only mild effects. The observations of Denny-Brown (4) show the most severe deficits resulting from colliculus lesions, but the lesions were large and in several cases invaded the pretectum and posterior-medial thalamus, among other regions. Schiller (21) also found deficits in the accuracy of saccades in three of four monkeys studied, but no histology was presented. Kurtz and Butter (13) found that monkeys with superior colliculus ablations were impaired in orienting their eyes toward targets presented 15–32° eccentrically. They suggested that the eye movement deficit depended on the size of the lesion, particularly the depth of the lesion, but the lesions also extended anterior to the colliculus. No enduring oculomotor deficits were observed by Pasik et al. (18). In their experiments, structures anterior to the colliculus in the pretectum were largely undamaged, although in one monkey there was bilateral damage to the pretectum and posterior commissure, and they noted paresis in upward gaze in that animal. After small lesions largely limited to the superior colliculus, Wurtz and Goldberg (30) and Mohler and Wurtz (17) found only slight deficits in the accuracy of saccades to visual targets, but they did not examine saccades to peripheral visual targets. Recent experiments by Schiller, True, and Conway (22, 23) did not find substantial deficits in the size of saccades following colliculus ablations. Similar results have also been found in humans (9).

The differences in the results between these studies might be explained by differences in the extent of the lesions and region of the visual field studied. First, large lesions might produce a disruption of saccades to visual stimuli either because they completely removed the superior colliculus or because such lesions invaded extracollicular structures. In contrast, restricted lesions either might have spared extracollicular structures or might have been incomplete and have left intact the posterior colliculus related to the peripheral visual field. Second, in most cases these studies examined only saccades to targets within the central 20° of the visual field.

Our strategy in the present experiments was to attempt to make large lesions of the colliculus that would certainly remove the entire visual and oculomotor representation of the visual field and thereby allow study of saccades to peripheral as well as to central targets. Our results suggest a resolution to the conflicting results following collicular ablation. We find that nearly complete but restricted lesions of the colliculus do not produce significant deficits in the accuracy of visually guided saccadic eye movements to either central or peripheral targets. However, colliculus lesions that include damage to more anterior structures in the pretectum and posterior-medial thalamus result in long-lasting oculomotor deficits. Furthermore, we find that this oculomotor deficit is related to the position of the eye in the orbit rather than a deficit in the direction or amplitude of saccades. This deficit can be more readily understood using a model of oculomotor control based on a spatial coordinate system, such as that of Robinson (20), than on a model based on a retinal coordinate system. A brief report of these experiments has appeared previously (1).

The report of these experiments is divided into two parts. This paper concentrates on

the oculomotor position deficits dependent on ablations of extracollicular structures as well as the superior colliculus. The following paper (2) includes added visuomotor deficits, such as the detection of visual targets or the distracting effect of novel visual stimuli, that were related to ablation of the superior colliculus alone. The sequence in which the experiments in both reports were performed is presented in the METHODS of this paper.

METHODS

In outline, the experiments on four adult rhesus monkeys (*Macaca mulatta*) followed this sequence: initial behavioral training, surgical implantation of bolts for head restraint and of magnetic search coils or electrooculogram (EOG) electrodes for recording eye movements, preoperative behavioral testing to determine normal saccadic behavior, unilateral ablation of the superior colliculus, postoperative behavioral testing, and histological reconstruction of the lesion. A summary of procedures performed on each monkey is included in Table 1.

Initial behavioral training

Each monkey was first trained on a behavioral fixation task (29). The monkey sat in a primate chair and, by depressing a bar, turned on a fixation light that stayed on for a variable length of time between 1 and 3 s. The fixation light then dimmed for about 0.5 s and, if the monkey released the bar during that time, he was rewarded with a drop of artificial fruit juice. The fixation light was small enough and the dimming of the light subtle enough so that the monkey generally maintained a steady gaze during the fixation period. The bar was disconnected for several seconds following each release of the bar; early or late release of the bar was not rewarded but not otherwise punished.

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

Surgical procedure

After this preliminary training but before collecting preablation data, the splenium of the corpus callosum was transected in two monkeys (Table 1) to permit ablation of the superior colliculus in a second operation. The double-step procedure served as a control by permitting the collection of preablation data after the corpus callosum had been sectioned but before the colliculus lesion. Section of the callosum alone produced no deficits in saccade accuracy in our tests.

While the monkeys were anesthetized with ketamine hydrochloride (Parke-Davis) and Nembutal (sodium pentobarbital, Abbott), a small unilateral parietal bone flap was removed under aseptic conditions. A dural flap was then cut and the hemisphere was gently retracted, exposing the splenium of the corpus callosum. The posterior 5–7 mm of the splenium was transected with a small-tipped glass sucker, exposing the superior colliculus on that side. In these two monkeys, the dura was then closed with silk sutures, the bone flap replaced, and the incision closed. In the two remaining monkeys (*Haw* and *Pec*), the callosum was cut at the time of the collicular ablation in a one-stage surgical procedure.

In a subsequent surgical session four bolts were implanted into the skull (5). The bolts were connected to a socket that was later used to connect the monkey's head rigidly and in a reproducible position to the primate chair. In two monkeys (*Haw* and *Pec*) a search coil was implanted under the conjunctiva according to the method of Judge et al. (10). Since this method does not involve surgical manipulation of the extraocular muscles, it minimizes the risk of producing strabismus. Care was taken to insure that implantation of the

TABLE 1. *Summary of experimental procedures*

Monkey	Eye Movement Measurement	Transection of Splenium	Side of Collicular Ablation	Day of Contact Occluder Removal
Mou (E740)	EOG	Before ablation	Right	8
Ner (E742)	EOG	Before ablation	Left	8
Pec (G293)	Eye coil (right eye)	Single step	Right	10
Haw (E909)	Eye coil (right and left eye)	Single step	Right	13

eye coil did not restrict the oculomotor range of the monkeys. By anesthetizing the animals and mechanically manipulating the eye, we could determine that the eye was not tethered by the eye-coil wires. As a further precaution, the postoperative saccade performance of monkey *Haw* was monitored with coils in the right and left eye to insure that tethering of the eye by the lateral lead wire could not account for the saccade inaccuracies that we observed. In the two other monkeys (*Mou* and *Ner*), nonpolarizable electrooculogram electrodes were implanted into the orbital bone at the outer canthi and above and below one orbit to permit recording of the horizontal and vertical components of the eye movements (17); no tethering of the eye could occur with the EOG since the orbit was not invaded.

After completion of preoperative testing, the monkeys underwent either the second stage of the two-stage procedure or the one-stage surgical procedure referred to above. The membrane covering the colliculus was cauterized and the subjacent tissue was removed by suction to a depth that varied from about 4 mm laterally and posteriorly to about 2 mm anteriorly and medially.

Before the monkey recovered from anesthesia, translucent contact occluders (16) coated with antibiotic ophthalamic ointment (bacitracin-neomycin-polymyxin, Pharmaderm, Inc.) were inserted into the eyes. This was done to prevent visual experience during the postsurgical recovery period. Every 3–7 days the animals were sedated with ketamine and a muscle relaxant (Rompun, xylazine, Haver-Lockhart), the occluders were removed, the eyes were examined for signs of infection or inflammation, and the contact occluders were again coated with antibiotic ointment and replaced. The eyes did not show signs of infection or irritation and the corneas remained clear and unabraded. The cage behavior of the animals did not indicate that they suffered discomfort from the occluders. Monkeys were allowed varying periods of postoperative recovery before removal of the occluders (Table 1) in order to test for the effect of visual experience on detection of visual stimuli (2).

Behavioral tests

The monkeys were examined on two tests of saccadic accuracy: the first tested saccades of various amplitudes and directions initiated from primary eye position; the second tested saccades of constant amplitude and direction initiated from both primary and eccentric eye positions.

SACCADES FROM PRIMARY EYE POSITIONS. The animals were trained and tested on a saccade task previously described by Mohler and Wurtz (17). The monkey initiated a trial by pressing a bar, and on 25% of the trials (fixation trials) a light appeared in the center of a tangent screen and dimmed after a variable delay. If the monkey released the bar during the 500-ms dim period he was given a liquid reward. Approximately 73% of the trials were saccade trials; the fixation light appeared and then after a delay it was turned off and a peripheral light was turned on at some unpredictable location on the tangent screen. Typically, the monkey then made a saccadic eye movement from the fixation light to the peripheral light in order to detect the dimming of that peripheral light. The timing, sequence, and positions of the stimuli were pseudorandomly varied under computer control so that the monkey could not guess where or when a peripheral target light would appear. Each of over 80 saccade targets was tested 8 times in a session. Saccade lights were 20′ of arc in diameter, with a brightness of 10 cd/m^2 on a background of 1 cd/m^2. The incandescent bulbs reached two-thirds brightness in about 10 ms.

The monkey faced the tangent screen, which was 28 cm in front of his eyes. Primary eye position (0°) refers to the eye position as the animal looked directly ahead at the fixation point. Since the head was held rigidly and in a reproducible position relative to the primate chair and tangent screen, this eye position remained constant over successive test days. We found that this primary eye position roughly corresponded to the center of a horizontal oculomotor range of ±40°. The full vertical oculomotor range could not be determined since the primate chair interfered with testing of the inferior visual fields.

SACCADES FROM ECCENTRIC EYE POSITIONS. This saccade test permitted study of saccades of constant size and direction that were initiated from primary and eccentric eye positions. The monkey's head was positioned 58 cm from a horizontal perimeter that held incandescent bulbs placed 10° apart. A fixation light appeared at 0, 10, 20, or 30° of horizontal eccentricity and the saccade target then appeared 10 or 15° more peripheral or more central than the fixation light. Primary position (0°) on the perimeter was identical to primary position on the tangent screen, and all other stimulus and timing parameters on this test were the same as described for the saccade task. The eccentric eye position task did not require any specific training; the monkeys readily transferred the training they had received on the tangent screen.

Eye movement measurement and calibration

In two monkeys (*Haw* and *Pec*) eye movements were recorded using the magnetic search-coil technique developed by Robinson (19) and Fuchs and Robinson (6). The monkey chair was placed inside two pairs of magnetic-field coils, a horizontal pair and a vertical pair, oscillating in phase

quadrature. The signal that was induced in a coil of wire chronically implanted under the conjunctiva of the monkey's eye (10) was fed into a phase detector (CNC Engineering), which produced two voltage signals proportional to horizontal and vertical eye position.

In two other monkeys, *Ner* and *Mou*, eye movements were monitored using implanted silver-silver chloride electrodes, as described previously (17), to record the electrooculogram (EOG). The EOG signal was amplified and filtered with a low-pass filter of DC to 25 Hz. The EOG was calibrated by requiring the monkey to fixate a target straight ahead and to either side or, after the ablation, to the ipsilateral side. It is well known that beyond 30° eccentricity the EOG is markedly nonlinear. However, we were able to evaluate saccade accuracy using the following measurements: *1*) the difference between the average amplitude of saccades made pre- and postoperatively to each target position, and *2*) the difference between the average variability in amplitude of pre- and postoperative saccades for each target position. Film records of the digitized eye movements were also examined for gross saccadic errors, that is, saccades made to the wrong quadrant of the visual field and for corrective saccades greater than 2°. The vertical component of the EOG was frequently contaminated by artifacts, either blinks or jaw movements, and this vertical EOG data was excluded from analysis; horizontal components of the saccade were included. Identification of the onset of a saccade in the EOG records was made from the horizontal component of the saccade.

The horizontal and vertical components of the eye movements, whether measured by the magnetic search-coil technique or EOG, were sampled every 4 ms and stored by a digital computer controlling the experiments (PDP-11 or PDP-12, Digital Equipment Corp.). Two-second segments of the eye movement traces and stimulus events were displayed on an oscilloscope and photographed on-line by a Grass camera. The display and eye movement measurements were derived from the digitized data and so had a temporal resolution of 4 ms. The resolution of the digital quantization of saccade amplitude on the linear range of the recording systems was 25′ of arc in the magnetic search-coil records and 30′ of arc in EOG records.

For identifying the onset of saccades in EOG records, the computer identified the start of an eye movement in one EOG channel when the deflection in the record was greater than 1.5° within two consecutive samples (8 ms). Termination of the saccade was identified by failure to detect a difference greater than 1.5° within two successive samples. For magnetic search-coil records, the principle used for identification of the beginning and end of the saccade was the same but a difference in samples greater than 50′ of arc was required in two samples taken 12 ms apart.

For each saccade trial the computer calculated and stored four parameters of the eye movements for off-line analysis (as shown schematically in Fig. 1): *1*) initial position—eye position at the onset of the saccade target, *2*) saccade amplitude—the change in eye position at the end of the initial saccade, *3*) final position— eye position at the beginning of the dim period after any corrective saccades, and *4*) saccade latency—the time between the onset of the saccade target and the beginning of the eye movement.

Eye movement measurements and film records were examined off-line. Measurements were excluded from calculations if film records revealed that there was an error in measurement due to an artifact, such as a blink, or if the animal released the bar prematurely or did not fixate. In preoperative records using the search-coil method about 10% of the trials were eliminated. In postoperative records both artifacts and behavioral errors increased and about 20% of the trials were eliminated by these criteria. Errors by the monkeys were not consistently related to a particular

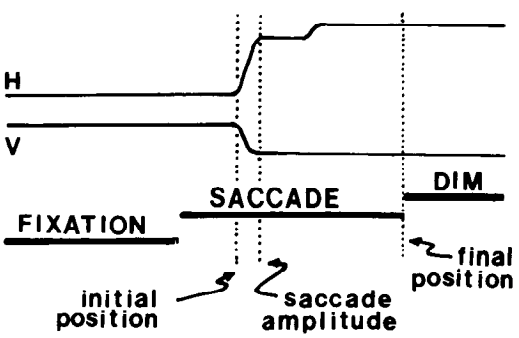

FIG. 1. Schematic drawing showing eye movement parameters measured in a saccade trial. The heavy solid lines indicate the sequence of stimulus events: *1*) fixation—when the fixation light was on, *2*) saccade—when the saccade target came on, and *3*) dim—when the saccade target dimmed signaling the beginning of the reward period. The eye movement traces are shown by finer lines labeled H (horizontal) and V (vertical). After a variable interval, the fixation light is switched off and simultaneously a peripheral stimulus is turned on at an unpredictable location. The monkey makes a saccade from the fixation light to the stimulus in order to detect the dimming of this light. If this saccade falls short of the saccade target, the monkey will often generate a second corrective saccade (as on the horizontal trace). Four measures are taken: initial eye position, saccade amplitude, final eye position, and the latency of the saccade after the saccade target comes on.

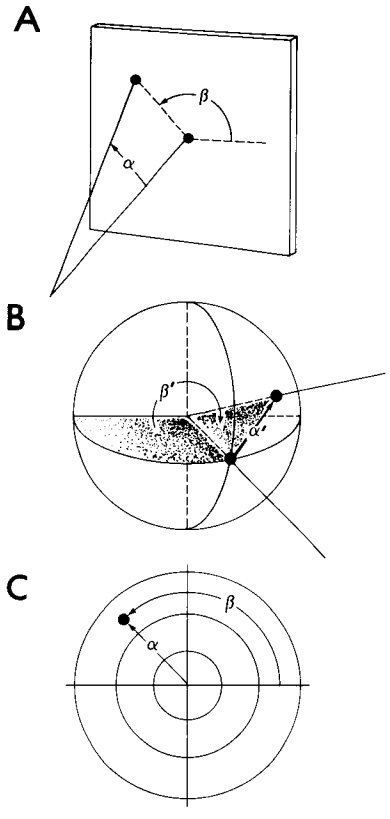

FIG. 2. Representation of stimulus position and eye position on a polar map. *A*: representation in polar coordinates of the position of a stimulus on the tangent screen. Eccentricity of the stimulus, α, defines the angular distance from the primary visual axis of the eye to a peripheral stimulus. Direction β describes the polar angle of a visual stimulus from the vertical and horizontal visual axes. *B*: schematic drawing in which the position of the eye in the orbit is defined by angles defining the eccentricity (α') and direction (β'). *C*: a polar map in which α and α' are shown related to distance from horizontal axis.

target position whether ipsilateral or contralateral to the lesion.

Calculation of stimulus and eye position

Polar coordinate systems were used to represent both visual target position and eye position. When the eye is in primary eye position, the position of a stimulus light can be represented in a polar map, as diagrammed in Fig. 2*A*. The position of a light on the tangent screen was converted into polar angles representing visual eccentricity, α, and visual direction, β, by the formulas

$$\alpha = \arctan \frac{\sqrt{x^2 + y^2}}{r}$$

and

$$\beta = \arctan \frac{y}{x}$$

where x and y represent the horizontal and vertical distance of a screen light from the fixation point on the tangent screen, and r represents the shortest distance of the eye from the screen.

The position of the eye in a polar-coordinate system is shown in Fig. 2*B*. Orbital eccentricity (α') refers to the angular deviation of the eye in the orbit from the visual axis at primary eye position. The orbital direction (β') refers to a polar angle from 0 to 360° where, for example, 90° would be vertical and 180° would be leftward from primary orbital position. Orbital eccentricity and direction were determined by the relations

$$\alpha' = \arcsin \sqrt{(g_y V_y)^2 + (g_x V_x)^2}$$

and

$$\beta' = \arctan \frac{g_y V_y}{g_x V_x}$$

where V_y and V_x are the recorded voltages related to vertical and horizontal eye position. The scale factors g_y and g_x are used to adjust the gains of the eye-coil signals. Derivation of these equations, which were used to convert the search-coil voltages to the data plotted on all figures, is shown in the APPENDIX.

The polar-coordinate system allows both visual target position and eye position to be represented on one polar map, as shown in Fig. 2*C*. An advantage of the polar-coordinate system for representing the position of visual targets is that it describes stimulus position in terms of angular deviation from the primary visual axis when the eye is in primary position (Fig. 2*C*). This method avoids complications due to use of tangent screens. An advantage for eye-position description is that derivation of eye position from eye-coil voltages is greatly simplified: eccentricity and direction are computed directly and may be easily compared to the position of visual targets in space. This is particularly important for eccentric orbital positions where eye-coil voltage and eye position are nonlinearly related.

Histology

After variable periods of postoperative testing, ranging from 2 to 6 mo, the animals were given an overdose of Nembutal and perfused through the heart with normal saline followed by 10% Formalin. The skull was opened and the cortex was exposed and photographed. Coronal sections (50 μm) were taken through the entire extent of the colliculus lesion and at least 4 mm anterior to the colliculus. Every 10th section was stained with thionine for cell bodies and adjacent sections were stained according to the method of Weil for

myelinated axons. Drawings and reconstructions were made from sections magnified and projected onto paper.

RESULTS

Three of the four monkeys with unilateral ablations of the superior colliculus displayed a deficit in visually guided saccadic eye movements. Histological analysis revealed that these behavioral deficits were correlated with the extent of the lesion: colliculus removal was essentially complete in all monkeys, but in the three with severe deficits there was also extensive damage anterior to the colliculus in the region of the pretectum and posterior-medial area of the thalamus. Because extracollicular lesion size appears to be the critical difference between monkeys, we will first consider the histological findings and then describe the effects on the accuracy of saccadic eye movement initiated from primary and eccentric eye positions.

Histological results

The extent of the lesions is shown in Fig. 3. The coronal sections through the midbrain

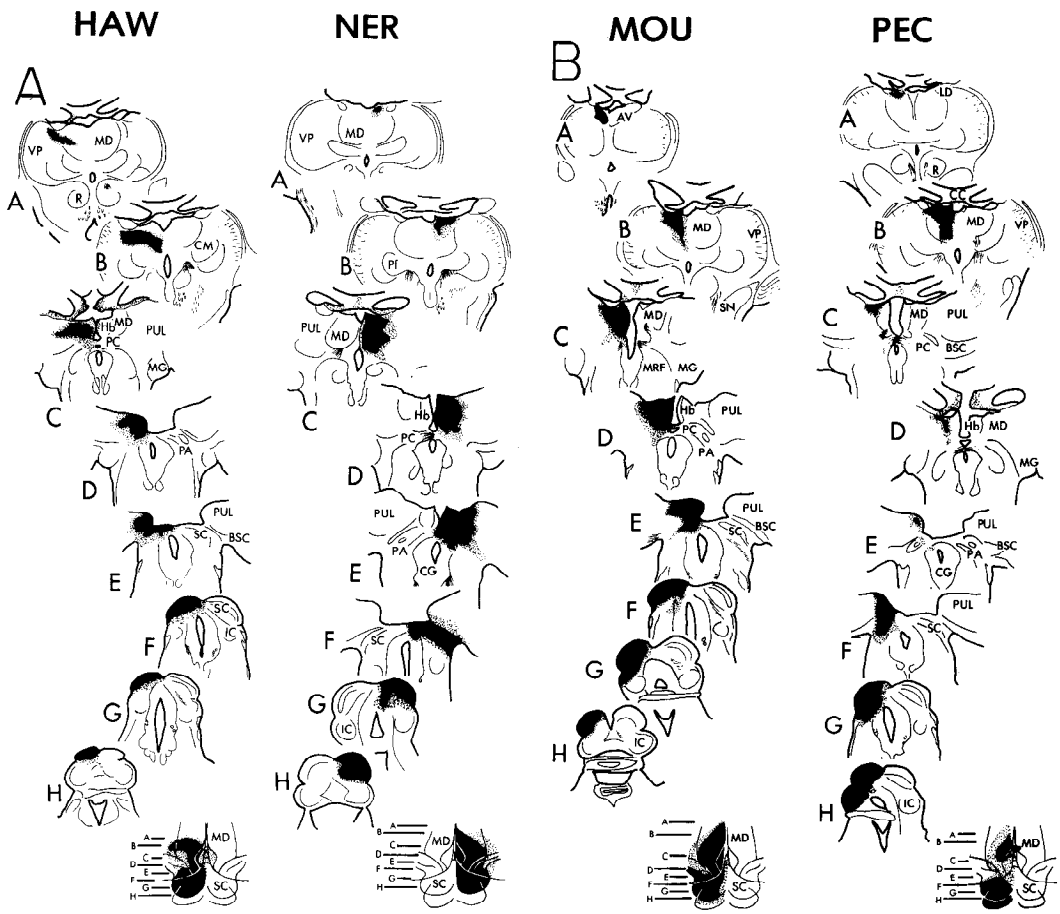

FIG. 3. Histological reconstruction of unilateral lesions in *Haw, Ner, Mou,* and *Pec.* The coronal sections illustrate the extent of the damage (black, missing tissue; stippling, gliosis) through the posterior-medial thalamus (sections A–D) mesodiencephalic junction (C–E) and midbrain (sections E–H). Ablations were large, as intended, and removed virtually all of the superficial and deep layers of the colliculus. An infarct rostral to the colliculus extended the ablations into the mesodiencephalic junction and posterior-medial thalamus (sections A–E) and three animals (*Haw, Ner,* and *Mou*) were more severely damaged in these regions. The dorsal view reconstructions at the bottom of each column show the full anterior-posterior extent of the damage in each monkey (black, severe damage; stippling, mild damage). Note that lesions in *Haw, Ner,* and *Mou* are more extensive anteriorly than is the lesion in *Pec* (sections A–E). Drawings of coronal sections were produced by tracing projected sections taken every 500 μm. Missing tissue (black area) was approximated by comparison with intact side. Dorsal view reconstructions were made from drawings of coronal sections.

and thalamus illustrate the depth and medial-lateral extent of tissue that was removed (black) or was gliotic (stippled). The unilateral ablations of the superior colliculus (Fig. 3, coronal sections E–H) were nearly complete. All lesions included the superficial and deep layers and extended to the central gray medially, to the external capsule and brachium of the inferior colliculus laterally, and to the mesencephalic reticular formation rostrally.

Three of the four monkeys (*Haw, Ner,* and *Mou*) had the most extensive damage anterior to the midbrain in the region of the mesodiencephalic junction and the posterior-medial part of the dorsal thalamus (Fig. 3, coronal sections A–F). This damage was due presumably to an infarct produced by cauterization of the blood vessels overlying the colliculus. The structures involved in the mesodiencephalic junction were the pretectal nuclei, posterior commissure, and the brachium of the superior colliculus, and in the posterior-medial portion of the dorsal thalamus they were the medial and lateral portions of the dorsomedial nucleus, medial pulvinar, habenula, and habenula-peduncular tract. The internal medullary lamina and intralaminar nuclei were invaded but to an unknown extent since shrinkage and distortion made these nuclei difficult to define in either Nissl- or fiber-stained sections. All these thalamic structures together will be referred to as the posterior-medial thalamus.

In two monkeys, *Mou* and *Pec*, the damage also extended posterior to the superior colliculus (Fig. 3, coronal sections G, H). These monkeys had the deepest lesions, which transected the brachium of the inferior colliculus and severely damaged the external capsule and central nucleus of the inferior colliculus.

Other structures damaged to varying degrees in different monkeys were the lateral dorsal nucleus, ventral posterior nucleus, central gray, inferior pulvinar, and mesencephalic reticular formation. In two monkeys given the two-stage surgical procedure (*Ner* and *Mou*), parietal and cingulate cortex also were slightly affected, presumably due to reopening the dura and resectioning the hemispheres in the second-stage operation.

The dorsal-view reconstructions at the bottom of Fig. 3 show the anterior-posterior extent of complete damage (black) and partial damage (stippled) through the midbrain and dorsal thalamus. The three larger lesions in *Haw, Ner,* and *Mou* extended rostrally into the dorsomedial nucleus and laterally to the internal medullary lamina (Fig. 3, dorsal view A–D). In contrast, the damage in monkey *Pec* was more limited anteriorly and laterally. The dorsomedial nucleus was spared; the pretectal area was affected only in a small, medial region, and only superficially. All monkeys had in common nearly complete unilateral ablations of the superficial and deep layers of the superior colliculus.

Saccade tasks

All the monkeys showed a transient deficit in their ability to detect the stimuli used as targets for saccades, as described in the following paper (2). The detection deficit had recovered before saccade tests were begun.

SACCADES FROM PRIMARY EYE POSITION. The monkeys were first tested for the accuracy of saccades initiated from primary eye position to peripheral visual targets. Figure 4 shows the pre- and postoperative results in two monkeys (*Haw*, with more extensive anterior lesion, and *Pec*, with a more restricted anterior lesion). Each polar map indicates the position of the fixation point (center cross), saccade targets (heavy crosses), and 10° of visual angle (concentric circles). The filled circles indicate the average eye position after the first saccade, the solid lines end at final eye position after corrective saccades, and the dotted lines indicate a remaining error in eye position. Before the ablation (upper polar plots in Fig. 4) the accuracy of the initial saccades declined with increasing eccentricity. Within the central 20° of the visual field the initial saccade usually brought the eye to a position within 1–2° of target position. Beyond the central 20°, the initial saccade generally fell short of the target. A second, corrective saccade brought the average eye position to within 2° of the target. In our practiced monkeys saccades to a given target were stereotypic; standard deviations in eye position rarely exceeded ±1.5° (the radius of the filled circles).

Unilateral ablation severely affected the accuracy of eye movements to contralateral visual targets in *Haw* but not *Pec* (shaded

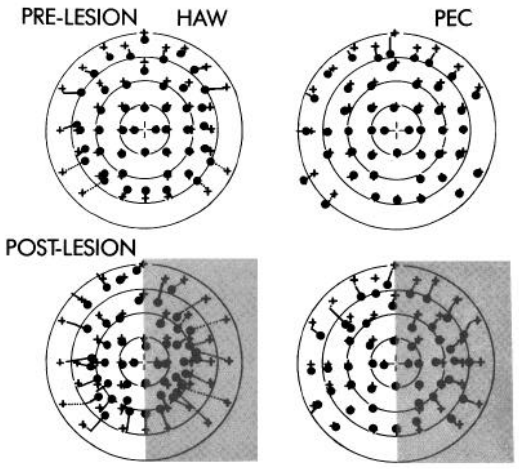

FIG. 4. Loss in saccadic accuracy to contralateral visual targets in *Haw* but not *Pec* after unilateral colliculus ablation. On each of the polar maps, the center cross indicates the fixation point and the heavy crosses indicate the position of the saccade targets. Each concentric circle demarcates 10° of visual angle. Filled circles show the average eye position at the end of the initial saccade while the solid lines connect this position with final eye position after corrective saccades. Dotted lines indicate any error in final eye position. Each point represents the average of eight saccades measured using the magnetic search-coil method. In the prelesion maps, saccades of both *Haw* and *Pec* are accurate, as shown by the proximity of the initial saccades to the saccade targets. Saccades to peripheral targets typically fell short and were corrected by second saccades. In comparison, the postlesion maps reveal a striking deficit for large saccades to peripheral visual targets in the visual field contralateral to the ablation (shaded side) in *Haw* but not *Pec*. Postoperative data obtained from *Haw* on day *28* and *Pec* on day *12*.

area on the lower polar plots of Fig. 4). In *Haw* the postoperative accuracy of the initial saccades to targets within 20° of the fixation point was only mildly affected, but beyond 20° the initial saccades fell short of the target. This deficit in accurately reaching the visual targets with a horizontal eccentricity greater than 20° affected oblique saccades, although the horizontal component of the saccades was more affected than the vertical components. For example, in the affected hemifield (Fig. 4, lower left) the final positions of saccades to targets within 20° of the vertical were within 2° of the visual target, but saccades with larger horizontal components fell short of the target by 10°. The maximum amplitude of the horizontal component of saccades was limited to about 25°.

In contrast to *Haw, Pec*'s saccades to contralateral targets (Fig. 4) were only slightly shorter on the average than either preoperative or ipsilateral saccades. These relatively small errors were readily corrected by second saccades, which were more frequent on the contralateral side (30% of the saccades had correctives) than on the ipsilateral side (15% of the saccades had correctives).

The loss of corrective saccades to targets in the contralateral visual field was perhaps the most striking feature of the deficit. Figure 4 shows that for monkey *Haw* the errors (dotted lines) that remained after the initial saccade to the contralateral side were not corrected by second saccades while corrective saccades to the ipsilateral side (solid lines) still occurred and in some cases caused the eye to overshoot the target. This figure also shows that the loss of corrective saccades corresponds to the region with shortened saccades; correctives never occurred for saccades to horizontal or near horizontal targets, whereas corrective saccades to vertical targets still occurred.

The postoperative performance of *Ner* and *Mou* studied with EOG recording corroborated the results obtained from *Haw*. Because the EOG did not provide a reliable means of measuring eye position, we were limited to measurement of the amplitude of the initial saccade. Both animals showed a decrease in the average amplitude of the horizontal component of the initial saccade from preoperative values for targets beyond 20°. Figure 5 shows polar maps for these two animals, with plus signs indicating points tested, and the shaded portion of the field indicating the region in which elicited saccades were short (by 3° or more in *Ner* and 2° or more in *Mou*) of average preoperative values. In general, the differences between preoperative horizontal EOG values and postoperative values were greater for more eccentric saccade targets, indicating that saccades to more peripheral targets were more severely affected. Like *Haw*, both animals studied with EOG also showed a loss of corrective saccades to eccentric targets. Changes in saccadic accuracy ipsilateral to the ablation was variable; *Mou* showed a consistent tendency to overshoot peripheral targets with the initial saccade but *Ner* did not. *Haw* fell short of peripheral targets but corrected this error with a second saccade.

The deficits in the accuracy of saccades

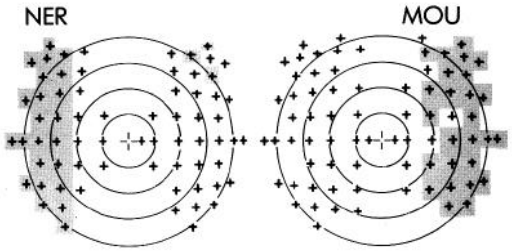

FIG. 5. Shortening of the horizontal component of initial saccades to peripheral visual targets contralateral to the ablation in *Ner* and *Mou*. The plus signs indicate points tested both pre- and postoperatively. The shaded portions of the polar plots indicate the position of visual targets that elicited shortened initial saccades. The difference between pre- and postoperative values of the average amplitude of the horizontal component was computed from EOG records. Target areas were shaded when the difference was greater than 2° in *Mou* and 3° in *Ner*. Both monkeys showed a decrease in the amplitude of the initial saccade for peripheral visual targets contralateral to the lesion (*Ner*, right superior colliculus lesion; *Mou*, left superior colliculus lesion). Records are from postoperative day *13* for *Ner* and day *19* for *Mou*.

seen in *Haw* and *Mou* were followed for more than 4 mo without an indication of recovery. Moreover, the performance of these animals actually deteriorated with time; saccade amplitudes became more variable and, on the average, less accurate. *Ner* did show an improvement in the amplitude of the initial saccade to peripheral contralateral targets after about a month, but he appeared to also develop an increased amplitude for ipsilateral targets as well. It is unclear whether these changes reflect a neural reorganization in response to injury or whether these changes reflect a change in strategy in response to continued testing. There is no indication that any long-term adaptive changes compensate for the deficit. Examination of the histological sections indicated that this deterioration did not result from any abscess or damage other than that described as part of the lesion.

SACCADES FROM ECCENTRIC EYE POSITIONS. We next determined whether the deficit was related to large-amplitude saccades or to saccades made to peripheral targets. We tested the monkeys on a perimeter that required the monkey to fixate at 10, 20, or 30° to the right or left of primary eye position and to make saccades to targets 10 or 15° more peripheral or more central to the fixation point.

The results for *Haw* and *Pec* are shown in Fig. 6 for centrifugal saccades. Each arrow shows the average eye position during fixation on several trials (base of the arrow) and the average final eye position (head of the arrow) after a saccade to the target. The preoperative saccades in both animals (Fig. 6, upper graphs) were accurate; errors were less than 5% for most points with a standard deviation of less than 1°.

In contrast, the postoperative performance of *Haw* (Fig. 6, lower left graph) revealed errors that were roughly proportional to target eccentricity on the side contralateral to the lesion. Saccades made from 0 to 10° were only slightly affected, but saccades from 10 to 20° and from 20 to 30° had larger errors. This performance was consistently in error since the postoperative variability increased only slightly (maximum SD, ±1.5). Saccades made from eye positions ipsilateral to the lesion matched expected values. Postoperative performance in *Pec* (Fig. 6, lower right graph) was comparable to preoperative behavior.

Corrective saccades were rare or very small (less than 2°) on this task in preoperative trials, presumably because the initial saccade was accurate. However, in spite of the large errors in *Haw*'s postoperative performance and the ample time to correct the error, we still did not observe corrective saccades in the film records made of each trial. In addition, in the quantitative measure of eye position, no significant differences were detected between eye position at the end of the saccade and final eye position at the end of the trial, which indicates the paucity of any small corrective saccades or slow eye movements. We attempted to study more eccentric saccades (greater than 30° orbital eccentricity), but we found that trials requiring more eccentric visual targets appeared to be frustrating for the monkey. As many as 75% of the postoperative trials were eliminated because the monkey developed the strategy of aborting the trial when the target appeared in the periphery, suggesting that the animal was performing the task extrafoveally. If the animal was not very thirsty, continued testing on peripheral target positions would have the effect of ending the day's testing.

Both the reduction in amplitude of saccades to peripheral targets and the failure

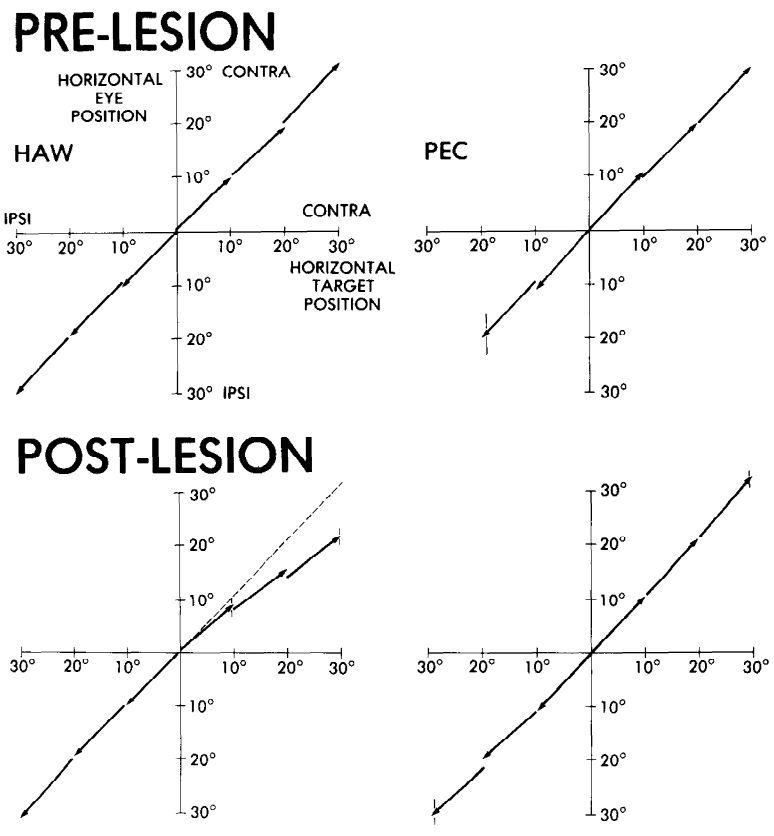

FIG. 6. Loss of accuracy of fixations and saccades with eccentrically directed saccades in *Haw* but not *Pec*. Each graph plots average horizontal eye position against horizontal target position for 10° saccades initiated from 0, 10, and 20° to peripheral targets. Prelesion graphs show that eye position when fixating, indicated by the origin of the arrow, and final eye position when on target, indicated by the head of the arrow, matched target position: the largest mean error was about 1.5°. The postlesion graphs show that both initial eye position and final eye position for peripheral fixation and target points contralateral to the ablation are grossly inaccurate in *Haw* but not affected in *Pec*. Each point on the graph is the mean eye position on about eight trials; standard deviation at each point is indicated by a vertical line unless it is no larger than ±0.50. Dotted line in *Haw*'s graph indicates accurate performance. Postlesion data taken from *Haw* on day *50* and *Pec* on day *13*.

to make corrective saccades to reach the targets indicate that the deficit was related to the accurate matching of eccentric eye positions to target positions. This notion is further supported by the observation that eye-position errors were also independent of the direction of the eye movement; centrifugal and centripetal movements resulted in comparable errors. The effect of the lesion on centripetal saccades of monkey *Haw* is shown in Fig. 7. Before the lesion, centripetal saccades were accurate even for peripheral eye positions. Errors were less than 1° for both fixation and final position. After the lesion, ipsilateral saccades were still quite accurate, but contralateral eye positions were dramatically affected. A particularly striking example is the case where the monkey is required to make a saccade from a far eccentric target. For a saccade from a target 30° in the contralateral field to one at 20°, the monkey began the saccade at an average position of about 20° and went to 14°. This is a curious result since the eye was closer to the target before the saccade than after. In this case, the best strategy for the monkey would have been to make no saccade at all.

The errors during fixation and saccades were roughly proportional to target position. In Fig. 8 average eye positions during fix-

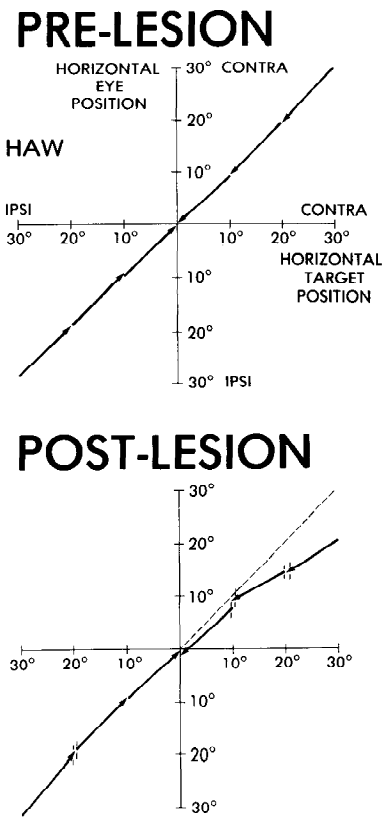

FIG. 7. Loss in eye position accuracy for centrally directed saccades from eccentric eye positions in *Haw*. Preoperative records show little error in eye position for centrally directed saccades from peripheral eye position. Postoperative saccades were shortened contralateral to the ablation and began and ended at inappropriate eye positions while ipsilaterally more eccentric saccades are slightly longer. Symbols same as in Fig. 6. Postoperative data from day *50*.

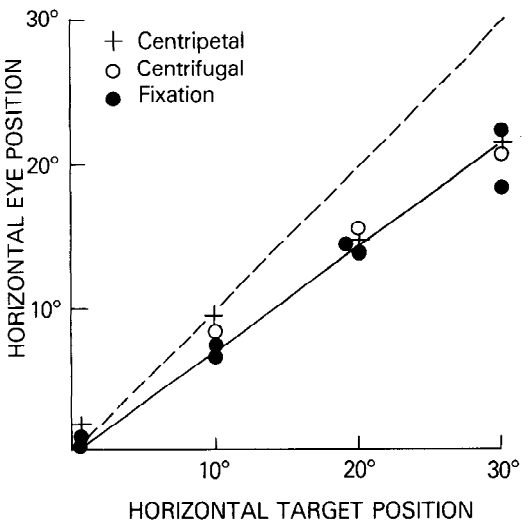

FIG. 8. For *Haw*, the error in eye position is proportional to target position. Average eye position during contralateral fixation and centrifugal and centripetal saccades is plotted in relation to target position. This graph shows that the error in eye position grew in relation to target eccentricity. Errors could be fitted to a line with a slope of 0.72 (solid line). The dotted line with a slope of 1.0 indicates what would be perfect performance. Data are derived from Figs. 6 and 7 except for the position after a centripetal saccade to 30°; this point on the graph was obtained from saccades in which the monkey attempted to fixate on a point 40° contralateral from the fixation point.

ation (when saccades were made from a variety of eye positions) and after centrifugal and centripetal saccades (when saccades were made from specified eye positions) for monkey *Haw* are plotted in relation to target position. The relation between eye position and target position is described by a line with a slope of 0.72 (correlation coefficient = 0.99).

Qualitatively similar results were found in monkeys *Ner* and *Mou* using the EOG to monitor eye movements. Since the EOG was unsuitable as a method of monitoring absolute eye position, we compared the amplitudes of 15° saccades that were initiated from ipsilateral eye positions to the amplitudes of saccades initiated from contralateral eye positions. Saccades made to targets from 30 to 45° in *Ner* and 15 to 30° in *Mou* did show a consistent deficit: peripheral saccades contralateral to the lesion were smaller than ipsilateral saccades.

Taken together the results of these experiments indicate that the oculomotor deficit is not specifically related to the amplitude or direction of the saccade but rather orbital eccentricity. The errors that remained were not corrected by subsequent saccades. In one monkey studied with the eye coil, we were able to show that the amount of eye-position error was roughly proportional to target eccentricity.

Optokinetic and vestibular stimulation and eccentric eye position

In order to determine whether the lesion produced a restriction of eccentric gaze un-

der conditions other than visually guided saccades, we tested the monkeys during optokinetic and vestibular stimulation. Unfortunately, reliable observations were obtained from only one monkey (*Haw*) that had both the deficit and the eye coil. We found that optokinetic and vestibular stimulation could drive the eyes to eccentric orbital positions. During optokinetic nystagmus, using a full-field drum, *Haw*'s eye position exceeded 28° on the contralateral side. During caloric stimulation, irrigation of the ear with cold water ipsilateral to the lesion produced nystagmus with a fast phase toward the contralateral side, driving the eye to contralateral positions beyond 30°. These limited results suggest that optokinetic and vestibular stimulation are capable of driving the eyes to eccentric eye positions while tasks that require the monkey to reach eccentric eye positions with visually guided saccades are affected.

We also attempted to train *Haw* to pursue targets to eccentric positions. We had found previously that pursuit to eccentric positions is a difficult task for a normal monkey, and in *Haw* we were unable to obtain consistent measures of performance to draw firm conclusions.

Latency for saccade initiation

Previous experiments have shown that the latency for the initiation of saccades to visual targets increases after collicular ablations (17, 29), and we examined whether this latency deficit was related to eye position.

After the lesion, increases in the latency of saccades were found throughout the visual field. Contour maps of average saccade latencies in three monkeys are shown in Fig. 9. Before the lesion (Fig. 9, left column), each monkey exhibited a pattern of latency distributions that was approximately horizontally symmetrical and that was different in detail for each monkey. In general, more eccentric targets were associated with longer latencies (lighter shading in Fig. 9), but within the central 30° ranged between 170 and 230 ms. After the lesion, shorter latency contours were shifted toward the ipsilateral side, indicating that average saccade latencies increased for targets contralateral to the lesion. The clearest effect is seen in *Pec* when, on the contralateral side, average latencies were increased by 85 ms (with an average standard deviation across all targets of 20 ms). The increases in average saccade latency were less dramatic in *Mou* and *Haw*; the average increase in latency was 28 ms

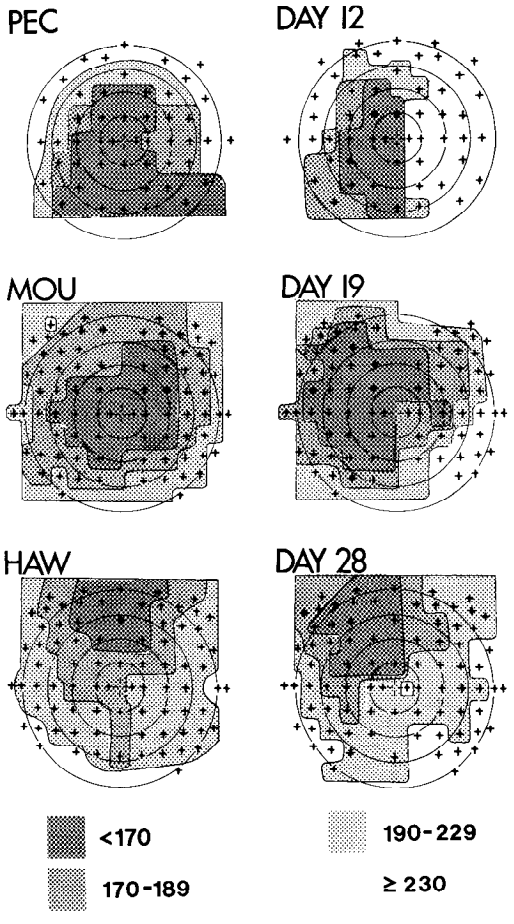

FIG. 9. Elevation in average saccade latencies to targets in the visual field contralateral to the ablation. Each polar plot illustrates interval contour maps based on average saccade latencies for saccades initiated from primary eye position. Dark regions contain tested points with saccade latencies that fell within the shortest latency intervals, and progressively lighter regions indicate longer latencies. The preoperative maps (left column) reveal that patterns of interval-latency contours are not necessarily radially symmetric. After the lesion (right column), the three animals are affected in a consistent manner: lowest latency-interval contours now occur on the side ipsilateral to the ablation (left) only; latencies for saccades to the contralateral side are elevated. *Pec* is more severely affected on day *13* in comparison to *Mou* on day *19* and *Haw* on day *28*.

(standard deviation, 22 ms for *Mou*, 17 ms for *Haw*). For the fourth monkey, *Ner*, the pattern of latencies was not different from those shown here but latencies were more variable and averaged 20–30 ms longer. The clear asymmetry in saccade latencies to the ipsilateral and contralateral side recovered during the first postsurgical month.

The increase in the latency for contralaterally directed saccades did not depend on eye position. Figure 10 shows average latency measurements for 10° contralaterally directed saccades initiated from eye positions ranging from 20° ipsilateral to 20° contralateral. In the preoperative records the latency of saccades varied with eye position; longer latencies were again associated with saccades to eccentric eye positions even for 10° saccades. After the lesion, there is a considerable increase in average saccade latencies for contralaterally directed saccades initiated from all eye positions.

The increase in the latency of saccades was not dependent on deficits in eye position. First, all four monkeys showed an increase in saccade latency for targets in the contralateral visual field while only three showed the eye-position deficit. Second, the latency deficit was not related to eye position. Third, the latency deficit diminished during the first postoperative month while the eye-position deficit persisted.

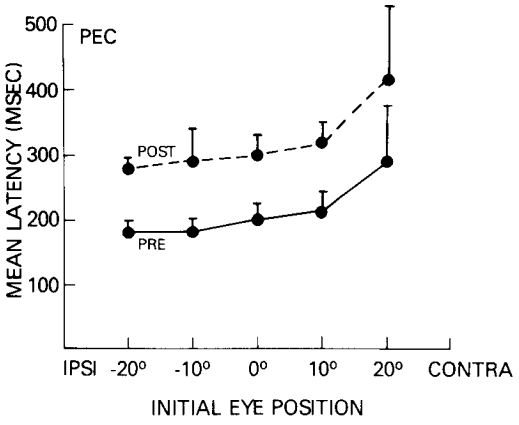

FIG. 10. Increase in mean saccade latency for contralaterally directed 10° saccades initiated from several eye positions. Average preoperative saccade latencies were longer for 10° saccades when they were initiated from more eccentric eye positions. However, an increase in postoperative saccade latencies occurred for 10° saccades across all eye positions. Postoperative data from day *12*.

DISCUSSION

Two salient observations emerge from these studies of superior colliculus ablation. First, deficits in the accuracy of visually guided eye movements are found when the ablation extends anterior into the pretectum and posterior-medial thalamus. Second, the oculomotor deficit is related to eye position rather than to either amplitude or direction of the saccade.

We will discuss three major points. First, we will compare the deficits that result from extension of the lesion to include structures anterior to the superior colliculus and deficits that result from colliculus damage alone. Second, we will show that our results are easily explained by models that assume a saccade is generated from a signal coding the position of a target in a spatial reference system. Finally, we will relate the present results to known anatomical connections involving the frontal eye fields and superior colliculus.

Eye-position deficits due to invasion of anterior structures

Although all four monkeys had nearly complete colliculus lesions, only three of the monkeys were found to have severe deficits in the accuracy of visually guided eye movements. Extracollicular structures were involved in all cases, but in the three monkeys with severe deficits, the lesions included extensive regions of the pretectum and posterior-medial thalamus. The fourth monkey, which was without such deficits, had the deepest colliculus lesion but also had the smallest amount of pretectal and thalamic damage. Thus, careful examination of the histology indicates that the severe oculomotor deficit is not related to the size or depth of the colliculus lesion but to the amount of anterior damage. These observations suggest a resolution to the conflict regarding saccade deficits resulting from colliculus ablation; severe oculomotor deficits are not dependent on the extent of colliculus damage, but rather these deficits are a result

of extracollicular damage, particularly in anterior structures.

Our results suggest that an increase in the latency of a visually guided saccade and an increase in the size and number of corrective saccades are a result of collicular damage alone. An increase in the latency for saccades made to targets throughout the visual field was found in all four animals. The severity of this effect was not correlated with the amount of extracollicular damage. A slight shortening of initial saccades was found in *Pec*, the monkey with the most restricted but complete colliculus lesion. Larger amplitude saccades to peripheral targets were particularly affected. The shortened initial saccades were compensated by second corrective saccades, and these second saccades tended to be larger and more frequent. Colliculus lesions in man also have a similar latency effect (9).

Our results, indicating deficits of an increased saccade latency and increased frequency and size of corrective saccades, are consistent with those of previous studies in which the ablation was also restricted to the colliculus (17, 30). While the lesion was nearly limited to the superior colliculus in only one monkey (*Pec*), the present study shows that these deficits extend throughout the visual field once served by the ablated colliculus, whereas in previous experiments with partial lesions these deficits were limited to a fraction of the visual field. Since the same deficits occur following nearly complete ablations of the colliculus, the more subtle deficits involving saccade latency and corrective saccades cannot be regarded as merely the result of partial lesions.

The deficits in the accuracy of saccades resulting from the larger lesions that invaded anterior areas were qualitatively different from the deficits resulting from colliculus damage alone. In these monkeys with the anterior lesions, the amplitude of the initial saccade was decreased. It was not simply large saccades that were affected, but smaller saccades as well when they were initiated from eccentric eye positions. Unlike the colliculus-related deficit of increased latency for saccades, this deficit is not retinotopically organized, since it does not correlate with the distance of the target from the fovea or with saccade amplitude. Instead, the deficit is related to eye position, since the error in performance grows as a function of orbital eccentricity whether the monkey attempts to fixate the stimulus or make a saccade toward it. The most striking aspect of the deficit is the reduction rather than increase in corrective saccades in spite of the large errors that remain after initial saccades.

Eye-position deficit and models of oculomotor function

The traditional explanation of how visual information is transformed into a signal used to guide a saccade assumes that a retinal error is computed by taking the difference between target position and the fovea. In such a system, damage in transmission of the retinal error to the saccade generator should produce errors in the amplitude of saccades. For example, if the retinal error signal was smaller than needed, saccades would fall short of the target. However, after the saccade, the eye would again sample the target position relative to the fovea and generate another corrective saccade to minimize the remaining error. These deficits, a shortened initial saccade, and an increase in frequency of corrective saccades are quite unlike the position deficits we have found in our monkeys with extensive anterior damage. Therefore, our behavioral results are not easily explained by a simple retinal error model.

Our results can, however, be explained when it is assumed that the signal used to generate a visually guided saccade indicates the position of the selected target in space rather than a retinal error. A model of oculomotor control proposed by Robinson (20) uses such a signal to guide the eyes to a visual target. According to this model, shown in simplified form in Fig. 11, the nervous system generates a retinal error (re) signal by combining at junction A information concerning target position (T) and eye position (E). At a subsequent step, an internal copy of eye position (ep) is combined with the retinal error at junction B to produce a signal representing the position of the selected target in space (tp). It is this target-position signal that is used by the saccade generator (sg) to move the eye to its new position.

For example, suppose the eye is at primary

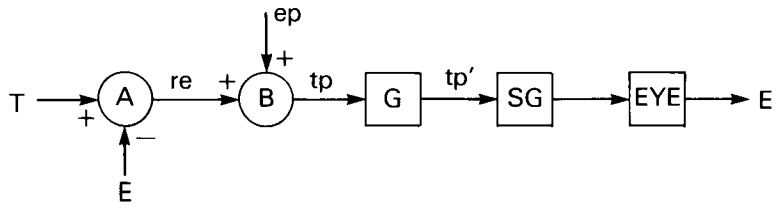

FIG. 11. A simplified model of oculomotor control that predicts the observed eye-position deficit. At A, target position in space (T) and eye position in space (E) are combined to compute retinal error (re). At junction B, retinal error is added to a copy of eye position (ep) to reconstruct an internal copy of target position in space (tp). After passing through a gain element (G), this target position signal (tp′) is the input to the saccade generator (sg). The characteristics of the saccade generator are described by Robinson (20) and, for present purposes, it is sufficient to indicate that the saccade generator acts to reduce the difference between eye position (E) and an internal representation of target position in space (tp′) to zero. The Robinson model does not account for the presence of corrective saccades in the normal animal, and our model makes no attempt to do so either. It should also be noted that the model is a unilateral one; only eye movements on one side are considered.

position (E = 0) and a target appears at 30° eccentricity (T = 30). A retinal error (re) of 30° results. The retinal error is added to eye position (ep = 0) to produce an internal copy of target position (tp) equal to 30°. If the gain (G) of the system is one, then an internal copy of target position (tp′) of 30° is supplied to the saccade generator (sg). The saccade generator moves the eye until the difference between eye position and this internal representation of target position is zero; in this case, the eye moves to 30°. In this scheme, eye position would always match target position, and in the normal case, the slope of this relationship would also be one.

Now, if we impose the effect of the lesion on this signal by assuming a gain (G) equal to two-thirds rather than one, then the internal representation of the target position space (tp′) reaching the saccade generator is only 20°. The result is that the eye makes a hypometric saccade of 20°. After the saccade there is a residual retinal error of 10° which, when added to an internal copy of eye position, gives an internal representation of target in space (tp) of 30°. But this 30° is reduced by the gain element to a tp′ of only 20°, which is where the eye already is. Thus, the saccade generator receives no new command to move the eye. In short, after the lesion final eye position would remain proportional to target position and, since the gain of the system is less than one, eye position would always be less than target position, as seen in Fig. 8.

When it is assumed that the gain acting on the target position in space is reduced, this simplified model of visual-oculomotor processing predicts the four major observations from our experiments: *1*) the eye position achieved is less eccentric than the target position; *2*) fixations as well as centrifugal and centripetal saccades are affected; *3*) the errors are proportional to orbital eccentricity; and *4*) these eye position errors are not corrected by subsequent saccades. Limitations of the model are indicated in the legend of Fig. 11.

In contrast, the effects of lesions of the superior colliculus alone are consistent with an attenuation of the retinal error signal before it is added to eye position at junction B in Fig. 11. Our results, as well as those of others, suggest that colliculus ablation alone produces shortened initial saccades that are compensated by second corrective saccades; these second saccades tend to be larger and more frequent (9, 17). In summary, the model suggests that lesions of the superior colliculus affect an earlier stage of processing than do lesions of the pretectum and posterior-medial thalamus.

Anatomical organization of visual-oculomotor circuits

Recently, Schiller, True, and Conway (22, 23) showed that while lesions in either the frontal eye fields or the superior colliculus alone produce only mild disturbances in saccades on a task requiring the monkey to reach for bits of apple on a board in front of it, damage to both structures produces a

severe reduction in the frequency and amplitude of saccades. Following these joint lesions, the monkey made saccades only in the central 10–20° of the visual field. They concluded that a loss of saccadic eye movements results from combined frontal eye field and colliculus ablation and that these two oculomotor areas must function in parallel to produce saccades. While Schiller et al. (23) emphasized the deficit in amplitude of saccades, they noted that saccadic eye movements remained but occurred over a limited range and that the monkeys did not accurately look at stimuli. The narrowed range could, however, indicate an inability of their monkeys to achieve eccentric orbital positions so that, like our monkeys, there was no deficit of saccades per se but rather a deficit in the ability to reach eccentric orbital positions accurately with a saccade. On the basis of currently available data, it seems reasonable to suggest that the deficits in their experiments and ours may be qualitatively similar.

There are at least two possible oculomotor circuits that may explain the relationship between combined frontal eye field and superior colliculus lesions and the present results (Fig. 12). One explanation of the present results is that the midbrain-thalamic lesions in our monkeys disrupted two direct pathways to the saccade generator (Fig. 12*A*). This is consistent with the hypothesis of parallel pathways proposed by Schiller (23). Anatomical studies have shown that the superior colliculus and frontal eye fields have direct descending connections to the paramedian pontine reticular formation, one presumed site of the saccade generation (3, 7, 8, 11, 14, 15). Our lesions may have interrupted these direct connections from the frontal eye fields and superior colliculus by eliminating the colliculus itself and by interrupting those fibers from the frontal eye fields running via a dorsal route through the thalamus. One problem with this explanation is that both pathways from the superior colliculus and frontal eye fields appear to carry signals related to retinal error rather than target or eye position. The deficit produced by disruption of a retinal error signal, as we have noted, is not consistent with the position deficit we have found in our monkeys.

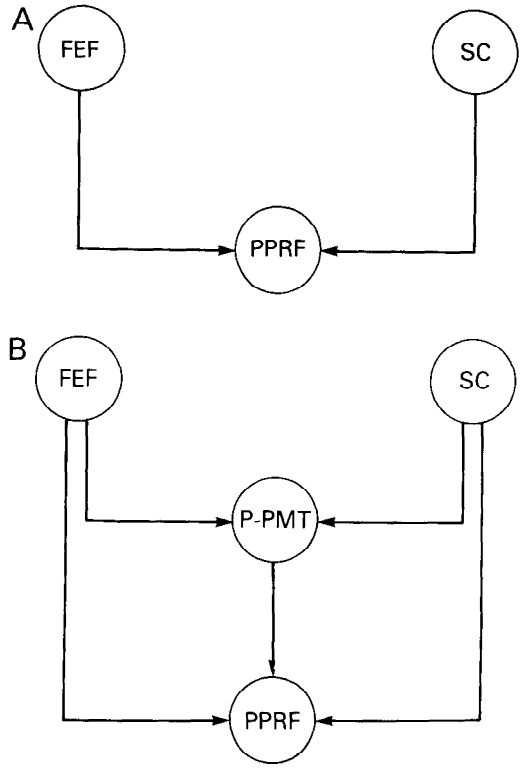

FIG. 12. Schematic drawing of two possible anatomical circuits related to saccade generation. In *A*, two direct pathways extend from the frontal eye fields (FEF) and the superior colliculus (SC) to the area of the pons related to saccade generation, the paramedian pontine reticular formation (PPRF). In *B*, an intervening area, possibly the pretectal posterior medial thalamus (P-PMT), lies in one pathway from FEF and SC to the PPRF. Deficits in matching eye position to target position that result from more extensive colliculus ablations involving areas in the pretectum and posterior-medial thalamus suggest an intermediate stage of processing between superior colliculus, the frontal eye fields, and the PPRF. Therefore, these deficits are consistent with the circuit in *B* rather than *A*.

Another possible explanation of our results is that the midbrain-thalamic lesion interrupted an indirect pathway that involves an intervening step before the saccade generator (Fig. 12*B*). This intervening area could be the posterior-medial thalamus, since it receives input from both the colliculus and frontal eye fields. Some cells in one area of the posterior-medial thalamus, the internal medullary lamina, discharge in

relation to saccadic eye movements in both cat and monkey (24–28). Some cells in this area also respond to visual stimuli but are modulated by the position of the eye in the orbit. The three monkeys with the most extensive anterior lesion had damage that extended laterally to the internal medullary lamina but probably not sufficiently rostral to include all of this region. However, the more lateral portions of these lesions would have involved fiber systems innervating this area and, therefore, may have deafferented or deefferented these areas. The lesions of Schiller et al. (22, 23) may have acted to deafferent this area with combined frontal eye field and superior colliculus lesions or may have included it with collicular lesions, as was the case in our experiments. If the hypothesis shown in Fig. 12*B* is correct, then future experiments should show that the pretectal-posterior medial thalamic area contains structures that are by themselves critical in the guidance of saccades to visual targets.

APPENDIX

The relationship between the output voltage of the search coil and the position of the eye in space (relative to the magnetic fields) can be simply computed in polar coordinates. In the schematic drawing in Fig. 13, the eye (E) begins a saccade from the origin (O) to an eccentric target (T) on the tangent screen. H represents the point along the horizontal axis of the tangent screen such that OHT and EHT are right angles. The primary axis EO is perpendicular to the tangent screen. Thus the angles EOH and EOT are right angles and θ, ϕ, α, and β represent azimuth, elevation, eccentricity, and direction, respectively.

From Robinson (19) we know that the vertical voltage output (V_y) and the horizontal voltage output (V_x) are related to azimuth (θ) and elevation (ϕ) by the formulas

$$g_y V_y = \sin \phi \qquad (1)$$

$$g_x V_x = \sin \theta \cos \phi \qquad (2)$$

where g_y and g_x represent scale factors used to adjust the gain of the voltage signals. These gain factors as well as any offsets due to angular deviation of the eye were estimated empirically by having the monkey fixate at several eye positions. In these equations the angles ϕ and θ represent the sum of the angles of the eye and the offset of the coil on the eye during prelesion testing.

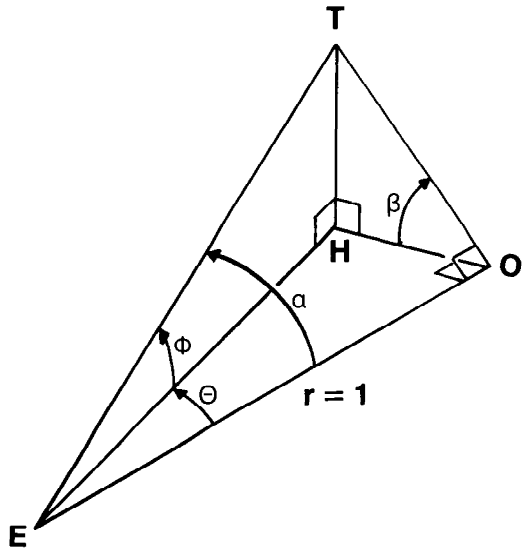

FIG. 13. Geometrical relations used to convert search-coil voltages to polar coordinates. See APPENDIX for details.

Using these relationships we can determine the relationship of α and β to the voltage output of the search coils. To determine α we are given that

$$\cos \alpha = \frac{\text{EO}}{\text{ET}}$$

From identities $\cos \theta = \text{EO}/\text{EH}$ and $\cos \phi = \text{EH}/\text{ET}$

$$\cos \alpha = \cos \phi \cos \theta$$

Squaring both sides and substituting

$$1 - \sin^2 \alpha = \cos^2 \theta \cos^2 \phi$$

$$\sin^2 \alpha = 1 - (\cos^2 \theta \cos^2 \phi)$$

$$\text{Substituting} = 1 - ((1 - \sin^2 \theta) \cos^2 \phi)$$

$$\text{Multiplying} = 1 - \cos^2 \phi + \sin^2 \theta \cos^2 \phi$$

$$= \sin^2 \phi + \sin^2 \theta \cos^2 \phi$$

Substituting *equations 1* and *2*

$$\sin^2 \alpha = (g_y V_y)^2 + (g_x V_x)^2$$

$$\alpha = \arcsin \sqrt{(g_x V_x)^2 + (g_y V_y)^2} \qquad (3)$$

To determine β we are given that

$$\tan \beta = \frac{\text{TH}}{\text{OH}}$$

From identities $\sin \theta = \text{OH}/\text{EH}$ and $\sin \phi = \text{TH}/\text{ET}$

$$\tan \beta = \frac{\text{ET} \sin \phi}{\text{EH} \sin \theta}$$

Substituting the identity $1/\cos \phi = ET/EH$

$$\tan \beta = \frac{\sin \phi}{\sin \theta \cos \phi}$$

Substituting *equations 1* and *2*

$$\tan \beta = \frac{(g_y V_y)}{(g_x V_x)}$$

$$\beta = \arctan \frac{(g_y V_y)}{(g_x V_x)} \qquad (4)$$

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