Activity of Superior Colliculus in Behaving Monkey. IV. Effects of Lesions on Eye Movements

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WE HAVE DESCRIBED a series of cells in the monkey superior colliculus related to visual stimulation and eye movement (4, 5, 17). In a vertical penetration through the superior colliculus the visual receptive fields and the movement fields of these cells are all topographically organized and all lie in the same area of the contralateral visual field. In the present experiments we destroyed a small volume of superior colliculus related to one area of the visual field in order to see if it was involved in the transfer of visual information to the eye-movement system. We recorded from cells in the colliculus with a microelectrode, located the receptive fields of these cells in the visual field, and had the monkey make saccades to spots of light within this area of the visual field. Then we ablated this part of the colliculus by passing current through the same microelectrode and determined how well the monkey could make saccades to points in the part of the visual field which was no longer represented in the superior colliculus.

METHODS

Four rhesus monkeys weighing between 3.6 and 11.8 kg were used. The monkeys had been trained to fixate a spot of light projected on a tangent screen in front of them and, if that spot of light disappeared, make a saccade to another spot. They had been prepared for single-cell and electrooculogram (EOG) recording as described previously (4).

Eye-movement measurement

The monkey's normal ability to make saccades to various parts of the visual field was carefully measured before any lesions were made using the following procedure. The monkey was trained to make a saccade to spots of light on a tangent screen 57 cm in front of him. The points were located on a 5° x 5° or 10° x 10° grid; the spots of light were 0.2° in diameter and 0.5 log unit above a background of 1.0 cd/m^2. The monkey first fixated a central light for a varying number of trials. This fixation light was on the tangent screen at a point on the vertical meridian 10° above eye level. During one of the trials the center light went off, another spot of light appeared in the periphery, and the monkey made a saccade to that point. The next three to five trials required saccades to the same peripheral point and then the next several trials required the monkey to fixate the central point again without making saccades. Fixation trials were then interrupted, the monkey's view of the screen was blocked, and the target point of the saccade was changed to another point on the grid of points. The monkey first saw the target point when he had to saccade to it. The order in which the grid points were presented varied from day to day. This set of eye movements, which were measured by EOG recording and stored on magnetic tape, provided a normal control with which we could compare the subsequent eye movements.

Focal lesions

In two monkeys a microelectrode penetration was made into the superior colliculus and the position and area of the visual receptive fields of cells in the penetration were determined. The microelectrode was advanced until it was at least 2 mm below the surface of the colliculus. The monkey's prelesion eye movements were measured according to the protocol outlined above. Both the area of the receptive fields of cells in the penetration and the reflection of that area across the vertical meridian were covered by the target grid. A lesion was then made, without further movement of the microelectrode, by passing 400 μa of direct current for 20 min with the microelectrode as the anode. The current removed the glass insulation for

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300–700 μ from the tip of the platinum microelectrode. The monkey evinced no discomfort during the lesioning procedure nor was any detectable eye movement evoked by the electrical stimulation. Immediately after the lesion was made, with the microelectrode still in place, the same set of eye movement measurements, which had been done just before the lesion, was repeated. The electrode was then removed. The eye-movement series was repeated daily for the first week and then at less frequent intervals.

Large lesions

In two additional monkeys the superior colliculus was located using microelectrode recordings, but larger lesions were made later with macroelectrodes. In one monkey an electrode was inserted serially at the corners of a 1 x 1.5 mm rectangle around the recording site, and 600 μa of direct current were passed at each point for 20 min. The second monkey was prepared under phencyclidine (1.0 mg/kg), anesthetized with pentabarbitol, and placed in a stereotaxic instrument; lesions were made on a similar grid using a radio-frequency lesion maker. At each point the lesions were considered complete when a thermistor at the tip of the lesioning electrode registered 70 C. Eye-movement measurements were obtained both the day before the lesion and the day after, and on subsequent days.

Three monkeys (one with a microelectrode lesion and two with macroelectrode lesions) had been used in previous experiments; a few microelectrode penetrations and several small electrolytic marking lesions such as those shown in the preceding paper (ref 17, Fig. 1) had been made in the superior colliculus contralateral to the side of the large lesion.

After the last series of eye-movement measurements, each monkey was anesthetized and perfused with saline and formalin, the brain examined histologically, and the extent of the lesion reconstructed from serial sections.

RESULTS

Focal lesions

We were unable to detect any deficit by simply observing eye movement or general behavior in either of two monkeys following focal superior colliculus lesions. When eye movements were measured both monkeys had a longer latency for a visually guided saccade, that is, the time between the appearance of the target for a saccade and the start of the saccade was greater after the lesion than it was before. This increased latency was limited to saccades made to points in the contralateral visual field. Figure 1 shows an example of such an increased latency. Figure 1*A* shows the horizontal EOG for a set of saccades to a point about 10° to the left of midline (top traces) and 10° to the right of midline (bottom traces) immediately before a lesion. Figure 1*B* shows the saccades to the same points on the day following a focal lesion in the left superior colliculus. The saccades to the left, the ipsilateral side, were unaffected; the

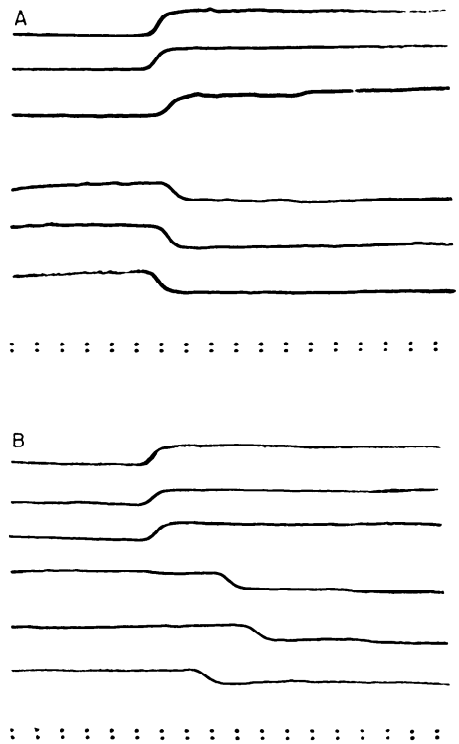

FIG. 1. Longer latency for saccades to visual field area where cells damaged by a focal lesion had their receptive fields. Eye-movement records in *A* were made before the lesion and show the EOG of the first three consecutive eye movements made to a point 10° to the left and 5° down, and then the first three eye movements made to the symmetrical point on the right. On the day after a lesion in the left superior colliculus the eye movements shown in *B* were made to the same point on the left (ipsilateral to the lesion, top three traces) and right (contralateral to the lesion, bottom three traces). Records are from the day after the lesion shown in Fig. 3. In this and subsequent figures, each trace is a horizontal EOG with a downward deflection indicating an eye movement to the right, up to the left; the time between successive dots on the time line is 50 msec.

saccades to the right, the contralateral side, now had a latency of 100–200 msec longer than the saccades to the left side. For both of these monkeys with focal lesions in the superior colliculus, the increase in latency for the saccade to a new fixation point was between 150 and 300 msec on the day following the lesion (an indication of this range for one monkey is given in Fig. 6). Occasionally the monkey did not make a saccade at all during the first trial using a new target point.

The accuracy of the eye movements was not altered by the lesion. In Fig. 1, for example, there are no more corrective saccades nor greater variation in amplitude of the saccade after the lesion than before. In addition the shape of the EOG of the eye movement, which would reflect the speed of execution of an eye movement, was not changed in the example shown in Fig. 1. The eye movements were sometimes slowed slightly (as for example, in Fig. 4*C*). This slowing of the execution of the saccade was inconstant and was gone within 2 days after the lesion while the latency deficit persisted. Thus in spite of a lesion in the superior colliculus, the monkey can accurately locate a point in space in order to make a normal saccade to that point; the monkey is just slow in doing it.

The deficit was found for eye movement to large areas of the contralateral visual field immediately after the lesion. However, by the second day after the lesion the deficit was limited to movements to that area of the field where the visual receptive fields of the destroyed cells had been located, and the size of that area remained about the same for a number of days. To determine the area of the deficit more exactly, we had the monkey make saccades to a series of points on both the ipsilateral and contralateral sides. Since the monkeys did not make perfect eye movements every time, not even on the prelesion days or in the control directions, we were forced to use an arbitrary scoring system to identify the deficit. On a given day for a given target, we compared the first three eye movements to a point in the ipsilateral field to the first three eye movements to the symmetrical point in the contralateral field. We defined the deficit as unequivocal when the latency of each of three saccades to the contralateral side was greater than all three saccades to the ipsilateral side; Figure 2*A* shows such a deficit. We regarded the deficit as marginal when the first contralateral saccade was longer than all three ipsilateral saccades (Fig. 2*B*); and we scored other sets of eye movements as showing no deficit (Fig. 2*C*). The area of the visual field which was associated with an unequivocal deficit (L, outlined by solid and dashed line in Fig. 2) was approximately the same area which had the receptive fields of cells ablated by the lesion (RF, outlined by dotted line).

The deficit is related to the retinotopic location of the target point, not to its position in the real world. As long as the monkey generated a saccade to a target in the

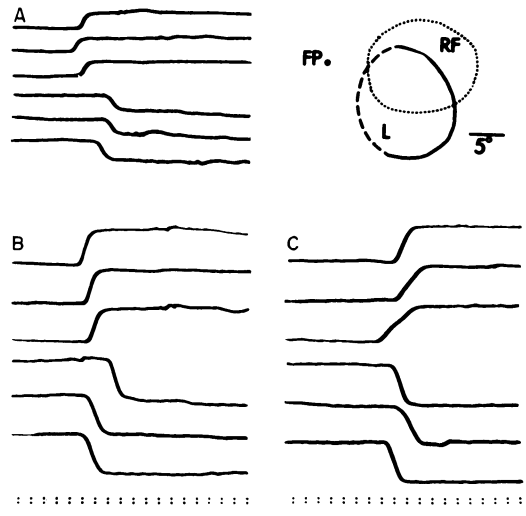

FIG. 2. Area of the visual field associated with the deficit due to the focal superior colliculus lesion shown in Fig. 3. The eye-movement records show consecutive eye movements to the ipsilateral side (top three traces in *A*, *B*, and *C*) and to the contralateral side (bottom three traces). *A* shows an unequivocal deficit, *B* shows a marginal deficit, and *C* shows no deficit. In the drawing the solid line outlines the area of the unequivocal deficit due to the lesion (L); the medial edge is indicated by a dashed line since too few points were available to determine this border exactly. The marginal points were beyond the solid line shown. The dotted line outlines the receptive field area (RF) of cells in the penetration determined before the lesion. This receptive field area was for cells studied 1–2 mm above the point of the lesion. The map of the lesion-effect area (L) was made on the second day after the lesion; traces in *A* are from the same point as is shown for the first postlesion day in Fig. 1.

retinal deficit area, the saccade showed an increased latency regardless of the initial position of the eye.

If the size of the target spot of light was increased from the usual 0.2° in diameter to a spot 0.7° in diameter, the size of the latency deficit remained unchanged regardless of spot location. The monkey had not been trained to recognize spots any larger than this as saccade targets.

Figure 3 shows the histological sections through the lesion which produced the deficit shown in Figs. 1 and 2. The damage is almost exclusively limited to the right superior colliculus, involves layers as deep as the intermediate white in the colliculus, does not extend into either pretectal or midbrain reticular areas, but does damage a tip of the hippocampus. The lesion in the other monkey was about the same size and was also limited to the superior colliculus except for a slight intrusion into the central gray and the corpus callosum.

Larger unilateral lesions

The selective effect of the focal lesions on the latency of a visually guided saccade might have resulted from the small size of the lesion. Since superior colliculus neurons have large receptive fields, receptive fields of undamaged cells might have overlapped the receptive fields of cells that were damaged by the lesion. To test this possibility, larger lesions were made in the superior colliculus of two monkeys. The effect of these larger lesions was like that of more focal lesions, and differed only in the extent of the visual field associated with the deficit. Figure 4 shows the eye movements on the day after radio-frequency lesions which destroyed nearly all of the right superior colliculus. There was an unequivocal increase in latency for saccades to all points in the contralateral visual field on the day after the lesion and on subsequent days. The accuracy of the eye movement was not affected, and the speed of eye movements was minimally affected; again the speed deficits recovered quickly, long before the latency deficits.

Figure 5 shows the extent of this lesion, which spared only a small amount of posterolateral superior colliculus, but which also invaded pretectum, central gray, and hippocampus. The other large lesion of the superior colliculus also damaged pretectal area, posterior thalamus, and corpus callosum anteriorly, and slightly invaded central gray, hippocampus, and cerebellum posteriorly; but the deficit in eye movements was the same.

Gross inspection of these monkeys' eye movements also clearly revealed abnormalities for several days following the lesion. One monkey (with the lesion shown in Fig. 5) showed only a paucity of spontaneous eye movements to the contralateral side. The other monkey tended to deviate his eyes to the side ipsilateral to the lesion, and made few eye movements to the contralateral side. These obvious effects disappeared within a few days in both cases, leaving only the latency deficits.

Time course of lesion deficit

The deficits were not permanent, but waned at times from 1 to 7 weeks after the lesion. Figure 6 shows the time course of recovery of the longest-lasting deficit, the one illustrated in Figs. 1, 2, and 3. As the latency deficit declined, the eye movements to areas of the visual field whose cells would be expected to lie at the borders of the lesion improved first. Thus, in the monkey with the large lesion deficit shown in Fig. 4 eye movements to the lower part of the visual field improved first, as one would predict since cells in the lateral superior colliculus were preserved, and these (see Fig. 5) have receptive fields in the lower visual field (4).

The exact duration of the deficit was difficult to determine since for some monkeys the eye movements to both sides of the field improved with practice (Fig. 6), while in other monkeys the eye movements to both sides became slightly less reliable.

DISCUSSION

In previous papers (4, 5, 17) we demonstrated a series of neuronal types in the monkey superior colliculus which fell along the continuum from pure sensory response to pure behavioral precursor. Cells in the superficial gray and optic layers all had large visual receptive fields. The response of roughly half of these cells was enhanced when the monkey was going to fixate a

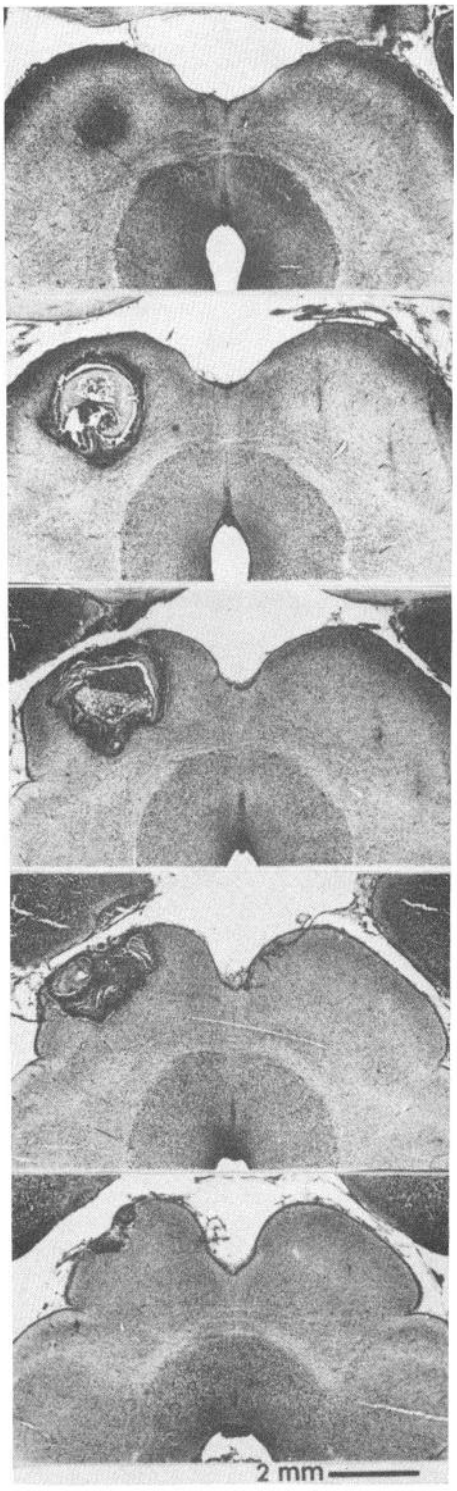

FIG. 3. Focal lesion of the superior colliculus made with microelectrode. The lesion was confined to the superior colliculus except for slight damage to the tip of the hippocampus. Coronal sections are cresyl violet-stained, and the five shown are 500–600 μ apart. The lesion extended a maximum of 150 μ anterior to the top section and 250 μ posterior to the bottom section.

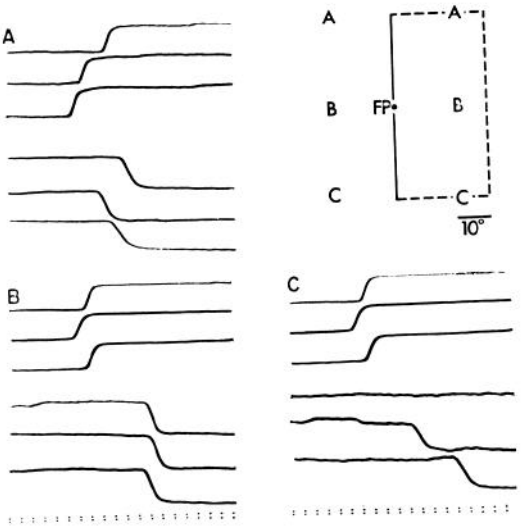

FIG. 4. Long-latency eye movements associated with a large unilateral lesion of the superior colliculus. All records are from the day after a large lesion was placed in the left superior colliculus. The top three lines in each section show eye movements to ipsilateral points *A*, *B*, and *C* shown in the drawing. The next three lines show records of eye movements to the symmetrical points on the right; there is a consistently longer latency for eye movements to these contralateral points. *A*, *B*, and *C* are 20° lateral from the fixation point (FP). *A* is 25° up and *C* is 25° down from the fixation point. In *C*, the monkey did not make a saccade to the point on the first presentation. Although the eye movements appear worse to *C* than to *A*, this is due more to variability in eye movements than to differences in the severity of the effect for these different places in the visual field. In fact the deficit recovered totally at *C* before it did at *A*.

stimulus in the receptive field. These cells had either no response to eye movement in total darkness or, for some, the background discharge was suppressed during saccades in any direction. In the intermediate layers cells with visual receptive fields also responded before eye movements to a given area of the visual field (the movement field) even in total darkness. Some cells, usually deeper in the intermediate layers, had only the movement precursor response. All of these collicular cells had a topographic organization in the colliculus such that not only were visual receptive fields mapped onto the contralateral colliculus in a point-

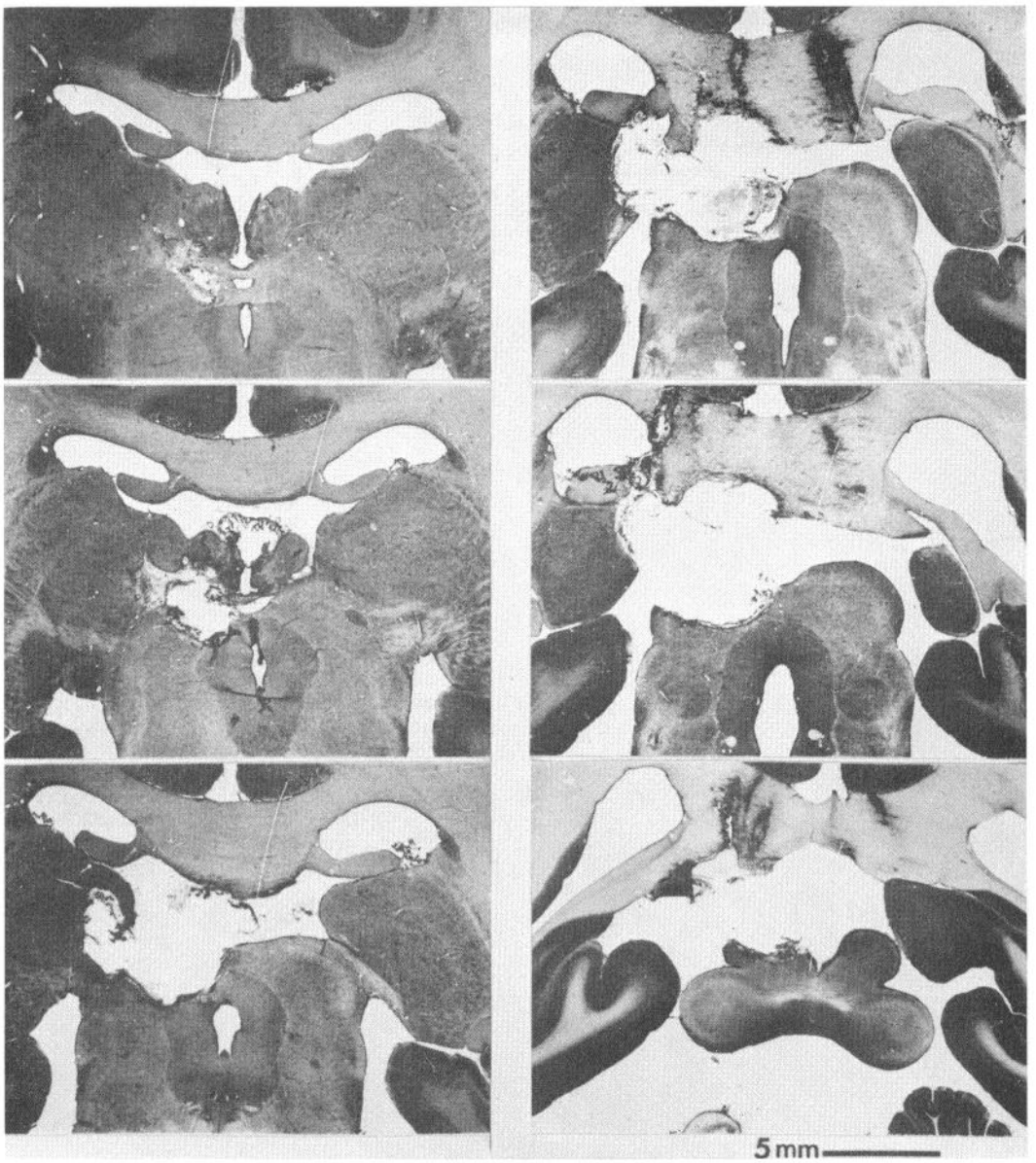

FIG. 5. Unilateral lesion of superior colliculus. Sequence of 50-μ sections reads down left column and then down right column. In addition to the superior colliculus the lesion extended into pretectum and posterior thalamus, hippocampus, and central gray. Sections are 700–1,700 μ apart.

to-point fashion, but also the target space of the eye movements. The two maps were congruent.

The presence of this ordered series of cells related to sensory input and motor output led us to agree with the old hypothesis that the colliculus had a function in the control of eye movements (17). Indeed, stimulation of the superior colliculus causes eye movement and the maps of target points of eye movements evoked by stimulation are similar to maps of the visual field (2, 10). However, the nature of the relationship of these neurons to the control of eye movements is not at all evident from the characteristics of their responses. The receptive fields are too broad to be of much use for exact localization of saccade targets in space

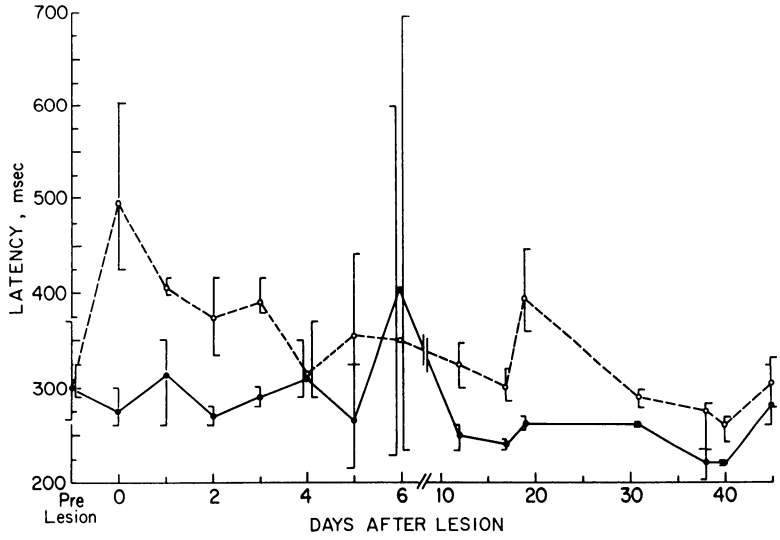

FIG. 6. Change of latency of eye movements with time following the focal lesion in the superior colliculus shown in Fig. 3. Average latency for eye movement to a point 10° over and 5° down on the side contralateral to the lesion are indicated by open circles; values for eye movements to a point 10° over and 5° down on the ipsilateral side are indicated by closed circles. Each mean is derived from values for the first three eye movements to each point and the range of these three movements is shown for each point. The contralateral target point was in the middle of the area associated with the deficit.

(4); the movement cells have far too large movement fields to be useful themselves in specifying saccadic eye movements (17). In order to test what function these cells might have, we examined the eye movements of monkeys in which lesions had been placed in the superior colliculus in an area which already had been identified by microelectrode recording as being concerned with a certain part of the visual field.

Monkeys with focal collicular lesions were able to make saccades to any point in the visual field immediately after the lesion; however, their eye movements to the contralateral visual field had a longer latency of onset than eye movements to the ipsilateral visual field. Within two days this deficit was limited to saccades to the area of the receptive fields of the cells that had been destroyed. Both the accuracy and the speed of the eye movements, once they had been initiated, were identical with both the prelesion controls and the ipsilateral eye movements.

The limited effect of the focal lesions was not due to the small size of the lesion which may have allowed survival of cells with fields lying in the deficit area. Monkeys with lesions destroying most of the superior colliculus had deficits in saccade latency that extended throughout the entire contralateral hemifield, but they also had no differences in the quality of the eye movement once it had been initiated.

Although in the laboratory a few hundred-millisecond deficit of latency for an eye movement may seem so trivial as to be negligible, the disadvantage of having no superior colliculus may be more significant in the wild where the object so inefficiently fixated might be a swinging branch or a looming predator.

These lesion experiments show that the monkey superior colliculus is not required for the accurate visual guidance of eye movements. Although it is still possible that the colliculus could contribute to such accurate guidance in conjunction with other systems, there is no evidence that it does so.

While the superior colliculus may have a number of functions in determining the behavior of a primate, we think our single cell and lesion data enable us to identify one central role related to a shift of attention and facilitation of eye movement. The primate fovea is by far the most accurate

part of the retina and is used to analyze objects in detail. By examining the path of successive fixations one can accurately establish what objects in the visual field a primate attends to (18). A saccade which changes the object of fixation must therefore be preceded by a shift of attention, so that the stimulus to be fixated becomes important to the animal before the eye movement occurs. If one considers that the phenomenon of attention is the selection of some sensory stimuli to be further analyzed at the expense of others, then the superior colliculus could be viewed as participating in the determination of which area of the visual field the monkey attends to. Since the superior colliculus has cells which change their discharge pattern according to the behavioral importance as well as the physical parameters of the stimuli (5), the colliculus may very well participate in indicating which part of the visual field is important. This specification of an area of the visual field need not be terribly exact, and the collicular neurons with large visual receptive fields could perform this function very well. Thus we can interpret the delay in fixating the target after a collicular lesion as a delay in the monkey's "noticing" the target; i.e., a difficulty in the monkey's shifting attention from objects in the area of the visual field being currently fixated to that which must be fixated next.

But the colliculus not only roughly locates the important objects, it helps initiate the movement necessary to fixate the objects. The movement cells which also have large fields would facilitate the oculomotor precursor neurons not for a specific movement, but for movements to a general area of the visual field. The brain must have other systems which can better specify movement parameters and stimulus location. In our model, the colliculus would roughly limit the area of the visual field in which these finer systems must act, and therefore limit the total time necessary to search out and specify the movement parameters necessary for fixation. In fact, destruction of the superior colliculus does not alter the speed or accuracy of an eye movement, it only lengthens the time necessary to initiate the eye movement.

This facilitatory function of the superior colliculus is analogous to a coarse focusing wheel on a microscope. Without the coarse focusing device, an observer can still use the microscope, but it takes him longer to focus on a slide because of the detailed analysis that must be wasted using the fine focuser. Similarly, without the superior colliculus, a monkey can make eye movements and fixate objects in the visual field, but it takes him longer to initiate the movement because the prior analysis to specify the target is less efficient in the absence of the colliculus.

The primate superior colliculus does not just have visual input nor does it just have oculomotor output. The monkey superior colliculus is known to receive somatosensory (6) and auditory afferents (7), and to have output to centers influencing neck musculature (8). In the cat superior colliculus Wickelgren (16) has found cells with both auditory and visual localizing features, and in the monkey we have found a few cells with both auditory localization fields and visual receptive fields but have not studied them in detail. It is therefore quite possible that the superior colliculus participates not only in shifting attention between objects in the visual world, but also in shifting fixation—and hence visual attention—to objects first appreciated as acoustic or tactile stimuli. Although we have only studied the superior colliculus during eye movements, it is probable that the information about shifting the visual receptors goes not only to systems concerned with eye movements, but with head and neck systems that are also concerned with the orientation of the visual receptors.

This formulation of a role of the colliculus in attention and movement is consistent with a number of previous experiments. Pasik, Pasik, and Bender (9) and Anderson and Symmes (1) found no deficits in spontaneous eye movements or the eye movements of optokinetic or vestibular nystagmus within a week of large bilateral collicular lesions. Our monkeys also soon lost any deficits which could be determined by informal observations of the eye movements while the latency deficit remained. While Denny-Brown (3) described monkeys

that "did not fix and looked approximately in the direction of the object," the lesions in these monkeys were not limited to the colliculus and the monkeys also had behavioral disorders which our monkeys with lesions limited to the colliculus did not have.

Schiller and Koerner (11) recently hypothesized that the superior colliculus is ". . . involved in the mechanism of foveation involving both target acquisition by saccades and target maintenance during smooth pursuit" and claimed that ". . . ablation of the superior colliculus in the primate results in the persistent inability of animals to accurately acquire targets in the visual world by saccadic eye movements . . ." Our conception is different; we think the colliculus is involved in a shift of attention and facilitation of movement to an area but not the coding of the exact location of the eye movement target, for two sets of reasons: one is that our monkeys with lesions can acquire the target perfectly but only take longer to start. The other is that properties of the cells are more consistent with an attention-shifting role than with a fine-guidance role.

Cats with unilateral lesions in the superior colliculus show a striking neglect of the contralateral world (15). They ignore stimuli in the contralateral visual field, and they walk in circles away from the damaged field. This is not a fixed deficit, however, because if the contralateral visual cortex is destroyed, the animal begins to look more normal (14). From the data on the collicular lesions Sprague and Meikle (15) postulated that the deficit in the animals with collicular ablations is one of attention, that the animal attends to the hemiworld overseen by the intact colliculus, and neglects the other. The monkey with a unilateral collicular lesion obviously does not show such a great neglect, but one can certainly interpret the latency deficit as a form of attention deficit.

Schneider (12, 13) recently showed that golden hamsters with bilateral collicular lesions were unable to locate and to orient to objects in space but could perform pattern discriminations which did not involve the animals' specifying where the correct pattern was. In contrast, monkeys with collicular lesions clearly have the ability to locate tiny objects in space and no ultimate deficit in orientation is apparent. However, they shift their attention between objects and orient more slowly than normal animals, and this more subtle deficit may be evolutionarily related to the more global deficit seen in the rodent.

SUMMARY

Two monkeys were trained to make a saccade from a central fixation light to a spot of light in the periphery. The visual receptive fields of cells along a microelectrode penetration in the colliculus were then determined and a lesion was made by passing current through the recording microelectrode. These focal lesions were largely confined to the superior colliculus and did not invade the pretectum, the inferior colliculus, or the opposite superior colliculus.

The lesion produced an increase in the latency for a saccade to a spot of light which fell in the area of the contralateral visual field where the lesioned cells had their receptive fields. The increase in latency was generally about 150–300 msec in the days following the lesion. No deficit in the accuracy of the saccades and only a transient change in speed of eye movement was observed. No changes in spontaneous eye movements or general behavior were detected.

Larger electrolytic lesions were made unilaterally in two additional monkeys; the same increase in latency of saccades with no loss of accuracy and slight effect on speed of eye movement was observed. The deficit was associated with eye movements to the entire contralateral visual field.

The magnitude of the increased latency and the area of the visual field to which eye movements were deficient was clear, and declined over time following either the focal or the larger unilateral lesions.

On the basis of our single cell and lesion experiments, we propose that the superior colliculus participates in shifting attention and facilitating eye and head movements responsible for orienting the visual receptors. In our model, the superior colliculus

would roughly limit the area of the visual field in which finer systems must act, and therefore limit the total time necessary to search out and specify the movement parameters.

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