Reversible Inactivation of Monkey Superior Colliculus. II. Maps of Saccadic Deficits

CHRISTIAN QUAIA, HIROSHI AIZAWA, LANCE M. OPTICAN, AND ROBERT H. WURTZ
Laboratory of Sensorimotor Research, National Eye Institute, Bethesda, Maryland 20892-4435

Quaia, Christian, Hiroshi Aizawa, Lance M. Optican, and Robert H. Wurtz. Reversible inactivation of monkey superior colliculus. II. Maps of saccadic deficits. J. Neurophysiol. 79: 2097–2110, 1998. Neurons in the superior colliculus (SC) are organized as maps of visual and motor space. The companion paper showed that muscimol injections into intermediate layers of the SC alter the trajectory of the movement and confirmed previously reported effects on latency, amplitude, and speed of saccades. In this paper we analyze the pattern of these deficits across the visual field by systematically comparing the magnitude of each deficit throughout a grid of targets covering a large fraction of the visual field. We also translate these deficits onto the SC map of the visual/movement fields to obtain a qualitative estimate of the extent of the deficit in the SC. We found a consistent pattern of substantially increased saccadic latency to targets in the contralateral visual hemifield, accompanied by slight and inconsistent increases and decreases for saccades to the ipsilateral hemifield. The initial and peak speed of saccades was reduced after the injection. The postinjection amplitude of the saccades were either hypometric or normometric, but rarely hypermetric. Although errors in the initial direction of the postinjection saccades were small, they consistently formed a simple pattern: an initial direction with minimal errors (a null direction) separating regions with clockwise and counterclockwise rotations of the initial direction. However, the null direction did not go through the center of the inactivated zone, as would be expected if the SC alone were determining saccade direction, e.g., with a population code. One hypothesis that can explain the misalignment of the null direction with the lesion site is that another system, acting in parallel with the SC, contributes to the determination of saccadic trajectory.

INTRODUCTION

The superior colliculus (SC) is one of the sites in the brain involved in the planning and execution of saccadic eye movements. Neurons in the intermediate layers of the SC discharge in association with both the appearance of a target (visual activity) and the movement that directs the fovea toward the target (motor activity) (see review by Sparks and Hartwich-Young 1989). Moreover, the collicular map is topographically organized, meaning that adjacent positions in the visual field correspond to adjacent loci in the SC. The saccadic vector is spatially coded in the SC; thus the SC locus activated, and not the firing rate of the neurons, encodes the position of the target in retinotopic coordinates (Sparks et al. 1976).

The companion paper showed that reversible lesions in the intermediate layers of the SC alter saccadic trajectory, even in cases where the saccades remained accurate (Aizawa and Wurtz 1998). These injections also produced deficits in the latency, peak speed, and amplitude of the saccades as reported previously for muscimol or lidocaine injections into the SC (Hikosaka and Wurtz 1985, 1986; Lee et al. 1988; Schiller et al. 1987; Sparks et al. 1990).

Whereas the companion paper dealt with how individual saccades were affected by the injections, in this paper we focus on the pattern of deficits observed across the visual field. Because the monkey made a series of saccades to a grid of targets covering a large part of the visual field, we had enough points to construct a map of these deficits. This map could be constructed in either visual field coordinates or collicular coordinates. However, such a map would look different depending on the coordinates used, because of the logarithmic warping of the collicular map (Robinson 1972). For example, large eccentricities are represented on a smaller portion of the SC map than are small eccentricities. As pointed out by McIlwain (1991), this would make the behavioral effects of inactivating the same size regions in the caudal and rostral SC appear quite different when mapped onto the visual field. Thus plotting the extent of the deficits in visual field coordinates is somewhat arbitrary, whereas plotting them in collicular coordinates more closely associates them with the effect of the lesion on the colliculus. Thus we decided to remap these deficits from the polar map of the visual field (Fig. 1A) onto the warped, logarithmic map of visual/movement fields of neurons in the SC (Fig. 1B).

As reported by Hikosaka and Wurtz (1985), but in contrast to other reports that found hypermetric as well as hypometric saccades (Lee et al. 1988; Sparks et al. 1990), we found a preponderance of hypometric saccades. We also found a consistent pattern of change in initial saccadic direction: the initial direction is rotated clockwise on one side of a null direction and counterclockwise on the other side. We will discuss these observations in relation to the SC center-of-activity hypothesis for determining the goal of the saccade, and to hypotheses of the contribution of the SC and other parallel systems to saccade generation.

METHODS

This report is based on the alterations in saccadic eye movements observed after muscimol injections into the SC of two rhesus monkeys (monkeys P and A) described in the companion paper (Aizawa and Wurtz 1998). All experimental procedures were described in that paper. The experimental protocols used were approved by the Institute Animal Care and Use Committee and complied with Public Health Service Policy on the humane care and use of laboratory animals.

Experimental data

In the present paper we analyzed the pattern of deficits for visually guided saccades made to a grid of targets distributed over a
Control saccades to the targets were recorded in sessions just before, or one or two days after, the injection. Statistically robust estimates of saccadic parameters for each amplitude and direction were obtained by pooling data from all of the control sessions (8 sessions for *monkey A*, 18 for *monkey P*). The number of trials for each saccade (after the data were pooled) ranged from 23 to 87, with an average of 60 trials/saccade. Targets at eccentricities of 2 and 50° had the fewest trials; targets between 5 and 40° always had >60 trials.

**Data analysis and visualization**

We constructed the maps of the changes of saccadic characteristics by interpolating between the sampled locations on the grid of target points using a two-dimensional linear (i.e., bilinear) algorithm. The final interpolated map was displayed by using grayscale (black, maximum value; white, minimum value; linear interpolation in between) images. To emphasize the difference between positive and negative values for changes in latency, occasionally a color scale was used (red, positive values; white, 0; blue, negative values).

A map of the change in saccadic characteristics was made for latency, peak speed, amplitude, initial speed, and initial direction for each injection. Latency was measured as the time between target onset and the initiation of the saccade. The beginning and end of the saccade were recognized using a template matching method (Waitzman et al. 1991). Latency changes were the difference between latency after and before the injections, expressed as a percentage of the preinjection (control) latency; thus a positive value represents an increase in latency. Saccadic amplitude was taken as the difference in the eye position at the beginning and end of the saccade, excluding any corrective saccades. Eye velocity was obtained by digital differentiation of the eye position signal (Usui and Amidor 1982). Peak speed was taken as the largest magnitude of the velocity during a saccade. Initial velocity was determined by first fixing the onset time of a saccade using a speed criterion (20°/s for 2° saccades, 40°/s for 5° saccades, and 50°/s for larger saccades), and then taking the instantaneous vectorial velocity at a time 2 ms after the onset time. The initial direction was defined as the direction of the velocity vector at the same time (i.e., 2 ms after the onset time).

**IPSILATERAL SACCADES.** The amplitude of the saccades to the ipsilateral hemifield was unreliable after the injection because of an artifact induced by the experimental paradigm. In this paradigm, each saccade started from a point that was in the visual hemifield opposite to the target, to make saccades over the center of the field (see Aizawa and Wurtz 1998). When the final target was in the hemifield ipsilateral to the injection, the monkey first had to make a saccade to the fixation point in the contralateral hemifield; this first saccade was controlled by the injected SC and was generally dysmetric. Thus saccades to a target ipsilateral to the injection started from a different location than saccades to the same target before the injection, i.e., the retinal error was different in the two cases. Now, the goal of this work was to study the effect of partial inactivation of the SC on saccadic eye movements, provided that the subject was attempting to make the same move before and after chemical injection. This was especially important for parameters that were sensitive to initial position (i.e., amplitude and initial direction). Therefore the changes in these two parameters were not analyzed for ipsilateral saccades.

**ESTIMATE OF SIGNIFICANCE OF BEHAVIORAL CHANGES.** As previously pointed out, because of the nonstationary effect of the muscimol on collicular activity, we were able to record only a few (no more than 5) postlesion trials for each target. Under such conditions it is clearly impossible to carry out any statistically meaningful test to determine whether a given saccadic parameter

---

**FIG. 1.** Mapping between visual and collicular reference frames. A: left visual hemifield in polar coordinates. Eccentricities from 2.5 to 50° are drawn at polar angle intervals of 30° for the left hemifield. B: projection of left visual hemifield on to the right superior colliculus (SC), based on a parametric fit by Ottes et al. (1986) to Robinson's (1972) stimulation map. The same eccentricities and polar angles are shown as in the visual hemifield map in A. Solid curves represent constant amplitude (eccentricity in degrees) on both maps; dashed lines represent constant elevation (polar angles in degrees). On the SC map, the abscissa represents distance along the rostral-caudal axis of the SC (in mm), and the ordinate represents the distance along the medial-lateral axis of the SC (in mm). Medial (corresponding to upward saccades) is plotted as a positive distance, and lateral (corresponding to downward saccades) is plotted as a negative distance. The polar coordinates of the visual hemifield are warped logarithmically onto the SC (see Appendix). The SC map is plotted with the rostral end (representing small saccades) at zero on the abscissa, and with upward directions corresponding to positive ordinate values.

---

A large fraction of the visual field rather than just the deficits for individual saccades. With one animal (*monkey P*) the grid used was particularly complete, with targets placed along radii at 15° intervals spanning 360°. The amplitude of the saccades ranged from 2 to 50° along each radius (although in some experiments the saccades to either the farthest or closest target locations were not recorded).

In 18 experiments on *monkey P*, enough trials were completed successfully to allow an analysis of the muscimol effects across the visual field [listed in Table 1 of the companion paper (Aizawa and Wurtz 1998), experiments 1–2, 5–8, 19–28, 35, and 37]. We did a similar analysis on eight experiments from the second animal, designated *monkey A* (in Table 1 of the companion paper, experiments 12–15 and 31–34). Although we were able to confirm in *monkey A* the observations made in *monkey P*, the distribution of target points for *monkey A* was much more limited, and the maps did not span as many directions as those for *monkey P*.

In the experiments with *monkey P*, which were the most extensively analyzed, the number of trials per target ranged from two to seven, with an average of five. However, as documented in the companion paper, the spread of muscimol throughout the experiment made the system nonstationary over time. Thus the effects of muscimol on the first and last trials studied (which were separated by 30–60 min) were different, with the deficits more pronounced in the last trials. To show the clearest change, we constructed the maps of the deficits averaging the parameters of the saccades only over the last three trials. A random order of target presentation ensured that affected and nonaffected saccades where produced evenly over the time course of the experiment.

---

**FIG. 1.** Mapping between visual and collicular reference frames. A: left visual hemifield in polar coordinates. Eccentricities from 2.5 to 50° are drawn at polar angle intervals of 30° for the left hemifield. B: projection of left visual hemifield on to the right superior colliculus (SC), based on a parametric fit by Ottes et al. (1986) to Robinson's (1972) stimulation map. The same eccentricities and polar angles are shown as in the visual hemifield map in A. Solid curves represent constant amplitude (eccentricity in degrees) on both maps; dashed lines represent constant elevation (polar angles in degrees). On the SC map, the abscissa represents distance along the rostral-caudal axis of the SC (in mm), and the ordinate represents the distance along the medial-lateral axis of the SC (in mm). Medial (corresponding to upward saccades) is plotted as a positive distance, and lateral (corresponding to downward saccades) is plotted as a negative distance. The polar coordinates of the visual hemifield are warped logarithmically onto the SC (see Appendix). The SC map is plotted with the rostral end (representing small saccades) at zero on the abscissa, and with upward directions corresponding to positive ordinate values.

---
was significantly affected by the muscimol injection. Consequently, we took a qualitative approach, analyzing the pattern of the deficits across several injections and looking for consistent effects. Nonetheless, we have attempted to provide an indication of the effects that could be considered as “normal variability” and the effects that should be imputed to the muscimol on collicular activity. We accomplished this by providing an indication of the variability present in the control trials.

We pooled the data collected during the different control sessions, grouping together the data for each target. We then computed for each group the mean (AVG) and the standard deviation (STD) for latency, initial speed, and peak speed. Throughout the paper we always analyze, for each target, the relative change between the mean value of the control (AVG) and mean value of the postinjection data (AVGP), i.e.

$$\text{Relative Change} = \frac{\text{AVG} - \text{AVGP}}{\text{AVG}} \times 100\% = \frac{\text{Change}}{\text{AVG}} \times 100\%$$

Consequently, to compare the value of the relative change between the control and the postinjection data with some measure of the variability in the control data, it is necessary to compute the relative standard deviation (RSTD), i.e., the STD as a percentage of the AVG

$$\text{RSTD} = \frac{\text{STD}}{\text{AVG}} \times 100\%$$

At this point we have a value of the RSTD for the saccades made by the monkey toward each target in the grid. To have a single measure for the complete set of controls, we then found the smallest value that is larger than the RSTD for 95% of the targets, and we called this the relative standard deviation for all the control saccades (RSTDC).

For monkey P we found that the RSTDC was equal to 26% for the latency, to 19.6% for the peak speed, and to 26.1% for the initial speed. For monkey A we did not pool the control data, because each experiment was targeted to a different and limited part of the visual field. Consequently, for monkey A different values of RSTDC were computed for each data set and are reported in the captions of the figures that use them.

On every figure in which changes in latency, initial speed, or peak speed are reported, we inserted a contour line that identifies the target locations for which the change between the pre- and the postlesion data are larger than the RSTDC for the corresponding parameter. This line is green in the color figure and black in the gray-scale figures. We want to emphasize that this contour line does not divide the map into significant and not significant changes, because that would require a statistical analysis on both the control and the postlesion data; the contour line only gives an indication of the variability in control trials.

This kind of measure was not provided for amplitude and initial direction because the vectorial nature of these parameters makes an indication of the variability even more arbitrary.

**Mapping saccadic deficits onto the SC**

To compare the effects of injections at different sites on the SC, we plotted the deficits seen for each variable onto a polar coordinate map of the visual hemifield (Fig. 1A). This visual map was then translated onto the warped, logarithmic coordinates of the SC (Fig. 1B), that had been determined by Robinson (1972) by correlating the site of collicular stimulation with the direction and amplitude of the movement evoked. We used a parametric map of the SC (Oates et al. 1986) to refer the saccadic deficits back to a site in the intermediate layers of the SC (see Appendix). As indicated in the Introduction, we presume that the deficits in saccades observed are related to the inactivation of a restricted part of the SC. Under this assumption, the use of the collicular map is clearly better suited to convey the extent of the SC affected by the injection. For example, suppose that 1 mm by 1 mm of the SC is altered by an injection; clearly the extent of the area affected mapped on the visual field will change depending on the location of the injection, because of the logarithmic distortion. Thus two identical lesions (same quantity and spread of the muscimol) centered at two different loci, would appear to produce a different effect if the analysis is performed in the visual field; this problem would clearly be avoided by performing the analysis in SC coordinates.

A further, even more important, benefit of remapping the data on the collicular map is related to the fact that saccades are accompanied by activity across a large part of the colliculus. To understand the effects of the muscimol, it is necessary to appreciate that both the lesion and the collicular activity associated with saccades are distributed across a population of cells. In both cases, it is reasonable to hypothesize that there is a central region that is maximally inactivated (or activated) and a surrounding area whose cells are progressively less inactivated (or activated). The overlap of these two distributions determines the extent of the deficits caused by the lesion. Thus even when saccades are directed to targets associated with maximal collicular activity at sites outside the area directly affected by the injection, part of the population normally activated may overlap the injected area. The effect of this overlap is that the region across which behavioral effects are observed is larger than the extent of the area affected by the injection.

To illustrate this concept, in Fig. 2A we plot, using a gray-scale map, a hypothetical distribution of muscimol on a collicular map. The amount of muscimol at collicular coordinates $(x, y)$ is assumed to have a Gaussian profile, centered at $(x_0, y_0)$

$$L(x, y) = K_s \exp \left[ -\frac{(x - x_0)^2 + (y - y_0)^2}{2\sigma_s^2} \right]$$

where $\sigma_s = 0.3$ mm. At the center of the affected area, the inhibitory effect exerted by the muscimol is set equal to $K_s = 800$ spikes/s to represent a substantial inhibition. To see how this injection would affect the global activity in the burst layer, we can now use the profile of activity reported by Munoz and Wurtz (1995) for burst neurons to derive the reduction in the global activity associated with saccades encoded by every collicular site. We used the following Gaussian profile to approximate the profile of burst neurons’ activation at different collicular sites, defined by the coordinates $(x_s, y_s)$

$$A(x, y, x_s, y_s) = K_i \exp \left[ -\frac{(x - x_s)^2 + (y - y_s)^2}{2\sigma_a^2} \right]$$

where $\sigma_a = 0.45$ mm and the peak value of activation is $K_i = 600$ spikes/s. The dashed circle in Fig. 2A represents the area activated $(A > 10$ spikes/s) during the movement associated with the center of the circle. The relative reduction in activation, $\Delta$, is determined by the overlap between the muscimol distribution and the spatial profile of activity associated with each saccade; $\Delta$ is defined for each point $(x_s, y_s)$ as

$$\Delta(x_s, y_s) = 1 - \int \int [A(x, y, x_s, y_s) - L(x, y)]^+ \, dx \, dy$$

where

$$[x]^+ = \begin{cases} x & x \geq 0 \\ 0 & x < 0 \end{cases}$$

A plot of $\Delta$ is shown in Fig. 2B. To better compare the relative extent of the muscimol and the reduction in neural activity, in Fig. 2C we superimpose a one-dimensional slice through the horizontal meridian of Fig. 2, A (dashed line) and B (solid line). Note that
the muscimol also affects saccades associated with sites not directly affected by the muscimol (e.g., the position of the circle in Fig. 2B) because of the large extent of the collicular region activated during saccades (dashed circle in Fig. 2A).

Now, as shown by Munoz and Wurtz (1995), the size of the region activated for different targets (e.g., the dashed circle in Fig. 2A) is essentially invariant in SC coordinates (although not in visual field coordinates). For this reason, the relation between Fig. 2A and B, would not change if the same injection were centered at a different locus on the colliculus; contrarily, this would not be true if the analysis were performed on the visual field, making the comparison of different experiments (in terms of the extent of the affected area) impossible. The analysis of Fig. 2 shows that from the reduction in global activation across the SC (e.g., Fig. 2B), the extent of the lesion can, at least qualitatively, be estimated; it is centered at the center-of-gravity of the deficit but has a smaller extent (e.g., Fig. 2A).

**Behavioral estimate of lesion extent**

Unfortunately, we have no way to directly measure the global activation of the SC; however, an estimate of the activation may be inferred from known relationships between SC activity and saccade parameters like latency and velocity. There is experimental support for the notion that the latency of saccades and the activation of the colliculus are negatively correlated. First, collicular injections of muscimol (Hikosaka and Wurtz 1985; Schiller et al. 1987) or lidocaine (Hikosaka and Wurtz 1986; Lee et al. 1988; Sparks et al. 1990), known to inhibit neuronal activity, induce an increase in latency for saccades associated with sites close to the injection. Conversely, Hikosaka and Wurtz (1985) showed that collicular injections of bicuculline, which disinhibits neuronal activity, produce a decrease in latency. Furthermore, increasing the frequency of electrical stimulation in the SC of either cat (Paré et al. 1994) or monkey (Stanford et al. 1996) decreases the latency of the evoked movements. Finally, there is a negative correlation between the level of activation of single neurons in the intermediate layers of the SC and the latency (Dorris et al. 1995, 1997). Although the observations are considerably less extensive, similar results have been obtained when peak velocity is considered: muscimol (Hikosaka and Wurtz 1985) and lidocaine (Hikosaka and Wurtz 1986; Lee et al. 1988; Sparks et al. 1990) produce a decrease in peak velocity, whereas bicuculline (Hikosaka and Wurtz 1985) induces an increase in peak velocity. Furthermore, an increase in stimulation frequency increases the velocity of the electrically evoked movement, although it also increases the movement’s amplitude, up to a maximum dependent on the site of stimulation (Paré et al. 1994; Stanford et al. 1996). Finally, there is a positive correlation between the velocity and the level of activation of SC neurons in the cat (Berthoz et al. 1986). Although either velocity or latency could be used to estimate the area of the SC involved in the muscimol lesion, we used latency as the indicator in our analysis because the relation between neuronal activity and latency has been more extensively documented.

These analyses were performed using MATLAB (The Mathworks, Natick, MA) programs running on a Challenge-L computer (Silicon Graphics, Mountain View, CA).

**FIG. 2.** Scheme illustrating the reduction of collicular activity as a function of the distribution of muscimol and of the activity profile of SC neurons. A: gray-scale map representing a hypothetical distribution of muscimol on the colliculus (see METHODS). Dashed circle represents the collicular region activated when the saccade associated with the center of the circle is made. A Gaussian profile is used to approximate the profile of burst neurons’ activation reported by Munoz and Wurtz (1995) (see METHODS). B: the gray-scale map represents the reduction in activity for saccades associated with any collicular site. For example, the value in the small circle in B is determined by the overlap of the collicular activity represented by the dashed circle and the distribution of muscimol shown in A (see METHODS). C: muscimol distribution (dashed line) and relative reduction in activity (solid line) along the horizontal axis (y = 0 in the SC map) are shown.
RESULTS

We found consistent patterns in the deficits in saccade generation following muscimol injections into the SC, and we have plotted these deficits onto a map in collicular coordinates. We first present these maps for changes in saccadic latency, peak speed, and accuracy, deficits that have been reported previously. The maps of these deficits, particularly the map of the latency deficits, then serve as an estimate of the extent of the injection’s effect (see METHODS). Subsequently, we plotted the newly recognized deficits in initial saccadic speed and direction in the same reference frame.

Saccadic latency

Change in latency after the injections was calculated as the difference between the average latency for saccades to each target after the injection and the average latency in control trials to the same target, expressed as a percentage of control latency. In Fig. 3 this difference is plotted as a pseudo-color image, where the color of a region corresponds to the change in the latency of saccades after the injection (see color scale). In this case, we used shades of red for increases in saccade latency and shades of blue for decreases in latency. Figure 3A shows the change in latency plotted on the map of the visual field, for targets between 5 and 50° eccentricity, after an injection in a site of the left SC corresponding to an eccentricity close to 10° in the right visual hemifield (cross). Figure 3B shows the corresponding collicular maps for the same injection. In both panels, the green line indicates the relative standard deviation of the control (RSTDC; see METHODS), which for this monkey corresponds to 26%.

In agreement with previous muscimol injection experiments, there were latency changes in both hemifields (Hikosaka and Wurtz 1985, 1986; Schiller et al. 1987). In the hemifield contralateral to the injection (right hemifield), there was a marked increase in latency for saccades to targets associated with collicular sites close to the site of the injection (cross), with smaller increases in latency for targets related to sites at greater distances from the injection. This contralateral increase in latency was large and consistent for all the injections. In the ipsilateral visual hemifield there were both increases and decreases in latency; however, they were always relatively small and did not form consistent patterns. In all the experiments these changes were smaller than the RSTDC.

The distributions of latency changes on the maps varied from experiment to experiment. Figure 4 shows the range of variation in the latency increases for the contralateral visual field and gives an indication of the largest (Fig. 4A) and the smallest (Fig. 4B) latency deficits observed. In the largest deficit the area of increased latency includes the injection site and extends almost to the most caudal region of the SC studied. The small injection (small or large injections refer to the observed behavioral effects, and not the quantity of muscimol) shows a latency deficit that is only slightly larger than the RSTDC. Figure 4C shows a widespread latency deficit even for an injection that was located in the rostral SC, as judged from the saccade evoked by electrical stimulation (cross). It should be noted that rostral injections were not directed at regions where fixation neurons were recorded, as was the case in a previous study (Munoz and Wurtz 1993b). Figure 4D shows a sample of a latency deficit for the second monkey that shows the same central area of
increased latency but over a smaller region because a smaller grid of targets was used. In this case no RSTDC line is present because for all the data points plotted the changes are higher than the RSTDC level.

To allow a comparison of the effect of muscimol on the different saccadic parameters, we will show the injection’s effect on each saccade parameter for the same injection as that shown in Fig. 3 (i.e., experiment pc12f).

**Reduction in peak speed**

As previously reported (Aizawa and Wurtz 1998; Hikosaka and Wurtz 1985, 1986; Lee et al. 1988), saccades made to targets associated with SC sites close to the site of the injection were slower than control saccades to the same targets. In Fig. 5A we plotted the decrease in peak speed for postinjection saccades relative to the peak speed for control saccades to the corresponding targets. The black line indicates the RSTDC for the peak velocity, which for this monkey is equal to 19.6%. The white line encircles the region(s) characterized by a change larger than 50%. A large area around the injection site was affected, whereas no consistent changes in the peak speed for saccades to the ipsilateral visual hemifield were observed (not shown).

One confounding factor in interpreting the extent of the decrease in peak speed is that most of the saccades after the injection were smaller than normal (as reported in the companion paper Aizawa and Wurtz 1998), and smaller saccades are normally slower than larger saccades (e.g., Bahill et al. 1975). To distinguish the slowing due to reduction of saccadic amplitude and the direct effect of the injection, we compared peak speed after the injection with peak speed of control saccades that were the same size as the postinjection saccades (instead of control saccades directed to the same target). In Fig. 5B we plot the changes relative to equal amplitude saccades; it is clear that the slowing of peak speed is partially accounted for by the slowing expected from the corresponding reduction in size but, as the white contour line (the 50% change line) reveals, this is primarily true for small saccades. Larger saccades are still much slower than expected for their size. Thus, even if the reduction in amplitude is taken into account, saccades are slower than normal after the injection. We found this to be the case for all of the injections analyzed.

The comparison of Fig. 5, A and B, with Fig. 5C shows that, as expected, there is an almost complete overlap between the effect on latency and on peak speed.

**Errors in saccadic amplitude**

Previous studies on collicular lesions have reported changes in the amplitude of saccades (Hikosaka and Wurtz 1985, 1986; Lee et al. 1988; Sparks et al. 1990); in the present experiments we confirm these dysmetrias and compare their extent to that of the latency deficits. Figure 6 shows the difference in the amplitude of control ($C$) and postinjection ($P$) saccades directed to the same targets. For clarity of the illustration, we plotted the control saccade ($C$) at the collicular site corresponding to the target location ($\tilde{T}$), and the postinjection saccade ($\tilde{P}$) at coordinates $T - (\tilde{C} - \tilde{P})$ (i.e., both symbols are slightly displaced, but without changing their relative position); we connected the two symbols with a straight line. For reference in Fig. 6, the map of the latency deficit is indicated by the gray background and roughly corresponds to the changes in saccadic amplitudes. To improve the contrast of the amplitude...
error vectors, and given the solely reference function of the latency plot, the gray scale used in this figure is lighter than that used in Fig. 5C.

The lines from the circles to the crosses in Fig. 6 are almost all directed toward the rostral SC, indicating that the postinjection saccades were consistently hypometric with respect to the control saccades. The same conclusion (i.e., predominant hypometria) can be drawn by looking at Fig. 7A, which also shows a substantial area of the SC affected by the injection as indicated by the latency deficit.

A previous study using lidocaine tested saccades within ~10° of the goal specified by the site of an SC injection (Lee et al. 1988; Sparks et al. 1990) and found that the saccades associated with SC sites rostral to the injection site were hypometric. In contrast, saccades associated with more caudal sites were hypermetric, and saccades associated with the site of the injection itself were normometric. In this study we found only one such pattern of hypometria-hypermetria (Fig. 7B). In all other experiments (17 of 18 for monkey P), only hypometria was observed. This result was confirmed in monkey A; however, we must point out that in four of eight data sets, the lesions were caudally located, and so no hypermetria was expected. Of course, many postinjection maps contain the occasional hypermetric saccades to some targets, but they do not form a consistent pattern relative to the site of the injection. Furthermore, those hypermetrias that are seen are much smaller in magnitude on the SC map than the hypometrias.

**Reduction in initial saccadic speed**

A novel finding in this study was the striking deficit in initial saccadic velocity that included a change in both initial speed and initial direction of the saccade. In the example shown in Fig. 8A, the distribution of the deficit in initial speed shows a pattern that is very similar to that of the deficit in peak speed for the same injection (Fig. 5A). In Fig. 8 the black line indicates the RSTDC, which was equal to 26.1%; the white contour line encircles the region(s) where the change in initial speed was >40%. Also in this case, the correction for the change in amplitude of the saccade (as in Fig. 5B for peak speed) accounts for part of the change in speed, but not for all, especially in the caudal part of colliculus (Fig. 8B). Again, no consistent changes were observed for saccades to targets ipsilateral to the injection site.

**Systematic error in initial saccadic direction**

The companion paper (Aizawa and Wurtz 1998) showed examples of curved saccades following muscimol injections; the saccades started in the wrong direction and then curved back toward the target. To quantify this change in trajectory for saccades made over the entire visual field, we compared the initial saccadic direction after the injection (Fig. 9A to the initial saccadic direction of the control (Fig. 9C). By comparing the postinjection initial direction to the control initial direction rather than to the overall direction of the saccade (Fig. 9, arrow B), we took into account any curvature in the preinjection saccades.

In Fig. 10 we plot the change in initial saccadic direction,
showing a gray-scale representation of the latency deficit for reference. The differences in direction are not quite as large as would be expected from the trajectories shown in the companion paper (Aizawa and Wurtz 1998), because the deficits reported here are the average over three saccades (see METHODS), whereas the first paper showed individual trajectories. The arrows around the circumference of the visual field show the average tipping along each direction; the arrow’s length subtends the difference between the control and postinjection saccades’ directions. Although initial direction errors were relatively small, they consistently formed a simple pattern. Within a range of directions, the initial direction for postinjection saccades (red arrows) was consistently tipped clockwise or counterclockwise with respect to the preinjection saccades (black arrows), as indicated by the circumferential arrows. A direction with minimal or null tipping was found between the regions with clockwise tipping and the regions with counterclockwise tipping, and we will refer to this as the null direction (Fig. 10A, cyan line).

To our surprise, the null direction did not pass through the center of the area affected by the injection (e.g., as indicated by the latency deficit map or the site of the injection). In Fig. 10A the null direction is only ~5° down and to the right, whereas the center of the affected region is lower in that quadrant. The change in initial direction in the contralateral visual hemifield (Fig. 10A, right) is mapped onto the left SC in Fig. 10B, and the average tipping vectors are plotted along the edge of the map. This display shows the large difference between the site of the injection (cross) and the null direction (cyan line). The initial directions of saccades to the visual hemifield ipsilateral to the SC injection were also altered (Fig. 10A, left visual hemifield), but these saccades were affected by the same problem described for saccadic amplitude, i.e., the saccades were made across the center, so that the first saccade was itself affected by the injection (see METHODS).

Figure 11 (A–C) shows similar deviations with injections in other parts of the SC. Thus the effect is not limited to a particular site. In Fig. 11D we show an example from the other monkey (A), where, even though the distribution of the targets is more limited, the effect is the same.

We found the lack of correspondence between the null direction and the center of the inactivated region in all the experiments, as shown in Fig. 12. Figure 12A shows the saccade direction corresponding to the center of the affected area, as determined by the effect on latency (△ for injections in the left SC, ◊ for injections in the right SC), and the null direction (○ for left SC, ● for right SC). The experiments are sorted using the position of the center of the affected area. Two conclusions can be drawn from this graph. First, the null direction is a function of the area affected by the muscimol, because nearby affected areas are associated with similar null directions. Second, in all but one case, the null direction is more horizontal than the direction associated with the center of the affected region (i.e., the circles in Fig. 12A are closer to the horizontal zero axis than the corresponding △ or ◊). This second observation is clearer in Fig. 12B, where the abscissa is the direction associated with the injection (inactivation direction), determined either by cellular activity or electrical stimulation at the injection site (×) or by the center of the affected area as judged from saccadic latency (△), and the ordinate is the null direction. If the null direction corresponded to the direction associated with the injection, the points in the scatter plot should lie along a line with unity slope (−·−·−·). In contrast, if the null direction is more horizontal than the injection direction, the line that best fits the data should have a slope smaller than 1, and this is what we found. When the injection site is used (×) to determine the inactivation direction, the line

FIG. 6. Deficits in saccadic accuracy. Errors in amplitude and direction are the differences between the amplitude of the control (○) and the amplitude of postinjection (+) saccades. The difference in these positions are indicated by the connecting lines. For clarity of the plot, each of these lines is displaced so that the origin of the line (○) corresponds to the target location (see text). Gray-scale image indicates magnitude of the latency deficit as an estimate of the extent of the injection; cross marks site of the injection as determined by stimulation. In this and subsequent maps, the scale for the latency deficit is not shown because it is used only for reference. The most common pattern of errors is a hypometria around the site of the injection, and normometria elsewhere (same injection as Figs. 3 and 5).
saccades to the visual hemifield contralateral to the SC injected with muscimol, and mild effects on latency for saccades to the ipsilateral hemifield. There were substantial decreases in initial and peak saccade speed. The saccadic amplitudes after the injection were almost exclusively hypometric or normometric throughout the visual field. Errors in the initial saccadic direction showed a systematic pattern of rotation, or tilt, of the initial direction away from a null direction. This null direction was related to, but not aligned with, the site of the injection.

We will first show how it is possible to reconcile the difference in the results regarding amplitude deficits obtained in this and previous studies (Lee et al. 1988; Sparks et al. 1990). We will then outline some hypotheses regarding the contribution of the SC to saccade generation that can

![Amplitude errors – pb03b](image1)

**FIG. 7.** Different patterns of dysmetria. *A*: injection with a smaller extent as judged from the latency deficit. Note that this example still shows a pattern of almost exclusively hypometric saccades. *B*: the only example of an injection showing consistent, but small, hypermetria for saccades to targets with vectors corresponding to SC sites more caudal than the injection site.

that best fits the points has a slope of 0.37 \( (r = 0.81) \) \( (---) \), whereas when the center of the affected area is used \( (\triangle) \) the slope is 0.40 \( (r = 0.76) \) \( (---) \).

Thus all injections showed a striking pattern of changes in the initial direction of the saccade such that the directions systematically rotated away from a null line. This null line, when plotted on the SC map, was more horizontal than the center of the injection, as indicated by either the injection site or a measure of the injection’s effect based on the latency deficit.

**DISCUSSION**

In this paper we have compared the spatial extent of the deficits in saccadic latency, amplitude, peak speed, initial speed, and initial direction following SC muscimol injections. We have found substantial increases in latency for

![Amplitude errors – pb16h](image2)

**FIG. 8.** Reductions in initial saccadic speed. Gray scale indicates relative deficit in percent. Black contour line indicates the RSTDC level (26.1%), whereas the white contour line indicates the 40% level. The cross marks the site of the injection as determined by stimulation. *A*: difference between control and postinjection saccades as a percentage of the control speed. *B*: changes in initial speed when the postinjection saccades are compared with control saccades of the same amplitude, as described for Fig. 5. When the white contour lines in *A* and *B* are compared, it is clear that the change in amplitude accounts only partially for the change in velocity.
crease. When the balance of activity shifts from fixation to burst neurons, the OPNs shut off and the saccade begins. In contrast, when a saccade stops, the balance between burst and fixation neurons must reverse again, and the OPNs reactivate to prevent saccadic oscillations (Zee and Robinson 1979). Thus the reactivation of the OPNs requires a mecha-

**FIG. 9.** Definition of initial and overall saccade directions. The circle is the starting point for the saccade; the square is the final eye position. Lines show sample trajectories for ideal (●●●), control (−−−), and postinjection (——) saccades. Initial direction for postinjection saccade is shown by arrow A, overall direction by arrow B, and initial direction for control saccade by arrow C.

account for the misalignment of the null direction with the center of the affected area.

**Determination of saccadic metrics**

The type of dysmetria seen in this study was almost always a hypometria (cf. Fig. 6), in agreement with that observed in other studies reporting the effect of collicular muscimol injections (Hikosaka and Wurtz 1985, 1986). In contrast, Sparks and colleagues (Lee et al. 1988; Sparks et al. 1990) reported that a pattern of hypometria and hypermetria is observed when small amounts of lidocaine are injected in the SC. In a brief report they showed that saccades made to targets corresponding to the injection site were normometric, saccades encoded by sites rostral to the lesion were hypometric, and saccades encoded by sites more caudal than the lesion were hypermetric. In other words, they found that the saccadic amplitude errors were distributed systematically, as if the center-of-activity of the population were used to determine the desired displacement. While our failure to find the pattern of hypometria and hypermetria surrounding normometric saccades could be taken as evidence against this center of activity mechanism, we think that such a conclusion is not warranted and that other, more parsimonious, explanations are more likely.

A possible explanation for the predominant hypometria observed after muscimol injections into the SC could be related to the mechanisms that start and stop the saccade. Saccades are generated by a pulse of activity in medium lead burst neurons (MLBNs) in the pons (see review by Van Gisbergen and Van Opstal 1989). The MLBNs are strongly inhibited by omnipause neurons (OPNs), which fire tonically between saccades and pause during saccades. Fixation neurons in the rostral SC also fire tonically between saccades, pause during saccades, and project to the OPNs (Büttner-Ennever and Horn 1994; Gandhi and Keller 1996; Munoz and Wurtz 1993a,b; Paré and Guitton 1994). It has been suggested (Optican 1994) that OPNs are controlled by the push-pull interaction of the fixation and burst neurons in the SC. Thus, to start a saccade, the burst neurons’ activity must increase, and the fixation neurons’ activity must de-

**FIG. 10.** Pattern of shifts in initial direction after injections. A: initial direction for postinjection (red radial arrows, A in Fig. 9) and control (black radial arrows, C in Fig. 9) saccades plotted on the visual field map. Initial direction is the instantaneous saccadic direction 2 ms after saccade detection (see METHODS). The base of the arrows is at the site of the corresponding saccadic target, and the arrow has an arbitrary length. After the injection, initial saccade direction tips away from the control direction in a consistent pattern. The arrows around the circumference of the visual field show the average tipping along each direction. The length of this arrow was calculated by taking the difference between control and postinjection directions (A and C in Fig. 9) for each of the 6 targets along each radius on the grid of visual targets, and taking the mean of these 6 values after excluding the outlier (the one value of the 6 values that maximized the variance). The base of the circumferential arrow is on the same radius around which the tipings are averaged. The direction of minimal tipping is called the null direction (cyan line). The gray-scale image represents the increase in latency, and the cross represents the site of the injection based on stimulation. Note that the null direction does not pass through the center of the affected area, as would be predicted by a population coding hypothesis. B: data from the contralateral (right) hemifield in A, mapped onto the left superior colliculus (SC). Arrows at the caudal end of the SC are the circumferential arrows plotted in A. Gray-scale image is latency map. Same injection as Fig. 3.
nism that inhibits the burst neurons synchronously with the end of the saccade. This mechanism must be able to account for normal variations in the level of burst neuron activation. After an SC lesion, if we assume that the reduction in burst neuron activity is larger than the range of activity levels that the system normally handles, then it is possible that the depressed burst neuron activity is not sufficient to keep the fixation neurons off long enough, allowing the OPNs to turn on and thus stopping the saccade too early. In this case, even if the saccadic goal were determined by a center-of-activity mechanism, saccades would be hypometric, simply because they would not reach the programmed goal.

This explanation could account for the different results observed by Lee et al. (1988), and the results reported by Hikosaka and Wurtz (1985, 1986) and essentially confirmed in the present study. In fact, the lidocaine injections of Lee et al. (1988) produced limited effects (the change in latency they observed was around 50–60%), were short acting, and possibly did not markedly affect the mechanism of activation of the OPNs. In contrast, our muscimol injections had much more dramatic effects (change in latency >100%) and might have disrupted the aforementioned OPN mechanism. Thus, although the desired ocular displacement may be determined by a center-of-activity computation, in our muscimol injection experiments this may have been obscured by interactions with other saccadic mechanisms (such as the reactivation of OPNs). If such a view is correct, when small (in terms of behavioral effects) muscimol lesions are considered one would expect to observe a pattern similar to the one reported by Lee and colleagues. In fact, in one experiment (Fig. 7B), in which the maximum change in latency was 60% (in the range of Lee et al. data), we observed a similar pattern of hypo- and hypermetria. Thus this experiment can constitute a bridge between the two data sets, which would thus form a continuum: the larger the lesion, the larger the disruption of the normal mechanism for saccade generation (which includes control of the OPNs), and the larger the predominance of hypometria over hypermetria.

To further support such an interpretation, it has to be pointed out that even in the Lee et al. (1988) study the hypometria dominates, even though to a lesser degree, over the hypermetria. Because the spatial extent of burst neurons’ activity is invariant on the SC, if saccade amplitude is determined by the position of the center-of-activity on the SC, the degree of hypometria/hypermetria should be ascertaind by plotting the data, as we did, in collicular coordinates, as first suggested by McIwain (1991) and as used by Lee et al. (1988) in describing the rationale for their experiment (see their Fig. 1). Now, when the data of Lee et al. (1988) is replotted on a collicular map, the symmetric pattern of dysmetria seen in the visual field must become asymmetric (as in our Fig. 7B). In particular, the degree of hypermetria must be smaller than the degree of hypometria, because of the logarithmic compression of the SC map, and thus the pattern of dysmetria observed looks similar to those reported in the present study (i.e., is not symmetric, contrary to the predictions of a theory based exclusively on the center-of-activity mechanism).

An alternative explanation of the hypometria could be based on interactions between the two colliculi. If mutual inhibition between the two colliculi (D. P. Munoz and H. Aizawa, personal communication) normally keeps the opposite colliculus off, then after the injection the noninjected colliculus might become active due to disinhibition. In this...
reaching the programmed goal. The effect of the size of the injection would still be the same: the larger the injection, the weaker the inhibition of the other colliculus, and the stronger the hypometria.

Determination of initial saccadic direction

The lack of alignment between the site of the injection and the null direction for the errors in initial direction was a surprise. As pointed out in the companion paper (Aizawa and Wurtz 1998), the output of the SC cannot be just a position signal, such as motor error or the desired eye displacement, because muscimol injections cause saccades to become curved. The SC must also control, or at least influence, the direction and speed of the saccade. However, if the SC were the sole provider of excitatory input to the brain stem burst neurons, one would expect muscimol injections to affect the initial direction of saccades in a very simple way. If the saccade is directed to a target corresponding to an SC site a little above the injection, it should be pushed further up; if the site to be activated is a little below the injection, it should be pushed further down, etc. In other words, the null direction, where there is no shift, should correspond to the site of the injection. This clearly would hold under the assumption that the chemical spreads symmetrically from the injection site; when this assumption does not hold, predicting the position of the null direction becomes more complicated. However, it is safe to say that, to a first approximation, the null direction should pass through the center of activity of the affected region (not necessarily the injection site), estimated using the effect of the injection on saccade parameters (e.g., latency, velocity).

There are several different hypotheses that can account for our finding that this is not what happens. One possible explanation is that the SC produces not only a position signal, but also a velocity signal, which we call a directional drive. Suppose now that the SC is not the sole provider of directional drive, and that there is another pathway that contributes to the velocity signal. These signals would be combined, perhaps as a weighted sum, to actually drive the saccades.

![Diagram](image URL)

**Fig. 12.** Shift of null-direction toward the horizontal direction. A: saccade direction corresponding to the center of the affected area, as determined by the effect on latency (△ for injections in the left SC, ◊ for injections in the right SC) and null direction (○ for left SC, ● for right SC) for each experiment are reported. Experiments are sorted using the center of the affected area (as determined by the effect on latency), so that nearby injections are displayed together. Note that injections in nearby sites are associated with similar null directions and that in all but one case the null direction is more horizontal than the direction associated with the injection site. B: the null direction is plotted vs. the direction associated with the injection site (inactivation direction), determined either by the injection site (×) or by the center of the affected area (△). The line that best fits the data has a slope smaller than 1, when either the injection site (———) or the center of the affected area (---) is used.
One interesting observation regarding the position of the null direction is that in 17 of 18 experiments it is tilted in the same direction, i.e., toward the horizontal direction (Fig. 12). To obtain this result with the parallel pathway scheme, we should suppose that, in normal conditions, the SC vector is always tilted toward the horizontal direction. In other words, the SC should, in normal conditions, provide more drive to the horizontal system than to the vertical system; the parallel system would then compensate for this bias, providing more drive to the vertical than to the horizontal system. Thus this scheme would resemble the model proposed by Van Opstal and Van Gisbergen (1989), which exhibited a horizontal bias in the motor drive provided by the SC.

There are other explanations for the misalignment of the null direction with the center of the affected area. One possibility is that the tipping of the null direction is due to the activation of the SC opposite to the injection site. For nearly vertical movements (i.e., those with upward or downward components larger than the horizontal component) both colliculi become active. The balance between the two determines the initial direction of the drive. For example, suppose a saccade is going up and slightly to the right (say, with an 80° elevation). This would require large upward drives from both colliculi, and nearly balanced leftward and rightward drives from the right and left colliculi, respectively. Then an injection into the left SC would remove some of the rightward horizontal drive, leaving the leftward drive from the right SC to dominate. Thus the postinjection saccades would have their initial directions twisted toward the vertical, which corresponds to a shift in the null direction toward the horizontal. This hypothesis depends on the outputs of the two SCs being summed downstream. One problem with this hypothesis is that for nearly horizontal movements, there would be no rotation of the saccade direction, because the opposite colliculus would not be active. However, it is possible that removal of the mutual inhibition between the two colliculi allows the opposite colliculus to become active (see above). This hypothetical mechanism for rotating the null vector away from the injection site could be tested by recording from neurons in the opposite SC after injections, and seeing whether their activity is consistent with the tipping of the null direction toward the horizontal.

Another possible explanation for the position of the null direction is compatible with the scheme recently proposed by Nichols and Sparks (1996). In their scheme, they proposed that the SC provides two outputs: the first is the position of the center of activity on the SC, which determines the goal of the saccade. The second output is a measure of the level of activation, and it is used to modulate the gain of the burst neurons in the brain stem. Now, if one supposes that the injection of muscimol affects, through this second signal, the gain of horizontal burst neurons more than the gain of their vertical counterparts, the tilting of the null direction toward the horizontal direction would naturally arise. The major problem with this explanation is that this would mean that the horizontal gain is always more affected than the vertical gain, regardless of the location of the injection site, and there is no reason to expect this. Thus, although there are other possible explanations, we think that the most likely explanation for the difference between the null and the inactivated region is the parallel systems hypothesis.

Conclusions

In conclusion, the saccadic dysmetria found in this study is compatible with the results of Hikosaka and Wurtz (1985, 1986) and Lee et al. (1988) by assuming that after an injection, saccade amplitude is determined both by a center-of-activity mechanism that selects the goal, and a pause-cell gate that terminates saccades. Furthermore, the shift away from the injection site of the null for the tilt in initial saccade direction suggests that a center-of-activity code in the SC is not sufficient to specify the initial saccade direction. Rather, another parallel path through the saccadic system may also

---

**FIG. 13.** Superposition of velocity commands from SC and another parallel system (PS) can explain pattern of errors in initial saccadic direction (ID). Arrows indicate vectorial contributions to the saccadic drive of the SC (green) and the PS (magenta), and their sum (black). In the normal case, the initial directions are correct, but this could be because both the SC and PS give the correct signal (e.g., upward direction), or because a bias in the PS vector is corrected by the SC (e.g., rightward direction). Note that the sum of the SC and PS vectors (ID) is about twice the length of either alone. After an SC injection (gray disk), the SC contribution becomes small and is tipped away from the locus of the injection, according to a population code. The green dashed arrows indicate tipping away from the null for the affected area (green dashed line). However, the PS still has a directional bias, so the initial direction vectors (black arrows) are tipped away from a null direction (blue dashed line) that is the sum (blue dashed arrows) of the tipping directions of the SC and PS. Note that the resultant initial direction is half the length of the normal resultant. The null direction for the shift (blue dashed line) does not go through the center of the injection site (green dashed line) because of the underlying bias of the parallel system.
contribute to the specification of initial direction. Thus many of the deficits seen after muscimol injections in the SC may depend on the interaction of the SC with other parts of the saccadic system.

APPENDIX

Suppose that visual space is mapped in polar coordinates, \((R, \Theta)\), in degrees (Fig. 2A), and the intermediate layers of the colliculus are mapped in cartesian coordinates, \((X, Y)\), in millimeters (Fig. 2B). The mapping from eccentricity in visual space onto the rostral-caudal axis of the SC is essentially logarithmic, probably reflecting the logarithmic warping of the cortical visual system (Schwartz 1980). An idealized SC mapping from visual space to the SC can be derived from Robinson's (1972) stimulation map of the primate SC. In the anisometric version used by Ottes et al. (1986), the equations relating collicular coordinates and retinotopic coordinates are

\[
X = B_x \log_2 \left( \frac{\sqrt{R^2 + 2AR \cos(\Theta)} + A^2}{A} \right) \tag{A1}
\]

\[
Y = B_y \tan \left( \frac{R \sin(\Theta)}{R \cos(\Theta) + A} \right) \tag{A2}
\]

Ottes et al. (1986) chose the parameters \((A=3.0^\circ, B_x=1.4 \text{ mm}, B_y=1.8 \text{ mm})\) to give the best fit to Robinson's (1972) stimulation map of the SC cortex.

Inverse conversion from millimeters on the collicular surface to degrees in visual space

\[
R = A \left( e^{2x} - 2e^x \cos(y) + 1 \right) \tag{A3}
\]

\[
\Theta = \tan \left( \frac{e^x \sin(y)}{e^x \cos(y) - 1} \right) \tag{A4}
\]

where \(x = X/B_x\) and \(y = Y/B_y\); \((x, y)\)-coordinates can be considered an isometric remapping of the anisometric \((X, Y)\)-coordinates.

We thank Dr. M. A. Basso for suggesting the hypothesis that errors in initial direction could be due to disinhibition of the noninjected colliculus. We also thank two anonymous reviewers for helpful comments about the manuscript.

Address for reprint requests: R. H. Wurtz, Building 49, Room 2A50, National Eye Institute, NIH, Bethesda, MD 20892-4435.

Received 20 June 1997; accepted in final form 2 December 1997.

REFERENCES


