Discharge of Saccade-Related Superior Colliculus Neurons During Saccades Accompanied by Vergence

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Submitted 2 October 2002; accepted in final form 22 March 2003


It has long been believed that the superior colliculus (SC) is involved in the production of saccades but plays no role in the generation of vergence eye movements. However, results from several recent studies suggest that it may be worthwhile to examine the role of the SC in saccade-vergence interactions. Specifically, the available literature suggests two questions: do saccade-related neurons in SC have threedimensional movement fields and is the slowing of saccades by vergence attributable, in part, to changes in the level of activity in SC? Single-unit data were recorded from 51 saccade-related neurons in rhesus monkey SC during saccades without vergence, saccades accompanied by convergence, and saccades accompanied by divergence. Most cells (78% for convergence, 86% for divergence) showed a significant reduction in peak spike density when the saccade was accompanied by vergence. A minority of cells (16% for convergence, 2% for divergence) increased their firing rate for saccades accompanied by vergence. Three cells were found that discharged in association with saccades, vergence, and the combination of the two. There were no cells that exhibited the pattern of discharge that would be expected of a cell tuned for saccades with divergence. Thus the present results do not support the hypothesis that saccade-related SC neurons are, as a rule, tuned in three dimensions. Small, but significant, differences in firing rate were often found for saccades without vergence at near and far distances. Approximately half of the cells showed a significant relationship between spike activity and saccade velocity, but the correlations tended to be very weak. This suggests that the decreased neuronal activity of SC neurons has only a limited effect on saccade velocity. For some cells, the movement field shifted for saccades with vergence. These shifts were highly variable from one cell to another.

1124 0022-3077/03 $5.00 Copyright © 2003 The American Physiological Society www.jn.org
between peak spike density and the firing rate of individual saccade-related burst neurons (SRBNs). On the other hand, Sparks and Mays (1980) showed that identical discharges (for a given SC neuron) may be associated with a wide range of saccades. Taken together, these results suggest that the level of activity in SC influences, but does not explicitly encode, saccade velocity (Goossens and Van Opstal 2000b). These observations suggest the possibility that the slowing of saccades with vergence might be associated with a decrease in the level of activity of SC.

**Role of superior colliculus in saccade—vergence interactions**

The traditional view has been that the SC is involved in the generation of saccadic eye movements but is not involved in the production of vergence (Wurtz 1996). Microstimulation of SC produces conjugate saccades but not vergence (Robinson 1972). Even if both SCs are stimulated simultaneously, the result is not vergence but a conjugate saccade that represents a vector average of the saccades that would have resulted from the activation of either site alone (unpublished observations). Although two studies in monkeys (Judge and Cumming 1986; Mays et al. 1986) found vergence-related neurons in the pretectum and one study in the cat has reported finding neurons in the rostral SC that modulate with pure vergence (Jiang et al. 1996), no unambiguous evidence exists for the existence of vergence-related neurons in the primate SC.

In recent years, however, a number of lines of evidence have suggested that this view of SC may at least be oversimplified. There is evidence from several sources suggesting that distance-related visual information may be sent to SC. At least four studies (in cat: Bacon et al. 1998a,b; Berman et al. 1975; opossum: Dias et al. 1991) have shown that superficial layer cells are sensitive to binocular disparity. Superficial to deep connections within SC have recently been demonstrated in tree shrew (Lee et al. 1997) and rat (Ozen et al. 2000).

The evidence for depth-related visual input from cortical visual areas to deep layer SC neurons is stronger. The lateral intraparietal area (LIP) is part of the so-called “where” visual pathway that encodes both the direction and distance of visual targets. This area also contains cells that appear to be involved in saccade planning. Gnadt and Mays (1995) recorded from single LIP neurons that were selective for eye movements to specific locations in three-dimensional space. This area sends dense projections to both frontal eye fields and SC (Cavada and Goldman-Rakic 1989a,b; May and Andersen 1986). Gnadt and Beyer (1998) were able to antidromically activate depth-related LIP neurons from deeper layers of SC. These authors suggested that saccade-related neurons in SC might be tuned in three dimensions.

Chaturvedi and Van Gisbergen (1997) have demonstrated that the saccadic system is capable of depth-specific adaptation. Using the intra-saccadic target displacement paradigm, these authors were able to induce opposite gain adaptation for targets closer to, or farther away from, the plane of fixation. Previous studies have provided evidence that the mechanism underlying saccadic adaptation involves structures upstream from the medium-lead burst neurons (MLBs) (Deubel 1987; Frens and Van Opstal 1994; Lemij and Collewijn 1992). On the basis of these studies and their own results, Chaturvedi and Van Gisbergen (1997) suggested that saccade-related neurons in SC and cerebellum might be tuned in depth.

Chaturvedi and Van Gisbergen (1999) found that stimulation of the intermediate layers of the caudal portion of primate SC during combined saccade+vergence movements caused an interruption of the vergence component. After the end of the pulse train, the vergence movement resumed until the eyes were on target. Once again, the possibility was suggested that saccade-related neurons in SC might be tuned in depth. According to this idea, microstimulation fails to elicit vergence movements because these cells are not topographically organized with respect to the vergence component. Stimulation of any given area in SC would activate roughly equal numbers of “saccade+convergence” and “saccade+divergence” cells, resulting in a net command for zero vergence change. Chaturvedi and Van Gisbergen (2000) showed that stimulation of the rostral portion of SC suppresses vergence movements with or without accompanying saccades.

Thus the available literature suggests two questions with regard to SC and saccade-vergence interactions: is the slowing of saccades with vergence reflected in a suppression of the discharge of saccade-related SC cells and are these cells tuned in depth? The present study was designed to address these questions.

**METHODS**

**Animals**

Five juvenile Macaque monkeys (Macaca mulatta) were used in this study, including four males and one female. Approval for this study was granted by the Institutional Animal Care and Use Committee at the University of Alabama at Birmingham.

**Surgical procedures**

Each monkey first underwent a 3- to 4-wk initial training period during which it learned to enter a primate chair and take juice from a drinking tube. After this initial training period was completed, animals underwent a series of four aseptic surgical procedures to prepare them for electrophysiological recording. Intramuscular injections of ketamine (Ketalar) were used to immobilize each monkey during preoperative procedures. During surgery, animals were intubated and maintained on 1–2% isoflurane. Heart rate, blood pressure, body temperature, and respiration were monitored constantly for the duration of each surgery. Analgesics were given for 1–3 days after surgery to minimize postoperative discomfort.

The first surgery consisted of attaching a metal post to the skull to allow the animal’s head to be fixed during training and recording sessions. First, two stainless steel strips were adjusted to match the curvature of the animal’s skull and then affixed with bone screws. Once these strips were in place, dental acrylic was used to attach the metal post to the upturned ends of these strips. In the second surgery, following procedures similar to those of Judge et al. (1980); an eye coil was implanted underneath the conjunctiva of the right eye to allow for the precise measurement of eye movements using the magnetic search coil technique (Fuchs and Robinson 1966). After this second surgery, animals were trained in simple target step conjugate saccade trials. Once the animals had been given enough practice to become proficient at this task, an eye coil was implanted in the left eye. After recovery from this third surgery, each monkey was trained to perform pure vergence eye movements as well as saccades accompanied by vergence. Finally, in the fourth surgery, recording cylinders were positioned bilaterally over 15-mm holes drilled into the skull. Dental acrylic was used to hold the head post and recording cylinders...
in place. The recording cylinders were each centered around a point 14.5 mm lateral to the midline and 1 or 2 mm rostral of ear bar zero, at a 20° angle with respect to the sagittal plane.

**Behavioral protocol and visual display**

Visual targets consisted of red Maltese crosses, subtending 1.2° of visual angle, displayed on computer monitors (90-Hz vertical refresh rate). The angular subtense of the monitors was ±20° horizontally and ±15° vertically. Target disparities of ±10° were generated through the use of a dichoptic system of mirrors (see Fig. 1). Accommodative demand was controlled through the use of movable, servo-driven lenses. To eliminate the problem of mechanical delays associated with the movement of these lenses, separate monitors were used for prestep and poststep stimuli for each eye (for a total of 4 monitors). The monitors were aligned so that corresponding points on different monitors were superimposed in the monkey’s field of view. Two cameras were aimed so that the animal’s eyes could be simultaneously centered in two video monitors to ensure proper positioning of the animal for the visual display.

During recording sessions, animals sat in a primate chair with their heads immobilized. An auditable tone signaled that a trial was about to begin. All trials began with the appearance of a fixation target, which the monkey was required to fixate within 500 ms. For all trial types, the fixation target remained on for 800–1,200 ms, during which time the animal had to keep its eyes within a imaginary, square, 10 × 10° window centered on the target. At the end of this time, the fixation target was extinguished at the same time that a peripheral target appeared. The horizontal and vertical coordinates of the peripheral target were manipulated to allow the movement field of each cell to be mapped. For each cell recorded, the amplitude and direction of the required saccades were selected to ensure that the range of data collection would be sufficient to obtain a good representation of the cell’s movement field. This was necessary because preliminary data analysis showed that movement fields may shift for saccades accompanied by vergence. Thus to ensure that the data points used for comparisons came from the same part of the cell’s movement field in each condition, it was necessary to plot movement fields for each condition for each cell. Due to limitations of the visual display system, recording sites were chosen with the goal of finding cells that preferred saccades of ±20°.

To properly evaluate the possibility that SC burst neurons might be tuned in three dimensions, it was necessary to be able to plot movement fields for saccades without vergence, saccades with convergence, and saccades with divergence. For example, if only two trial types (saccades without vergence and saccades with convergence) had been used, a cell preferring saccades with divergence would be indistinguishable from a cell that is suppressed by all vergence movements. It was also necessary to include saccade-only trials at both near distance and far distance. On saccade + divergence trials, the eyes start out converged and then diverged. If this had been the only trial type used in which the eyes start out converged, the monkeys would quickly learn to begin to diverge their eyes even before the target step. The inclusion of saccade-only trials at near distance avoids these anticipatory responses. In addition, it is useful to determine if there is an effect of static vergence angle. Such an effect might cause cells to fire more or less vigorously for saccades with vergence, even if the vergence movement itself has no effect.

**SACCADE-ONLY TRIALS AT FAR.** Both the fixation and peripheral targets appeared at optical infinity. Thus this trial type required the animal to make a saccade without vergence. (Hereafter, when the phrase “saccades without vergence” is used, it refers to this trial type.)

**SACCADE-ONLY TRIALS AT NEAR.** Target disparities of 8° (for monkey 254) or 10° (for all other monkeys) were matched with the appropriate accommodative demand so that fixation and peripheral targets appeared at near distance and at the same apparent distance. This trial type required the animal to make a saccade without vergence.

**SACCADE-CONVERGENCE TRIALS.** Target disparity and accommodative demand were manipulated so that the monkey was required to converge the eyes while making a saccade. For monkey 254, a vergence demand of 8° was used. For all other monkeys, vergence demand was 10°.

**SACCADE-DIVERGENCE TRIALS.** Target disparity and accommodative demand were manipulated so that the monkey was required to diverge the eyes while making a saccade. Here also, a vergence demand of 8° was used for monkey 254 and 10° for all other monkeys.

For a cell to be accepted for data analysis, ≥40 correctly performed trials had to be present for saccades without vergence and a minimum of 40 correctly performed trials from at least one of the two saccade + vergence trial types. Sufficient data were obtained for 40 cells for saccades without vergence and for both saccade + vergence conditions. Cells for which sufficient data were obtained in only one of the two saccade + vergence conditions (11 cells) could still be useful to investigate some of the issues relevant to this study; therefore these cells were included in most data analyses (exceptions will be described in the following text).

**Data acquisition and electrophysiological recording**

All data were acquired using custom software running on a PC 486 DOS-based system. Horizontal and vertical eye-position data from both eyes were acquired through the use of the magnetic search coil technique and sampled at 1 kHz. Single-unit activity was recorded from 51 saccade-related neurons in SC, using low-impedance (0.1–0.3 MΩ) tungsten microelectrodes (Microprobe). Unit data were filtered single-unit data >5 kHz. Unit data were passed through a window discriminator and stored for offline analysis. An additional channel was used to store the 20-kHz analog signal from the electrode. Electrodes were insulated with polyamide tubing and mounted in 26-gauge steel tubing. To penetrate the dura, 21-gauge hypodermic needles were used as guide tubes. Electrodes were held by an X-Y positioner and advanced by a hydraulic microdrive. High-frequency magnetic interference from the field coils was eliminated by filtering single-unit data >5 kHz. Unit data were passed through a window discriminator and stored on compact disks for offline analysis.

The criteria used to verify that the electrode was located in the SC were as follows: 1) neurons were found that displayed a high-frequency burst of spikes associated with saccades of a particular vector; 2) saccadic eye movements could be elicited by microstimulation at 20 μA; 3) electrode tracks and/or marking lesions could be observed in SC sections during histological reconstruction.

**Data analysis**

Data were analyzed off-line using custom software and SigmaPlot for Windows 2000. Calibration errors and nonlinearities in the search coil system were corrected by fitting eye-position data with a second-order polynomial.

For each correctly performed trial, the first saccade to the peripheral target was manually selected for data analysis. The custom software...
used allowed the following parameters to be measured and exported to SigmaPlot for further analysis: the size of horizontal and vertical components of the saccade for each eye; peak cyclopean eye saccade velocity, peak vergence velocity, number of spikes, peak spike density (described in the following text), burst duration, latency from burst onset to saccade onset, and latency from target onset to saccade onset.

If SC neurons are tuned in three dimensions, this should be reflected in the discharge characteristics of the cell for trials near the movement field peak. Various curve fitting approaches were tested, including three-dimensional Gaussian functions and modified three-dimensional Gaussians. These methods gave a poor fit to the data for some cells on saccade+ vergence trials. Because the movement field peak was of primary importance to the issues this study was designed to address, the data analysis focused on trials near the movement field peak. In general, independent groups t-test were used to compare data between the saccade without vergence at far and each of the other conditions.

For number of spikes, these comparisons were performed on the 15% of trials in each condition with the largest number of spikes. For all other parameters, comparisons were performed on the 15% of trials with the highest peak spike densities.

The inverse distance three-dimensional smoothing function in Sigma-Plot was used to generate movement field plots for the cyclopean eye for each condition for each cell. These plots were used to verify that the range of data collection had been sufficient to include the movement field peak.

Many of the analyses performed required a precise quantification of the timing of saccade onset and/or offset. Saccade onset was defined as the point at which cyclopean eye saccade velocity first exceeded 15°/s, whereas saccade offset was defined as the point at which this value first fell to 25°/s. For each trial, spikes were counted within a time window beginning 30 ms before saccade onset and ending at saccade offset. The size of this window was selected to exclude as much preburst activity as possible while still being large enough to include the entire burst for each trial. Independent groups t-test were used to perform pair-wise comparisons using data from the 15% of trials with the largest number of spikes.

Spike-density functions were calculated for each trial by dividing the measuring period into 1-ms time bins and computing a gaussian pulse for each bin in which a spike occurred. The Gaussian pulse for the k-th bin is given by the following equation:

$$g(k) = \frac{1}{\sqrt{2\pi}\sigma} \exp \left( -\frac{k^2}{2\sigma^2} \right)$$

(1)

These Gaussians were then convolved to produce a continuous function representing spike density

$$s(k) = \sum_{i=0}^{N} f(i) g(k - i)$$

(2)

where N is the number of time bins in the perisaccadic interval, and f(i) is the gaussian function for the ith time bin. A fixed value of 3 ms was used for the width of the gaussians. Spike-density functions have been commonly used as a means of obtaining a smoothed estimate of spike frequency (for the original reference, see Richmond and Optican 1987).

The spike-density function was used as a basis for the calculation of number of spikes and average peak spike density for the 15% of trials with the highest peak spike densities for saccades without vergence. As noted in the preceding text, Collewijn et al. (1995) showed that horizontal saccades are slower, and of longer duration, in humans when the saccade is accompanied by vergence. This suggests the possibility that SC neurons might show prolonged bursts with lower spike frequencies. To test this possibility, burst duration was calculated as follows: burst onset was defined as the last point in time before the peak at which spike density rose to a threshold of 50% of peak spike density. Burst offset was defined as the first point in time after the peak at which spike density fell to 50% of its maximum value. Thus burst duration was equal to the number of milliseconds between these threshold defined points. Burst duration for the 15% of trials with the highest peak spike densities for saccades without vergence was then compared with burst duration for the top 15% of trials in each of the other conditions.

### Results

**Reduction in neuronal activity for saccades with vergence**

The primary objective of this study was to investigate the role of the SC in saccade-vergence interactions. It was possible to obtain sufficient data from 45 cells (of 51 total) to characterize the neuronal response properties associated with saccades with convergence. Compared with saccades without vergence, 35/45 cells (78%) showed a statistically significant decrease, and 7/45 (16%) showed a significant increase in the average peak spike density for the 15% of trials with the highest peak spike densities ($P < 0.05$). For saccades with divergence, it was possible to obtain sufficient data from 44 cells, of which 38 (86%) showed a significant decrease in mean peak spike density ($P < 0.05$). Only one cell (2%) showed a significant increase in peak spike density for saccades with divergence. Averaging across all cells, saccades with convergence were associated with a 30% reduction in peak spike density, while saccades with divergence were associated with a 25% reduction. Figure 2 shows contour plots displaying movement fields for cell 01120.254 for saccades without (Fig. 2A) and with (Fig. 2B) vergence. The contour lines in this figure indicate peak spike density. Figure 3 shows single trials from cell 00430.713 for saccades without (A) and saccades with (B) convergence. The number of spikes and peak spike density were both suppressed for this cell for saccades with vergence. As reported in the following text, the movement fields of some neurons shifted for saccades with vergence. However, this cell showed no movement field shift for saccades with vergence; both trials are taken from the movement field peak. Figure 4A compares each cell’s response for saccades with convergence to its response for saccades with divergence. Data are expressed as a percentage of no vergence (far distance) control. The horizontal and vertical reference lines indicate 100% (or no change) for divergence and convergence, respectively. From this figure, it is clear that the overwhelming majority of cells showed lower peak spike densities for both convergence and divergence. The only cell that showed a significant increase for saccades with divergence showed no change for saccades with convergence.

A similar suppression of discharge was observed using number of spikes as a measure of cell activity. For saccades with convergence, 33/45 (73%) showed a significant reduction in number of spikes compared with 9/45 (20%) that showed an
increase. For saccades with divergence, 31/44 (70%) showed a reduction in number of spikes. Again, only one cell (2%) showed a significant increase (this was the same cell that showed a significant increase in peak spike density for saccades with divergence). Averaging across all cells, saccades with convergence were associated with a 31% reduction in the number of spikes, whereas saccades with divergence were associated with a 23% reduction. In general, as shown in Fig. 4B, the data closely resembled the results seen for peak spike density. The major difference between the results for number of spikes and peak spike density was that the minority of cells that showed enhanced responses forvergence movements tended to show greater increases in number of spikes than in peak spike density. This tendency primarily reflects the fact that the small number of cells that showed increased activity for saccades with vergence also tended to show weak or moderate increases in preburst activity on some saccade+vergence trials. However, there were three cells (described in the following text) that fired vigorously for vergence movements even in the absence of saccades.

In an attempt to investigate the reasons for this suppression of neuronal activity, it is useful to search for evidence of a relationship between peak spike density and various other factors, such as saccade velocity, saccade latency, intrasaccadic vergence, peak vergence velocity, and vergence motor error at the time of saccade onset. One potential problem was the fact that the relationship between saccade vector and peak spike density is so strong that weaker effects are likely to be hard to detect. To overcome this problem, it was necessary to statistically remove (as much as possible) the effect of saccade vector on peak spike density. The procedure used takes advantage of the fact that there is a roughly linear relationship \( r = 0.7 \) between the peak discharge rate of SC neurons and the distance of the endpoint of each saccade from the movement field peak (see Fig. 5, A and B). The data were then fit with a linear regression line. For a few cells, the range of data collection extended far enough beyond the edge of the movement field that a floor effect was observed. Therefore if peak spike density was \( <150 \) for all trials over a range of \( \geq 2^\circ \) along the abscissa (distance from peak), trials over this range were not considered for purposes of fitting the regression line for computation of normalized peak spike density. For each trial, normalized peak spike density was calculated by the following equation

\[
\text{Normalized peak spike density} = \frac{S}{S_{\text{predicted}}} \times 100 \quad (3)
\]

where \( S \) is the peak spike density for each trial, and \( S_{\text{predicted}} \) is the value of peak spike density predicted by the regression analysis. If a cell did not have enough trials for saccades with convergence, saccades with divergence were used for this analysis. This procedure could not be performed on one cell because the cell’s discharge was too weak for both convergence and divergence. Thus it was possible to calculate normalized peak spike density for 50 cells.

An added benefit of this procedure was that it provided an independent means of measuring the suppression of neuronal
activity in SC. To do this, the preceding normalization procedure was repeated for saccades with vergence with the following difference: peak spike density for each trial was divided by the value of peak spike density that would have been expected (based on distance from peak) for a trial in the no vergence condition. In other words, predicted peak spike density was taken from the regression analysis for the peak spike density–distance from peak relationship for the no vergence condition. This made it possible to express peak spike density for each trial as a percentage of what would have been expected for saccades without vergence. Calculating the mean of this value across all trials gave a measure of the amount of suppression of neuronal activity for the cell across the entire movement field. Figure 6 shows that this measure agreed well with data obtained by averaging data for the 15% of trials in each condition that showed the highest peak spike densities for each cell. On the x axis is plotted the suppression index for each cell; this was calculated by dividing the mean peak spike density for the top 15% of trials in the vergence condition (usually saccades + convergence) by the mean peak spike density for the top 15% of trials in the no vergence condition and multiplying...
the result by 100. On the y axis is plotted the average value (for each cell) of peak spike density, normalized with respect to saccades without vergence. For this analysis, the correlation coefficient was 0.75.

Effect of static vergence angle

It is possible that the suppression of neuronal activity on saccade + vergence trials might, in part, reflect a dependence on static vergence angle. To test this possibility, data were collected from 28 cells for saccades without vergence at far distance (optical infinity) and near distance (eyes converged by 8° for monkey 254 and 10° for all other monkeys). Fourteen cells (50%) showed significant reductions and 8 cells (29%) showed significant increases in peak spike density for saccades at near distance. Results for number of spikes were similar to those obtained for peak spike density: 9/28 cells (32%) fired significantly fewer spikes for saccades at near distance, and 6/28 (21%) fired significantly more spikes. The average change in peak spike density was 14%, and the average change in number of spikes was 22% for saccades at near distance. Figure 7 compares movement fields for cell 01201.254 for saccade-only trials at far (A) and near (B) distance.

Burst duration for saccades with and without vergence

Comparisons of burst duration for each cell showed that the suppression of discharge in SC neurons is not compensated for by an increase in burst duration. On the contrary, the tendency was for burst duration to decrease for saccades accompanied by vergence. In the saccade+convergence and saccade+divergence conditions, the discharge of some neurons was suppressed to the extent that the cell no longer exhibited saccade-related bursts. Therefore analyses of burst duration were only conducted on cells for which peak spike density >400 spikes/s for a minimum of 15% of trials in each condition. For saccades with convergence, 22 cells met this criterion. Of these, 10 (45%) showed significant reductions in burst duration. Two cells (9%) showed increased burst duration. These data are summarized in Fig. 8. For saccades with divergence, burst duration data were analyzed for 26 cells. Five of these (23%) showed significant decreases in burst duration. There were no cells that showed significant increases in burst duration for saccades with divergence. Static vergence angle had relatively little effect on burst duration. For this measure, 17 cells met the criterion for inclusion, with significantly shorter bursts seen in 3 cells (18%) and significantly longer bursts in 1 cell (6%).

SC activity related to saccadic velocity

As noted in the preceding text, Collewijn et al. (1995) have shown that horizontal saccades in humans are slowed by vergence. A similar reduction in saccadic velocity in monkeys may be due, in whole or in part, to the reduction in colliculus activity seen for saccades with vergence. The first objective was to determine if saccades are slowed in monkeys by comparing main sequence data for saccades with and without vergence. Results were highly variable from one animal to another and, in some cases, even from day to day, but generally
saccades tended to be slower on saccade + vergence trials. To quantity this, main sequence relationships were first plotted for saccades without vergence, saccades with convergence, and saccades with divergence. Data for each condition were fitted with a second-order regression equation and then normalized with respect to saccades without vergence, using the following equation

\[
\text{Normalized saccade velocity} = \frac{V}{V_{\text{predicted(nv)}}} \times 100
\]

where \(V\) is the peak saccade velocity for each trial, and \(V_{\text{predicted(nv)}}\) is the value of peak saccade velocity predicted by the regression analysis for the no vergence main sequence. This procedure allowed a direct comparison of data for the no vergence and saccade + vergence conditions. Table 1 shows these data for saccades with vergence for each monkey. For monkeys 941, 254, and 713, horizontal and vertical saccades were analyzed separately. For purposes of this analysis, saccades were considered to be horizontal if the direction was within 2° of horizontal; a saccade was considered to be vertical if the direction was within 2° of vertical. For monkeys 21 (Fig. 9A) and X01 (Fig. 9B), only a few cells were collected. Therefore for these animals, data from all saccades were analyzed together, regardless of direction.

Because saccadic velocity is so strongly related to saccadic size, it was necessary to use our normalization procedure to remove the effect of saccade size before it was possible to

### Table 1. Average normalized saccade velocity

<table>
<thead>
<tr>
<th>Monkey</th>
<th>Horizontal CV</th>
<th>Vertical CV</th>
<th>Horizontal DV</th>
<th>Vertical DV</th>
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<td>0.97</td>
<td>0.93</td>
<td>0.93</td>
<td>0.90</td>
</tr>
<tr>
<td>713</td>
<td>0.88</td>
<td>0.89</td>
<td>0.92</td>
<td>0.94</td>
</tr>
<tr>
<td>941</td>
<td>0.93</td>
<td>0.82</td>
<td>0.88</td>
<td>0.82</td>
</tr>
<tr>
<td>X01</td>
<td>0.84</td>
<td></td>
<td>0.93</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>0.90</td>
<td></td>
<td>0.98</td>
<td></td>
</tr>
</tbody>
</table>

Values are average peak saccade velocity, normalized with respect to saccades without vergence. CV, saccades with convergence; DV, saccades with divergence. Data for monkeys X01 and 21 are for saccades in all directions. Data for monkeys 254, 713, and 941 are given separately for horizontal and vertical saccades.
determine if normalized peak spike density was related to saccade velocity. This was done using the following equation

\[
\text{Normalized saccade velocity} = \frac{V}{V_{\text{predicted}}} \times 100
\]  

(5)

where \(V\) is the peak saccade velocity for each trial, and \(V_{\text{predicted}}\) is the value of peak saccade velocity predicted by the main sequence for that condition (no vergence, saccade+convergence, or saccade+divergence). Saccades with convergence were used for this analysis because the greater variability in saccade velocity reduced the likelihood of an effect being overlooked. A significant positive correlation was found between normalized saccade velocity and normalized peak spike density for 24/50 cells (48%). One cell (2%) showed a significant negative correlation. Figure 10A shows an example of the resulting data for one cell that showed a strong relationship between saccade velocity and peak spike density, cell 20926.21 \((r = 0.65\) for this cell). Across all cells, the average value was \(r = 0.22\).

These results show that decreases in saccade velocity are often associated with decreases in peak spike density. Because saccades are slower when accompanied by vergence, this suggests the possibility that this effect might explain the suppression of neuronal activity on saccade+vergence trials. However, the reduction in peak spike density was, in general, substantially greater than would be expected on the basis of the reduction in saccade velocity. To test this observation quantitatively, the relationship between normalized peak saccade velocity and peak spike density (normalized with respect to saccades without vergence) was plotted for saccades with and without vergence. This analysis was only performed on cells that showed a significant suppression of peak spike density for saccades with vergence \((n = 37)\). The two regression lines were found to be significantly different for 73% of cells on which this analysis was performed. Thus the suppression of SC activity was usually greater than would be predicted on the basis of the velocity effect alone. Figure 10B shows these data for one typical cell.

**Relationship between vergence parameters and reduced activity**

The preceding results indicate that factors other than saccade velocity must be largely responsible for the reduction in spike activity on saccade+vergence trials. It is possible that input from the vergence system might inhibit SC neurons. If this is the case, then the amount of suppression observed on any given trial should be related to vergence motor parameters. The relationship between peak vergence velocity and normalized peak spike density was investigated. Peak vergence velocity, however, is correlated with peak saccade velocity (C. Busettini and L. E. Mays, personal communication). An example of this relationship is shown in Fig. 11. To determine if peak vergence velocity is related to peak spike density, it was necessary to compute normalized peak vergence velocity, using the following equation

\[
\text{Normalized peak vergence velocity} = \frac{V}{V_{\text{predicted}}} \times 100
\]  

(6)

where \(V\) is the peak vergence velocity for each trial, and \(V_{\text{predicted}}\) is the value of peak vergence velocity predicted by the regression analysis. Of the 50 cells for which normalized peak spike density could be calculated, 11 (22%) showed a significant relationship between normalized peak vergence velocity and normalized peak spike density. This correlation was negative for nine cells (18%) and positive for two cells (4%). Overall, there was little evidence to suggest that peak spike density is related to vergence velocity for the majority of SC neurons. Across all cells, the average value was \(r = -0.08\).
The possibility of a relationship between intrasaccadic vergence (the vergence change during the saccade) and peak spike density was also investigated. Because the disparity of the visual target was held constant between trials, a dependence of spike activity on intrasaccadic vergence would provide evidence that the SC is, either directly or indirectly, inhibited by input from the vergence system. A significant negative correlation was found for 12/50 cells (24%). Two cells (4%) showed a significant positive correlation. Across all cells, the average value was \( r = -0.12 \). Thus the relationship between intrasaccadic vergence and firing rate appears to be very weak. For saccades with convergence, an average of 34% of the vergence change occurred during the saccade; for saccades with divergence, intrasaccadic vergence accounted for an average of 39% of the total vergence change.

Another possibility is that the activity of SC burst neurons might be affected by vergence motor error at the time of saccade onset (C. Busettini, personal communication). (Vergence motor error is equal to the difference between current and desired vergence angle.) The relationship between this variable and normalized peak spike density was examined. A significant positive correlation was found for 8/50 cells (16%), and a significant negative correlation was found for 3/50 cells (6%). Across all cells, the average value was \( r = 0.05 \). Thus the relationship between vergence motor error at saccade onset and neuronal activity was weak.

**Neuronal activity related to visual factors**

Although the data provide evidence that the activity of some SC neurons is related to vergence motor parameters, no evidence was found to suggest that this is the case for the majority of cells. This suggests the possibility that the observed suppression of activity in SC neurons might primarily reflect the fact that the visual target is outside the plane of fixation, instead of the occurrence of a vergence movement, per se. If this is the case, most SC neurons should fire less vigorously whenever the task calls for a vergence change regardless of whether a vergence movement coincides with the saccade. On the other hand, if the suppression of discharge is exclusively due to inhibition from vergence-related areas, then SC neurons should show no difference in peak spike density when there is no difference in intrasaccadic vergence regardless of where the visual target is. Although the monkeys are generally very good at performing these eye-movement tasks, there were some trials for which the eye movement that occurred was inappropriate for the trial type. For example, the animals would occasionally make a conjugate saccade on a trial that called for a combined saccade+vergence movement. In other trials, the animal would make both a saccade and a vergence movement but at different times. In either case, the saccade ended up being a conjugate saccade not the combined saccade+vergence movement that was required. This allowed a comparison of normalized (with respect to saccades without vergence) peak spike density for trials in which there were differences in the disparity and accommodative demand of the visual target but no difference in the vergence change during the saccade. To do this, data in each condition were sorted by intrasaccadic vergence. If the range of intrasaccadic vergence for the saccade+vergence condition overlapped with that for the no vergence condition, then the largest region of overlap for which the difference in mean intrasaccadic vergence was \( \leq 0.5^\circ \) was selected as the region for comparison (across all 15 cells, the average difference in mean intrasaccadic vergence for the selected regions of overlap was 0.2°). If this region of overlap consisted of six or more trials in each condition, then \( t \)-tests were performed on the mean normalized (with respect to saccades without vergence) peak spike density. Care was taken to ensure that the saccadic component of the movement was accurate even though the vergence component was not. If the suppression of SC activity is primarily due to the fact that the visual target is outside the plane of fixation, there should be a fairly good match between the amount of suppression found for this missed trials analysis and the amount of suppression found for all trials. As Fig. 12 shows, this is the case. The suppression index is calculated by dividing the average value of normalized (with respect to saccades without vergence) peak spike density for saccades with vergence by average normalized peak spike density for saccades without vergence and multiplying the result by 100. On the x axis is plotted the suppression index for the missed trials; on the y axis is plotted the suppression index for all trials. The correlation was equal to 0.75 (\( P = 0.0012 \)). As this figure shows, the suppression of neuronal activity for most SC burst neurons occurs when the visual target is outside the plane of fixation regardless of whether or not the saccade is accompanied by vergence.

**SC neurons that discharge for vergence in the absence of saccades**

We recorded from three cells that appeared to have both saccadic and vergence responses on the same cell. These cells burst for saccades and, as shown in Fig. 13, had saccade-related movement fields similar to those of other SC cells. All
three cells displayed very similar discharge properties. Figure 14 shows single trials from cell 00310.713. In Fig. 14A, the monkey makes a saccade without vergence. In such trials, the cell’s response was typical of saccade-related burst neurons in SC: a crisp, high-frequency burst of spikes begins 20 ms before saccade onset. Figure 14B shows a trial in which the animal was required to make a convergence movement without a saccade. The cell began to discharge shortly before the onset of the vergence movement. Peak spike density tended to be

![Graph showing suppression index for all and missed trials](image1)

**FIG. 12.** Comparison of data from missed trials to data from all trials. Suppression index is equal to the peak spike density for saccades with vergence divided by the peak spike density for saccades without vergence multiplied by 100. Peak spike density is suppressed if the visual target is outside the plane of fixation, regardless of whether or not the saccade is actually accompanied by vergence.

![Movement field for cell 00310.713](image2)

**FIG. 13.** Movement field for cell 00310.713. In addition to having a saccade-related movement field, this cell discharges for convergence movements, even in the absence of saccades.

![Graph showing single trials from a cell that discharged for both saccades and vergence](image3)

**FIG. 14.** Single trials from a cell that discharged for both saccades and vergence. A: on saccade only trials, this cell was indistinguishable from any other burst neuron in SC. B: the cell fired continuously during convergence movements, even in the absence of saccades. C: little or no discharge was observed when the animal made divergence in the absence of saccades. D: on saccade + convergence trials, the cell’s response was a combination of that seen in A and B. HR, horizontal position of the right eye; HL, horizontal position of the left eye; V, vertical eye position; VA, vergence angle; SV, saccade velocity; VVerg, vergence velocity.
∼200 spikes/s. The offset of this discharge was loosely associated with the offset of the vergence movement. As Fig. 14C shows, the cell’s response was weak or absent when the animal diverged its eyes without a saccade. When the task called for a combined saccade+convergence movement, the result was a combination of the responses shown in Fig. 14, A and B—the cell shows both a saccade-related burst and the convergence response, although the saccade-related burst is significantly suppressed (Fig. 14D).

**Movement field shifts**

Movement field shifts were sometimes observed for saccades with vergence. For most cells, these shifts were small and not significant. Average horizontal movement field shift was 1.4° for saccades with convergence and 1.0° for saccades with divergence. However, as Fig. 15 shows, shifts of several degrees were observed for some cells. Although the shifts were highly variable, some possible trends could be seen. When the horizontal shift was significant, the direction of shift was toward a larger horizontal component for 10/16 cells for saccades with convergence and 7/9 cells for saccades with divergence. Significant movement field shifts in the vertical dimension were found for 6 cells for saccades with convergence and 11 cells for saccades with divergence. These shifts were in the upward direction for 5/6 cells for saccades with convergence and 10/11 cells for saccades with divergence. No relationship was found between the suppression index and the size of the movement field shift ($r = 0.04$). The average eccentricity of movement field peaks for the no vergence condition was $8.3 \pm 4.3$° (mean ± SD) and ranged from 1.9 to 19.8°.

In addition, an attempt was made to determine if each neuron was monocular or binocular by analyzing movement field shifts separately for the two eyes in each condition. The goal was to determine if the movement fields for most cells were consistently associated with one eye or the average of the two (cyclopean eye). Unfortunately, the variability in move-
ment field shifts made it impossible to reliably classify individual neurons as monocular or binocular.

**DISCUSSION**

**SC movement fields in three dimensions**

One of the primary goals of the current study was to investigate the possibility that the primate SC might encode, by number of spikes and/or peak spike density, the depth component of re fixations in three-dimensional space. This idea implies that individual cells should fire their most vigorous bursts only for saccades of a particular vector, accompanied by particular vergence changes. If this was the case, then the current study should have found a mixture of cells preferring saccades without vergence, saccades with convergence, and saccades with divergence. Although several cells were found that burst more strongly when the saccade is accompanied by convergence, there were no cells that displayed the pattern of discharge that would be expected of a saccade+divergence cell. One cell, 00106.941, fired more strongly for saccades with divergence, but this cell also showed an enhanced discharge for saccades with convergence. The lack of any saccade+divergence cells in the present data argues strongly that saccade-related neurons in primate SC are not, as a rule, tuned in depth.

This would mean that another explanation would have to be found for the finding of Chaturvedi and Van Gisbergen (1999) that stimulation of deep layers of SC during combined saccade+vergence movements stops the vergence component for the duration of the stimulation. One possibility is suggested by the three saccade-related neurons that were found that also discharged during vergence even in the absence of a saccade. Although all three such cells described in the present study discharged during convergence and not divergence, C. Busettini (personal communication) has encountered cells that discharge during divergence (and not convergence) in the absence of a saccade. Thus although the typical saccade-related neuron in SC does not appear to be tuned in depth, it may be that microstimulation of deep layers may specify a command for “no vergence change” by activating equal numbers of convergence and divergence cells.

We also found that most SC burst neurons show significant changes in number of spikes and peak spike density associated with differences in static vergence angle, although this effect was somewhat inconsistent and tended to be smaller than the suppression of neuronal activity for saccades with vergence. The fact that some cells burst more vigorously and others less vigorously when the eyes are converged indicates that the suppression of SC activity for saccades with vergence is not simply a consequence of an effect of static vergence angle. Because data were only collected for two values of target distance for the initial fixation target (far distance and near distance), it was not possible to perform a detailed evaluation of the sharpness of tuning of saccade-related SC neurons with respect to static vergence angle. However, the fact that differences of 8 or 10° vergence angle usually were associated with small differences in spike activity implies that burst neurons are very broadly tuned with respect to static vergence angle. Nonetheless, it appears that some neurons burst more vigorously when the eyes are initially converged, and others burst more vigorously when the initial fixation point is near optical infinity, which suggests the possibility that the SC may carry signals related to static vergence angle.

**SC activity and the slowing of saccades with vergence**

SC burst neurons exhibit, on the average, approximately a 30% reduction in both number of spikes and peak spike density for saccades with convergence and approximately a 25% reduction for saccades with divergence. This effect was not compensated for by an increase in burst duration. This reduction in activity was weakly correlated with reductions in saccade velocity. Data from studies involving pharmacological inactivation of SC suggest that decreased activity of the burst neurons tends to cause a slowing of saccades (Aizawa and Wurtz 1998; Hikosaka and Wurtz 1985,1986; Lee et al. 1988; Quaia et al. 1998). In addition, decreasing the frequency of stimulation pulses results in a decrease in the velocities of stimulation-induced saccades (Stanford et al. 1996). Thus it seems very likely that the slowing of saccades with vergence is partially attributable to decreased output from SC. On the other hand, there are several reasons to suspect that this effect is largely due to events in other structures. First, it should be pointed out that this suppression of burst neuron activity occurred on many days when no slowing of saccades was observed. Second, correlations between normalized peak spike density and normalized saccade velocity, while sometimes highly significant, were not particularly strong. Finally, it should be kept in mind that lidocaine or muscimol injection might be expected to suppress SC far more than the 25–30% reduction in activity described in the present study. The present results, then, are consistent with the suggestion of Goossens and Van Opstal (2000) that, while changes in the activity of SC can influence saccade velocity, the actual metrics of the saccade are determined by other structures. Thus future studies should focus on other structures, such as the pontine parame- dian reticular formation (PPRF) and the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF), for a possible neurophysiological explanation for the slowing of saccades with vergence.

**Causes of the suppression of SC activity**

There are several possible reasons why saccade-related bursts might be less vigorous for combined saccade+vergence movements. Many neurons in SC have both visual and motor responses in the same cell (Mays and Sparks 1980). One possibility is that the visual responses might be less vigorous if the visual target is not in the plane of fixation. This would mean that burst neuron activity might be suppressed with vergence because of a partial or complete loss of the visual component of the response. However, this is unlikely to be a major cause of the observed reduction in activity for two reasons. First, the latency of the visual response of visuomotor cells is usually different from the latency of the motor response. Visual responses begin ~70 ms after target onset and are usually over within 120 ms. Saccade latencies in the present data were rarely <150 ms and were often ~200 ms. Because the measuring period began 30 ms before saccade onset, it would be rare for any visually evoked spikes to be included in the spike count. Second, robust reductions in spike
activity were often observed for cells that showed no evidence of a visual response.

A second possibility is that the activity of SC neurons might be influenced by saccade velocity. If so, then it might be that burst activity is suppressed with vergence because one or more saccade-related areas that project to SC are calling for slower saccades. According to this idea, the suppression of SC activity is only indirectly attributable to the presence of a vergence movement. If the SC is within the local feedback loop, then it is possible that events occurring downstream might influence the level of activity in SC. While this possibility cannot be completely ruled out, the data show that this explanation is unlikely to be the primary explanation for the result reported in this study. The suppression of neuronal activity was usually too large to be solely attributable to a velocity effect. A consistent relationship was found between saccade velocity and normalized peak spike density, but the aforementioned pharmacological studies suggest that the slowing of saccades is more likely to be an effect, rather than a cause, of the suppression of SC activity.

A third possibility is that the saccade-related bursts of SC neurons may be less intense if the visual target is outside the plane of fixation. This is not the same thing as suggesting that the suppression is due to the loss of a visual response. Mays and Sparks (1980) described a class of cells they referred to as visually triggered motor cells. As noted in the preceding text, these cells display saccade-related motor responses but only if a visual target is present. It should be mentioned that these cells have been consistently reported to represent a minority of saccade-related neurons in SC (Mays and Sparks 1980; Mohler and Wurtz 1976), whereas the present study found suppressed activity in ~75% of cells for saccades with vergence. Mays and Sparks (1980); for example, found only 3/53 burst neurons that did not burst at all unless the saccade was to a visual target and 8/53 cells that discharged weakly for saccades in the absence of a visual target. However, most previous studies have used strict classification criteria for classification of a cell as a visually triggered motor cell. Cells are usually only classified as visually triggered motor cells if they fire weakly or not at all for saccades in the absence of a visual target. Edelman and Goldberg (2001) found that the saccade-related discharge of many SC neurons is less vigorous when no visual target is present. The level of activity for the majority of these cells was found to be related to the latency from the offset of a visual target to the onset of the saccade. These authors suggested that the visually triggered motor cells described by Mays and Sparks (1980) might be at one extreme of a continuum of visual dependence. This suggests the possibility that the bursts of SRBNs and build-up cells might also be influenced by characteristics of the visual target, such as blur, disparity, size, and motion. If so, then the suppression of neuronal activity on saccade+vergence trials might be more attributable to a dependence of saccade-related activity in SC on accommodative blur and/or disparity than to the presence of a vergence movement per se.

Neuronal activity was still suppressed on trials calling for a combined saccade+vergence movement even if there was no difference in the intrasaccadic vergence between these trials and trials calling for saccades without vergence. This fact, combined with the lack of a consistent relationship between vergence motor parameters (including intrasaccadic vergence, which is a relatively direct measure of saccade-vergence interaction) and peak spike density, suggests that the activity of saccade-related neurons is suppressed on saccade+vergence trials primarily because these neurons are not activated as strongly if the visual target is outside the plane of fixation.

A fourth possibility is that the motor regions of SC might receive inhibitory input from vergence-related areas. If this is the case, then peak spike density might be corre...
found for only 12/50 cells. Even for these 12 cells, the correlation tended to be weak. While negative results should always be interpreted with caution, there are good reasons to believe that the data analysis procedures used in this study would have shown a stronger relationship between vergence parameters and neuronal activity if this was the primary reason for the decrease in number of spikes and peak spike density on saccade-vergence trials. First, it was possible to show a consistent relationship between saccade velocity and peak spike density despite the fact that the available literature suggests that this is a relatively weak dependence. Second, the results of measures of neuronal activity involving normalized peak spike density agreed well with the results obtained by simply comparing the mean peak spike density for the 15% of trials with the highest peak spike densities in each condition (Fig. 6). In contrast, analysis of data from 15 cells showed that the decreases in peak spike density for saccade-vergence trials were still present even when the intrasaccadic vergence was no different from that found in saccade-only trials. These results suggest that although the suppression of activity of SC neurons may be partially due to inhibitory input from the vergence system, this effect is likely to be primarily a consequence of the fact that the visual target is outside the plane of fixation.

Movement field shifts

The location of the movement field peak for some cells shifted for saccades with vergence. This phenomenon remains unexplained. Horizontal shifts were more commonly observed than vertical ones, suggesting the possibility that the shifts may be partially caused by downstream interactions with the vergence system. Overall, however, the size and direction of movement field shifts was highly variable from cell to cell. This made it impossible to reliably classify individual cells as monocular or binocular.

One possible reason for the inconsistency is that the size and direction of movement field shifts might be affected by more than one factor. This idea is supported by the observation that significant vertical (usually upward) shifts were observed for some cells, a result that is difficult to explain in terms of saccade-vergence interactions. Schiller and Sandell (1983) and Sparks and Mays (1983) reported that decreases in current strength caused decreases in the amplitude of saccades evoked by stimulation of any given site in SC. This implies that a decrease in the level of neuronal activity in SC might have the effect of slightly decreasing the amplitude of saccades resulting from activation of a given location in the collicular map. However, this would cause movement fields for the cyclopean eye to shift toward smaller saccades for combined saccade-vergence movements. This is in the opposite direction from the most commonly observed shift of the cyclopean eye movement fields.

Another possibility is suggested by the results of Stanford and Sparks (1994). These authors found that SRBN movement fields were shifted (often upward) for saccades to remembered targets. Systematic saccadic errors are known to occur on memory saccade trials (White et al. 1994). On such trials, Stanford and Sparks (1994) found that the activated region of the SC motor map predicted an accurate saccade not the erroneous saccade that actually occurred. It was concluded that systematic errors on memory saccade trials are the result of signals added or subtracted downstream from SC. In other words, the saccade that occurs is not necessarily the same as the one requested by SC. When saccadic and vergence signals are combined downstream from SC (at the level of the motoneurons), the eye movement in one eye becomes larger, whereas the eye movement in the other eye becomes smaller. If this effect is not equal in the two eyes, the result would be that movement fields for the cyclopean eye would shift for SC neurons. This could cause the actual saccade to be slightly different from the one requested by SC. This would be expected to produce a movement field shift for the cyclopean eye. Similarly, if slow vergence commands are combined with saccadic commands downstream from SC, a monocular cell’s movement field would be expected to shift slightly in opposite directions for saccades with convergence and saccades with divergence. Therefore it may be that the inconsistency in movement field shifts was the result of a combination of factors, including small size of the shifts, the extent to which any given cell is monocular or binocular, and the extent to which the amplitude and/or direction of the saccadic component of the movement is affected by downstream interactions with the vergence system.

Conclusions

The purpose of this study was to examine the possibility that the SC might play a more complex role in saccade-vergence interactions than simply providing the conjugate saccadic command. The available literature suggested two questions: do saccade-related neurons in SC have three-dimensional movement fields and is the slowing of saccades by vergence attributable, in part, to changes in the level of activity in SC? The present results indicate that most neurons in this structure are not tuned in three dimensions. Although a few cells might be considered to be very broadly tuned saccade-vergence cells, there were no neurons that showed the pattern of discharge that would be expected of a saccade-vergence cell. The great majority simply fire less vigorously for a saccade accompanied by any vergence movement. Although it is likely that this suppression plays some role in the slowing of saccades by vergence, the present data imply that the contribution of the SC to this effect is small.

Overall, the current data suggest that the SC mostly provides a conjugate saccadic command, even when the saccade is accompanied by vergence. The activity of some neurons in this structure is weakly related to vergence motor parameters, and a small number of cells were found that discharge for vergence movements in the absence of saccades. However, it is likely that the behavioral effects that are characteristic of saccade-vergence interactions have their neurophysiological basis primarily in other structures.

We thank S. Hayley for computer programming and J. Millican for technical assistance.

DISCLOSURES

This research was supported by National Eye Institute Grant to L. Mays (R01 EY-03463) and Core Grant P30 EY-03039

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