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Fixation Cells in Monkey Superior Colliculus I. Characteristics of Cell Discharge

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SUMMARY AND CONCLUSIONS

1. We studied the role of the superior colliculus (SC) in the control of visual fixation by recording from cells in the rostral pole of the SC in awake monkeys that were trained to perform fixation and saccade tasks.

2. We identified a subset of neurons in three monkeys that we refer to as fixation cells. These cells increased their tonic discharge rate when the monkey actively fixated a visible target spot to obtain a reward. This sustained activity persisted when the visual stimulation of the target spot was momentarily removed but the monkey was required to continue fixation.

3. The fixation cells were in the rostral pole of the SC. As the electrode descended through the SC, we encountered visual cells with foveal and parafoveal receptive fields most superficially, saccade-related burst cells with parafoveal movement fields below these visual cells, and fixation cells below the burst cells. From this sequence in depth, the fixation cells appeared to be centered in the deeper reaches of the intermediate layers, and this was confirmed by small marking lesions identified histologically.

4. During saccades, the tonically active fixation cells showed a pause in their rate of discharge. The duration of this pause was correlated to the duration of the saccade. Many cells did not decrease their discharge rate for small-amplitude contraversive saccades.

5. The saccade-related pause in fixation cell discharge always began before the onset of the saccade. The mean time from pause onset to saccade onset for contraversive saccades and ipsiversive saccades was 36.2 and 33.0 ms, respectively. Most fixation cells were reactivated before the end of contraversive saccades. The mean time from saccade termination to pause end was −2.6 ms for contraversive saccades and 9.9 ms for ipsiversive saccades. The end of the saccade-related pause in fixation cell discharge was more tightly correlated to saccade termination, than pause onset was to saccade onset.

6. After the saccade-related pause in discharge, many fixation cells showed an increased discharge rate exceeding that before the pause. This increased postsaccadic discharge rate persisted for several hundred milliseconds.

7. The discharge rate of fixation cells was not consistently altered when the monkey actively fixated targets requiring different orbital positions.

8. Fixation cells discharged during smooth pursuit eye movements as they did during fixation. They maintained a steady tonic discharge during pursuit at different speeds and in different directions, provided the monkey looked at the moving target.

9. We hypothesize that the fixation cells in the rostral pole of the monkey SC provide a signal related to active visual fixation. Activation of these cells would inhibit the activity of saccade-related cells in the rest of the SC and the saccade premotor circuitry in the brain stem. A prerequisite step in the generation of a saccade would be the reduction of activity in these collicular fixation cells.

INTRODUCTION

The monkey superior colliculus (SC) is critical for the generation of saccadic eye movements (for a review see Sparks and Hartwich-Young 1989). Neurons that discharge in relation to saccades (Schiller and Koerner 1971; Sparks 1978; Sparks et al. 1976; Wurtz and Goldberg 1971, 1972) are concentrated in the intermediate layers of the SC (Ma et al. 1991). These neurons are organized into a retinotopically coded motor map with the amplitude and direction of a saccade determined by the spatial distribution of neural activity on this map (Lee et al. 1988; McIlwain 1991; Sparks 1989; Sparks et al. 1976). Microstimulation of the monkey SC revealed a detailed motor map on the two colliculi (Robinson 1972) that is illustrated in Fig. 1. On this map the intermediate layers of each SC contains a representation of saccades to points in the contralateral visual field. Stimulation of the caudal SC elicits large-amplitude saccades, and movement of the stimulating electrode rostrally evokes smaller saccades.

The SC of the cat has recently also been shown to be involved in the control of visual fixation (Munoz and Guitton 1989, 1991; Munoz et al. 1991; Peck 1989). In the cat a subpopulation of collicular cells are maximally active when the cat fixates a target of interest. These cells are located in the rostral SC where the central visual field is represented and have an axon projecting into the crossed tectoreticulospinal tract via the predorsal bundle (Munoz and Guitton 1989, 1991; Munoz et al. 1991).

In the present experiments we have investigated the role of the SC of the monkey in the control of fixation. We identified a subset of neurons in the rostral pole of the SC (marked by a question mark in Fig. 1) that discharge tonically when the monkey actively fixates a target spot and pause during saccadic eye movements. In this paper we describe the discharge characteristics of these fixation-related cells. We hypothesize that these cells provide a fixation-related signal that can be used to suppress saccades. In the following article (Munoz and Wurtz 1993) we test this hypothesis by artificially activating and deactivating cells in this region with electrical stimulation and injection of GABAergic drugs.

Some of the findings described here have been reported in preliminary form (Munoz et al. 1990; Munoz and Wurtz 1992).

METHODS

We recorded from neurons in the rostral SC of three male rhesus monkeys (*Macaca mulatta*) that weighed between 5 and 12 kg. In

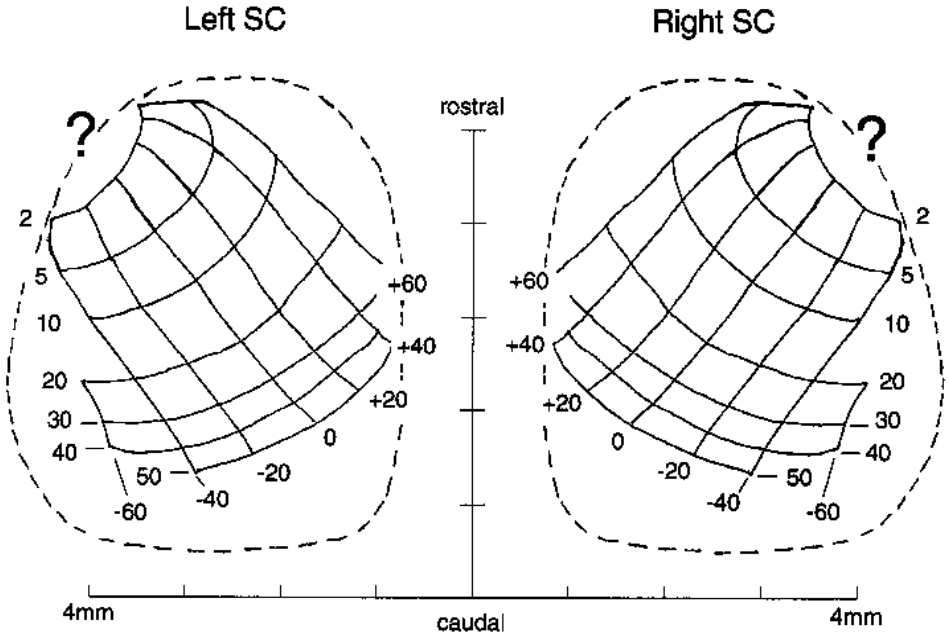


FIG. 1. Schematic motor map of the intermediate layers of the monkey superior colliculus (SC). Maps of the right and left SC show isodirection lines running from rostrolateral to the caudomedial SC (positive numbers are for upward directions, negative for downward), and isoamplitude lines. The question mark at the rostral pole shows the location of cells with a clear relation to fixation. Adapted from Robinson (1972).

the first two monkeys we studied the fixation- and saccade-related responses of cells encountered in the rostral pole of the SC. In the third monkey we verified the pursuit-related responses of fixation cells that were studied in only one of the first two monkeys. All experimental protocols 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.

Physiological procedures

The monkeys were prepared for experiments in a single surgical session. Anesthesia was induced with an intramuscular injection of ketamine and valium, and the monkeys were then intubated and maintained on isofluorothane anesthesia for the duration of surgery. Heart rate, respiratory rate, and body temperature were monitored. The surgical procedures for the implantation of eye coils to measure eye movements and the construction of a head implant have been published previously (Judge et al. 1980; Komatsu and Wurtz 1988). The implant included a stainless steel head holder for restraint of the head during the experiments, a stainless steel chamber for microelectrode recording, and the plugs from the eye coil leads. The recording chamber was tilted top backward 38° from the frontal plane, and centered over the midline between the two colliculi. After surgery, monkeys were monitored until they were fully awake, given Banamine for analgesia, and returned to their home cage. Monkeys were given 1–2 wk for recovery before the start of behavioral training.

Behavioral paradigms, visual displays, and storage of data were under the control of a PDP 11/73 computer running a UNIX-based real-time data acquisition system (REX) (Hays et al. 1982). The position of visual targets was controlled by the computer via D to A converters controlling an X-Y mirror galvanometer (General Scanning). Eye movements were recorded by the use of the magnetic search coil technique (Fuchs and Robinson 1966) with a resolution of 0.1°. Horizontal and vertical eye position channels were digitized at 500 Hz.

Single neurons were recorded by the use of tungsten microelectrodes (Frederick Haer) with impedances of 1–5 MΩ. Electrodes were driven through stainless steel guide tubes (23 gauge) by a micromanipulator (Narishige, MO-95) attached to the recording chamber. The guide tubes were held in position by a delrin grid that was fixed to the recording cylinder (Crist et al. 1988). Single-cell discharges were collected at 1 kHz via a window discriminator that produced a pulse for each valid spike that met both amplitude and time constraints.

Behavioral paradigms

The animals were trained to perform several behavioral tasks for liquid reward including saccades, smooth pursuit, and prolonged periods of fixation. Schematics of these tasks are illustrated in Fig. 2. During the experiments, monkeys were seated in a primate chair with their heads restrained for the duration of the experiment (3–6 h). A tangent screen was positioned 86 cm in front of the animal. The monkey had an unobstructed view of 70 × 70° of the screen (±35° from center in any direction). Each behavioral trial lasted ~2–3 s and was preceded by an initial 2- to 3-s period in which the screen was diffusely illuminated (1.0 cd/m^2) and the monkey was not required to fixate. At the start of each behavioral trial, the background lighting was extinguished, and the task was performed in total darkness except for the presence of the back-projected target spots produced by red light emitting diodes (LEDs; 0.3 cd/m^2). This light/dark cycle prevented the animal from becoming dark adapted. In each trial the background light was extinguished, and the monkey was left in darkness for 250 ms until a target spot, referred to as the fixation point (FP), came on at a point on the screen. The monkey had to look at the FP and maintain steady gaze (Fig. 2*A*). At this point the paradigms diverged into those requiring the monkey to maintain fixation (Fig. 2, *B* and *C*), that requiring the acquisition and pursuit of a second target spot that was moving at a constant velocity across the visual field (Fig. 2*D*), and those requiring the generation of a saccade to a second LED target spot (Fig. 2, *E–H*).

In the visual fixation paradigm (Fig. 2*B*) the monkey had to maintain fixation on the FP for up to 1,500 ms. In the fixation blink paradigm (Fig. 2*C*) the FP was momentarily blinked out (300–600 ms) on some trials to produce active fixation without a visual stimulus present.

In the smooth pursuit paradigm (Fig. 2*D*) the FP was turned off, and a second target (T) appeared either 1 or 2° away from the FP and immediately began to ramp back toward the location of the FP at either 8 or 16°/s, respectively, for 1.2 s. The monkey was required to look at the moving target and continue to follow it to receive the reward.

In the saccade paradigms the cue to initiate the saccade was the offset of the FP. In all of these paradigms, the monkey was re-

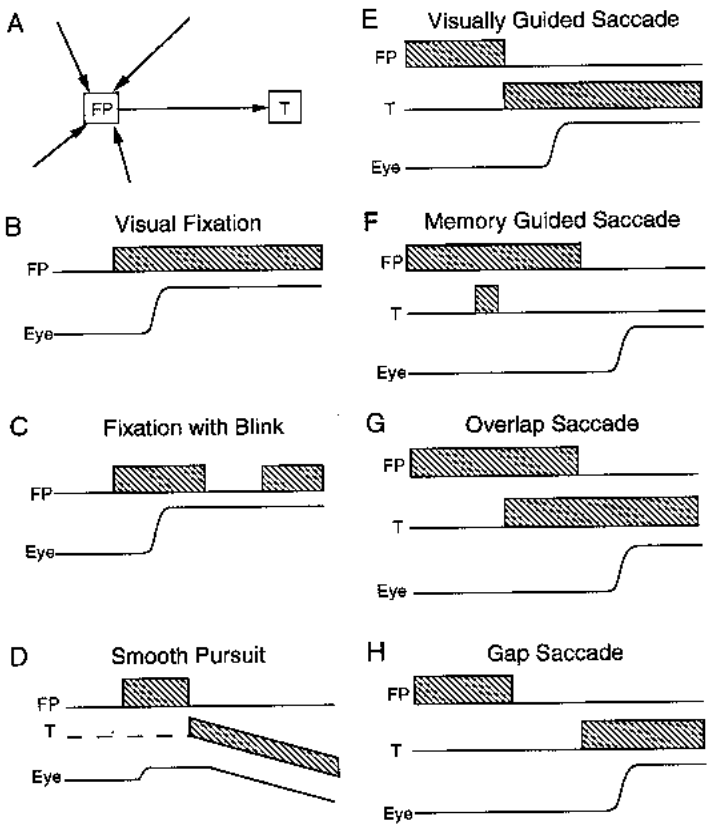


FIG. 2. Behavioral paradigms employed. In all paradigms the monkey faced a stationary tangent screen that had visual targets projected on it (*A*) and was rewarded for performance of the task. The 1st target to appear, referred to as the fixation point (FP), initiated the behavioral trial. Then a 2nd target (T) appears in some paradigms. In the visual fixation paradigm (*B*) the FP appeared, and the monkey had to look at it for up to 2 s to obtain a reward. In the fixation blink paradigm (*C*) the monkey had to 1st look at the FP, which was then turned off for a random period of time (300–600 ms), and the monkey had to maintain fixation. In the pursuit paradigm (*D*) the monkey had to 1st look at the FP and then, when it was turned off, follow the T, which stepped to one side of the fovea and ramped back across the screen at either 8 or 16°/s. In the saccade tasks the monkey had to 1st look at the FP and then, when it was turned off, look to the location of the T. In the visually guided saccade paradigm (*E*) the T appeared at the same time as the FP was turned off. In the memory-guided saccade task (*F*), the T was flashed on for 50–80 ms while the FP remained lit. After the FP was turned off, the monkey had to look at the remembered location of the T flash. In the overlap saccade task (*G*), the T came on and stayed on concurrent with the FP for 500–1,000 ms. In the gap saccade task (*H*), the FP was turned off for 200–400 ms before the T came on, and the monkey had to initiate the saccade only after T onset.

quired to maintain fixation until after the FP went off. In the visually guided saccade paradigm (Fig. 2*E*) a new target (T) appeared in the peripheral visual field at the same moment that the FP was turned off, and the monkey had to look at T immediately. In the memory-guided saccade paradigm (Fig. 2*F*) the T was flashed for 50–80 ms while the FP remained illuminated. After a randomized period of time (400–800 ms), the FP was turned off, and the monkey had to saccade to the remembered location of the T flash. In the overlap saccade paradigm (Fig. 2*G*) the T came on while the FP remained illuminated. After a random period (500–1,000 ms) the FP was turned off, and the monkey then had to look to the visible T. In the gap saccade paradigm (Fig. 2*H*) the FP was turned off, but the T was not illuminated until after a period of darkness ranging from 200 to 400 ms. The monkey had to make the saccade to T only after it came on.

In all of these tasks, the monkey was required to maintain its gaze within a computer-controlled window of ±1 to 3° during the periods of required fixation and smooth pursuit to obtain the liquid reward. The smallest window was used in the fixation tasks (Fig. 2, *B* and *C*) and the largest window for the saccade tasks (Fig. 2, *E–H*). If the monkey's gaze drifted out of this window, the trial was aborted, and the monkey received no reward on that trial. In the saccade paradigms the monkey was usually given up to 500 ms to initiate the saccade after receiving the final cue to go (FP offset in Fig. 2, *E–G*; T onset in Fig. 2*H*). If the saccade was not initiated in this time, then the trial was aborted, and no reward was delivered.

The visual fixation and fixation blink paradigms were randomly interleaved, and the position of the FP on the visual screen was randomly varied. The saccade tasks were interleaved, and the T was positioned at one or two different locations. In other blocks of trials, only the visually guided saccade paradigm was run, but the T position could be randomly varied among eight different positions. The fixation tasks (Fig. 2, *B* and *C*), pursuit task (Fig. 2*D*), and the saccade tasks (Fig. 2, *E–H*) were never presented together within a single block of trials.

A monkey would typically perform between 1,500 and 3,000 trials in a 3- to 6-h experimental period, working to satiation, after which the monkey was returned to its home cage. Careful records were kept of the weight and health status of the monkeys, and supplemental fruit and water were provided as needed. The monkeys were under the care of the institute veterinarian.

Data analysis

To evaluate the relation between cell discharge and specific events (such as target onset or eye movement), we produced rasters and a continuously varying spike-density function (Richmond et al. 1987) aligned on these events. To generate the spike-density function, a Gaussian pulse of specified (fixed) width was substituted for each spike, and then all of the Gaussians were summed together to produce a continuous function in time. The time from peak to $1/e$ for each Gaussian pulse was defined as σ. For all figures and analysis, σ had a value of either 4 or 10 ms. The wider Gaussian pulse permitted greater smoothing of the discharge envelope and was preferred unless a sudden change in discharge rate was being shown, in which case the narrower Gaussian pulse was used. Large values of the spike-density function represent a greater probability of the occurrence of a spike, and the peak of the function represents the peak discharge of the cell. A mean spike-density function was computed by averaging the spike densities over a series of trials. Saccades were identified during off-line analysis, with the use of a previously described computer program that identified the onset and termination of each saccade using velocity and acceleration threshold criteria (Waitzman et al. 1991).

Histology

Toward the end of recording, we made small marking lesions (10 μA, 30 s) where fixation cells had been recorded. After all the experiments were completed, the monkeys were deeply anesthetized with pentobarbital sodium and perfused through the heart. The brain was fixed by the use of procedures recently described (Ma et al. 1991). Sections through the sagittal plane were stained with thionen for cells and with the Gallyas stain (Gallyas 1979) for fibers and were examined microscopically.

RESULTS

Defining characteristics of fixation cells

We identified fixation cells by their tonic discharge pattern when the monkey looked at the fixation point. Figure 3

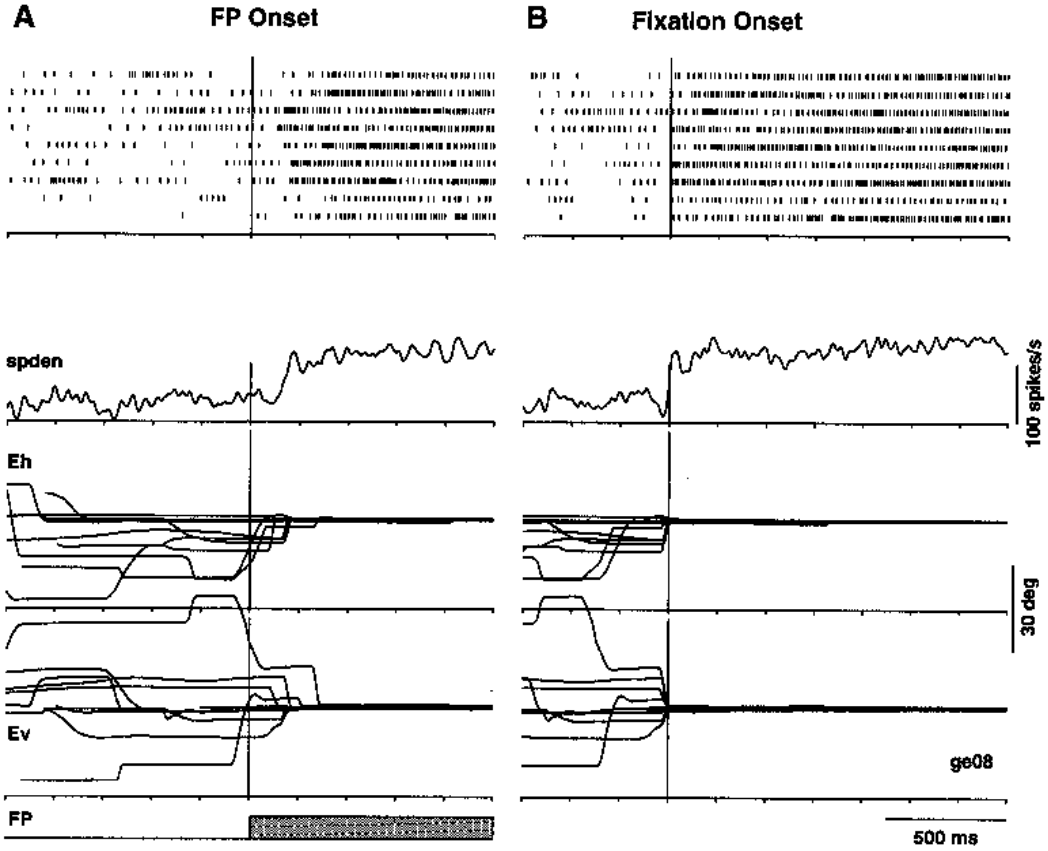


FIG. 3. Activity of a fixation cell in the rostral left SC during visual fixation. Rasters are aligned on the onset of the fixation target (*A*, fixation point onset) and the time when the eye entered the computer controlled fixation window (*B*, fixation onset). In this and subsequent figures the traces shown from top to bottom are the individual rasters with cell number (*ge08*), the spike-density function (spden), and the horizontal (Eh) and vertical (Ev) eye position traces. The Gaussian width for the spike-density calculation had a σ of 10.

shows the activity recorded from a fixation cell while the monkey looked at the fixation point at the center of the tangent screen (i.e., central gaze position). The rasters, spike density, and horizontal and vertical eye position traces are aligned on the onset of the fixation point (Fig. 3*A*), and on the time at which the eye entered the computer-controlled fixation window (Fig. 3*B*). Before the onset of the fixation point (Fig. 3*A*), the monkey was free to look anywhere, and the cell was only sporadically active. Shortly after the fixation point came on in the center of the screen, the monkey looked at it, and the activity of the cell immediately increased to a steady tonic rate of ~100 spikes/s (Fig. 3*B*), which continued for the next 1.5 s while the monkey maintained steady fixation.

When the monkey spontaneously fixated between saccades made across the dimly lit visual screen during intertrial intervals (e.g., *left side* of Fig. 3*A*), fixation cells were sporadically active. Because we have no control over the monkey's behavior during these spontaneous fixations, we could only speculate on the significance of fixation cell discharge and on the control of fixation during this period. We did not, therefore, quantify aspects of cell discharge during this period. Instead, we have quantified the effect of fixation cell activity on spontaneous saccades in the accompanying paper when we manipulated activity with GABAergic drug injections (Munoz and Wurtz 1993).

We tested whether the sustained response of the fixation cells was dependent on the fixation point acting as a visual stimulus on the fovea. When we momentarily turned off the fixation point during fixation (fixation blink paradigm, Fig. 2*C*) and required the monkey to maintain the same gaze position to obtain the reward, some tonic activity persisted (Fig. 4). Note that, although there was some drop in the cell's level of activity during the blink, the cell largely maintained its tonic discharge during the blinking off of the fixation point indicating an extraretinal input to the cell. At the end of the blink, the cell discharged a weak phasic burst, indicating that it also had a foveal visual input.

To compare quantitatively the discharge with the fixation point present and absent, we calculated the average firing frequencies of fixation cells during fixation of a visible target (visual fixation paradigm, Fig. 2*B*) and during the blinking off of the fixation point (fixation blink paradigm, Fig. 2*C*). Figure 5 plots the mean discharge rate when the target was visible against the rate during the blink for all of the 101 cells that we studied in the rostral SC of the two monkeys. Cells that increased their discharge rate during the blink lie above the dotted line, and cells that decreased their rate during the blink lie below the line. Thirty-five cells were more active during the blink than when the fixation point was visible, whereas 66 cells were less active. Cells lying below the horizontal dashed line were excluded from further analysis because they had discharge rates that were so low during the blink of the fixation point (<10 spikes/s).

For the rest of our experiments, we defined fixation cells

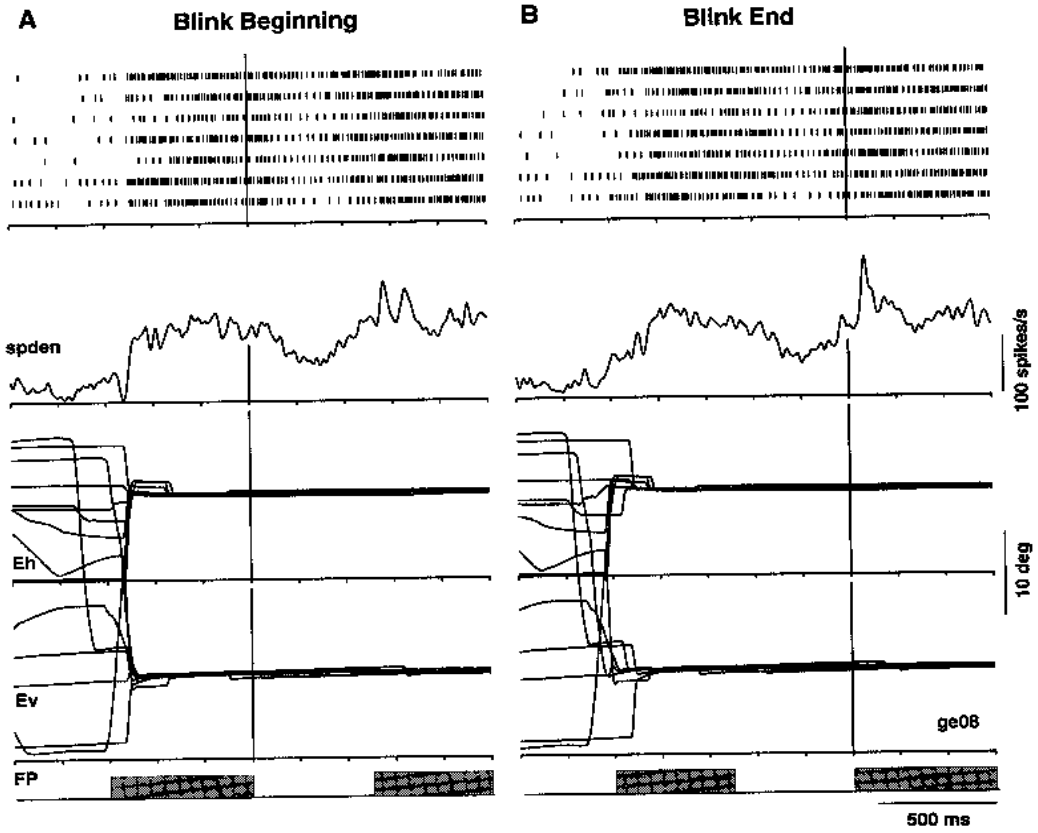


FIG. 4. Continued activity of a fixation cell in the absence of the fixation target. The FP blinked off for 300–600 ms as in the fixation blink paradigm (Fig. 2C). The duration of the blink was randomized. Rasters are aligned on the blink beginning (*A*) and blink end (*B*). The Gaussian width for the spike-density calculation had a σ of 10.

as those cells lying within the rostral pole of the SC that discharged tonically during active fixation and whose discharge did not drop below 10 spikes/s during the blink of the fixation point. The average discharge rate of the 74 fixation cells so identified was 49.2 ± 27.1 (SD) spikes/s when the monkey fixated the visible target spot, and 43.7 ± 28.0 spikes/s when we blinked off the fixation point.

We represented the change in discharge between fixation of a visible stimulus and fixation in darkness with a fixation index: the cell's average firing rate during the fixation point blink, divided by the cell's response in the presence of the fixation point. Figure 6 shows the distribution of this index for the 74 fixation cells, and Fig. 7 illustrates the spike-density profiles of 4 cells whose fixation indexes covered this range. The *top three cells* in Fig. 7 were classified as fixation cells: the cell in Fig. 7*A* almost doubled its firing rate during the blink; the cell in Fig. 7*B* showed no change during the blink; whereas the cell in Fig. 7*C* had a considerable reduction in discharge during the blink. The cell shown in Fig. 7*D* was not classified as a fixation cell because it stopped firing altogether during the blink. For comparison, the cell shown in Figs. 3 and 4 had a fixation index of 0.75.

Location of fixation cells

We found fixation cells in the rostrolateral pole of the SC. When we moved the guide tube >0.5 mm posterior or medial, we recorded only parafoveal visual cells and cells related to small saccades. When we moved the guide tube anterior or lateral, we did not encounter fixation cells. We did not find any fixation cells outside the rostral SC.

We identified the depth of the fixation cells within the rostral pole of the SC by noting the sequence of cell types encountered in each penetration and the distance of each cell type from the dorsal surface of the colliculus. Figure 8*A* shows the depth below the SC surface for 60 cells recorded in 12 electrode penetrations from 1 guide tube. Each vertical dotted line corresponds to a single electrode penetration through the rostral pole of one SC. We encountered cells with foveal and parafoveal visual receptive fields in the dorsal-most 1 mm of electrode travel through the SC. These cells responded phasically to the onset and offset of a visual stimulus and had little or no tonic activity during fixation of a visible target. Just below those, we found visual cells with tonic activity that was eliminated during the blink of the fixation point. Beneath the visual cells, we encountered saccade-related burst cells that had parafoveal visual receptive and movement fields with centers <2° from the fovea. These cells were found between 0.8 and 1.7 mm below the dorsal surface. Immediately ventral to these burst cells, we encountered fixation related cells that were tonically active when the monkey fixated a visible target spot. These fixation cells extended from ~1.1 to 3.0 mm below the dorsal surface of the SC, with most concentrated between 1.5 and 2.5 mm below the surface. Figure 8*B* shows the mean depth (±SD) of visual, saccade burst, and fixation cells from the

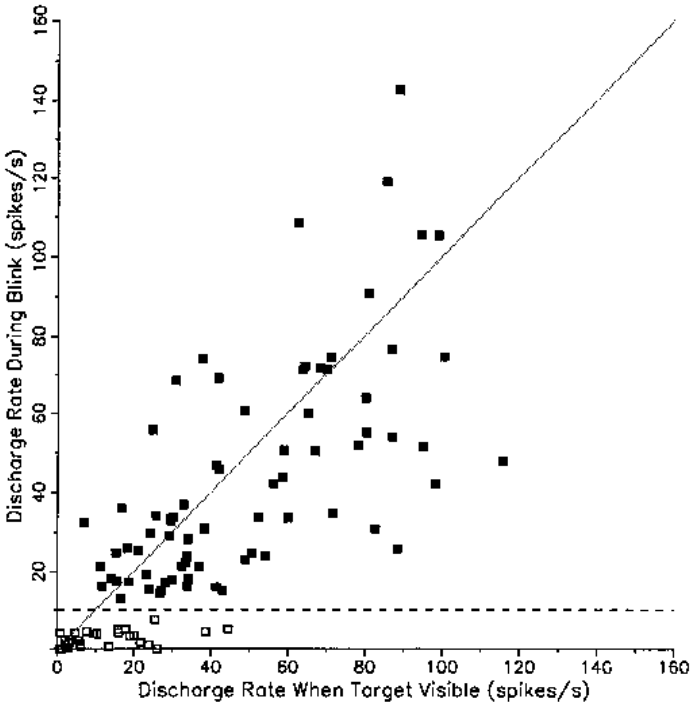


FIG. 5. Plot of firing rate during visual fixation (abscissa) vs. firing rate during the blink (ordinate) for 101 cells located in the rostral SC of 2 monkeys. Firing rate during visual fixation was calculated for the interval spanning from 0.7 to 1.2 s after FP onset in the visual fixation paradigm (Fig. 2*B*). Firing rate during the blink was calculated for the interval spanning the last 200 ms of the FP blink in the fixation blink paradigm (Fig. 2*C*). This 200 ms began at least 100 ms after onset of the blink so that it was not influenced by any transient response that may have been related to the FP going off. The dotted oblique line has a slope of 1.0. Cells lying below the horizontal line had discharge rates <10 spikes/s during the blink of the fixation point and were excluded from our analysis.

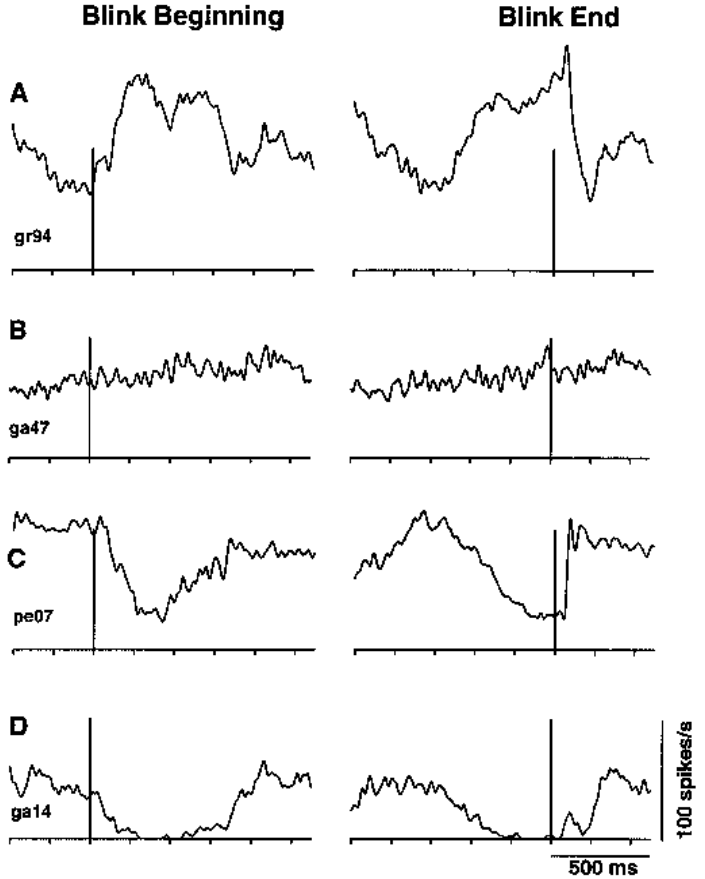


FIG. 7. Range of response to blink of the fixation point. Spike-density profiles from 4 cells at the beginning and end of the blink in the fixation blink paradigm. Fixation indexes for the 4 cells are 1.60 (*A*), 1.05 (*B*), 0.29 (*C*), and 0.04 (*D*). The Gaussian width for the spike-density calculation had a σ of 10.

same guide tube position shown in Fig. 8*A*. We saw a similar distribution of cells on another guide tube reconstructed in the second monkey (Fig. 8*C*). Fixation cells were found at a comparable depth in both animals.

To confirm this physiological localization of the fixation cells, we made a marking lesion at the site of fixation cells in each monkey. Figure 8*D* shows a sagittal section through the SC of one monkey with a marking lesion at the location of a fixation cell. This cell was located ~1.7 mm below the dorsal surface of the SC, which is consistent with the range of depths seen on the penetrations illustrated in Fig. 8, *A–C*.

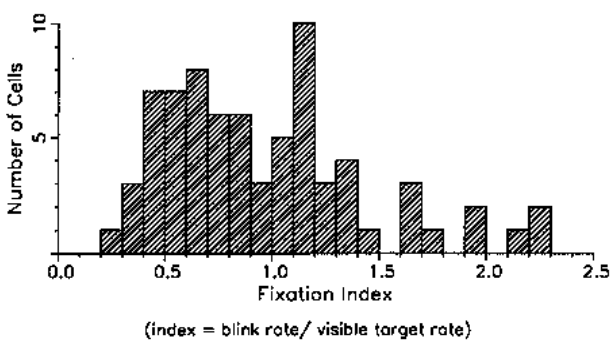


FIG. 6. Fixation indexes for 74 fixation cells. Only cells that were in the rostral pole and had discharge rates >10 spikes/s during the blink of the FP are included in this histogram. Calculation of discharge rates as in Fig. 5. One cell with an index of 4.64 is omitted from this histogram.

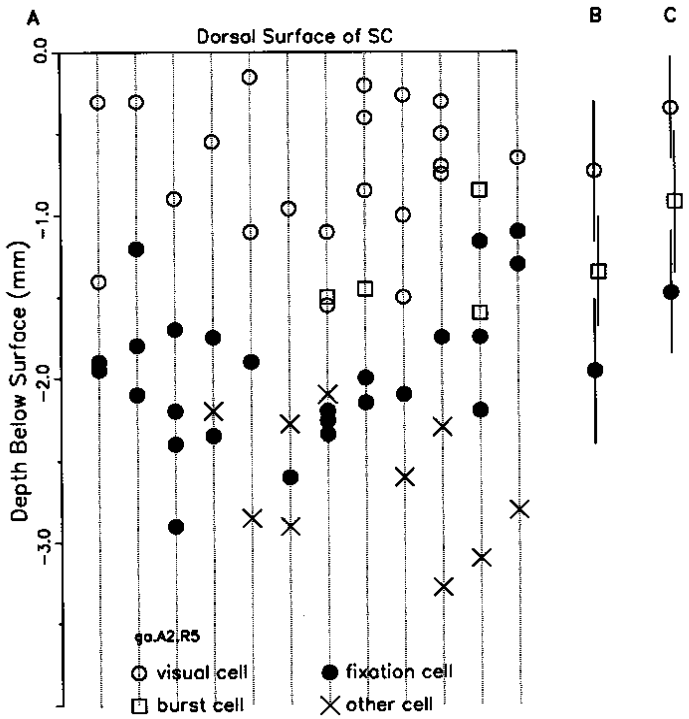
The mark falls at the border of the intermediate gray and white layers of the SC.

Saccade-related responses of fixation cells

The discharge rate of fixation cells decreased during large saccadic eye movements regardless of the direction of the saccade. Figure 9 shows the pause in discharge of a fixation cell during centrifugal saccades made in the visually guided saccade paradigm (Fig. 2*E*) to four different target locations. The saccade-related pause was omnidirectional; the cell paused immediately before the onset of each saccade and resumed firing at the time of saccade termination regardless of saccade direction.

Some fixation cells did not decrease their discharge rate for small-amplitude saccades. Figure 10 shows the activity of a fixation cell, located in rostral right SC, that paused during 30° saccades to the left or right (Fig. 10, *A* and *D*), and during 1.5° saccades to the right (Fig. 10*C*) but had a burst of activity for small contraversive saccades to the left (Fig. 10*B*).

We varied saccadic direction and amplitude to quantify their influence on the discharges of fixation cells. Figure 11, *A* and *B*, shows the mean firing frequency of two cells (illustrated in Figs. 9 and 10, respectively) during different amplitude ipsiversive and contraversive saccades, after subtracting the discharge rate during fixation. The curve illus-



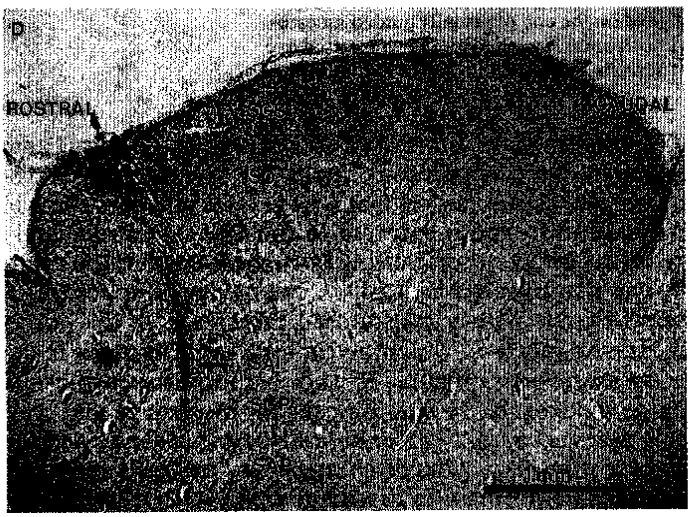


FIG. 8. Location of fixation cells within the rostral SC. *A*. distribution of all fixation cells recorded in 12 electrode penetrations within 1 guide tube within the rostral SC of *monkey g*. Penetrations (vertical dotted lines) were made at an angle 38° posterior to vertical and were all directed into 1 rostral pole at the same anteroposterior and mediolateral coordinate. Cells are plotted relative to the depth of the 1st multiunit visual activity detected (○, visual cells, $n = 21$; □, visuomotor burst cells, $n = 4$; ●, fixation cells, $n = 25$; ×, other cells). Of the 25 fixation cells identified in this guide tube, we collected data from 14 of them. *B*: mean depth (±SD) of the visual, saccade burst, and fixation cells from the 12 penetrations shown in *A*. *C*: mean depth (±SD) of the visual, saccade burst, and fixation cells from 16 penetrations within 1 guide tube in the rostral SC of *monkey p*. *D*: Nissl-stained sagittal section through the SC of 1 monkey showing a small marking lesion (arrow) at the site of a fixation cell. To the left is the rostral SC, to the right, the caudal SC. Location of layers were estimated primarily from adjacent Gallyas-stained sections. The mark is at the caudal edge of the fixation zone. SGS, superficial gray layer (stratum griseum superficiale); SO, optic layer (stratum opticum); SGI, intermediate gray layer (stratum griseum intermedium); SAI, intermediate white layer (stratum album intermedium); SGP, deep gray layer (stratum griseum profundum); SAP, deep white layer (stratum album profundum).

trated in Fig. 11*A* is from a cell that paused for all saccades. For very small saccades, to targets centered around 0°, the pause was minimal. The curve illustrated in Fig. 11*B* is from a cell that did not pause for contraversive saccades <15° in amplitude. Figure 11*C* shows the curves obtained from 6 other fixation cells that were representative of the 20 cells in which we tested a wide enough range of amplitudes (4–16 amplitudes in 2 horizontal directions). For very small ipsiversive saccades, some cells had less of a decrease in firing rate or no decrease at all. For small contraversive saccades, most cells showed an increase in discharge. The same trends were observed for oblique saccades. Thus cells such as those in Fig. 11, *B* and *C*, have an increased discharge during fixation, but they also increase their discharge for small contraversive saccades as do the saccade cells lying adjacent to them in the SC.

We studied the saccade-related responses of 34 fixation cells. Ninety-one percent (31/34) of these cells paused for saccades >15° in amplitude in any direction. The remaining three cells paused for ipsiversive but not contraversive saccades. The saccade-related pauses of fixation cells were present for both centrifugal and centripetal saccades (not shown). Of the 20 cells whose movement fields we were able to estimate, 35% (7/20) paused for all saccades (e.g., Figs. 9 and 11*A*), 60% (12/20) paused for all saccades except small-amplitude contraversive movements (e.g., Figs. 10 and 11*B*), and 1 cell did not pause for contraversive saccades.

Temporal relation of pause and saccade

The relation of the pause in discharge of the fixation cells and the occurrence of a saccade is reminiscent of the brain stem omnipause neurons (OPNs) that discharge tonically for all fixations and pause for all saccades (Keller 1974; Luschei and Fuchs 1972). The duration of the OPN pause is well correlated to saccade duration, and this correlation applies to collicular fixation cells as well. Figure 12 shows the correlation between the pause duration and the duration of the saccade for ipsiversive (Fig. 12*A*) and contraversive (Fig. 12*B*) saccades for the fixation cell illustrated in Fig. 10. The slope and correlation values of the linear regression line were 1.20 and 0.92, respectively, for ipsiversive saccades and 1.12 and 0.88, respectively, for contraversive saccades. The regression lines obtained from four other fixation cells with sufficient data to be analyzed were similar to those shown in Fig. 12, *A* and *B*.

We also determined the temporal relation between the pause and the saccade for 31 fixation cells. Figure 13, *A* and *B*, show the mean time (±SD) from pause onset to saccade onset for each cell for ipsiversive and contraversive saccades. Every cell paused before saccade onset. The mean time from saccade onset to pause onset was −36.2 (range, −113 to −11 ms) and −33.0 ms (range, −103 to −6 ms), respectively, for ipsiversive and contraversive saccades. The difference between ipsiversive and contraversive saccades was not significant (*t* test, $P > 0.05$). Figure 13, *C* and *D*, shows the time from pause end to saccade end. The mean time from saccade termination to pause end for ipsiversive and contraversive saccades was 9.9 (range, −9 to 62 ms) and −2.6 ms (range, −23 to 26 ms), respectively. This dif-

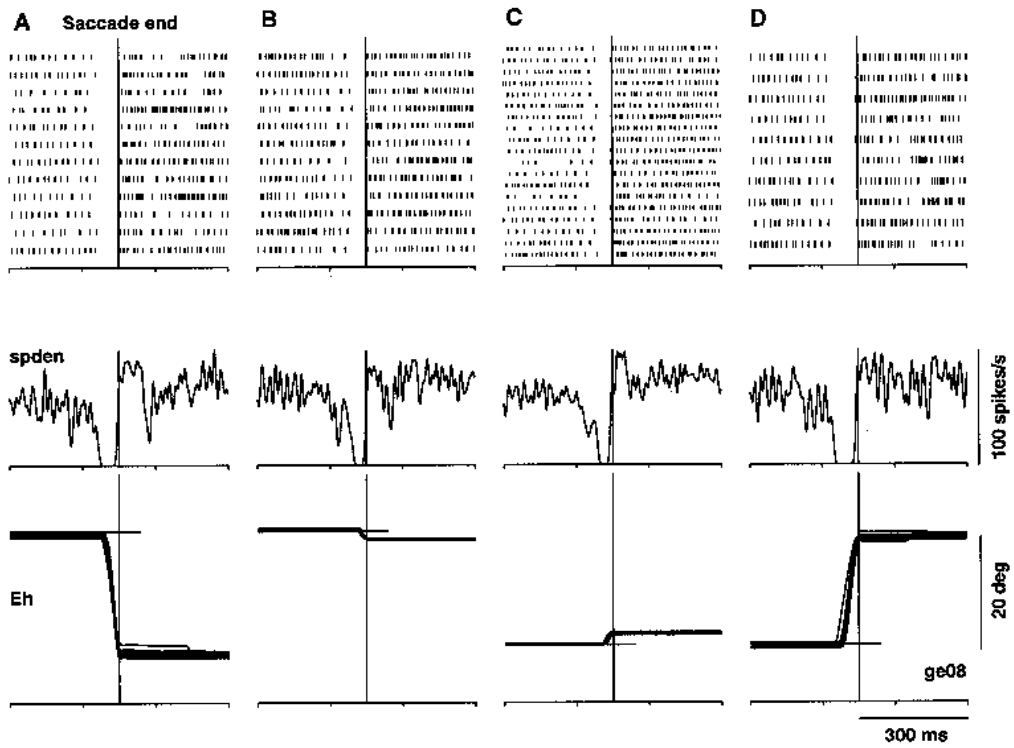


FIG. 9. Pause in discharge rate of a fixation cell during saccades. Traces are aligned on the end of visually guided saccades to targets located 20° left (*A*) or right (*D*) or 2° left (*B*) or right (*C*). The short horizontal line aligned with the initial eye position traces in this and subsequent figures indicates the center of the tangent screen. The Gaussian width for the spike-density calculation had a σ of 4. Cell was located in the rostral left SC.

ference was highly significant (t test, $P < 0.005$). Fixation cells therefore were reactivated sooner for contraversive, as compared with ipsiversive, saccades.

The most temporally synchronized measure was the time from pause end to saccade end. The average standard deviation for this relationship was 9.8 and 7.4 ms for ipsiversive and contraversive saccades. In contrast, the time from pause onset to saccade onset was far more variable: the average standard deviations were approximately doubled to 17.0 and 17.2 ms for ipsiversive and contraversive saccades.

Increased postsaccadic discharge rate

An additional characteristic of fixation cells was that some cells discharged at a higher rate for the first 200 ms after a saccade. Figure 14 illustrates this for a fixation cell that paused for saccades of all amplitudes and directions. Note that the postsaccadic discharge rate of this cell was higher than that before saccade onset. This postsaccadic increase in discharge rate was not dependent on saccade amplitude (tested in 20 cells) or final orbital position (tested in 31 cells).

The postsaccadic increase in discharge was also not dependent on the visual stimulation provided by the saccade target, because it was present when the monkey made saccades in the absence of the visual stimulus in the memory-guided saccade task (tested in 28 cells). For example, Fig. 15 shows the postsaccadic increase in discharge rate for saccades made to the right and the left in the memory-guided saccade task. In these cases the increase was present, but at the time of the saccade the target was not present.

To quantify the amount of increase in the postsaccadic discharge, we computed an index by dividing mean discharge rate in the period from 50 to 150 ms after saccade termination by the mean rate in the 100 ms immediately preceding target onset. During both these periods the monkey was fixating a visible LED. A cell having an index of 1.0 meant that it had no postsaccadic increase in discharge, whereas an index > 1.0 signified at least some increase in the postsaccadic rate. Figure 16 shows the postsaccadic discharge indexes for 31 fixation cells for ipsiversive and contraversive saccades. Notice that only some cells showed the increase in postsaccadic discharge. Very few cells showed a postsaccadic decrease in discharge rate.

Effect of orbital position on fixation cells

Fixation cells were not sensitive to position of the eyes in the orbit. Figure 17 shows the spike-density profiles obtained from a fixation cell (same cell as in Figs. 3 and 4) when the fixation point was located at five different positions on the screen (center position and 20° up, down, left, and right). The time course and magnitude of this cell's response appeared unrelated to eye position during active fixation. A similarly negative finding was also obtained in the fixation blink paradigm when orbital position was varied (not shown).

We found virtually no orbital position sensitivity on any of the 29 fixation cells tested. Figure 18 shows the mean discharge rate, computed during visual fixation, plotted against horizontal gaze angles for 29 cells and vertical gaze angles for 26 cells. Note that the discharge rates of almost all

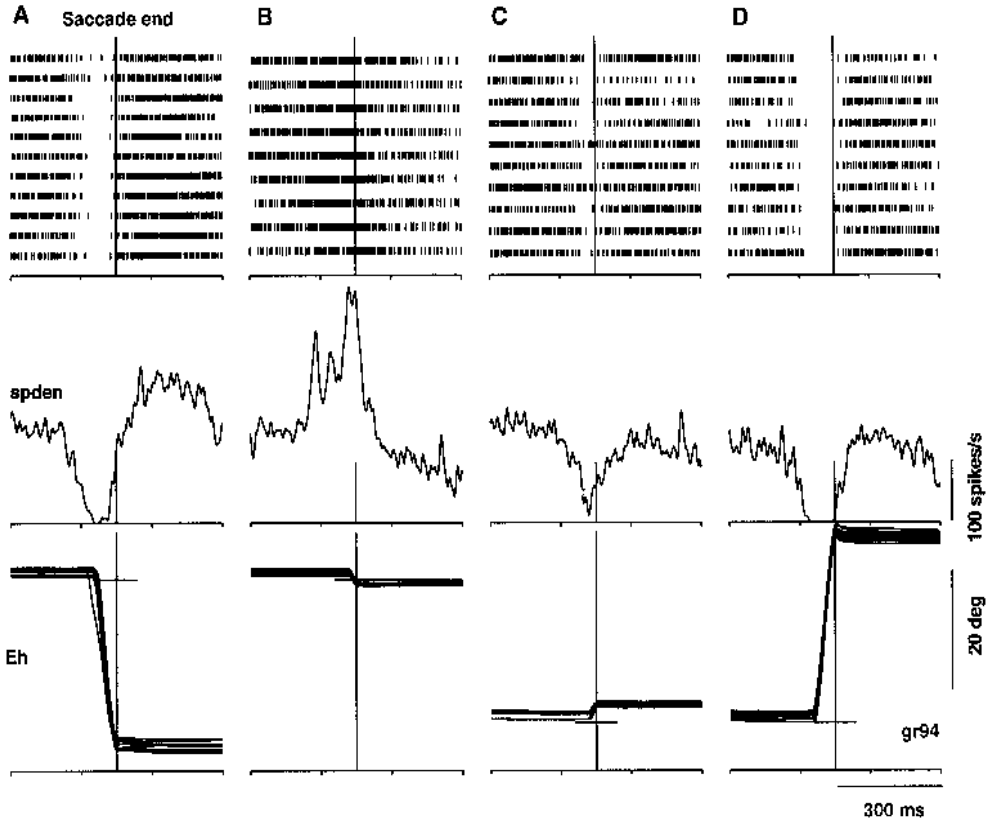


FIG. 10. Continued discharge of a fixation cell during small contraversive saccades. Traces are aligned on the end of visually guided saccades to targets located 30° left (*A*) or right (*D*) or 1.5° left (*B*) or right (*C*). Horizontal line marks center of the tangent screen. The Gaussian width for the spike-density calculation had a σ of 4. Cell was located in the rostral right SC.

cells showed no consistent change with orbital position. Fixation cells paused similarly for both centripetal and centrifugal saccades so that the saccade-related pause also had no orbital position dependency.

Response of fixation cells during pursuit

We also recorded the discharge of some fixation-related cells when monkeys made smooth pursuit eye movements. Figure 19 shows the responses of a fixation cell, located in the rostral left SC, when the monkey pursued a visual target moving smoothly at 16°/s to the right (Fig. 19, *A* and *B*) or left (Fig. 19, *C* and *D*). The cell was active during pursuit in both directions. Note also that this cell paused for small ipsiversive (leftward) but not contraversive (rightward) saccades. We recorded pursuit-related responses from eight fixation cells in two monkeys. All of these cells were active during both ipsiversive and contraversive pursuit, provided the visual axis was aligned with the target.

We also tested a few fixation cells when the monkey actively fixated a stationary spot during passive head rotation, a condition in which vestibularly driven slow phase eye movements were required to keep the eyes aligned with the target. We noticed no changes in the discharge rate of the fixation cells.

DISCUSSION

We have identified a subset of neurons in the intermediate layers of the rostral SC of the monkey, which we have called fixation cells. These cells discharged tonically when the monkey actively fixated a visual target and produced a weaker more sporadic discharge when the monkey looked spontaneously around the dimly lit laboratory. Fixation activity continued when the target was momentarily blinked off, indicating that the discharge was not simply a visual response. These characteristics are consistent with fixation cells recently identified in the rostral SC of the cat (Munoz and Guitton 1989, 1991; Munoz et al. 1991; Peck 1989) and with collicular neurons reported to be tonically active when a monkey fixates a visible target (Goldberg and Wurtz 1971; Sparks and Mays 1980). In addition to the basic characteristics of increased discharge during active fixation, we also have been able to establish other characteristics of these cells in the monkey: *1*) a pause in discharge associated with saccades in any direction; *2*) the duration of the pause increased with the duration of the saccade; *3*) a closer relation of pause duration to the end of the saccade than the beginning; *4*) an increase in discharge after this pause; *5*) no orbital position dependency; and *6*) a continued discharge during the initiation and maintenance of smooth pursuit eye movements.

Our hypothesis is that the fixation cells within the rostral pole of the SC are critical for maintaining active visual fixation and suppressing the generation of saccades. We propose that activation of fixation cells leads to suppression of activity of saccade-related cells in the SC and brain stem, thereby inhibiting the generation of saccades. We will first discuss the evidence supporting this hypothesis that is simi-

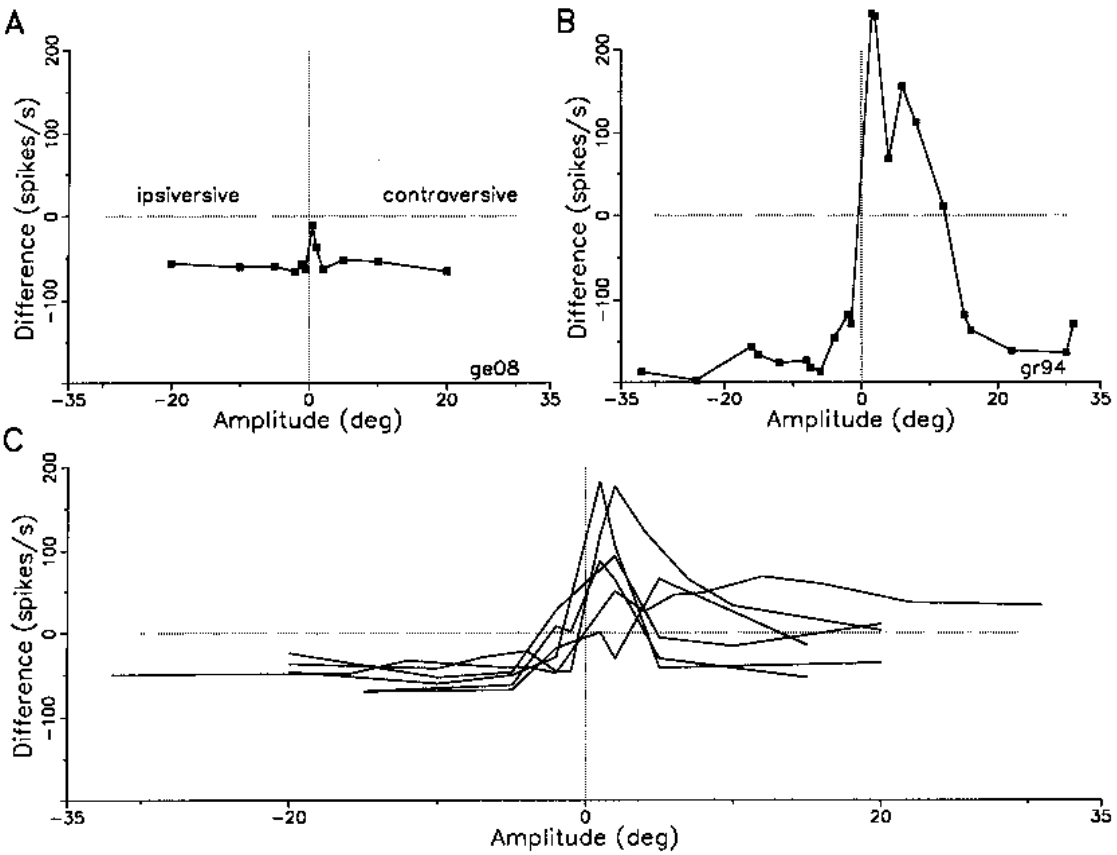


FIG. 11. Discharge of fixation cells with ipsiversive and contraversive saccades. Graphs show the saccade amplitude for ipsiversive (−) and contraversive (+) saccades (abscissa) plotted against the difference in discharge rate during the saccade and during fixation of the visual target (ordinate). The dotted horizontal line indicates the discharge rate during the 200 ms of fixation immediately before target onset, which was 56 spikes/s in *A* and 170 spikes/s in *B*. The plot in *A* is for the same cell shown in Fig. 9, which paused for all saccades but, as the graph indicates, less for small saccades. The plot in *B* is for the cell in Fig. 10 that paused for ipsiversive saccades but not for contraversive saccades with amplitudes of ≤15°. The plot in *C* shows the same curves for 6 other representative cells. Firing frequency for the saccade was measured from 8 ms before saccade onset to 8 ms before saccade end and during fixation immediately before target onset.

lar to that proposed by Munoz and Guitton (1991) based on their experiments in the cat. We will then discuss the descending influence of the SC fixation cells on the oculomotor system, and the possibility that the SC fixation cells are part of the final common path in a larger system related to visual fixation.

SC fixation cells and saccade generation

A number of characteristics of the fixation cells in the monkey justify regarding them as part of a fixation system. The most important observation is that cells in the rostral SC had a tonic discharge during visual fixation. The fixation had to be active, that is when the monkey looked at a known visual target to obtain a reward. This fixation activity was not dependent on the visual stimulation resulting from the fixation spot as has also been demonstrated for some cells in the cat (Peck 1989). In our experiments blinking off this spot modulated but did not eliminate the discharge of these cells during fixation, although the blink did eliminate the response of the visual cells lying just above in the SC.

When the monkey spontaneously fixated between saccades made across the dimly lit visual screen during intertrial intervals, these cells were more sporadically active. Because we have no control over the monkey's behavior during these spontaneous fixations, we can only speculate on the significance of the fixation cells and on the control of fixation during this period. Some of these spontaneous fixations were accompanied by an increased discharge rate of the fixation cells and may therefore be active fixations. Other fixations clearly were not accompanied by such an increase in discharge and may instead result from the combination of weak fixation cell activity and a lack of input to the saccade-generating system.

The pause in fixation cell discharge that accompanied saccades is also critical for the argument that activity of fixation cells suppresses the generation of saccades. All cells ceased firing before the onset of saccades that were >15° in amplitude, and many did so before smaller saccades as well. The pause was for saccades in any direction. We take both these characteristics to indicate that the fixation cells are related to the generation of all saccades, not just those to the contralateral side served by one SC. In this regard the monkey fixation cells are comparable with those in the cat (Munoz et al. 1991). Most importantly, the duration of the pause was linearly related to the duration of the saccade; as saccade duration increased, so did the pause duration of the

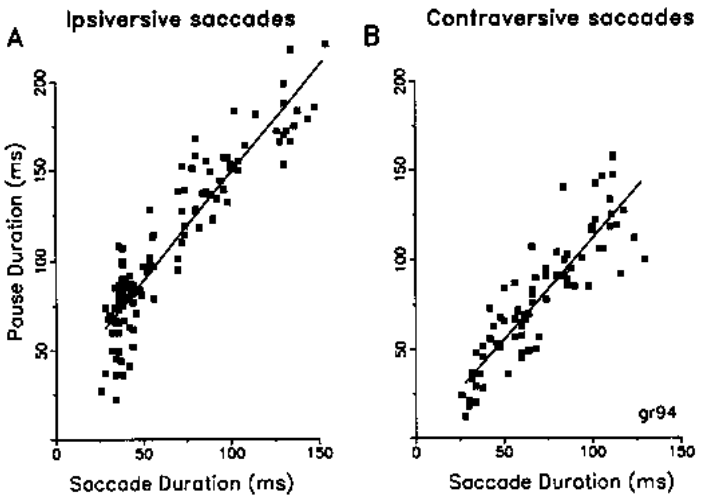


FIG. 12. Plot of pause duration vs. saccade duration for ipsiversive (*A*) and contraversive (*B*) saccades. Data from an individual fixation cell (same cell as shown in Fig. 10). Solid lines are least-squares regression lines through the data points. Slope and correlation values of the linear regression lines were 1.20 and 0.92 for ipsiversive saccades and 1.12 and 0.88 for contraversive saccades.

fixation cells. If the fixation cells are controlling the activity of saccade-related cells, this duration characteristic is a prerequisite.

The end of the pause in discharge of the fixation cells was better synchronized with the end of the saccade than was the beginning of the pause and saccade onset. Furthermore, several cells were reactivated *before* the end of the saccade, especially for contraversive saccades. This reactivation was not due to the visual stimulus falling into the visual receptive fields of fixation cells, because we observed a similar pattern of reactivation at the end of saccades in the memory-guided saccade paradigm where the saccade was generated in total darkness (e.g., see Fig. 15). These observations suggest that the start of the pause and its end may be under the control of different mechanisms. One possibility is that pause onset may signal the end of active visual fixation, whereas the end of the pause may be involved in terminating the saccade (Munoz et al. 1991).

Some fixation cells had an increased postsaccadic discharge for the first 200 ms after the saccade-related pause. A possible function of this postsaccadic activity is to reduce the probability of a saccade, because, by our hypothesis, fixation cell activity must be terminated before the execution of a saccade to a new target. This is consistent with the relatively refractory state of the saccadic system after a visually guided saccade (Becker 1989).

The increase in discharge rate with active fixation and the pause in discharge of the fixation cells during saccades occurred regardless of the orbital position of the fixation. This orbital independence is similar to that of the collicular saccade-related cells that are related to visually guided sac-

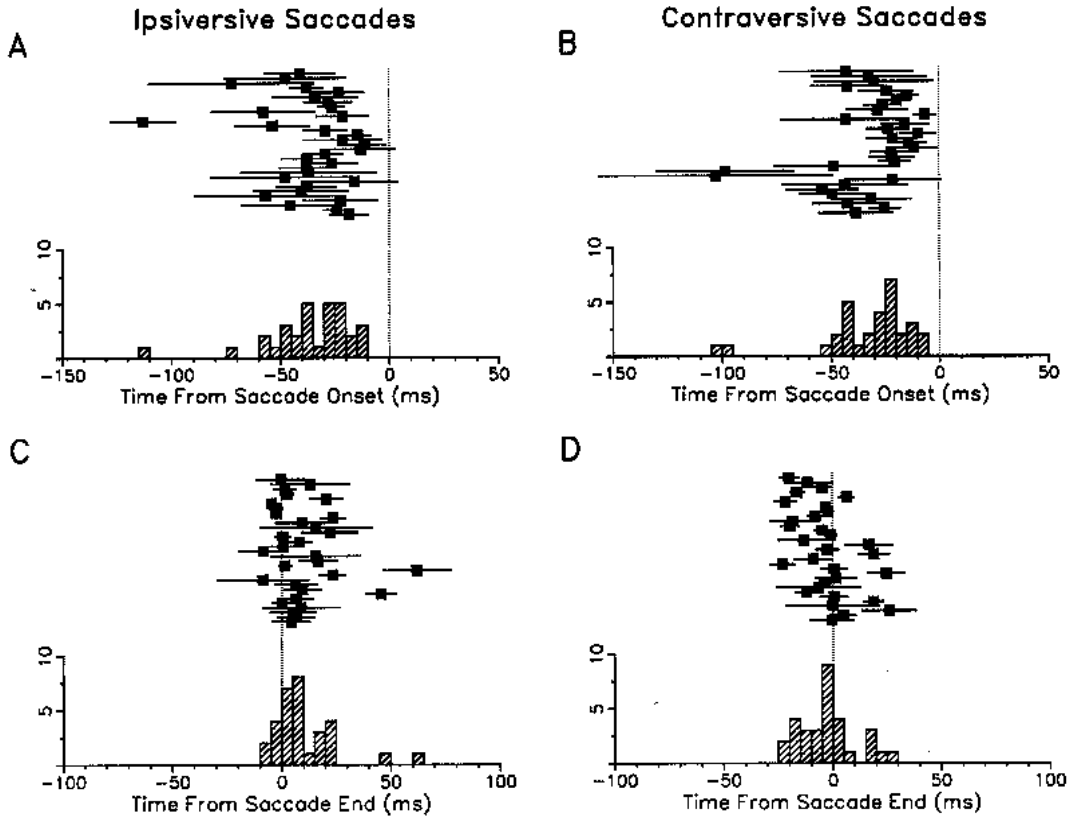


FIG. 13. Time from saccade onset to pause onset (*A* and *B*) and saccade end to pause end (*C* and *D*) for ipsiversive (*A* and *C*) and contraversive (*B* and *D*) saccades. Each horizontal line corresponds to data from a single cell, expressed as the mean latency (±SD). Cells used all had data from saccades that were between 10 and 20° in amplitude, and mean times were computed from these amplitudes. This range of saccades was used because larger saccades were occasionally accompanied by a small corrective saccade and smaller saccades were not always associated with a clear pause. Negative values imply that pause onset or end preceded saccade onset or end, respectively.

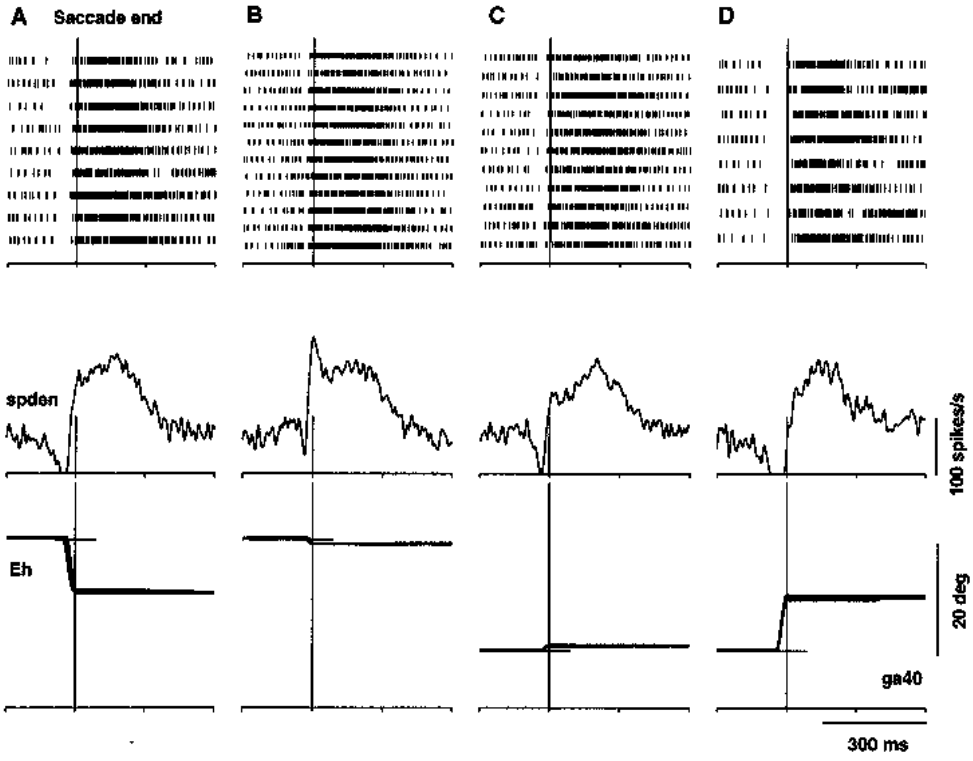


FIG. 14. Increased postsaccadic discharge rate of a fixation cell. Traces are aligned on the end of visually guided saccades to targets located 10° left (*A*) or right (*D*) or 1° left (*B*) or right (*C*). The Gaussian width for the spike-density calculation had a σ of 4. Cell was located in the rostral right SC.

cades of a given amplitude and direction regardless of the position of the eye in the orbit (Wurtz and Goldberg 1972). Therefore both fixation cells and saccade cells of the SC are primarily related to the *change* in eye position not what that position is in the orbit.

The activity of the fixation cells is tonic regardless of what other oculomotor activity occurs concomitantly with active visual fixation. Although we have not studied the fixation cells extensively during oculomotor functions other than visual fixation and saccades, we do have a few observations during the initiation and maintenance of smooth pursuit and passive head rotation that indicates that the fixation cells continue to discharge, provided the monkey tried to keep its visual axis aligned with the target. Put another way, it was not necessary for fixation cell activity to cease during the generation of these nonsaccadic oculomotor move-

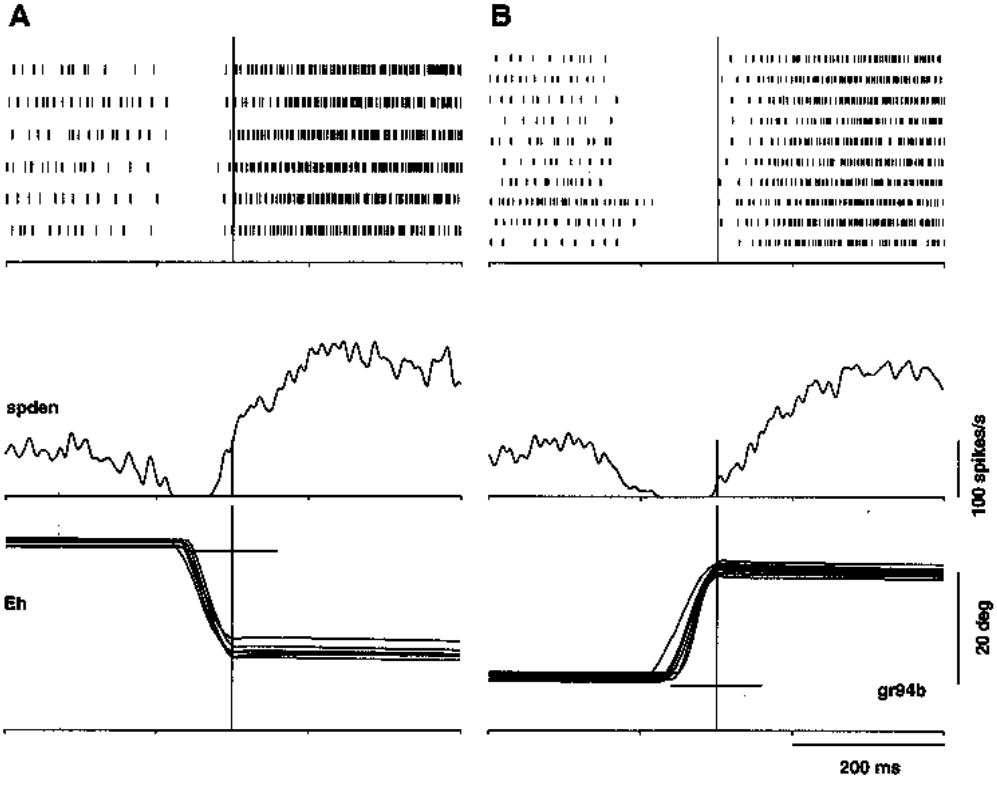


FIG. 15. Increased postsaccadic discharge rate of a fixation cell in the memory-guided saccade task in which no visual target was present at the time of the saccade. Traces are aligned on the end of the saccades to targets located 20° left (*A*) or right (*B*). The Gaussian width for the spike-density calculation had a σ of 4. Cell was located in the rostral right SC.

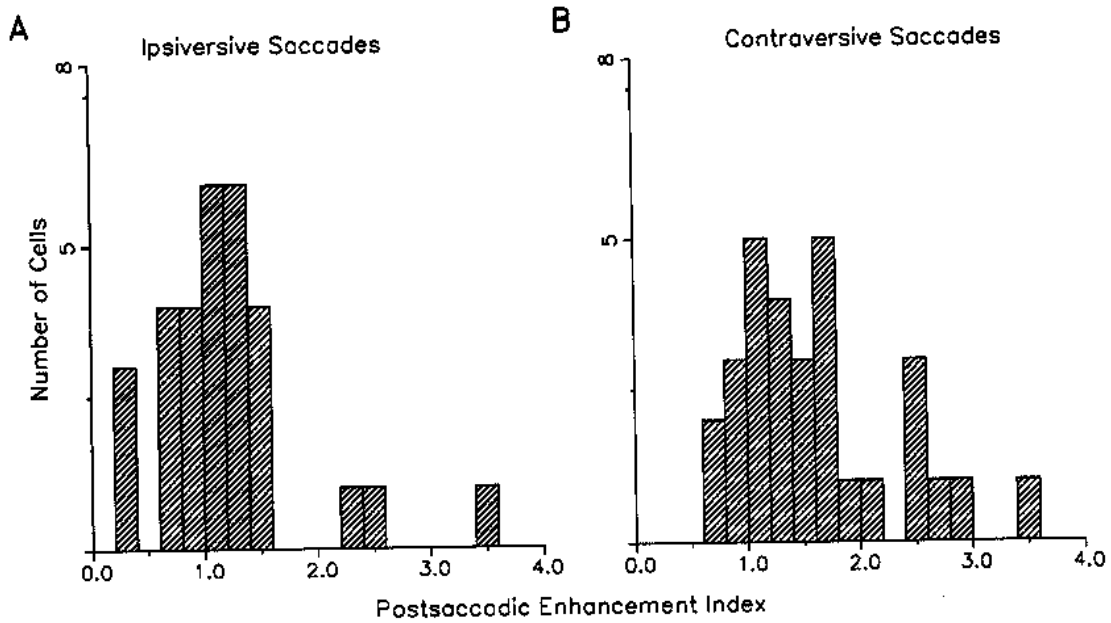


FIG. 16. Plot of postsaccadic discharge indexes calculated for 31 fixation cells for ipsiversive (*A*) and contraversive (*B*) saccades. Saccades were between 10 and 20° in amplitude made in the visually guided saccade paradigm. The index was calculated by dividing a cell's mean discharge 50–100 ms after a saccade by that in the 100 ms preceding target onset.

ments. Therefore fixation cell activity did not coincide with suppression of all eye movements, just saccadic eye movements.

Interactions between fixation cells and the saccade system

The interaction between fixation cells and the saccadic system could occur both within the SC and between the SC and the brain stem. Within the SC, we assume that the fixation cells would act to inhibit directly or indirectly the saccade-related cells. The discharge patterns of collicular fixation cells were very different from the classic description of saccade-related cells in the monkey SC (Schiller and Koerner 1971; Sparks 1978; Sparks et al. 1976; Wurtz and Goldberg 1971, 1972). Saccade-related burst cells, located in the intermediate layers of the monkey SC, are silent during fixation and discharge vigorously during contraversive saccades. This pattern of activity is reciprocally related to

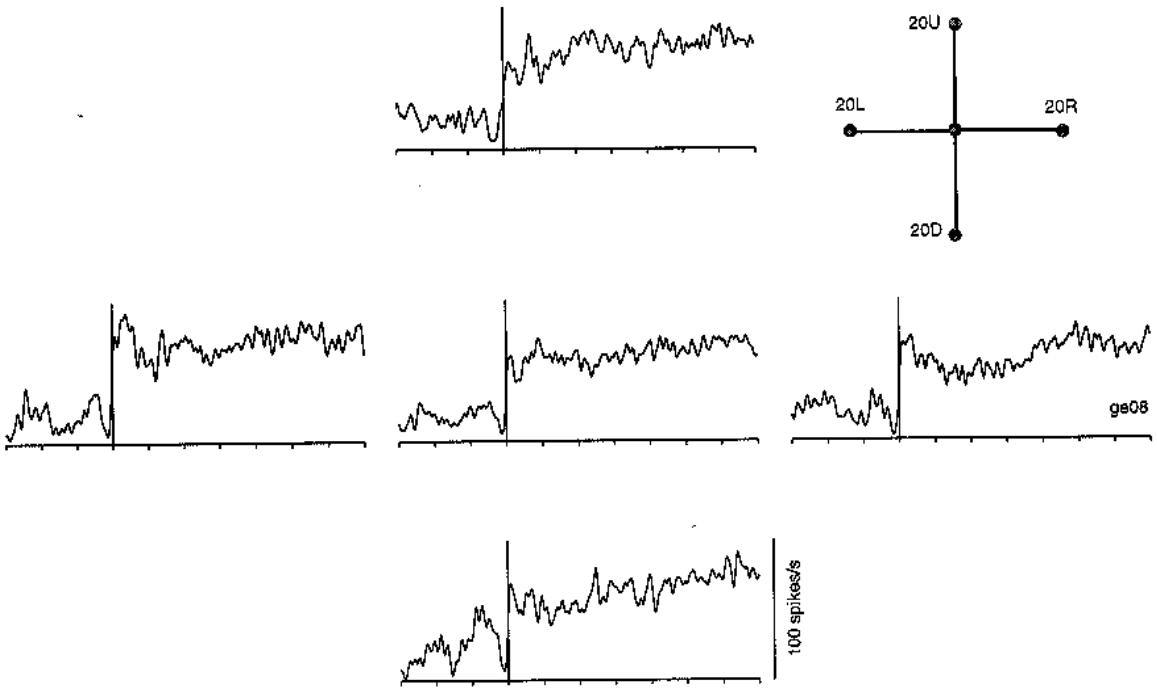


FIG. 17. Independence of fixation discharge and orbital position. Spike-density profiles are for 1 fixation cell while the monkey looked at the FP at 5 different orbital positions: center and 20° left, right, up, and down. Spike-density profiles are aligned on when the eye entered the fixation window in the visual fixation paradigm. The Gaussian width for the spike-density calculation had a σ of 10.

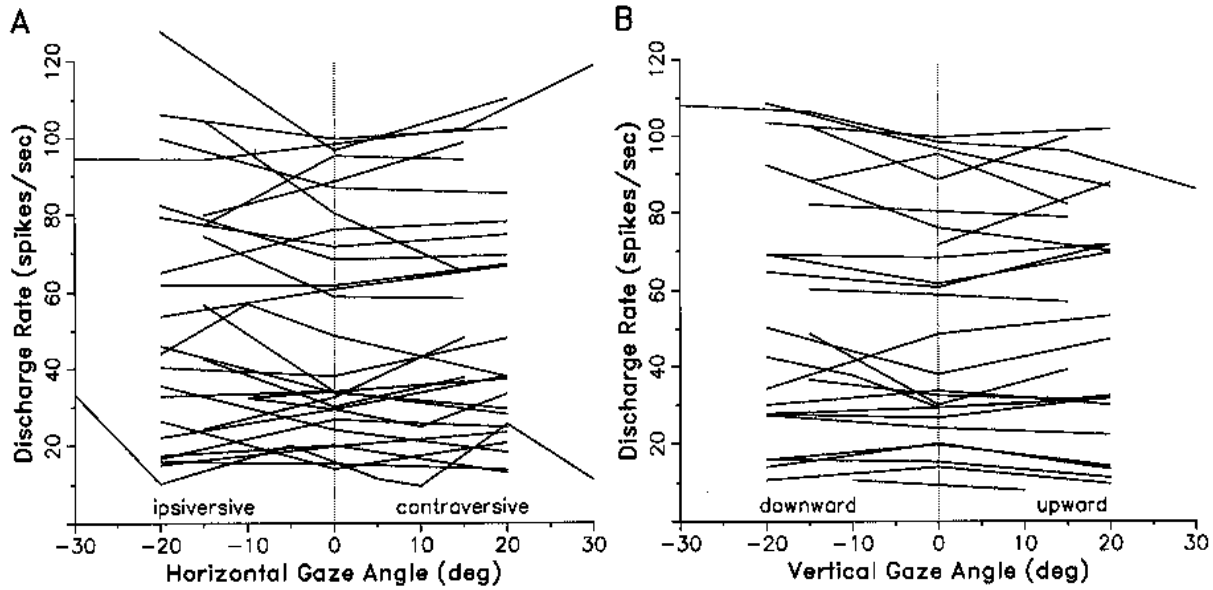


FIG. 18. Plots of average discharge rate vs. horizontal (*A*) and vertical (*B*) orbital position. Each curve represents data obtained from 1 cell.

the fixation cells, all of which pause with ipsiversive saccades, and some of which pause with all saccades. There is some evidence suggesting the presence of an inhibitory network within the SC of the cat, whereby activation of neurons at one collicular locus leads to inhibition of neurons at other loci located ipsilateral and contralateral to the activated zone (Douglas and Anderchek 1991; Douglas and Vetter 1986; Mascetti and Arriagada 1981). It is therefore possible that some fixation cells in the rostral SC and some saccade-related cells in other collicular regions may form part of an intracollicular inhibitory network. Figure 20 illustrates such a simple network with fixation cells in the rostral pole of the SC inhibiting saccade cells and vice versa. This network would be involved in maintaining only one locus of activity at a time on the SC motor map (Munoz and Guitton 1989, 1991). When the monkey keeps its eyes aligned on a visual target, fixation cells in the rostral poles would be active, leading to a suppression in activity of the saccade-related cells. When the monkey is about to make a saccade to a new target, an ensemble of saccade-related cells, located at the appropriate site on the SC saccade map, would be activated leading to the deactivation of the fixation cells.

Another area where interactions between the SC fixation neurons and the oculomotor system might occur is in the pons. The characteristics of the collicular fixation cells are reminiscent of brain stem OPNs, located in the midline raphe nuclei of the pons, which are tonically active for all

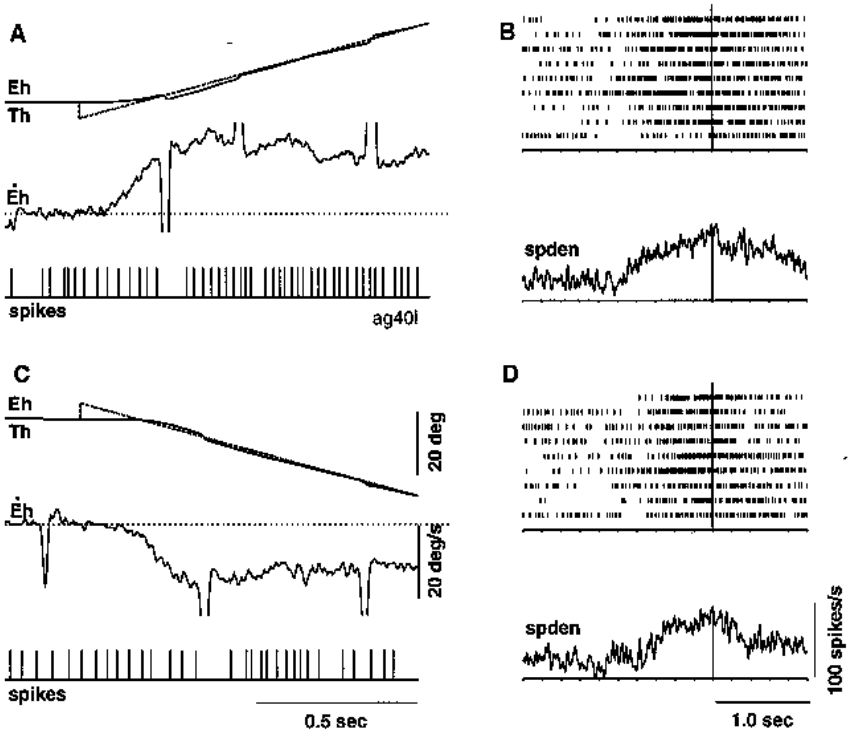


FIG. 19. Continued activity of a fixation cell during smooth pursuit eye movements. The pursuit target moved at 16°/s to the right (*A* and *B*) or left (*C* and *D*). The target first stepped 2° away from the fixation point and then ramped back, and the monkey acquired the target without making a saccade. Records are shown for single trials (*A* and *C*) and multiple trials aligned on the onset of pursuit target motion (*B* and *D*).

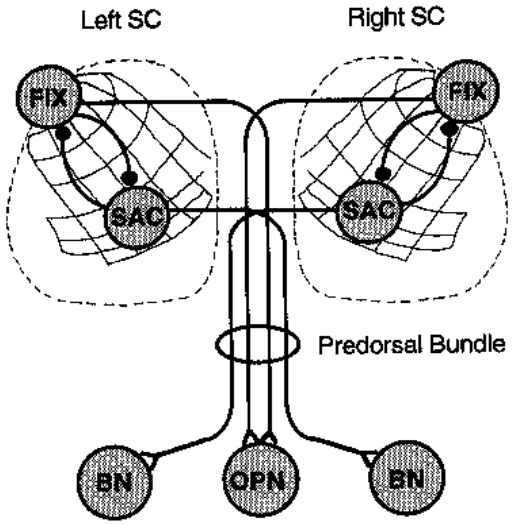


FIG. 20. Summary figure illustrating possible connectivity within the SC and between the SC and the brain stem that might be related to the action of fixation cells. Each SC has a fixation (FIX) and a saccade (SAC) cell that inhibit each other and also project to the brain stem to activate omnipause neurons (OPN) and burst neurons (BN), respectively. See DISCUSSION.

periods of fixation and pause during saccadic eye movements (Evinger et al. 1982; Keller 1974; Luschei and Fuchs 1972). There are, however, several differences between the collicular fixation cells and OPNs in the monkey. *1*) The typical discharge rates of monkey OPNs during periods of fixation, are higher (frequently >100 spikes/s) than those we found for SC fixation cells (usually <100 spikes/s; see Fig. 5). *2*) Monkey OPNs maintain their high tonic rate during spontaneous fixation, whereas SC fixation cell activity became more sporadic (see the leftmost regions of the trials illustrated in Figs. 3*A* and 4*A* where the fixation target had not yet appeared on the screen). *3*) Most monkey OPNs pause for saccades of all amplitudes and directions, whereas many collicular fixation cells did not pause for small contraversive saccades. As shown schematically in Fig. 20, the SC projects to the OPNs because stimulation of the SC leads to activation of OPNs with monosynaptic latencies in both monkey (Raybourn and Keller 1977) and cat (King et al. 1980). Furthermore, it has been demonstrated that fixation cells in the rostral pole of the cat SC have an axon that joins the predorsal bundle (Munoz and Guitton 1989, 1991; Munoz et al. 1991). Thus SC fixation cells may convey an excitatory signal directly onto the OPNs to prevent the occurrence of saccadic eye movements when the monkey is fixating on a target of interest.

The SC has also been shown to have direct control over the saccade generator circuitry. Stimulation of the SC in monkey leads to short-latency activation of the burst neurons (BNs) in the paramedian pontine reticular formation (Raybourn and Keller 1977). Figure 20 shows this signal arising from the saccade-related cells in the SC, because these cells in both monkey and cat project into the predorsal bundle (Berthoz et al. 1986; Moschovakis et al. 1988; Munoz and Guitton 1989).

In the scheme illustrated in Fig. 20, there are two ways in which collicular fixation cells could suppress the initiation of saccades. First, they could inhibit the activity of the collicular saccade-related cells through local connections within the SC. Second, they could activate the brain stem OPNs, which would then suppress saccade generation at the level of the burst neurons.

Fixation system within the brain

The fixation cells we have studied in the rostral pole of the monkey SC are similar to neurons in other areas of the brain that are also active during visual fixation. It seems likely that at least some of these areas are part of a larger system for visual fixation within the brain that interacts with the saccadic system at several levels.

Preeminent among cortical areas with neuronal activity that is modulated by active fixation is area 7a of posterior parietal cortex (Lynch et al. 1977; Mountcastle et al. 1975; Sakata et al. 1980). These visual fixation neurons are similar to the fixation cells in the SC in that they are only sporadically active when the animal spontaneously looks around its surroundings but increase their activity abruptly with fixation of a target to obtain a reward. Many parietal neurons pause with saccades from one target to another, they continue to discharge during smooth pursuit eye movements, and they do not require the presence of the visual stimulus for the fixation activity to occur. The parietal neurons differ from the collicular cells in that they do not pause for saccades in all directions and many cells do not discharge with fixation at all orbital positions. But such a restriction in the response of the parietal neurons might not be seen at the SC if a number of parietal neurons with different orbital position sensitivities projected to the same SC fixation cells. The reduction of the discharge of parietal neurons with saccades was not as closely related to the onset and termination of saccades as was that of the SC neurons, but the duration of the pause of SC fixation neurons might be determined within the SC. Thus, although there are significant differences between the fixation cells in these two areas, it is possible that the parietal fixation neurons and the SC fixation neurons are part of the same fixation system.

In the frontal eye fields, one population of identified corticotectal cells responded to foveal visual stimuli but did not change discharge rate with active fixation (Segraves and Goldberg 1987) as did the collicular fixation cells. The frontal eye field neurons seem less likely than parietal neurons to be involved in visual fixation in the same way as the SC. In other areas of frontal cortex, cells with increased activity during active visual fixation have also been identified, and, whereas less is known about these neurons, they might form part of a fixation system (Bon and Lucchetti 1992; Schlag et al. 1992; Suzuki and Azuma 1977).

Neurons in the basal ganglia discharge in relation to visual fixation but appear to be quite different from the fixation cells in the SC. In the substantia nigra pars reticulata (SNr), fixation-related neurons are dependent on the presence or absence of the fixation stimulus (Hikosaka and Wurtz 1983), whereas those in the caudate nucleus, which projects directly to the SNr, would seem to be more closely related to the expectation of reward than fixation alone (Hikosaka et al. 1989). In the subthalamic nucleus, neurons active during visual fixation also decrease their discharge when the fixation point is blinked off, indicating that

they too require the presence of the visual target (Matsumura et al. 1992). Thus, from what is known about these divisions of the basal ganglia, it seems unlikely that their input is relevant to the type of fixation activity seen in the rostral SC.

Few of the above-mentioned areas project selectively to the rostral pole of the SC where we found fixation cells. There is a clear projection from the posterior parietal cortex to the SC (Lynch et al. 1985), but it remains to be determined whether the parietal fixation neurons project selectively to the rostral SC. Projections from the frontal eye fields include but are not limited to the rostral SC (Komatsu and Suzuki 1985). One noteworthy exception of a projection exclusively to the rostral SC is the input from the deep cerebellar nuclei to the SC in monkey (May et al. 1990). Two separate projections were identified: one pathway arising in the caudal fastigial nucleus and terminating in the rostral pole bilaterally; and a separate projection arising from the posterior interposed nucleus and the adjacent posterior wing of the dentate and terminating throughout the ventral half of the intermediate and deep layers of the contralateral SC. The fastigial nucleus therefore appears to project specifically to the part of the SC where we identified fixation cells. The functional significance of this projection remains to be determined.

The fixation cells within the rostral pole of the SC may be part of the final common pathway from cortical fixation areas to the brain stem premotor circuitry. Saccades elicited by microstimulation of the SC (Sparks and Mays 1983), frontal eye fields (Goldberg et al. 1986), and posterior parietal cortex (Shibutani et al. 1984) have longer latencies and higher thresholds when the monkeys were actively fixating a visual target compared with being in darkness. Stimulation of the frontal eye fields (Azuma et al. 1986) and areas within the superior temporal sulcus representing foveal vision also delayed the onset of saccades (Komatsu and Wurtz 1989). Some of these effects may have been mediated via the SC fixation cells, which presumably would have had to be silenced before saccade initiation.

In net, we think that the properties of the fixation cells in the rostral pole of the monkey SC are consistent with the hypothesis that increased activity here suppresses the generation of saccades. In the next article (Munoz and Wurtz 1993), we test our hypothesis by altering activity of fixation cells in the rostral SC and then measuring the monkey's ability to fixate and make saccades.

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