The Relationship of Monkey Frontal Eye Field Activity to Saccade Dynamics

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SUMMARY AND CONCLUSIONS
1. In this study, we compared the temporal waveforms of the activity of monkey frontal eye field movement neurons with the dynamics of saccadic eye movements.

2. Movement neurons in the frontal eye field were selected according to previously published criteria. They had little or no response to visual stimuli in a fixation task, and equivalent activity before visually guided and memory-guided saccades. We studied corticocortical neurons and corticopontine neurons identified by antidromic excitation, as well as neurons whose projections were not identified.

3. These neurons had a peak activation at a mean of 13 ms before the saccade began. However, rather than falling off rapidly as the saccade ended, most neurons continued to fire after the saccade, returning to baseline at a mean of 93 ms after the end of the saccade.

4. We measured the decrement in activity for these neurons during the saccade. Although a few neurons showed decrements of >60% of their peak activity level, the average activity dropped only 16.9%, with some neurons actually showing a rise in activity during the saccade. If we ignored the latency between peak in activity and saccade start and measured the fall in activity for a period equal to one saccade duration after the peak, the average drop in activity was still only 34.9%. Thus, the activity of these neurons did not appear to be closely related to dynamic motor error, which falls from its maximum value to zero over the time course of a saccade.

5. These results suggest that a focus of movement activity within the topographic map in the frontal eye field specifies the amplitude and direction for an impending saccade, whereas the peak of movement activity signals the time to initiate a saccade.

6. Unlike the superior colliculus, the activity of frontal eye field movement neurons does not appear to be related to dynamic events that occur during the saccade, such as motor error.

INTRODUCTION
The frontal eye field has an important role in the control of saccadic eye movements. Microstimulation of this portion of frontal cortex produces saccades with short latency and low threshold (Bruce et al. 1985; Robinson and Fuchs 1969); its movement neurons discharge before purposive saccades, including those made to remembered target locations (Bruce and Goldberg 1985). Frontal eye field movement neurons project to both the superior colliculus and to oculomotor regions of the pons, including the region of omnipause neurons in the paramedian pontine reticular formation (Segraves 1992; Segraves and Goldberg 1987). Although it is likely to participate in the generation of all voluntary saccades, the frontal eye field appears to be essential for the generation of cognitively difficult forms of saccades, including saccades to remembered target locations (Deng et al. 1986) and antisaccades—saccades directed away from the spatial location of a visual cue (Guitton et al. 1985).

Within cerebral cortex, several regions are involved in the control of saccades besides the frontal eye field, including posterior parietal cortex (Andersen and Gnadt 1989; Barash et al. 1991a,b; Duhamel et al. 1992; Kurylo and Skavanski 1991), the supplementary eye field (Schall 1991; Schlag and Schlag-Rey 1987), and the cortex surrounding the principal sulcus (Boch and Goldberg 1989: Funahashi et al. 1991). These regions share many connections and appear to form a cortical network involved in the control of saccades. Recently, it has been reported that lesions of the frontal eye field or parietal regions alone do not have a substantial effect on visually guided saccades, whereas combined lesions reduce the accuracy and increase the latency for saccades to visual targets (Lynch 1992). These monkeys, however, recovered from the effects of these lesions after several weeks. Schiller and colleagues (1980) have shown that combined bilateral lesions of the superior colliculus and frontal eye field cause substantial reductions in the range of eye movements, as well as decreases in the frequency and velocity of saccades. These deficits do not diminish over time, and suggest that the frontal eye field generates an essential output from the cortical network.

The frontal eye field has many features in common with the superior colliculus. Both regions have neurons that discharge before movements; both can support saccades in the absence of the other; and of all cortical regions studied, only the frontal eye field can evoke saccades by electrical stimulation in the absence of the superior colliculus (Keating et al. 1983; Schiller 1977). Although they seem to have parallel functions in the generation of saccades, the frontal eye field does have the ability to influence the colliculus in the generation of saccades in the normal monkey. It sends a powerful presaccadic signal to the colliculus (Segraves and Goldberg 1987), and may also help to control the basal ganglia circuits, which themselves exert an influence on the colliculus (Hikosaka et al. 1989). It is believed to exert a cognitive influence on a more stimulus-bound colliculus, helping to select the appropriate eye movement to make from a variety of possibilities (Goldberg and Segraves 1987).

Many characteristics of saccade-related activity in the superior colliculus have been known for some time. The most prominent feature of the firing pattern of neurons with saccade-related activity is a burst of firing that begins ~20 ms before the start of the eye movement (Mays and Sparks...
1980; Mohler and Wurtz 1976; Sparks 1978). For some neurons, this burst is preceded by a gradual increase in firing rate beginning shortly after the signal to make a saccade—several hundred milliseconds before the beginning of the movement. Because the focus of this activity arises from a location within the movement map in the intermediate and deep layers of the colliculus, this activity could signal the amplitude and direction of an impending movement. The burst of activity occurs at a time appropriate to trigger brain stem saccade-generating circuitry. Recent evidence, however, suggests that the activity of subpopulations of collicular neurons helps to control dynamic aspects of the saccadic eye movement. Focal lesions in the superior colliculus result in decreased saccadic velocity as well as dysmetria (Hikosaka and Wurtz 1986; Lee et al. 1988), suggesting that the superior colliculus is involved in the actual motor programming of the saccade. Berthoz and colleagues (1986) showed that part of the collicular discharge resembles the velocity profile of the ongoing saccade, and Waitzman and colleagues (1988, 1991) have shown that the discharge of some collicular eye movement neurons decreases as a linear function of the distance remaining in the saccade, the motor error.

Because of its close relationship to the superior colliculus in the generation of movement, we thought it likely that the frontal eye field would also be involved in the control of saccade dynamics. Such a relationship would be particularly important in neurons that project to the pons, neurons that are likely to drive saccadic eye movements in the absence of the superior colliculus. In these experiments, we compared the relationship between neuronal discharge and saccadic eye movements to see if the frontal eye field, like the superior colliculus, was important in the dynamic programming of saccades. We studied three populations of movement neurons: identified corticocollicular neurons, identified corticopontine neurons, and neurons that could not be excited antidromically from either the superior colliculus or the pons. The presaccadic component of the activity of these neurons was similar to the original descriptions of saccade-related neurons in the colliculus. This activity began to rise above baseline ~100 ms before the beginning of the saccade, and peaked just before the start of the eye movement. This signal might 1) relay the amplitude and direction of an impending saccade to the superior colliculus and pons and 2) initiate the trigger to make a saccade. We failed to show a consistent relationship between saccade dynamics and neuronal discharge for these cortical neurons. In particular, frontal eye field neurons did not appear to emit a signal that could be related to motor error or velocity during the saccade. A preliminary report of these experiments has been published elsewhere (Segraves et al. 1990).

METHODS

The data described in this paper were obtained during single-unit recordings from four behaving rhesus monkeys (Macaca mulatta) identified as LSR30, LSR50, LSR51, and MASO1. All experimental procedures for monkeys LSR30, LSR50, and LSR51 were performed at the National Eye Institute. The experimental procedures for monkey MASO1 and subsequent data analysis for all four monkeys was performed at Northwestern University. The neurons described in this report are a subset of frontal eye field neurons encountered during experiments designed to identify and characterize neurons with projections to the superior colliculus and pons. The procedures for training, surgery, electrophysiology, and histology for these monkeys appear in the original reports (Segraves 1992; Segraves and Goldberg 1987).

Behavioral tasks

To obtain the data described in this report, behavioral tasks with and without saccades were used. 1) Visual no-saccade task: the monkey fixated the stationary light spot in the center of the tangent screen (FP in Fig. 1) for the duration of the trial. During the
FIG. 2. Activity in the visually guided saccade task. While the monkey was fixating, the fixation point was turned off and a target light (T) turned on for 50 ms at the same location used in the visual no-saccade task illustrated in Fig. 1. The rasters and histograms illustrate activity occurring 200 ms before and after the onset of the target light (A), the beginning of the saccade (B), and the end of the saccade (C). In this task, the saccade begins ~200 ms after the onset of the target, and so it is difficult to separate visual- from movement-associated activity. It is clear, however, that the activity is much stronger than when the monkey did not make an eye movement (Fig. 1), and that the increase in activity peaks near the beginning of the saccade.

Data analysis

Rasters and histograms were constructed with neuron activity aligned on important events within a trial.

We classified neurons on the basis of their activity during visuomotor tasks, using the criteria of Bruce and Goldberg (1985). Our present analysis was confined to neurons with movement activity. Movement neurons have little or no response to visual stimuli, but very strong activity before both visually guided and memory-guided saccades. Movement neurons in the frontal eye field have definable movement fields. Their peak discharge occurs for saccades of a specific amplitude and direction, with these parameters depending on the neuron’s location in the frontal eye field’s topographic map.

To facilitate the comparison between neuron activity and the changes in eye position and velocity during saccades, the static raster or histogram plot of activity was converted to a continuous spike density function (Richmond et al. 1987; Sanderson and Kobler 1976). This was done in two ways. For most neurons (n = 33), both neuron firing activity and eye position data during eye movement trials were stored at 250 or 1,000 Hz. A Gaussian curve with $\sigma = 8$ ms was fitted to the time of occurrence of each neuron spike during a trial. For each trial, Gaussian curves representing each spike were summed, providing a continuous function ex-
pressing the expected spike frequency for any point in time during the trial. This activity was then averaged across trials to produce the average spike density for the neuron in a particular task. For these data, the beginning and end of the saccade were defined as the times at which eye velocity exceeded and then decreased below 100°/s. For the remaining neurons (n = 15), continuous records of eye position during each trial were not available. For these cells, spike density functions were generated from rasters of neuron activity that contained timing marks to indicate the beginning and end of the saccade. These rasters were generated during the recording experiment with a sampling frequency of 250 Hz. The beginning and end of the saccade were determined on-line at the time of the experiment and corresponded to the times at which eye velocity exceeded and then decreased below 30°/s. For these data, a histogram of activity averaged across 16 trials was generated and then a spike density function, with σ again equal to 8 ms, was derived from the histogram. Although two different velocity criteria were used to distinguish the beginning and end of saccades, these times, as well as the durations of saccades obtained with both techniques, were very similar.

RESULTS

We examined the activity of a total of 48 frontal eye field movement neurons. These included 35 identified neurons: 26 corticocellular neurons and nine corticopontine neurons. In addition, our analysis included 13 movement neurons whose projections were not identified: one neuron that could not be antidromically excited by a collicular stimulating electrode, and 12 neurons that were not antidromically excited by a pontine electrode.

The activity of a corticopontine movement neuron is illustrated in Figs. 1–4. This particular neuron was chosen for illustration because the timing of the beginning, peak, and end of its saccade related activity was similar to the average times for the entire sample. During the visual no-saccade task (Fig. 1), this movement neuron gave only a few spikes per trial in response to the 50-ms flash of a spot of light in the center of its movement field. In contrast, it gave brisk bursts of activity that preceded and continued through the duration of both visually guided saccades (Fig. 2) and saccades made in darkness to the remembered location of the target spot (Fig. 3). Although the rasters show some variability from trial to trial, the histograms illustrate that, overall, the activity of this cell began to rise ~150 ms before the start of the saccade, peaked near the beginning of the saccade, and fell back to a baseline level ~100 ms after the end of the eye movement. The cell’s activity for both visually guided and memory-guided saccades was quite similar.

Figure 4 illustrates the mean spike density for the same neuron shown in Figs. 1–3. The mean spike density is superimposed on plots of vertical eye position and velocity. Velocity was computed by digital differentiation. The spike density is compared to saccades made to both visually (Fig. 4A) and memory-guided saccades (Fig. 4B).

A qualitative examination of the spike density profile confirms what was seen in the histograms of Figs. 2 and 3. The rise in spike density related to the saccade begins and ends ~100 ms before and after the saccade, and peaks near the start of the eye movement. Note that the spike density profile was similar for firing activity generated in both visually guided (Fig. 4A) and memory-guided (Fig. 4B) saccade tasks.

For each of the 48 neurons included in our sample, we made quantitative measurements of the timing of the rise in spike density relative to the saccade. Figure 4B illustrates how these measurements were made. The baseline level of spike activity was determined by examining the activity for ~1,000 ms before and after the start of the saccade. The beginning and end of the spike density rise were defined as the times at which spike density rose above and later returned to the baseline level of activity. The time for the beginning and peak of the rise in spike density was measured relative to the start of the eye movement. The time for the end of saccade-related spike density activity was measured relative to the end of the saccade.

Figure 5 illustrates the distributions of times for the beginning, peak, and end of the change in spike density associated with saccades. For 27 neurons, the times used for these distributions were measured from data obtained during the visually guided saccade task. For the remaining 21 neurons, sufficient data for visually guided saccades did not exist, and the times for these distributions were measured from data obtained during the memory-guided saccade task. These movement neurons began to increase their activity from 31 to ~200 ms before the start of the saccade (Fig. 5A). The mean time for activity to rise above the baseline level was 148 ms before the beginning of the saccade. Their activity peaked from 120 ms before to 93 ms after the start of the saccade (Fig. 5B), with 58% of the neurons having a peak of activity within 40 ms of the start of the saccade. The mean time for activity to peak was 13 ms before the saccade began. Some significant differences (t test, P < 0.05) were found for the mean times of peak activity for the different groups of neurons included in this study. The peak of activity for corticocellular neurons (~34 ms) occurred earlier than it did for both corticopontine (~2 ms) and unidentified (~20 ms) movement neurons. All neurons, except three corticocellular cells, finished firing after the saccade was completed (Fig. 5C). Most neurons continued firing for a substantial period of time after the end of the eye movement, with a mean of 93 ms after the end of the saccade.

In the superior colliculus, neurons that are believed to code dynamic motor error showed a sharp decrement in their firing activity during a saccade (Waitzman et al. 1991). These collicular neurons had peak activity at the beginning of a saccade, and their activity decreased nearly 100% during the course of the eye movement. We examined the decrement in activity during saccades for the frontal eye field neurons included in this study.

For each neuron, we determined the magnitude of the change in activity during the saccade and expressed this value as a percentage of the peak firing activity. A change in activity from peak to baseline level during the saccade was represented as a 100% drop. Increases in activity during the saccade were expressed as negative percent drops. For example, for the neuron illustrated in Fig. 4B, activity dropped an amount equal to 33% of the peak activity during the visually guided saccade task (Fig. 4A), and dropped 28% during the memory-guided saccade task (Fig. 4B). Figure 6 is a plot of these values for the 48 frontal eye field neurons included in this study. Each value is referenced to the latency of the neuron’s peak discharge to the beginning of the saccade. Overall, the activity of these neurons dropped 16.9 ±
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FIG. 3. Activity in the memory-guided saccade task. While the monkey was fixating, a target light was turned on for 350 ms and then turned off. Fifty milliseconds after the target was extinguished, the fixation point was turned off, signaling to the monkey that he could make a saccade. The saccade was made, in darkness, to the remembered location of the flashed target. The monkey was rewarded for making a saccade of appropriate amplitude and direction. Rasters and histograms are arranged as in Fig. 2. In this task, the onset of the target and the beginning of the saccade were separated by ~500 ms. As a result, it is easy to distinguish the faint visual response in A from the substantial movement-related activity shown in B and C.

31.3% (mean ± SD) during the eye movement. Note that only a few neurons showed drops >60%, and many neurons actually increased their activity during the saccade. The activity of unidentified movement neurons tended to drop less during the saccade than did other neurons, although this difference was only significant when comparing unidentified with corticocellular neurons (t test, P < 0.05).

We attempted to allow for delays between the drop in activity after the peak and the actual time of generation of the movement by measuring the drop in activity during a time period equal to one saccade duration immediately after the peak in discharge. For the neuron illustrated in Fig. 4, this value was equal to 40% for visually guided saccades (Fig. 4A) and 27% for memory-guided saccades (Fig. 4B). This procedure eliminates the generation of negative percent drops. Figure 7 plots the distribution of these values for all 48 neurons. This manipulation ignores the latency required for frontal eye field activity to have an effect on eye movement dynamics, and produces a narrower distribution of changes in activity. We found an average of 34.9 ± 15.4% (mean ± SD) drop in activity. Again, only a small number of neurons showed a drop in activity of >60% of the peak activity level. Corticopontine neurons tended to drop less quickly after the peak of activity, but this tendency was only significant when comparing the means for corticopontine with unidentified movement neurons (t test, P < 0.05).

DISCUSSION

We have examined the activity of movement neurons in the frontal eye field and have compared that activity with the dynamics of saccadic eye movements. The population of movement neurons that we studied included cells with identified projections to the superior colliculus or to the pons, as well as neurons whose termination sites were unknown. As a group, these neurons began to increase their activity >100 ms before the start of a saccade. Their activity peaked with a mean of 13 ms before the beginning of the saccade, and then gradually fell toward baseline levels. Their activity reached baseline 93 ms after the completion of the saccade. On average, the activity of these neurons fell during the saccade by an amount equal to only 17% of the peak activity level. There was a great deal of variability in this measurement, with a few neurons showing decrements of >60% during the saccade and a number of neurons showing increases in activity during the eye movement.

This discussion will consider the probable role of frontal eye field movement activity in the generation of saccades, and the relationship of that activity to feedback control models of the saccadic system, and will make comparisons with the role performed by the superior colliculus in this process.

Frontal eye field movement neurons signal the amplitude and direction of an intended movement, and the time to initiate that movement.

The scope of this study was restricted to an examination of neurons with movement-related activity. Movement neurons form the largest population of cells that project to the superior colliculus and pons (Segraves 1992; Segraves and Goldberg 1987). Thus their activity generates a major out-
put signal that is sent from frontal eye field to the brain stem. We have not included foveal neurons in this study. Frontal eye field foveal neurons are responsive to visual stimulation of the fovea and, in addition, have activity related to fixation (Bizzi 1968; Bruce and Goldberg 1985; Suzuki and Azuma 1977; Suzuki et al. 1979). Foveal neurons compose the second-largest group of neurons that project to the superior colliculus and pons. Their activity is believed to contribute to the maintenance and release of fixation (Segraves 1992; Segraves and Goldberg 1987).

Figure 8 summarizes our current understanding of the signals sent by frontal eye field movement neurons to both
FIG. 5. Timing of the beginning, peak, and end of the rise in spike density associated with the saccade. Measurements taken from plots such as those illustrated in Fig. 4 were used to produce histograms of the distribution of the times for beginning (A), peak (B), and end (C) of the rise in saccade-related spike density. These data were put into bins 20 ms wide. Note that for A and B, time = 0 marks the beginning of the saccade, and for C, time = 0 marks the end of the saccade. Time values less than −200 ms were included in the −200-ms bin; values greater than +200 ms were included in the +200-ms bin. Values for 48 neurons are included in these histograms.

the superior colliculus and pons. Our interpretation of the present results is that the movement neurons in the frontal eye field provide two signals relevant to the generation of a saccadic eye movement. 1) The peak of their activity signals the time to initiate the saccade. It is a trigger signal. The peak in movement neuron activity, at a mean of 13 ms before the saccade, occurs at about the time that omnipause neurons are turned off at 10–15 ms before the saccade (Henn and Cohen 1976; Keller 1974; Luschei and Fuchs 1972; Robinson 1975). On the basis of the latencies of corticopontine, corticotectal, and tectopontine cells, this signal could directly excite pontine cells within 1.4–4.0 ms and within 2.3–4.9 ms via a projection from cortex to colliculus to pons (Raybourn and Keller 1977; Segraves and Goldberg 1987). Because it is unlikely that the corticopontine neurons are directly inhibitory (Schwartz et al. 1985, 1988), if frontal eye field neurons turn off pause neurons, they probably do so via inhibitory neurons in the vicinity of the pause cells. 2) The sustained increase in activity output from a group of movement neurons signals the amplitude and direction for the impending saccade. This activity would originate from a restricted area within the movement map of the frontal eye field that represents large eye movements dorsomedially and small eye movements ventrolaterally (Bruce et al. 1985). Our study does not rule out the possibility that some frontal eye field neurons send a dynamic motor error signal to the midbrain and pons; our sample may have missed those neurons. The present analysis, however, makes it clear that the frontal eye field is sending both a trigger and a desired change in eye position signal to the midbrain and pons. These signals are very similar to those ascribed by Sparks and colleagues to saccade-related burst neurons in the superior colliculus. Those collicular neurons gave a burst of activity that began ~10–20 ms before the start of a saccade with optimal amplitude and direction. About one fourth of superior colliculus movement neurons showed a gradual rise during the period between the signal to make a saccade and the beginning of the saccade (Mays and Sparks 1980; Sparks et al. 1976). This collicular activity may arise, in part, from frontal eye field input, but is probably also due to the integration of input from many other sources. Concerning the slow rise in fron-
Frontal eye field does not contribute to feedback control or the specification of eye velocity during saccades

Feedback is an important component of the control system for generating saccades [see Van Gisbergen and Van Opstal (1989) for review]. Early experimental evidence for this feedback came from patients with spinocerebellar degeneration whose saccades were sufficiently slow to allow intrasaccadic modification of their trajectories (Zee et al. 1976). Other evidence came from normal human subjects (Becker and Jürgens 1979; Hallett and Lightstone 1976) and monkeys (Sparks and Mays 1983). Robinson (1975, 1981) proposed a local feedback control model where desired eye position is compared with an efference copy of actual eye position to yield a motor error signal. The motor error signal drives the burst neurons, which in turn produce the pulse of eye velocity that provides the major component of the motor neuron discharge. In subsequent modifications of the feedback model (Jürgens et al. 1981; Scudder 1988; Van Gisbergen and Van Opstal 1989), desired saccade amplitude is compared with the distance traversed by the current saccade to produce the motor error signal. The present results suggest that the frontal eye field does not participate in the feedback process, because its signals are relatively constant during the saccade. Thus the feedback loop must occur at a level downstream from the frontal eye field cortex. Scudder (1988) has proposed that the feedback occurs at the level of long lead burst neurons in the pons, with frontal eye field and superior colliculus providing similar inputs specifying the saccade target and the trigger to initiate the movement. The activity of many saccade-related neurons in the superior colliculus seems appropriate for this function, and is in many ways similar to that found in the frontal eye field (Mays and Sparks 1980; Mohler and Wurtz 1976; Sparks 1978). Recent experiments, however, provide evidence that would place the superior colliculus within the feedback loop. Waitzman and colleagues have demonstrated that some neurons in the monkey superior colliculus appear to have activity that codes dynamic motor error (Waitzman et al. 1988, 1991). They propose that the colliculus generates this signal by continually subtracting a current change in eye position signal from the initial desired change in eye position signal. The current change in eye position signal would be obtained by integrating the eye velocity command output by burst neurons (Waitzman et al. 1988, 1991). Munoz and colleagues have proposed that neurons in the cat superior colliculus lie within a gaze feedback loop and exhibit changes in activity that are related to the dynamics of movements of the eyes and head (Munoz et al. 1991). In this case, brain stem feedback is used to shift the center of activity across the collicular movement map, from the region coding the desired saccade amplitude and direction toward the fixation zone at the rostral pole of the colliculus. Their model also requires that the feedback signal pass through a neural integrator (Guitton 1991; Munoz et al. 1991).

The superior colliculus may also help to control the velocity of saccades (Berthoz et al. 1986; Iikosaka and Wurtz 1986; Lee et al. 1988). We did not, however, find evidence in this study for a relationship between eye velocity and the activity of movement neurons in the frontal eye field. In most cases, frontal eye field activity declined slowly during the saccade—an inappropriate signal for coding the dynamics of eye velocity. Waitzman and colleagues (1991) have pointed out that a steadily declining signal could not code the increase and decrease of velocity that occur during a saccade. If it did, equal velocities during the acceleration and deceleration phases of the movement would be coded by different levels of neuron activity. Unfortunately, our experiments did not allow us to address the possibility that frontal eye field activity might code the peak velocity for an impending saccade. Although it has been reported that the velocity of equal-length saccades can vary depending on the eye movement task (Rohrer et al. 1987), we noted very little variation in the velocity of saccades generated in our visually versus memory-guided saccade tasks. One possible explanation is that the gap between target disappearance and the cue to make a saccade tended to be relatively short in our memory task (≤250 ms).

Relative contributions of the frontal eye field and superior colliculus in the generation of saccades

Placing the superior colliculus, but not the frontal eye field, within a feedback loop involved in the dynamic control of saccades is difficult to reconcile with the finding that monkeys can make visually guided saccades after bilateral removal of the superior colliculus (Schiller et al. 1980). This lesion study emphasized the parallel nature of the participation of colliculus and frontal eye field in the generation of saccades. If, as recent findings suggest, the superior colliculus is within a feedback loop that supplies the oculomotor system with a dynamic motor error signal, how is this signal derived in the absence of the superior colliculus? One possibility is that the frontal eye field, as well as other oculomotor-related areas, modify their output in the absence of the superior colliculus and provide the needed motor error signal to the brain stem. Anatomic projections
from oculomotor cortex to the brain stem that bypass the superior colliculus are known to exist. Both the frontal eye field and supplementary eye field project directly to oculomotor regions of the pons, including the region of omnipause neurons in the nucleus raphe interpositus; the general vicinities of burst neurons in the paramedian pontine reticular formation, and nuclei forming relays with the cerebellum, including the nucleus reticularis tegmenti pontis and basal pontine nuclei (Huerta and Kaas 1990; Huerta et al. 1986; Leichnetz et al. 1984; Schnyder et al. 1985; Shook et al. 1990; Stanton et al. 1988). An alternative explanation is that the dynamic motor error signal observed in the superior colliculus is not necessary to make accurate saccades, but is perhaps part of a corollary discharge generated by the colliculus. It should be noted that the anatomic targets of the collicular dynamic motor error cells described by Waitzman and colleagues have not been identified. Finally, it may be possible that a desired change in eye position signal from the frontal eye field is sufficient for other structures such as the cerebellum to drive the saccade-generating mechanism in the absence of a motor error signal from the colliculus.

Although it is unclear how saccades are generated in the absence of the superior colliculus, under normal circumstances the superior colliculus and frontal eye field, as well as other regions including posterior parietal cortex, the supplementary eye field, and periprincipal cortex operate via both serial and parallel pathways to control the generation of saccades. Our examination of frontal eye field activity combined with the work of other laboratories investigating superior colliculus activity reveals a significant difference between the superior colliculus and frontal eye field in their roles in the generation of saccades. Whereas the colliculus appears to code signals that are related to dynamic aspects of the movement, like velocity and motor error, the frontal eye field functions at a higher level in the saccade generation process, signaling the target for the saccade and triggering the movement. This emphasizes a purported role for the frontal eye field as a link between cortical cognitive processing preceding the initiation of an eye movement and the more machine-like organization of the midbrain and brain stem designed to execute the motor command (Goldberg and Segraves 1987). As such, the frontal eye field may select the target for the next eye movement from a variety of external visual stimuli and internal cues, but it is not involved in the guidance of the movement once it has begun.

We are grateful to the following for skilful support: A. Hayes for computer software; A. Ziminsky and Northwestern’s Chemistry Electronics Shop for electronic hardware; C. Crist, T. Rutter, and Northwestern’s Physics Instrument Shop for machining; M. Smith for histology; C. Polchow for histology and photography, and A. Hanson, H. Macgruder, K. Powell, and the staff of Northwestern’s Center for Experimental Animal Resources for animal care. We are particularly grateful to M. Goldberg for providing the computer software to generate the spike density functions and for innumerable discussions and insights concerning this work. We thank D. Burman for his comments and discussion concerning this manuscript.

This work was supported by National Eye Institute Grant EY-08212, Division of Research Resources-Biomedical Research Development Grant-RR-07028, and The Sloan Foundation.

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Received 24 July 1992; accepted in final form 29 January 1993.

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