Superior Colliculus Neurons Mediate the Dynamic Characteristics of Saccades

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SUMMARY AND CONCLUSIONS

1. The locus of activity within the superior colliculus (SC) is related to the desired displacement of the eye. Current hypotheses suggest that the location of this locus of activity determines the amplitude of the saccade and that the level of activity at this locus determines eye velocity. We present evidence that suggests that, although the locus determines the amplitude of the saccade, the level of activity in the colliculus encodes dynamic motor error (the difference between desired and current eye displacement).

2. We categorized 86 neurons in the intermediate and deep layers of the superior colliculus of two rhesus monkeys by their activity in relation to the end of saccadic eye movements. In 36% of the cells (n = 31), activity was completely cut off by the end of the saccade (clipped cells). For 53% of cells (n = 46), the major burst of activity ceased by the end of the saccade, but activity continued for 30-100 ms after the end of the movement (partially clipped cells). The remaining 10% of the cells (n = 9) had no clear burst of activity (unclipped cells) but rather had activity that increased gradually before the saccade and then slowly decreased for up to 100 ms after the saccade. These categories were part of a continuum of cell types rather than discrete classes of cells.

3. We determined whether this new categorization of cells revealed a relation between the discharge of clipped and partially clipped cells and saccadic amplitude and peak velocity. As expected, we found a steady increase in spike count as saccadic amplitude increased up to the center of the movement field, and an increase in peak saccadic discharge as peak velocity increased up to a maximum radial eye velocity. Variability in the cell discharge was substantially greater than the variability of saccadic amplitude or peak velocity. We concluded that these single point or averaged measures did not reveal any new functional relationship of these cells.

4. We then examined the relationship of the temporal pattern of discharge of clipped and partially clipped cells to instantaneous changes in radial error and radial velocity. There was a monotonic delay in spike discharge with declining radial error. In contrast, there was a complex, multivalued relationship between spike discharge and radial velocity; collicular cells produced two different values of spike discharge for the same velocity, one during acceleration and the other during deceleration of the eye during a saccade. When saccadic duration and cell discharge were normalized within groups of clipped and partially clipped cells of the same amplitude range, these relationships of radial error and radial velocity to spike discharge were maintained.

5. When all amplitude groups were normalized and averaged together, the relationship of radial error to spike discharge remained very close to linear. However, the complex, multivalued relationship of radial velocity to spike discharge became much closer to single valued, because cells whose discharge either led or lagged the changes in velocity were averaged together.

6. On the basis of these observations, we propose a collicular feedback model of the saccadic system that places the SC inside the feedback loop controlling saccadic amplitude. This model can account for the dynamic relationships of collicular firing to both saccadic amplitude and velocity. It also provides insight into previous experiments showing that a change in the level of activity in the SC can change saccadic velocity. Our model shows explicitly how the superior colliculus participates in transforming the spatial locus of collicular activity into the rate of discharge needed to innervate eye muscles (the spatial-to-temporal transformation).

INTRODUCTION

That neurons in the intermediate and deep layers of the superior colliculus discharge before the onset of saccadic eye movements has been known for two decades (Schiller and Koerner 1971; Wurtz and Goldberg 1971, 1972). These neurons discharge before saccades made to a particular region of the visual field, referred to as the movement field of the neuron (Wurtz and Goldberg 1972). The movement fields of these neurons form a topographic map of desired eye displacement in intermediate and deep layers of the superior colliculus (Sparks et al. 1976; Sparks and Mays 1980; Wurtz and Goldberg 1972). Electrical stimulation of these layers yields saccades and a similar topographic map (Robinson 1972; Schiller and Stryker 1972; Sparks et al. 1976). Neurons in these layers project to the brain stem areas known to be related to the generation of saccades (Harting 1977).

In a recent review, Sparks and Mays (1990) summarized what is referred to as a dual coding hypothesis for the contribution of the superior colliculus to the generation of saccades. First, the amplitude of the impending saccade is determined by the locus of maximal activity in the topographic map within the intermediate and deep layers of the superior colliculus. This discharge encodes initial motor error, the direction and amplitude of the desired change in eye position, but not the movement to a particular orbital position. This relationship of saccadic amplitude to the locus of activity in the superior colliculus is based on the experiments cited above, and there seems to be general agreement on this aspect of collicular function. However, why that relationship should guarantee saccadic accuracy despite variations in saccadic velocity is not known.

The second tenet in this dual coding hypothesis is that "the rate of firing in the active population may be a determinant of the vectorial velocity of the ensuing saccade" (Sparks and Mays 1990). The relation of the superior colliculus to the velocity of a saccade was first suggested by several reversible lesion experiments: injection of either lidocaine or muscimol into the colliculus reduces the velocity of
the saccade (Hikosaka and Wurtz 1985, 1986; Lee et al. 1988). In addition, microstimulation of the superior colliculus produces acceleration of saccades (Munoz and Guitton 1987a; Munoz et al. 1992). Finally, several experiments show that the discharge rate of collicular cells is positively correlated with saccadic velocity (Berthoz et al. 1986; Lee et al. 1988; Rohrer et al. 1987). Although a relation between the superior colliculus and the velocity of the saccade is an experimental fact, we think that these experimental observations show that the activity of collicular neurons is correlated with saccadic velocity but the activity does not encode saccadic velocity. We will argue that collicular discharge encodes the dynamic motor error that in turn is used by midbrain structures to determine saccadic velocity. Thus collicular discharge is correlated with both dynamic motor error and velocity, but it encodes the former, which influences the latter.

In the present experiments we reexamined the relationship between the discharge of superior colliculus cells and both saccadic amplitude and saccadic velocity. We did this by comparing the discharge of the collicular cells over time with the dynamic changes in both saccadic amplitude and velocity. As we reported previously (Wattzman et al. 1988), the discharge of many collicular cells decreased during saccades and stopped, more or less abruptly, when the saccades ended. We have now extended this analysis to show that the activity of many collicular neurons is nearly linearly related to the dynamic motor error: the difference between desired and current eye displacement. On the basis of these observations and the model of saccadic control by Jürgens et al. (1981), we propose a collicular feedback model of saccadic control that places the superior colliculus inside a feedback loop. This loop provides the error signal needed to control saccadic amplitude by continually comparing the desired and current eye displacements. The results of such a comparison are consistent with the decline in discharge rate seen in many collicular neurons over time. Because the output of this comparison is envisioned in the model as the input to the saccadic velocity controller, the saccadic activity would be expected to correlate with saccadic velocity as well as desired change in eye position. However, only by making the colliculus the position comparator can saccadic amplitude be preserved despite the effects of lesions or stimulations that alter velocity.

METHODS

Physiological and behavioral procedures

We studied two female rhesus monkeys (Macaca mulatta) that had been prepared for single-cell recording under general anesthesia (intravenous pentobarbital sodium titrated to effect) and aseptic conditions. A head holder for restraint of the head during the experiments, a stainless steel chamber for microelectrode recording, and two eye coils for measurement of eye position were implanted as previously described (Komatsu and Wurtz 1988). The chamber for recording was tilted backward 38° from the frontal plane with its center directed toward the midline between the two colliculi and 1 mm posterior to the interaural line. After the surgery the monkey was monitored until it was fully awake, given pentothal (Nalwin) for analgesia, and returned to its home cage. The monkey was allowed 2 wk for recovery before physiological or behavioral testing. All experimental protocols were approved by the Institutional Animal Care and Use Committee and compiled with Public Health Service Policy on the humane care and use of laboratory animals.

The monkeys were trained in several behavioral tasks, including fixation and making saccades to visual targets. In brief, the monkeys were first trained to press a bar, fixate a pair of aligned 0.1° targets and, after a random interval (0.6-0.9 s), make a saccade within 0.4 s after a second light (0.25°) appeared and the fixation light went off (Wurtz 1969). Release of the bar when the target dimmed led to a liquid reward. If the monkey released the bar earlier or later, or broke fixation, the trial was aborted, and it received neither punishment nor reward. Eye movements were recorded with the use of a magnetic search coil technique, with a resolution of 0.1° (Fuchs and Robinson 1966; Judge et al. 1980). The monkey's eye position was continually monitored, and, if this position exceeded ±2.5° window around the visual target, the trial was aborted.

Single neurons were recorded with the use of tungsten microelectrodes (Frederick Haer) with impedances of 2-5 MΩ. Electrodes were introduced into the colliculus through stainless steel guide tubes by the use of a micromanipulator (Narishige, MO-95B) attached to the implanted chamber over the colliculus. The guide tubes were held in position by a grid (Crist et al. 1988) that contained holes positioned at 1-mm intervals. We usually made 5-10 penetrations with microelectrodes through each of the 7-9 guide tube positions available over each superior colliculus.

Experimental procedures

During the experiments the monkeys faced a tangent screen 86 cm in front of them while seated in a primate chair with their heads fixed by coiled wires to a support arm of the cranial fixation system (CNC-ENG Engineering) were lowered over the monkey and allowed an unobstructed 60° × 60° field of view. Visual stimuli were back-projected red light-emitting diode (LED) spots (0.35 cm²); the light from these spots passed through the implanted chamber and activation and when spontaneous saccades were collected. Between trials the room was dimly illuminated (1.0 cd/m²) to prevent the monkey from the ambient light. Behavioral performance 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 targets were positioned by the computer through D to A converters controlling an X-Y mirror galvansometer system (General Scanning). Horizontal and vertical eye positions were sampled every 2 ms. Single-cell activity was sampled every millisecond by means of a window discriminator that produced a ±5-V pulse for each spike that met height and time constraints. Events associated with the behavioral tasks, such as fixation point and target onset and offset, were also stored at 1-ms intervals.

As the microelectrode entered the superior colliculus, we estimated the receptive-field location of multicell visual activity and placed the target for visually guided saccades in the center of that field. While the monkey made saccades to this target, the electrode was lowered farther, and we inferred that the electrode had entered the intermediate layers when single neurons with saccade-related activity were isolated. We then determined the dimensions of the area in the visual field for which the cell increased its activity before a saccade, the cell's movement field (Wurtz and Goldberg 1972). The coordinates for the center (highest peak activity) and edges (up, down, right, and left) of the field were entered into the computer, and a five-point grid of target locations was calculated. The grid included the center and four points located halfway from the center to each edge of the movement field. Different target locations were interleaved and were presented in a pseudo-random order at each of these five locations. This ensured that variations
in cell responsiveness over time would be distributed evenly across all types of behavioral trials and target locations. The visual re-
sponse of the neuron at each location was determined by having the monkey maintain gaze steadily on the fixation light while tar-
targets were presented. A monkey typically would perform 2,000–
3,000 trials during a 4- to 6-h experimental period, 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.
In the first monkey (7271) we made 82 penetrations at 26 different
grid locations in the colliculus on both sides of the brain. In the second monkey (167C) we made 55 penetrations at 11 different
grid locations. Nine of these penetrations were on the right side.
During penetrations on the right side, we suspected (and later
confirmed on histological examination) that the monkey had expe-
rienced a hemorrhage, and recording was stopped at that time.
The monkey showed no discomfort but displayed some lethargy,
which continued over a period of 3-4 days. About 1 mo later, we
established that the monkey had normally accurate (by amplitude
criteria) eye movements to locations in both the right and left
visual fields. At that time we resumed recording from the opposite
(left) colliculus.
After all the experiments were completed, the monkeys were deeply anesthetized with pentobarbital sodium and perfused through the heart, and the brain prepared for histological examina-
tion by the use of procedures described recently (Ma et al. 1991).
Sections in the frontal plane were subsequently examined at low
and high magnification to verify that the penetrations were
through the superior colliculus.

Data analysis
SACCADE PARAMETERS. The beginnings and ends of saccades
were obtained offline by a computer program that used a two-
pass process. The first pass identified possible beginnings or end-
ings of a saccade with the use of velocity and acceleration thresh-
old criteria. In the second pass a template correlation method
was used to accurately position a fiducial mark. Templates for specific
types of saccades (visually guided or spontaneous) consisted of
~20 points in time and were made by averaging the beginning,
end, and overshoot of 10 nearly ideal saccades that were hand-
picked by the experimenter. The experimenter corrected any sac-
cade recognition failures made by the marking program.
The marking program calculated the radial magnitude and ra-
dial angle for each of the saccades in the use of horizontal and
vertical position and the Pythagorean theorem. The difference be-
tween the final and instantaneous radial magnitudes was defined
as the radial error of the saccade. Radial velocity was determined
by applying a differential finite impulse response (FIR) filter to the
radial magnitude trace (Oppenheim and Schafer 1975).

SINGLE-CELL ANALYSIS. Single-cell activity was displayed in ras-
ters aligned on specific events such as the end of a saccade. To
obtain a continuously varying measure of neuronal activity from a
discrete train of neuronal pulses, we used a spike-density function
that estimates the probability of spike occurrence over time (Sil-
verman 1986; Richmond and Optican 1987). The spike-density
trace for a single saccade was calculated by substituting a Gaussion
pulse of fixed width for each spike and then summing these (SD in
Fig. 1A). Thus large values of the spike density represent a greater
probability of the occurrence of a spike, and the peak represents
the peak discharge of the cell. The generation of a continuous function
was necessary because we wanted to compare a series of
discrete events, the cell’s spike train, with the continuously vary-
ing velocity and amplitude of saccades. The spike-density function
was used instead of the reciprocal interval function, because the
spike-density function is a linear measure of spike frequency,
whereas the reciprocal interval measure is nonlinear.
A mean spike-density function was computed by averaging the
spike densities over a series of trials, usually 8–10 as illustrated in
Fig. 1B. Using a fixed-width Gaussian pulse (kernel) to compute
spike densities blurs abrupt changes in spike frequency, such as
those occurring in the burst associated with saccades. Therefore
we used an adaptive method that varied the width of each Gaussi-
ian pulse in averaging the raster diagrams, as shown in Fig. 1B (η =
addy) (Richmond et al. 1990; Silverman 1986). The adaptive kernel
method changes the value of η for the Gaussian pulse according to
the firing rate of the cell. When the cell discharge rate is high, or is
very consistent across trials, a narrow kernel is used to replace each
spike. When the discharge rate is low, or is very inconsistent across
trials, a wider kernel is substituted. As shown in Fig. 1B, this gener-
ated a spike-density function comparable with that with a fixed η
of 4 ms but with slightly steeper rising and falling phases and a
slightly higher peak value. The adaptive kernel method empha-
sizes that changes seen in temporal aspects of the cell-firing pat-
terns are dependent on the cell firing and not the underlying ker-
nel used to represent that firing (Richmond et al. 1990).

SHIFITING TIME FOR PHASE PLATES. The comparison of the tem-
poral waveforms of neuronal discharges and eye movements (2
time-varying functions) was facilitated by plotting one variable
against another in a phase plane. To do this, we compensated for
the time delay between the appearance of neuronal activity and the
movement of the eyes by a relative time shift between the two
traces. The portion of the collicular cell discharge after the peak
discharge has been reached is most important for comparison with
saccadic velocity. Because the start of the burst varied from trial to trial, we aligned
the peak spike density of the cell with the peak velocity of the
saccade. We arbitrarily set a criterion threshold of 10% of the peak
discharge of the cell for the beginning of the burst. The end of
the burst was marked when discharge declined below 10% of peak
discharge or with the end of the saccade. This method truncated
activity that continued beyond the end of the eye movement and
as well as any prolonged build up of activity that occurred >50 ms
before the beginning of the saccade. What distinguishes this
method most from prior analytic methods, however, is that it
counted only those spikes before and during the saccade that could
possibly influence the burst discharges in the pons and thus
modulate the generation of that saccade.

RESULTS
We studied 86 superior colliculus neurons, the discharge
of which increased before and during visually guided sac-
cades. Of these cells, 16% (n = 14) had a burst of activity
during spontaneous saccades in the dark, and 69% (n = 59)
had a visual response to a stimulus placed in the movement
field of the cell.

Relation of discharge to end of saccades
Figure 1 shows the discharge of a cell that ceases by the
end of the saccade. Figure 1A compares the horizontal, ver-
tical, and radial magnitude of a single saccade (Fig. 1A, top)
with the train of spikes and their spike-density representa-
tion (Fig. 1A, bottom). Figure 1B (top) shows the spike
trains associated with eight similar saccades for the same cell. Each row of the raster is aligned on the end of the saccade (vertical line) as are the average spike-density traces shown below. Note that the cell discharge ceased by the end of the saccade (vertical line) in every trial.

We found that different cells varied in the extent to which neuronal activity extended beyond the end of the saccade, and Fig. 2 shows cells typical of the three major varieties of discharge we observed. The burst of spikes associated with the saccade for the cell shown in Fig. 2A was cut off by the end of the saccade (similar to the cell shown in Fig. 1), and we refer to these as clipped cells. We classified a cell as clipped if, for 80% of the trials at a given saccadic amplitude, no spikes were observed after the end of the saccade. Clipped cells made up 36% (n = 31) of our total sample. We refer to the activity of cells as partially clipped (Fig. 2B) if the major burst of activity declined rapidly before the end of the saccade, but a low level of activity continued beyond the end of the eye movement. These cells comprised ~53% (n = 46) of our sample. Unclipped cells (Fig. 2C) had almost no burst of activity during the saccade, but instead their discharge gradually increased before the saccade, continued at about the same level throughout, and then gradually decreased after the saccade (for up to 100 ms). These cells represented only ~10% (n = 9) of our sample.

For ease of comparison with the previous literature (Sparks 1978; Wurtz and Goldberg 1972), we aligned the discharge of the same cells on the beginning of the saccade in Fig. 2, D–F. With the use of the previous criteria to categorize collicular cells, the cells in Fig. 2, A and B, were visual-movement cells because they responded briskly on both visual and saccade trials. Fifty-two percent (16 of 31)
of clipped cells and 78% (36 of 46) of partially clipped cells were visual-movement cells. The remaining cells were saccade-related burst neurons that had little if any discharge in response to a visual target (as in the cell shown in Fig. 5A) (Sparks 1978; Sparks and Mays 1980). The decay in the saccade-related bursts in visual-movement and saccade-related burst cells could be either clipped or partially clipped.

**CONTINUUM OF CELL TYPES.** Figure 3 shows the method we used to determine whether collicular cells that we placed into the three categories also could be divided into distinct groups if we used a continuous quantitative measure. For each cell, we expressed the drop in activity before and during the saccade as a fraction, \( \gamma \), of the peak discharge. The value of \( \gamma \) would be one if the cell discharge had dropped to zero by the end of the saccade, and zero if there was no change between the peak and the end of the saccade.

Figure 4A shows the percent of cells in each class (clipped, partially clipped, unclipped) plotted against \( \gamma \). The overlap between classes in the histogram demonstrates that no separation into distinct classes is possible. We therefore regard the cell classes as examples from a continuum of response types, with varying amounts of saccade-related burst and decay, and our subsequent use of the terms clipped, partially clipped, and unclipped refers to our categorization of a particular cell as described above. We have concentrated on cells that had either clipped or partially clipped activity in our subsequent analysis.

We also examined whether distinct groups of cells, based on either their peak firing rate or optimal saccadic amplitute, could be distinguished. No clear separation of the types existed for peak activity (Fig. 4B) or optimal saccadic amplitude (Fig. 4C). However, unclipped cells tended to have lower peak discharge rates and smaller saccadic amplitudes than clipped or partially clipped cells.
FIG. 3. Method for calculating $\gamma$, which quantifies the drop in activity from peak discharge to the end of the saccade. Traces are for radial magnitude (RM), spikes (Spk), and spike density (SD) for a partially clipped cell, and the vertical dashed line indicates the end of the saccade. The absolute value of the difference between the spike-density function at its peak (SD$_p$) and at the end of the saccade (SD$_t$) is divided by the absolute value of the spike density at the peak to produce $\gamma$.

POSTMOVEMENT ACTIVITY ACROSS THE MOVEMENT FIELD

Our categorization of cells has been based on their discharge with saccades made to the center of their movement fields, i.e., those saccades that were associated with the strongest discharge of the cell. Figure 5, A–C, shows how activity changes with saccades to different parts of the movement fields for clipped, partially clipped, and unclipped cells, respectively. In these cells the peak response to visually guided saccades varied in some cases across the movement field, but the amount of postsaccadic activity remained the same across the movement field (i.e., the cells did not change category). This was true of 62 of 80 cells (clipped, partially clipped, unclipped) studied in this way. Only 2 of 28 clipped cells acquired postmovement activity for saccades to the edges of the movement field, whereas 15 of 45 partially clipped cells showed a change. Figure 5D shows a partially clipped cell, the activity of which became unclipped toward the edges of the movement field.

Relationship of cell discharge to saccadic amplitude and velocity

Our new categorization of saccade-related neurons opened the possibility that the discharge of one category of cell might be more closely related to either saccadic amplitude or velocity. We therefore compared the discharge characteristics of clipped and partially clipped cells with the amplitude and peak velocity of saccades as had been done in previous experiments (Berthoz et al. 1986; Sparks 1978; Rohrer et al. 1987).

To compare the total number of spikes with the amplitude of the saccade, we considered only saccades made along the optimal direction for a particular cell. We collected sufficient data for this analysis from 14 collicular neurons in one monkey (8 partially clipped and 6 clipped cells). The total spike count shown in Fig. 6A (a partially clipped cell) gradually increased up to the amplitude at which maximum discharge occurred (shown by the filled diamond at 1.4° on the abscissa) and then declined for saccades greater than -20°. This discharge was representative of cells with small circumscribed movement fields. In contrast, the cell in Fig. 6B (a clipped cell) had a large move-
FIG. 5. Variation in the post-saccadic activity of collicular cells across the movement field. Graphs in each panel (A-D) show the fixation point (x), the location of the center of the movement field (center x of the group of 5 x's), and show the target points located halfway between the center and the edges of the movement field (the remaining 4 x's). The group of 5 spike-density traces in each panel shows the response of a collicular cell to 10 visually guided saccades made to each target position. A-C: for these cells the end of the discharge showed little variation for saccades to different areas of the movement field. D: a cell, the activity of which appeared partially clipped in association with optimal (to the center of the field) or smaller amplitude saccades, but unclipped for larger saccades.

The spike count increased from the medial edge of the movement field to ~24° and remained about the same for larger saccades. Both cells show a range of saccadic amplitudes that are roughly proportional to the total number of spikes. This relation between spike count and saccadic amplitude up to the center of the movement field was consistent across all 14 of the analyzed cells, regardless of whether they had small or large movement fields or whether their activity was clipped or partially clipped. There was, however, a large variation in the spike count even for saccades of the same amplitude and direction. For example, at the optimal target amplitude of the cell shown in Fig. 6A, the range of cell discharge was 8.9–29.1 spikes (19.3 ± 5.3, mean ± SD; n = 50) with a standard deviation of ~27% of the mean value. The radial magnitude, however, ranged from amplitudes of 11.8–14.8° (13.5 ± 0.6°, n = 50) with a standard deviation of only ~5% of the mean value. Similarly, for the cell shown in Fig. 6B, a standard deviation of 4.5 spikes at the optimal saccadic amplitude was ~28% of the mean value, whereas saccades of that amplitude had a standard deviation of 1.2°, ~5% of the mean value. The variability in the neuronal activity of each of the 14 cells studied was more than five times the variability of the amplitude of the saccades.

We next compared the peak discharge frequency with the peak velocity of the saccade for the same cells. Figure 6C, for the partially clipped cell with the small movement field shown in Fig. 6A, shows a general trend for peak discharge frequency to increase with peak eye velocity up to a maximum of 700°/s and then to decline with faster movements. Figure 6D, for the clipped cell with the large movement field of Fig. 6A, shows a steady increase in peak discharge frequency with increasing peak velocity that saturates at a peak frequency of ~400 spikes/s. In both Fig. 6C and 6D the variability in saccadic velocity for the optimal target position is much less than the variability in the peak frequency. For example, in Fig. 6C, there is a two-fold change in peak firing rate that would encode only a 16% increase in velocity (620–720°/s) for saccades of optimal size (indicated by letter g). Figure 6D shows that, for the optimal saccadic amplitude (indicated by letter m), there is a 64% variation in peak frequency, whereas there is only a 27% change in peak velocity (range, 750–950°/s). This relationship was similar across the rest of the 14 cells studied.
FIG. 6. Relationship of spike count to saccadic amplitude (A and B) and peak discharge frequency to peak saccadic velocity (C and D). A: a partially clipped, small movement field cell. The number of spikes in an interval extending from ~30 ms before the saccade to the end of the saccade, plotted against radial amplitude (n = 226 saccades). The direction of the saccades used was restricted to within ±10° of the optimal direction, along a line connecting the fixation point with the center of the movement field. The filled diamond on the abscissa indicates the amplitude of the optimal saccade. The solid curve was the best-fit quadratic with equation \( A_n = A_0 + B_x + C \times x \), where \( A = -900.5, B = 3.0, \) and \( C = -8.3 \). (The root mean square error was 5.08.) B: a clipped, large movement field cell. The number of spikes plotted against amplitude (n = 133 saccades). The interval and direction restrictions are the same as in A. The data were fit best with a Hill function of the form \( y = k \times (A/x) \), where \( z = 1 + (A/x)^n \). The gain, A, was 1.80; the location of the break in the curve, \( \lambda \), was 15.8; the steepness of the rise, \( n \), was 5.4, and the offset, \( K \), was 1.0. (The root mean square error was 5.3.) Note that we might not have detected a decline in discharge with very large saccades because of the limited amplitude saccades we could elicit from this monkey. C: peak frequency plotted against the peak radial velocity for the same set of eye movements shown in A (n = 226). The best fit to the data was a quadratic with equation \( A_n = A_0 + B_x + C \times x \), where \( A = -0.0035, B = 4.72, \) and \( C = -1.17 \). (The root mean square error was 103.8.) The data points that are represented by the same small letter indicate repetitions of different saccades to the same target location. The target amplitude (T), average radial amplitude (RAM), average radial peak velocity (RPV), peak discharge frequency (P), and number of saccades (n) for each letter were as follows: a: T = 10.2, \( \text{RAM} = 9.7, \text{RPV} = 545.5, P = 368.6, n = 18; b: T = 8.2, \text{RAM} = 7.7, \text{RPV} = 465.1, P = 260.6, n = 19; c: T = 12.3, \text{RAM} = 11.5, \text{RPV} = 622, P = 381.5, n = 19; d: T = 16.1, \text{RAM} = 15.4, \text{RPV} = 714.9, P = 355.4, n = 20; e: T = 18.1, \text{RAM} = 16.3, \text{RPV} = 714.7, P = 307.6, n = 18; f: T = 20.1, \text{RAM} = 19.2, \text{RPV} = 817.6, P = 340.2, n = 20; g: T = 25.1, \text{RAM} = 23.0, \text{RPV} = 839.2, P = 262.1, n = 20; h: T = 30.0, \text{RAM} = 27.4, \text{RPV} = 832.9, P = 189.2, n = 20; i: T = 41.1, \text{RAM} = 13.6, \text{RPV} = 692.3, P = 456.7, n = 20; j: T = 14.1, \text{RAM} = 13.2, \text{RPV} = 670.9, P = 402.4, n = 20; k: T = 22.1, \text{RAM} = 20.4, \text{RPV} = 844.95, P = 416.2, n = 10; l: T = 14.1, \text{RAM} = 13.6, \text{RPV} = 690.5, P = 511.6, n = 10; m: T = 11.2, \text{RAM} = 11.0, \text{RPV} = 601.9, P = 506.8, n = 9; and n: T = 14.3, \text{RAM} = 13.9, \text{RPV} = 712.9, P = 370, n = 1. D: the peak spike frequency plotted against the peak radial velocity for the same saccades as in B (n = 133). Data were fit best by a Hill function of the form \( y = A_0 + K \), where \( z = 1 + (A/x)^n \). The gain, A, was 300.4, the location of the break in the curve, \( \lambda \), was 68°, the steepness of the hill, \( n \), was 12.3, and the offset was 19.6. The root mean square error was 89.9%. Values for each letter in this graph were: a: T = 29.2, \( \text{RAM} = 26.8, \text{RPV} = 839.4, P = 318.4, n = 15; b: T = 5.8, \text{RAM} = 5.4, \text{RPV} = 356.5, P = 6.72, n = 18; c: T = 11.7, \text{RAM} = 11.3, \text{RPV} = 645.4, P = 157.0, n = 17; d: T = 17.5, \text{RAM} = 15.0, \text{RPV} = 779.2, P = 343.0, n = 18; e: T = 23.3, \text{RAM} = 20.6, \text{RPV} = 856.7, P = 331.2, n = 16; f: T = 29.2, \text{RAM} = 29.4, \text{RPV} = 800.0, P = 293.1, n = 16; g: T = 35.0, \text{RAM} = 30.6, \text{RPV} = 848.3, P = 303.2, n = 12; h: T = 37.7, \text{RAM} = 32.3, \text{RPV} = 867.7, P = 441.1, n = 10; i: T = 30.9, \text{RAM} = 29.1, \text{RPV} = 900.4, P = 442.6, n = 10; and j: T = 15.0, \text{RAM} = 13.6, \text{RPV} = 528.7, P = 196.4, n = 1.
In net, for these clipped and partially clipped cells, the relationships between spike count and saccadic amplitude and between peak firing rate and peak velocity are similar. For saccades of less than optimal amplitude, there is a rise in the spike count and peak firing rate with increasing amplitude and velocity, respectively. This is not at all surprising given the previous analysis of collicular burst cells (Sparks and Mays 1980). There is much more scatter in the spike count than there is in the amplitude of the saccade and more scatter in the peak frequency than in the eye velocity. Such variability in the discharge of individual cells is also not surprising because it is generally agreed that it is the population of cells that is the significant output of the colliculus. But these results show clearly how difficult it is to determine whether collicular discharge is more closely related to the amplitude or velocity of the saccade when the data are based on single-point or averaged measurements, such as peak firing frequency or spike count.

**Dynamic relationship of cell discharge to radial error and radial velocity**

To see whether dynamic measures of saccadic amplitude or velocity would more clearly differentiate the role of collicular activity, we examined the relationship of the tem-

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**FIG. 7.** Comparison of cell discharge with radial error and radial velocity. Plots are derived from the mean responses of 10 visually guided eye movements of a partially clipped cell. The portion of each record represented by the solid line indicates time during execution of the saccade. A: the radial magnitude (the difference between current and initial eye positions) of this 3.5° saccade is plotted against time. B: the velocity of the saccade is symmetrical and is derived from the amplitude data with the use of a FIR filter differentiator that added a time shift of no more than 1 ms. C: the spike density of the cell on the same time axis as in A and B (i.e., there is no time shift). D: the covariance of the 2 dependent variables, spike density and radial error, shown as a trajectory. The start of the eye movement is indicated by a, and the end of the saccade by b. Time, the independent variable, is the distance along the trace and runs in the direction from a to b. The spike density of this cell required no shift to produce the maximum overlap of peak spike density and radial error. Note that the spike density is well correlated with radial error during the movement. E: the covariance between radial velocity and spike density shown as a trajectory. Time begins at top left of the curve and moves clockwise around it as indicated by the arrow. No shift is introduced between spike density and radial velocity. Note that there are 2 values of spike density for each value of eye velocity with an exception at peak eye velocity. F: the mildly curved trajectory of the saccade as a function of horizontal and vertical components. Time proceeds from the top left to the bottom right.
poral pattern of collicular discharge with the instantaneous changes in radial error and velocity. Figure 7 compares the spike density of a partially clipped cell with the dynamics of a saccade. The mean radial magnitude, radial velocity, and spike density of 10 similar saccades are plotted as functions of time in Fig. 7A, B, and C, respectively. The portion of the waveform in each panel that occurs while the eye is moving is shown by the solid line (defined from the position trace of A). Note the symmetrical appearance around the peak of saccadic velocity (Fig. 7B), as opposed to the asymmetrical shape of the burst (Fig. 7C).

Figure 7D shows the relation of spike density to the radial error, the difference between final and current radial positions. The initial, almost vertical rise of the trajectory is due to the increase in cell discharge before the start of the saccade. After the eye movement begins (a in Fig. 7D), there is a steady decline in the spike density function until the eye movement ends (b in Fig. 7D). As this is a cell with partially clipped activity, there is some postmovement discharge that declines to zero as the eye remains stationary (the 2nd vertical line closest to b). The portion of this trajectory between a and b (—) illustrates our major observation: there is a simple, monotonic relationship between the decline of cell discharge and declining radial error during a saccade.

Contrast this simple relationship of radial error with spike density to the curved trajectory seen when spike density is plotted as a function of radial velocity (Fig. 7E). The beginning of the saccade is associated with nearly the highest spike density. Time advances in a clockwise direction along the trajectory as indicated by the arrow. During the accelerating phase of the saccade, there is one value of spike density for each value of velocity, and during the decelerating phase there is a different value of spike density for each value of velocity. Thus the eye accelerates and decelerates through a sixfold change in velocity, whereas the spike den-

**FIG. 8.** Comparison of different time shifts of spike density with respect to radial error and radial velocity for a clipped cell. A: the average radial error and magnitude for 10 visually guided saccades of optimal amplitude (top), the average velocity (middle), and the average spike density of this neuron (bottom) shifted from 0 to 20 ms. B: trajectories for average spike density and radial error (left column) or radial velocity (right column). Successive graphs show the effect of increasing shift from no shift (top) to 20 ms (bottom). A 7 ms shift (3rd from top) produced the greatest linearity between radial error and the spike-density function, whereas the relationship between radial velocity and spike density remains curvilinear and double valued at all velocities. Time for radial velocity trajectories advances in a clockwise direction for all curves except after a shift of 20 ms.
sity remains almost constant during each phase. Most of the change in discharge rate occurs near peak velocity.

DELAY BETWEEN COLICULAR DISCHARGE AND SACCade. In plotting the trajectory in Fig. 7D no shift was required to produce the nearly linear decline in the spike density with declining radial error. However, the peak activity of other collicular cells could lead the start of the saccade by up to 12 ms. For these cells a shift was required to generate a nearly linear trajectory relating spike density and radial error. We next investigated whether this was a robust phenomenon. That is, did shifts in the spike density relative to the start of the movement result in a straight trajectory relating spike density to radial error and radial velocity? Figure 8A shows averages of the radial magnitude, radial error, radial velocity, and the spike-density response for a clipped cell. The solid line on the spike-density plot in Fig. 8A (bottom) indicates the unshifted (0 ms) location of the spike-density function, and the broken lines show the relative locations of the spike density as it is shifted for 5–20 ms. Figure 8B shows the effect of different shifts of spike density with respect to radial error (left column) and radial velocity (right column). A shift of 7 ms produced the straightest trajectory for radial error and spike density (between the beginning and end of the saccade). With the exception of shifts >15 ms, the radial error versus spike-density trajectory remained single valued (i.e., there was only one value of spike density for each value of radial error). In contrast, there were two different values of spike density for the same velocity, and the difference between the two values was reduced with shifts of 13–20 ms. For example, at a velocity of 400°/s, the spike density values were 0.25 and 0.35 for the decelerating and accelerating portions of the velocity trajectory, a difference of ~22% of the peak response of the cell.

FIG. 9. Distribution of intervals between peak spike-density and radial error (A) and radial velocity (B). Values are for all clipped and partially clipped cells in each monkey, 167C (left) and 727J (right). The optimal saccade vector was used for each cell. A: histograms of the interval between 90% of the peak discharge of the cell and the start of the saccade, when radial error was maximal. Zero on the abscissa indicates the start of the saccade, and positive intervals indicate that cell discharge preceded the start of the saccade. B: histograms of the interval between peak discharge of the cell and the peak velocity of the saccade. Note that mean intervals were much longer for velocity than for radial error.
This family of trajectories shows that the simplest radial error trajectory results when the peak of the spike density is shifted to the start of the saccade. Similarly, the radial velocity trajectory has the smallest loop when the peak of the spike density is shifted to the peak of the eye velocity. Thus we chose to plot trajectories by shifting the peak spike density to the start of the movement or to the peak of velocity, for each cell individually, rather than by a fixed amount. Figure 9 shows the magnitude of these shifts for radial error (Fig. 9A) and peak velocity (Fig. 9B) for each monkey. Figure 9A shows that the mean intervals between 90% of the peak of the spike density and the start of the saccade were 6.3 and 4.8 ms for all the clipped and partially clipped neurons from each monkey; shifts very close to 7 ms produced the straightest radial error trajectories. Figure 9B shows that the mean intervals between peak spike density and peak radial velocity were 15.4 and 15.6 ms, which are similar to the shift of 15 ms that produced the tightest loop in the velocity versus spike-density trajectories (Fig. 8).

**Radial Error Trajectory Across the Movement Field.** Figure 10 shows that the single-value nature of the radial error versus spike-density trajectory remains even when the saccade is not to the center of the movement field of a clipped cell. The monkey made saccades with a range of amplitudes, and the top row of Fig. 10 shows radial error for those eye movements, whereas the middle row shows the associated spike densities. The graphs of spike density versus radial error (Fig. 10, bottom row) demonstrate the characteristic feature of the radial error trajectories seen for clipped and partially clipped cells. For saccades up to the optimal saccadic amplitude within the movement field (Fig. 10, B–D), there was a fairly straight trajectory relating

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**FIG. 10.** Radial error vs. spike-density trajectories for saccades to targets across the movement field of a clipped cell. The top row shows the average radial magnitude of −20 to 20 ms (almost 40 in D) saccades along the line connecting the fixation point with the center of the movement field of this cell with amplitudes from 5° to 30°. Interrupted lines indicate the individual responses; solid lines indicate the averages of individual trials. The middle row shows the average spike density for the saccades shown just above. These spike-density traces extend from 90% of peak activity to the end of the saccade and have been shifted to align 90% of peak activity with the beginning of the saccade. (Even though this was a clipped cell, the shift causes the end of the spike density to occur after the end of the saccade.) The bottom row shows the trajectory of the decline of spike density (ordinate) with the decline of radial error (abscissa). Time (the independent variable) in this row runs along the trajectories from top right to bottom left of each trajectory. Note the similarity in range of decline for trajectories in B–D, whereas there is a plateau in the response for E. E: combined data from 3 sets of trials (each ~20 trials) collected near the beginning of the recording session for this cell. The target location in A was outside of the movement field of the cell. Mean saccade amplitudes, average shifts, and number of saccades were as follows, respectively: A: 5.4°, no discharge, n = 18; B: 11.2°, −0.63 ms, n = 19; C: 15.9°, 3.95 ms, n = 19; D: 20.4°, −1.1 ms, n = 18; E: 26.6°, 2.89 ms, n = 36.
the decline in radial error to the decline in spike density during the execution of the saccade. However, for larger saccades, the decline of spike density with declining radial error varied considerably (Fig. 10E). The simple, single-valued character of the relationship between spike density and radial error is maintained across the movement field, at least up to its center.

**VARIATIONS OF TRAJECTORIES ACROSS CELLS.** So far we have only considered the relationship of cell discharge to saccade dynamics for several illustrative cases. We found that the same observations held over the sample of cells we studied. Figure 11 shows the variations in the radial error and velocity trajectories among 16 cells with clipped activity in one monkey. On the basis of the assumption that a group of cells within the colliculus is activated for any visually guided saccade of optimal size and direction, we have grouped the trajectories of these 16 cells according to radial amplitudes. In Fig. 11A the radial error is plotted against spike density with time (the independent variable) running along the curve from right to left as radial error declines (△).

Although there were variations from a straight trajectory, all of the trajectories were single valued. The relationship of the average radial velocity to spike density (Fig. 11B) shows one or two velocity trajectories where the loops are quite small (e.g., 1st column, c7888.1), but the majority of cells have an area contained within the loop. This indicated a difference in spike density during the accelerating and decelerating portions of eye velocity.

Even after grouping cells within specific amplitude ranges, it is difficult to compare the trajectories of one cell with another because they vary widely in peak discharge rate, radial magnitude, and peak velocity. We therefore normalized the cell responses within a group to be a fraction of their peak discharge, normalized the radial errors to be a fraction of the saccade's amplitude, and normalized the radial velocities to be a fraction of the saccade's peak velocity. Figure 12A shows the normalization of spike density and saccadic amplitude for all the clipped cells in one monkey,

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![Diagram](image)

**FIG. 11.** Comparison of trajectories for spike density and radial error with those of spike density and radial velocity for all of the clipped cells from one monkey (167C). The 16 cells are grouped by the saccade amplitudes: 5-9.9° (n = 7); 10-14.9° (n = 5); 15-24° (n = 4). A: radial error and spike density. On the trajectories time progresses along the curve (from top right to bottom left). Each trajectory represents the average response of 1 of the 16 cells to at least 10 visually guided saccades to the center of its movement field. Spike density has been shifted in each case so that 90% of peak spike density coincides in time with movement onset. B: radial velocity and spike density. In these trajectories time runs to the right along the top curve and back to the left on the bottom curve. Peak spike density has been shifted in each trace to match peak eye velocity.
FIG. 12. Normalized trajectories of spike density and radial error. A: the maximum radial error and spike density for each of the 7 cells shown has been set to 1 and the minimum to 0. The abscissa shows radial error as a fraction of total amplitude. The ordinate shows the spike density as a fraction of the maximum discharge of the cell. Once the data from successions of different sizes were normalized to have the same duration, the time intervals between one data point and the next were no longer equal across movements. We fitted a cubic spline to the normalized data and resampled at 0.01-ms intervals to produce a set of equally spaced data points that could be averaged. Trajectories for the 7 clipped cells with optimal saccadic amplitude within the 5°-10° range shown in the left panel of Figure 11A. Note the increased variability in the final 10% of the radial error (0.0-0.16). B: the mean of the trajectories shown in A. C: the mean and standard error of the mean (SE, ···) of radial error over time for the 7 cells shown in A, D: the mean and SE for spike density over time for the 7 cells shown in A.

The optimal saccadic vectors of which were between 5 and 10° (i.e., normalized data of Fig. 11A, 1st column), and Fig. 12B shows the mean of these trajectories. (Fig. 12, C and D), show the normalized and resampled average radial error and average spike-density curves from which Fig. 12B was derived.

Figure 13 shows the results of this normalizing, resampling, and averaging for spike density and radial error across all clipped (Fig. 13, A and B) and partially clipped cells (Fig. 13, C and D) in both monkeys. All these normalized trajectories were single valued, and almost all had monotonically declining spike densities (Fig. 13, A–D). When we pooled together and averaged all of the trajectories from each monkey (Fig. 13, E and F), the functions were monotonically decreasing and nearly linear. It is interesting to note that most of the variability in the curves occurs within the last 10% of the plots (i.e., from 0.1 to 0.0 of radial error and spike density, especially in Fig. 13, B and C). This variability is even clearer in the individual traces of Fig. 12A and is due to wobble in the tail of the spike-density function. We think that, once the spike discharge has declined to 10% of its peak value, the rest of the decline is probably close to neuronal noise. This would make the tail of activity seen in the partially clipped cells less important for subsequent processing because that discharge is usually below 10% of the peak discharge of the cell.
FIG. 13. Linear relationship of spike density and radial error over all clipped and partially clipped cells studied in both monkeys. Results in A-D are grouped into separate amplitude ranges (see key). As in Fig. 12, the abscissa shows radial error as a fraction of total amplitude, and the ordinate shows spike density as a fraction of the maximum. A and B: trajectories grouped by amplitude for the clipped cells of the 2 monkeys, 167C and 7272. Note that there were no cells with amplitudes in the 0-4.9° range for monkey 167C. C and D: all the partially clipped cells grouped by amplitude. E and F: average over all amplitude ranges and both cell types for each of the 2 monkeys.

Figures 14 and 15 show the same normalization process for peak spike density and peak saccadic velocity as did Figs. 12 and 13 for spike density and radial error. In Fig. 14B, note that even the average trajectory of this group remains an open loop and does not become single valued. However, as shown by Fig. 14, E and F, we found that we were actually averaging two different varieties of responses.

In one type (shown in Fig. 14F), the spike discharge increased faster than velocity and produced a clockwise trajectory, but in the other type (shown in Fig. 14F) the radial velocity increased faster producing a counterclockwise trajectory. When these clockwise and counterclockwise trajectories were averaged together, the phase differences of the individ-
Fig. 14. Normalized trajectories of spike density and radial velocity for clipped and partially clipped cells in monkey 167C. A: the peak radial velocity and peak spike density for each of the 7 cells shown has been set to 1 and the minimum to 0. The abscissa shows radial velocity as a fraction of peak velocity. The ordinate shows the spike density as a fraction of the peak discharge. The cell trajectories are the normalized version of those shown for the 7 clipped cells in the 5–10° range shown in the left panel of Fig. 11B. Note the open loop trajectories for all but 2 of the cells. B: the mean of the trajectories shown in A. C: the mean and standard error of the mean (SE, ---) of radial velocity over time for the 7 cells shown in A. D: the mean and SE of spike density over time for the 7 cells shown in A. E: for cells in which the spike density increased faster than the radial velocity, a clockwise trajectory was produced. The mean of all (n = 15) the clockwise trajectories of the partially clipped cells in monkey 167C are shown. F: for cells in which the radial velocity increased faster than the spike density, a counterclockwise trajectory was generated. The mean of all (n = 13) the counterclockwise trajectories of the partially clipped cells in monkey 167C are shown.
FIG. 15. Disappearance of multi-valued trajectories for spike density and saccadic velocity by averaging all the clipped and partially clipped cells studied in both monkeys. Results in A–D are grouped into separate amplitude ranges (see key). As in Fig. 14, the abscissa shows radial velocity as a fraction of peak radial velocity, and the ordinate shows the spike density as a fraction of peak discharge. A and B: trajectories grouped by amplitude for the clipped cells of the 2 monkeys, 167C and 727J. Note that there were no cells with amplitudes in the 0–4.9° range for monkey 167C. The amplitude range of 0–4.9° group for monkey 727J had the only closed trajectory. C and D: all the partially clipped cells grouped by amplitude. Again the amplitude range of 0–4.9° group for monkey 727J had the only nearly closed velocity trajectory. The cells in C are the same cells as in Fig. 14, E and F, but now grouped by amplitude instead of by the direction of their trajectory. E and F: averaging over all amplitude ranges and both cell types for each of the 2 monkeys produces closed trajectories for radial velocity, so that there would be a single value of spike density for a single velocity (see text for explanation).
such neurons were minimized, and the areas within the velocity trajectory loops were sharply reduced (Fig. 15, A–D). When all cells from each animal were pooled together and averaged (Fig. 15, E and F), the trajectory shows a marked decline in the area of the velocity loop. From this we infer that the complex, multivalued relationship between cell discharge and eye velocity may be reduced (even to a single-valued function) by averaging over a very large number of cells, differing particularly in whether spike discharge leads or follows velocity. However, the loop cannot be eliminated when the discharge of an individual neuron is related to radial velocity (see Fig. 11B).

**Discussion**

Our results are consistent with the well-established idea that the focus of activity within the colliculus is related to the amplitude of the impending saccade. In addition, we have found that the change in activity of cells within that locus is related to the remaining instantaneous motor error during a saccade. In many collicular cells, we found a steady decay in spike discharge with decreasing motor error. The end of the burst of many collicular neurons, which we referred to as clipped cells, was cut off in synchrony with the end of the saccade. This characteristic cutoff of activity was a robust phenomenon that varied little with saccades to different points in the movement field of the cell. In addition, if we include partially clipped cells, the discharge of 90% of our sample of intermediate and deep layer collicular cells decreased as the saccade ended.

We think that this declining phase of collicular cell discharge is important because of the temporal relation of these superior colliculus cells to the presaccadic neurons in the pons. If we take the average delay between the start of medium-lead burst neuron (MLBN) activity and the start of the eye movement to be 8 ms (Scudder 1988), and the delay between collicular stimulation and perturbation of saccades as ~8 ms (Munoz and Guittion 1987; D. P. Munoz, H. A. Wartemann, and H. R. Wurtz, unpublished observations), there can only be a very short delay between collicular and MLBN activity. In contrast, the delay between onset of the rise in collicular discharge and saccade onset was ~20 ms. The portion of the collicular activity that occurs during the discharge of the MLBNs is the only portion that could contribute to shaping the temporal discharge pattern of the pontine neurons controlling saccadic waveform. We have therefore concentrated on this period of collicular discharge.

In discussing the relation of our observations to the generation of saccades, we first present a collicular feedback model that incorporates the superior colliculus into a local feedback loop that controls saccadic amplitude; then we compare the predictions of the model with the characteristics of the collicular cells we studied; and finally we contrast this model to several others that consider the role of the superior colliculus in saccade generation.

**Collicular feedback model of the saccadic system**

Our observation that the discharge of collicular cells decreases as the amplitude of the saccade decreases leads us to propose a model that places the superior colliculus within a feedback loop that controls the amplitude of the saccade. Such a proposal has been made previously by Keller (1979). Our model builds on the previous feedback models of Robinson (1975) and Jürgens et al. (1981). Like those models, our model relies on continual comparison of feedback of current eye displacement with desired eye displacement to determine whether the saccade has brought the eye onto the target. As in the model of Jürgens et al. (1981), the comparison of eye and target position in our model is made in retinotopic coordinates rather than in the spatial coordinates of the Robinson model.

Figure 16 shows a diagram of the brain stem circuitry that forms the basis of our model. For simplicity we will consider a one-dimensional model that can be thought of as generating radial saccades. The rectangle labeled SC outlines the superior colliculus. We envision the colliculus to operate in a place code with a map of desired eye displacements, and the activity at a specific locus of cells within the colliculus ($e_p$) as determining the amplitude of the impending saccade. The size of the eye movement to be executed is specified by the center of gravity of the active cells within that region of the colliculus (Duibel et al. 1984; du Lac and Knaudsen 1987), as summarized in the first point of the dual coding hypothesis of Sparks and Mays (1990). We regard the clipped (and possibly partially clipped) cells to be the output of the summing junction ($e_p$) that compares desired displacement ($\Delta E$) with feedback of current displacement ($\Delta E'$). The desired displacement signal ($\Delta E$) might be derived from cortical inputs including those from the frontal eye fields (Goldberg and Segraves 1989) and parietal cortex (Andersen and Gnadt 1989). We have not observed the $\Delta E'$ signal in the colliculus, and we assume that it is represented by fibers projecting to the colliculus. To generate this signal, an inverse temporal-to-spatial transform that maps brain stem temporal activity to a collicular place code is required.

A gain element ($a$) has been placed after the locus of activity ($e_p$) to indicate that the level of activity at the $e_p$
locus can be altered by many factors that affect saccadic velocity but not saccadic accuracy. The next step in the model is the conversion from a place code to a temporal code. We assume that this conversion occurs through appropriately weighted connections onto presaccadic burst neurons in the pons and midbrain (B in Fig. 16). The signals to the left of the vertical dashed line are in the topographical place code of the colliculus; signals to the right of this line are in the temporal code of the presaccadic neurons. The output of these presaccadic neurons is the burst of innervation needed to make a saccade ($V_s$). The net level of activity at the $e_n$ locus is represented by $e_{mn}$, the dynamic motor error, which is in a temporal code.

As in the other models of saccadic control, the velocity command ($V_s$) is integrated by the neural integrator (NI) to produce the new eye position ($E$) (Robinson 1975). More important for our model is the local feedback loop extending through a model integrator (MI) back to the colliculus. The velocity command ($V_s$) is integrated by this resettable integrator (MI) to determine current eye displacement ($\Delta E$), as proposed by Jürgens et al. (1981). This signal ($\Delta E$) is still in the temporal domain, however, and must be transformed back into a place code. Thus, its activity level may be subtracted from the discharge of the cells within the $e_n$ locus.

In net, our collicular feedback model incorporates the following features: 1) the colliculus is within the feedback loop that includes desired and current eye displacement to control the amplitude of the saccade; 2) the neuronal elements within the colliculus encode dynamic motor error; 3) this motor error in the place-coded map within the colliculus is transformed into a temporal code in the pons to drive the eye; 4) the temporal code is transformed back into a spatial code again in the loop feeding back the information on current eye displacement to the colliculus. Further details of the model are given in the appendix.

Applications of the collicular feedback model

Implementation of this model allows us to compare the discharge of the clipped collicular cells that we recorded with the variable in the model that we think reflects this discharge, motor error, $e_m$. Figure 17 compares the neuronal data (left column) with the predictions of the model (right column). Figure 17A shows a sample of the observed discharge spikes density from a cell on a single trial during the execution of a 12° saccade, and Fig. 17B shows the decline in the model variable, motor error ($e_m$), as the saccade progresses. Figure 17C plots spike density against radial error, and Fig. 17D shows the corresponding plot for the signal $e_m$. The model signal $e_m$ has a trajectory that matches the trajectory of the collicular neuron. This shows that the discharge of the clipped cells in the colliculus behaves in the way we would expect if it conveyed information about dynamic motor error.

The model also produces results consistent with the observations of a change in cell discharge with the velocity of the saccade. Figure 17E shows the spike density of the cell versus eye velocity, and Fig. 17F shows the corresponding trajectory for the spike discharge derived from the model plotted against eye velocity. The match between neuronal discharge and model prediction is quite good.

This model equates dynamic motor error with the output of the superior colliculus, it also generates changes in eye velocity that are consistent with those observed in single-cell recording. Thus, a single model, with the comparator of the local feedback loop within the superior colliculus, accounts for the observed relationship of collicular discharge to both radial error and radial velocity.

The prediction of the collicular feedback model with respect to velocity during the saccade also might explain several observations that have related the superior colliculus to the control of velocity. A relationship between cell discharge and saccadic velocity has been observed in both the cat (Berthoz et al. 1986) and monkey (Rohrer et al. 1987). Berthoz et al. (1986) shifted the eye velocity traces by up to 86 ms to produce a match between the peak activity of the histogram of cell discharge and the peak velocity, and suggested that one of the output signals of the colliculus was a temporal signal indicating saccade velocity. Rohrer et al. (1987) reported a positive correlation between mean and peak spike rate and peak eye velocity, and they suggested that the colliculus could send a velocity signal to burst neurons in the brain stem. These changes in saccadic velocity with change in cell discharge would be expected if the colliculus is within the feedback loop as described in our model.
Any change in activity at the $e_p$ locus of activity within the <br>colliculus would change the discharge rate at the brain stem <br>site represented by B, and this would change the velocity <br>of the saccade. The electrical microstimulation experiments <br>of Munoz and Guittion (1987a), which produced saccade <br>acceleration, also would be consistent with our model for <br>the same reason. Small, reversible collicular lesions induced by injecting <br>muscinol or lidocaine cause slowing and shortening of saccades <br>(Hikosaka and Wurtz 1983, 1986; Lee et al. 1988). <br>Because the amplitude and direction of a saccade is deter-<br>mined by the center of gravity of the collicular activity at <br>the $e_p$ locus, any shift in this locus would predict a shift in <br>the amplitude or direction of a saccade, and the reversible <br>lesions presumably would produce such a change of locus <br>(Lee et al. 1988). The velocity changes would result because <br>the collicular cells affected by such lesions would lie in the <br>feedback loop, and a change in the total output from the <br>locus of activity in our model would produce changes in the <br>bursters (B), which would lead to saccades of different veloc-<br>ities. Thus, whereas our collicular feedback model is designed to <br>control the amplitude of the saccade by placing the supe-<br>rior colliculus within a feedback loop, a consequence of <br>such a model is an understanding of the relationship of the <br>superior colliculus to saccadic velocity. Simulations of the <br>model reproduce simultaneously both the almost linear re-<br>lationship between spike density and radial error and the <br>complex and multivalued relationship between spike den-<br>sity and radial velocity. This model thus provides an expli-<bricit mechanism for the dual coding hypothesis of Sparks and <br>Mays (1990). <br>As an aspect of our model that is less well worked out is the spatial-to-temporal transformation between the superior <br>colliculus and the brain stem. Our conception of this is that <br>areas of the colliculus that are related to larger saccades <br>have a larger effect on their target cells in the pons or mid-<br>brain as has been suggested previously (Edwards and Hen-<br>kel 1978; Wurtz and Albano 1980). That is, cells that are <br>active before large saccades (in the caudal colliculus) would <br>have a significantly greater density of projections than <br>would cells in the rostral pole, the activity of which is re-<br>lated to smaller eye movements. For horizontal saccades <br>these target cells would be the vector long lead burst neu-<br>rons and directional long lead burst neurons of the pons <br>described by Hepp and Henn (1983), which in turn project <br>to the medium lead bursters (B in Fig. 16). There are proba-<br>bly a series of steps necessary to accomplish the complete <br>conversion from the spatial to the temporal domain (Tweed <br>and Villis 1990), and we are probably only outlining one of <br>the earliest steps, with subsequent and probably indepen-<br>dent operations occurring elsewhere in the brain stem <br>(Hepp and Henn 1983; Ottes et al. 1986). <br>Our collicular feedback model also requires that feed-<br>back or current displacement $\Delta e^*$, which is in a temporal <br>code, be transformed into a place code as an input to the <br>comparator in the superior colliculus. We do not know <br>where this transformation would be done, but one possible <br>area is the central mesencephalic reticular formation <br>(cMRF), because cells in that area respond primarily with <br>horizontal saccades (Cohen et al. 1986), a characteristic we <br>would expect as part of such an inverse transform. It is also <br>possible that the colliculus itself performs the inverse trans-<br>formation (Lefevre and Galiana 1990). <br>Comparisons with other models of collicular function <br>Two recent models of the saccadic system directly ad-<br>dress the issue of how the colliculus affects eye velocity. In <br>one model the output of the colliculus represents the de-<br>sired rate of change of the eye displacement and is the input <br>to a feedback loop in the pons (Scudder 1988). In this <br>model the colliculus, to a certain extent, encodes both eye <br>velocity and amplitude, and thus the peak discharge of a <br>collicular neuron should be proportional to the peak veloc-<br>ity of small saccades. As saccadic size increases, the peak <br>discharge increases, and peak velocity should also increase <br>because of the linear pontine burst neurons used in this <br>model. Thus this model would show no saturation of col-<br>licular discharge with increasing eye velocity, whereas our <br>data show such a saturation. The Scudder (1988) model <br>predicts that increased collicular activity would lead to <br>larger saccades and this would appear to be inconsistent <br>with single-cell studies (e.g., Fig. 6). It would also be diffi-<br>cult to apply this model to our data because no mechanism <br>was proposed for generating a collicular burst with the re-<br>quired shape. Thus this model could not explain our almost <br>linear decay of collicular activity with dynamic motor <br>error. <br>The other model suggests that the output of the superior <br>colliculus encodes the desired eye rotation in three dimen-<br>sions (Tweed and Villis 1990). Such an eye rotation may be <br>represented by quaternions, four-dimensional operators for <br>specifying rotations in three dimensions. The four compo-<br>nents of the quaternion provide enough redundancy that a <br>ratio of the components can be used to determine saccadic <br>amplitude. Increased collicular activity would increase the <br>speed of eye rotations, but not their amplitude, because the <br>components would be larger, but they would still have the <br>same ratio. This model could explain our results if the supe-<br>rior colliculus, rather than the pons, were the site of the <br>feedback. <br>Our experiments confirm the dual coding hypothesis of <br>Sparks and Mays (1990) and extend that hypothesis by pro-<br>posing a collicular feedback mechanism for its implementa-<br>tion. The locus of activity within the colliculus determines <br>saccadic amplitude, because a feedback loop maps saccadic <br>displacement onto the colliculus, which continues to fire <br>until the displacement matches the desired amplitude. The <br>level of activity within the active $e_p$ locus is related to the <br>differences between desired amplitude and current displace-<br>ment and thus encodes dynamic motor error. This dynamic <br>motor error signal indirectly drives burst neurons in the <br>pons, which control saccadic velocity. Because the error <br>computed by this feedback loop is a displacement, and not <br>a velocity error, the loop guarantees saccadic accuracy even <br>when saccadic velocity varies.

APPENDIX
Simulations for Fig. 17 were produced with the use of a one-di-<br>dimensional model of a radial saccadic system. The basic structure <br>was two integrators (Jürgens et al. 1981). One integrator is in the <br>final common path and converts the eye velocity command from <br>the burst generator into an effference copy of eye position (Rohn-
son (1973). The eye velocity command, efference copy, and neural slip, which cause the velocity command to die out exponentially, were fed into a one-zero, four-pole model of the ocular motor plant (Optican and Miles 1985). The parameters of the plant (time constants, external oscillation frequency, and damping factor) were adjusted empirically to match the actual eye movement shown in Fig. 17. The saccadic bursting, or velocity command, was obtained as the output of a nonlinear burst gain element that simulated a medium-lead burst neuron (MLBN) (Zee et al. 1976). The input to the MLBN was the difference between the desired eye displacement, ΔE, and the efferency copy of the eye's displacement since the beginning of the current saccade (ΔE0). This efference copy of saccade displacement was obtained from the second integrator (MI in Fig. 16). This integrator receives a copy of the velocity command and is reset at the beginning of each movement.

This simple model was sufficient to construct the dynamic motor error, eM. The purpose of the simulation was to show how the time course of eM varied with that of eye position and velocity. The simulation was necessary because of the nonlinear and dynamic nature of the model.

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