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Discharge Properties of Monkey Tectoreticular Neurons

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Rodgers, C. Kip, Douglas P. Munoz, Stephen H. Scott, and Martin Paré. Discharge properties of monkey tectoreticular neurons. J Neurophysiol 95: 3502–3511, 2006; doi:10.1152/jn.00908.2005. The intermediate layers of the superior colliculus (SC) contain neurons that clearly play a major role in regulating the production of saccadic eye movements: a burst of activity from saccade neurons (SNs) is thought to provide a drive signal to set the eyes in motion, whereas the tonic activity of fixation neurons (FNs) is thought to suppress saccades during fixation. The exact contribution of these neurons to saccade control is, however, unclear because the nature of the signals sent by the SC to the brain stem saccade generation circuit has not been studied in detail. Here we tested the hypothesis that the SC output signal is sufficient to control saccades by examining whether antidromically identified tectoreticular neurons (TRNs: 33 SNs and 13 FNs) determined the end of saccades. First, TRNs had discharge properties similar to those of unidentified SC neurons and a proportion of output SNs had visually evoked responses, which signify that the saccade generator must receive and process visual information. Second, only a minority of TRNs possessed the temporal patterns of activity sufficient to terminate saccades: Output SNs did not cease discharging at the time of saccade end, possibly continuing to drive the brain stem during post-saccadic fixations, and output FNs did not resume their activity before saccade end. These results argue against a role for SC in regulating the timing of saccade termination by a temporal code and suggest that other saccade centers act to thwart the extraneous SC drive signal, unless it controls saccade termination by a spatial code.

INTRODUCTION

Neurons in the intermediate layers of the primate superior colliculus (SC) have been shown to display activity necessary for regulating the production of saccadic eye movements (Paré and Hanes 2003; Schiller et al. 1980; Sparks 1978). Consistent with this role in saccade processing, many neurons in these SC layers are also known to send descending projections that contact neurons within the brain stem, including the paramedian pontine reticular formation (PPRF), which innervate cranial nuclei to control extraocular muscles (Scudder et al. 2002; Sparks 2002). Nevertheless, the specific activity of monkey tectoreticular neurons (TRNs) has not been characterized in more detail than to indicate that it is related to saccade production (Gandhi and Keller 1997; Moschovakis et al. 1988; Scudder et al. 1996a). Little is therefore known about the discharge properties of TRNs and whether they differ from those of unidentified SC neurons. The function of the SC beyond movement initiation remains highly debated (Anderson et al. 1998; Goossens and van Opstal 2000; Munoz and Wurtz 1995b; Port et al. 2000; Soetedjo et al. 2002; Waitman et al. 1991) because it is unclear whether its neuronal activity controls saccade trajectory and/or specifies an updated eye displacement signal during saccades that can effectively signal saccade termination. Here we studied the activity of antidromically identified TRNs to determine the nature of the SC projection to the brain stem saccade generator and whether it carries signals appropriate to specify saccade termination.

The intermediate layers of the SC contain two main populations of neurons involved in saccade control. First, saccade neurons (SNs) discharge a burst of activity that peaks around the time of saccade onset, which is thought to provide a motor command specifying the vector of an upcoming saccade (Sparks 1978; Sparks and Mays 1980; Sparks et al. 1976). This burst of activity occurs during saccades to a limited region of the visual field known as the neuron’s movement field or response field (RF; for review, see Sparks 1986). These RFs are topographically organized and the SC forms a saccade map, with command signals for large saccades being coded in its caudal portion and small saccades further rostrally (Ottes et al. 1986; Robinson 1972). Second, fixation neurons (FNs), located in the very rostral SC, are tonically active when the eyes are still and pause during saccades (Munoz and Guitton 1991; Munoz and Wurtz 1993a). This activity is thought to prevent intrusive saccades (Munoz and Wurtz 1993b).

We investigated the possible code in the SC output signals that could actively terminate saccades. Specifically, we tested two hypotheses by which the SC could temporally code the end of saccades by the activity of SNs and FNs. As first proposed by Waitzman et al. (1991), if the saccade burst from the majority of output SNs were highly attenuated (i.e., if they had “clipped” activity) at the time of saccade end, saccades could be terminated simply because of the absence of drive. In this scenario, the activity of SNs is required to fall below a certain threshold necessary to prolong a saccade. Accordingly, we would expect to see not only a significant decrease in SC activity at the time of saccade end but little variability in the activity level of individual SNs across a sample of neurons. Such a temporal code for saccade metrics signaling saccade termination has not been supported by previous studies examining the discharge from a general population of SC neurons (Frens and van Opstal 1997; Keller et al. 1996; Stanford and Sparks 1994), but it was not tested specifically in TRNs. The possibility exists that SNs with “clipped” activity selectively project to the brain stem saccade generator.

The second—alternative—hypothesis is that saccades are terminated by the reactivation of FNs that choke the saccade.
drive signal in the PPRF. Substantial evidence indicates a functional connection between the rostral pole of the SC and a major group of inhibitory neurons in the brain stem called omnipause neurons (OPNs) (Büttner-Ennever et al. 1999; Gandhi and Keller 1997; Paré and Guitton 1994). Like FNs, OPNs are tonically active during intersaccadic periods but pause during saccades. The tonic activity of OPNs acts as an inhibitory gate for saccade generation and a pause in OPN activity is required for saccade initiation (Keller et al. 1996). Bergeron and Guitton (2002) recently demonstrated that, during multiple-step gaze shifts in head-free cats, SC fixation neurons code gaze position error and increase their discharge during a saccade at a rate proportional to the distance between the current and the desired gaze positions. They propose that this progressive reactivation of FNs controls saccade termination by causing OPNs to resume their activity. Although the majority of monkey FNs resumes their activity only after the end of saccades (Everling et al. 1998), it is still possible that those FNs that do project onto OPNs are reactivated before the end of saccades.

In addition to their saccade-related burst of activity, a subset of SNs called visuomotor neurons (Mohler and Wurtz 1976) exhibit visually evoked responses. Previous studies have assumed that visual signals are sent by the SC to the PPRF and contribute to saccade processing (Dorris et al. 1997; Edelman and Keller 1996, 1998; Munoz et al. 2000; Paré and Munoz 1996; Sommer 1994). Physiological evidence suggests that visual and motor bursts of activity in individual SC neurons can merge to trigger short-latency, “express” saccades (Dorris et al. 1997; Edelman and Keller 1996) and shape averaging saccades made in response to two targets presented simultaneously (Edelman and Keller 1998). However, the anatomical substrate underlying this visual influence on saccade processing is unknown. While examining the discharge properties of TRNs, we also tested the hypothesis that the SC send visual signals to the brain stem saccade generator by output visuomotor neurons.

Our results reveal that visual signals are indeed sent from SC output neurons to the PPRF and confirm previous assumptions about the origin of visually evoked responses in downstream neurons. Importantly, we found that the majority of output SNs does not have “clipped” activity and there is large variability in their level of activity at saccade end. Finally, FNs were not consistently reactivated when saccades end. In summary, saccade termination does not seem to be coded temporally in the SC by a removal of drive signals from SNs or reactivation of FNs.

These results were previously reported in abstract form (Rodgers et al. 2003).

METHODS

The data described in this report were obtained from single neurons recorded in four monkeys (Macaca mulatta, 5–10 kg) trained to perform oculomotor tasks for a liquid reward. Details of surgical, gaze monitoring, and electrophysiological techniques were previously described (Munoz and Istvan 1998; Paré and Munoz 1996). All monkeys were implanted with one recording chamber centered on the midline and angled 38° posterior of vertical to access the SC. In two monkeys, an additional chamber was centered on the interaural axis and angled 25° lateral of vertical to access brain stem OPNs. Animals received both antibiotics and analgesic treatments during an extended posturogical recovery period. All animal care and experimental procedures were in accordance with the Canadian Council on Animal Care policies on use of laboratory animals and approved by Queen’s University Animal Care Committee.

Behavioral tasks

Monkeys were seated in a primate chair with their heads restrained for the duration of the experiments, which were performed in total darkness. Visual stimuli were red light-emitting diodes (0.03 cd/m²) back-projected onto a tangent screen positioned 86 cm from the monkeys’ eyes. The data described in this report were collected while the monkeys performed the step saccade and gap saccade tasks, which were described previously (Paré and Munoz 1996). All trials began once the monkey maintained fixation for 500–1,000 ms on a central fixation point. In the step saccade task, the fixation point was extinguished at the same time that an eccentric target was presented in the peripheral visual field. The monkey had 500 ms to initiate a saccade and required to fixate the target for an additional 300 ms. In the gap saccade task, a gap period of 0–800 ms was introduced between fixation point disappearance and target appearance, and the monkey was required to maintain fixation at the location of the extinguished fixation point.

Antidromic identification

We identified TRNs physiologically in two monkeys by recording SC neurons that were activated antidromically by stimulation delivered in the raphe interpositus (RIP) nucleus, which contains OPNs (Büttner-Ennever et al. 1988). This nucleus had already been identified in these two animals during a previous single-neuron recording study (Everling et al. 1998), and we used this information to position in each monkey the tip of a bipolar concentric stimulating electrode (Kopf SNEX-100) close to the midline between the two columns of cells (i.e., OPNs) that compose the RIP nucleus. With stimulating electrodes positioned medial and in close proximity to both predorsal bundles that contain descending axons of SC output neurons (Büttner-Ennever et al. 1999; Harting 1977; Moschovakis et al. 1988), antidromic action potentials and field potentials could be elicited in both SCs. The electrical stimulus used for antidromic identification consisted of biphasic pulses (0.1–0.3 ms) with varying intensity. Details of the parameters of electrical stimulation for individual neurons are given in RESULTS. The threshold intensity to evoke antidromic responses was defined as the current intensity required to evoke a response on roughly 50% of stimulation trials. To demonstrate antidromic activation, we relied on the constancy of the response latency (measured at 1.5 × threshold current) and the collision of orthodromic (self-generated) and antidromic (stimulated) spikes (Lipski 1981; Munoz and Istvan 1998).

Data analysis

We quantified neuronal activity with spike density functions aligned on target onset, saccade onset, or saccade end. To generate the spike density functions, a Gaussian pulse (σ = 4 ms) was substituted for each spike and all Gaussians were summed together to produce a continuously varying function in time.

SACCADE-RELATED ACTIVITY. TRNs were classified as SNs if they showed an increase in activity (>100 Hz above baseline) that peaked ±20 ms of the initiation of saccades made in the neuron’s RF (Dorris et al. 1997; Sparks et al. 1976). The peak saccade activity of each neuron was taken as the highest discharge rate associated with the optimal saccade vector. Activity at saccade end was taken as the discharge rate during ±1 ms of saccade end. All activity levels were corrected by subtracting the baseline. SNs were further classified according to properties described below.
CLIPPING ACTIVITY. To quantify the amount of activity at the time of saccade end relative to peak activity, an attenuation index (\( \gamma \)) was calculated from each neuron’s spike density function

\[
\gamma = \frac{SD_{p} - SD_{j}}{SD_{p}}
\]

where \( SD_{p} \) is the peak saccade activity and \( SD_{j} \) is the activity at the time of saccade end. Each neuron was assigned to one of the three categories defined by Waitzman et al. (1991): 1) “clipped” neurons had the majority of their activity cut off by the end of a saccade and <20% of peak activity remained at saccade end (\( \gamma > 0.8 \)); 2) “partially clipped” neurons had 20–50% of peak activity still present at saccade end (\( \gamma = 0.5–0.8 \)); and 3) “unclipped” neurons displayed >50% of their peak activity (\( \gamma < 0.5 \)) at saccade end.

RESPONSE FIELDS. SNs were also subdivided into two categories of RFs: “closed” and “open” (Munoz and Wurtz 1995a). To determine the RF shape of a neuron, we used the step saccade task and targets positioned randomly among one of eight eccentricities in the optimal direction. Each block of trials consisted of the target being presented in the optimal direction and amplitude, as well as two to four smaller and three to five larger amplitudes. For larger target eccentricities (>20°), the fixation point was positioned on one side of the visual screen and the target appeared on the opposite side to increase the testable visual angle. The maximum amplitude tested for each neuron was usually >50°. Neurons that discharged for all saccades in the optimal direction with eccentricities equal to or greater than the testable visual angle. The maximum amplitude tested for each neuron was usually >50°. Neurons that discharged for all saccades in the optimal direction with eccentricities equal to or greater than the testable visual angle. The maximum amplitude tested for each neuron was usually >50°. Neurons that discharged for all saccades in the optimal direction with eccentricities equal to or greater than the testable visual angle. The optimal were characterized as having open RFs. Neurons that showed no discharge during any saccade of greater than optimal amplitude were characterized as having closed RFs.

VISUALLY EVOKED RESPONSES. The step saccade task was used to determine whether SC neurons had visual activity. To determine background discharge for each neuron, baseline activity was calculated during a period of active fixation over 100 ms before target onset. To be consistent with previous studies (Everling et al. 1999; Paré and Munoz 2001) neurons were classified as having visual activity if they had a distinct increase in activity (>50 Hz above baseline) that peaked within 60–110 ms after the onset of a target presented at the optimal location.

FIXATION-RELATED ACTIVITY. TRNs were classified as FNs if they were tonically active (>10 Hz) during the gap period of the gap saccade task and exhibited a pause in activity during all ipsiversive and most contraversive saccades (Dorris et al. 1997; Everling et al. 1998; Munoz and Wurtz 1993a). Tonic activity, used as baseline, was measured in an epoch of 100 ms before target onset to ensure that the monkey was actively fixating during this time. To assess the contribution of FNs to saccade termination, we measured during the step saccade task the time of FN reactivation as the first spike after a saccade-related pause to target eccentricities that were large enough to induce a pause in activity. Results are presented for saccades of 10° because this amplitude consistently evoked the typical pause in FN activity.

RESULTS

We antidromically activated a total of 116 neurons in both SCs of two monkeys. Sufficient data were collected from 61 identified TRNs to fully characterize their discharge properties. Of this sample, 46 neurons had discharges modulated during the tasks: 33 were classified as SNs and 13 were classified as FNs according to the criteria outlined in METHODS. For comparison, an additional sample of nonidentified SNs (n = 210) was recorded in the same monkeys as well as from the SC of two other monkeys.

Characteristics of antidromic responses

Figure 1A shows the distribution of antidromic response latencies for the 61 TRNs that we studied in detail. These ranged from 0.5 to 2.6 ms, with a mean (±SD) of 1.2 ± 0.5 ms. They did not differ statistically (\( P > 0.01 \)) across the classes of TRNs: 1.1 ± 0.5 ms for the 33 SNs, 1.1 ± 0.6 ms for the 13 FNs, and 1.5 ± 0.5 ms for the 15 neurons whose discharges were unrelated to saccade or fixation behavior. Assuming a distance of 16 mm from the mid-SC to the PPRF, we estimated the conduction velocity of TRNs to range from 6 to 27 m/s and to average 13 m/s.

Figure 1B shows the distribution of activation threshold currents for each neuron. Current thresholds across the classes of TRNs ranged from 20 to 900 µA, with a mean ± SD of 290 ± 194 µA. Mean current threshold was 290 ± 197 µA for SNs, 244 ± 113 µA for FNs, and 360 ± 236 µA for the neurons whose discharges were not modulated during the tasks.

Visually evoked responses

Eighteen of the 33 identified output SNs (55%) had significant visually evoked responses. Figure 2A illustrates one representative example. The peak discharge rate of the visual responses of these 18 output visuomotor neurons ranged from 89 to 448 Hz, with a mean ± SD of 235 ± 102 Hz (Fig. 2B, top histogram). In comparison, 127 of the 210 (60%) nonidentified SNs had visual activity, a proportion not statistically
different from that of TRNs (χ², P > 0.05). The peak discharge rates of the visual responses of these nonidentified visuomotor neurons ranged from 81 to 895 Hz, with a mean of 185 ± 111 Hz (Fig. 2B, bottom histogram). The distribution of visual activity in those samples did not differ significantly [Kolmogorov–Smirnov (KS) test, P > 0.05].

Saccade-related activity

Figure 3 shows typical activity patterns of output SNs when aligned on saccade onset or end. We quantified the relative amount of activity present at the time of saccade end by calculating an attenuation index (γ) for each neuron and classified them as having “clipped,” “partially clipped,” or “unclipped” activity (see METHODS). Of the 33 output SNs, eight had “clipped” activity, 19 had “partially clipped” activity, and six had “unclipped” activity (Fig. 3, A, B, and C, respectively). The peak discharge rate of the saccade activity of these output neurons ranged from 199 to 918 Hz, with a mean ± SD of 478 ± 192 Hz (Fig. 4A, top histogram). Similarly, the peak saccade activity of the 210 nonidentified SNs ranged from 107 to 936 Hz, with a mean of 388 ± 195 Hz (Fig. 4A, bottom histogram). There was no significant difference between the distributions of saccade activity of these samples (KS test, P = 0.15).

The discharge rate of the 33 output SNs at the time of saccade end ranged from 38 to 571 Hz, with a mean ± SD of 174 ± 125 Hz (Fig. 4B, top histogram). In comparison, the 210 nonidentified SNs had activity at the time of saccade end that ranged from 0 to 442 Hz, with a mean ± SD of 111 ± 81 Hz (Fig. 4B, bottom histogram). Although there was a significant difference in the distribution of activity rates at the time of saccade end between output and nonidentified SNs (KS test, P = 0.03), the levels of activity were well above zero in both samples. Figure 4C shows that there was a large overlap in the distributions of the attenuation index of output and nonidentified SNs. The γ values of the output SNs ranged from 0.16 to 0.90, with a mean ± SD of 0.64 ± 0.18. The γ values of the nonidentified neurons ranged from 0.09 to 1.0, with a mean of 0.66 ± 0.22. These distributions were not significantly different (KS test, P = 0.45). We also found no correlation between the attenuation index of a neuron and the amplitude or direction of its optimal saccade vector (not shown), thereby indicating that a
neuron’s location on the SC motor map is not related to its clipping property.

Response fields

The 33 identified output SNs had RFs with amplitude tunings that were closed (n = 14), open (n = 9), or unclassified (n = 10); the ratio of neurons with closed to open RFs was thus 1.56. Examples of neurons with closed and open RFs are shown in Fig. 5, A and B, respectively. Figure 5C shows the distributions of RF types and clipping activity among those neurons. We found no relationship between the type of RFs of a neuron and its attenuation index. The distributions were similar for open and closed RFs despite a greater number of partially clipped neurons with closed RFs (KS test, P = 0.87). Mean ± SD γ values of neurons with closed and open RFs were 0.63 ± 0.19 and 0.66 ± 0.19, respectively. Neurons with open RFs all displayed “build-up” activity during the gap period of the gap saccade task, as previously reported by Munoz and Wurtz (1995a). Neurons with closed RFs did not show any build-up activity and as such resembled the burst neurons described by the same authors.

Fixation-related activity

The activity of a representative output FN is shown in Fig. 6A. The baseline tonic activity of our sample of 13 FNs ranged from 21 to 123 Hz, with a mean ± SD of 56 ± 30 Hz. For 12 of these neurons, the first consistent spike after their saccade-related pauses occurred only after saccades had ended. Spike density functions aligned on saccade end are shown for all neurons in Fig. 6B. Figure 6C shows how the resumed FN activity at the time of saccade end estimated from the spike density functions was only a negligible percentage of the baseline tonic activity: on average, 13.4%.

DISCUSSION

Similarity between identified and nonidentified SC neurons

Our data indicate that the discharge properties of output SNs (n = 33) showed little or no difference with those of a larger comparison sample of nonidentified SNs (n = 210). We found no significant difference between 1) the proportion of neurons with visually evoked responses; 2) the distribution of peak visual activity; 3) the distribution of peak saccade activity; and 4) the distribution of γ values. Also, we found that output SNs possess either open or closed RFs in similar proportions to previously reported results (see following text). As a consequence, the discharge properties of nonidentified SNs in our study, which were consistent with those reported in the literature, can be considered representative of the SC output signal sent to the brain stem saccade generator. Our findings may thus
validate the interpretation of previous SC studies, especially those with adequate sampling sizes. It may be that the sampling in most of these studies was biased toward large neurons, which are easier to record and most likely output neurons. A similar correspondence was observed between the discharge properties of identified corticotectal neurons and nonidentified neurons in the lateral intraparietal area (Pare´ and Wurtz 1997, 2001) and the frontal eye field (Segraves 1992; Segraves and Goldberg 1987; Sommer and Wurtz 2000, 2001). Neuronal identification studies nevertheless remain valuable because they provide conclusive answers regarding the functions of these oculomotor structures and can shed light on the processing performed by their intrinsic circuitry (e.g., Munoz and Itsvan 1998).

Visually evoked responses of SC output neurons

We showed that more than half of the output SNs in our sample had visually evoked responses. The existence of such responses has been well established in neurons located in the SC intermediate layers (Mohler and Wurtz 1976) and they are thought to originate from striate and extrastriate cortical areas (Sparks and Hartwich-Young 1989). Visually evoked responses with similar latencies have also been reported downstream of the SC: OPNs in the RIP nucleus (in cat: Evinger et al. 1982; King et al. 1980; in monkey: Everling et al. 1998), long-lead burst neurons (LLBNs) in the nucleus reticularis tegmentum pontis (NRTP) (Crandall and Keller 1985; Matsu- zaki and Kyohou 1997) and the PPRF (Kaneko 2006; Munoz et al. 2000), and even in neck muscles (Corneil et al. 2004). Our observations suggest that these responses likely arise from visuomotor neurons in the SC, which could innervate these target structures by descending projections that have been identified anatomically (Büttner-Ennever et al. 1999; Harting 1977; Langer and Kaneko 1984; Moschovakis 1988; Scudder et al. 1996a) and/or physiologically (Gandhi and Keller 1997; Kaneko and Fuchs 1982; King et al. 1980; Paré and Guitton 1994; Raybourn and Keller 1977).

What is the function of these visual responses in the SC output signal? One clue comes from the observation that, in the presence of elevated preparatory activity, the visual activity of visuomotor SNs appears sufficient to trigger short-latency (70–90 ms) “express” saccades (Dorris et al. 1997; Edelman and Keller 1996). The view held by many groups is not only that the visually evoked responses of SNs are responsible for the initiation of express saccades but that these seemingly sensory responses could be viewed as failed motor signals during regular latency saccades (Edelman and Keller 1996; Guitton 1991; Paré and Munoz 1996; Sommer 1994). Our data provide solid evidence for this visuomotor hypothesis.

Weak visual responses of TRNs preceding regular saccades may also serve to “warm up” or prepare downstream neurons for initiating these saccades. Raybourn and Keller (1977)
showed that subthreshold stimulation of the SC caused reverberatory firing of LLBNs in the PPRF that long outlast the stimulation train, after which OPNs concurrently decrease their activity. This observation suggests that LLBNs play a role in controlling OPN activity and ultimately saccade duration. Because high preparatory activity of LLBNs has been correlated with an increased probability of express saccades (Munoz et al. 2000), there must be a mechanism to prevent saccades from being prematurely initiated. It may be that the visual responses of OPNs (Everling et al. 1998) counteract the target-related activation of LLBNs. In summary, the visually evoked responses of TRNs may serve not only to warm up the saccade system by innervating LLBNs but also to prevent premature saccades by increased activation of OPNs.

**Response fields of SC output neurons**

We show that output SNs had both closed and open RFs in a ratio of 1.6:1. Although we did not characterize the RFs of our sample of nonidentified SNs, our results are comparable to the ratio of 1.7:1 previously reported by Munoz and Wurtz (1995a). This finding once again validates the approach of sampling nonidentified neurons to estimate the SC output signal. Future SC models of saccade control will have to account for our observations and include outputs of neurons with both closed and open RFs to downstream targets. Our data cannot speak on whether these projections form a single command signal (Quaia et al. 1999) or separate pathways with different functions (Optican 1995; Wurtz and Optican 1994). Nevertheless, because output SNs with closed and open RFs had similar attenuation indices, it seems unlikely that they constitute two functionally separate populations with respect to the control of saccade end.

**Role of SN activity in saccade termination**

Despite an undisputed role in saccade initiation (Dorris et al. 1997; Paré and Hanes 2003), the SC role in saccade termination is far more controversial. In this study, we demonstrated that SC saccade activity is dissociated from the actual saccade metrics because SC discharge outlasted the end of saccades. Previous studies of unidentified neurons have shown similar dissociation and have also used such evidence to suggest that the SC does not code the metrics of saccades (Frens and van Opstal 1997; Keller et al. 1996; Stanford and Sparks 1994). This dissociation, however, is in direct contrast to the study of Waitzman et al. (1991), who observed that SC activity subsides as the eyes arrive on target and concluded that SC activity reflects an updated feedback signal (dynamic motor error) corresponding to the remaining distance the eyes must travel to reach a desired endpoint. Although our results do not address the specifics of the feedback or motor error debate surrounding the SC, they established that the SC activity is unlikely to specify saccade end or code dynamic motor error. If SC neuronal activity reflected saccade motor error and signaled saccade end, we would expect it to have returned to baseline level or below a threshold level when current eye position matched desired eye position at the time of saccade end. This trend would have been represented in a skewed $\gamma$ distribution toward 1.0. Instead we observed that peak activity decreased by an average of only about 36% at the time of saccade end and the $\gamma$ distribution was skewed toward lower values.

Alternatively, SC activity may not have to be fully attenuated to control saccade end. The possibility exists that saccade termination is coded by the SC activity when it falls below a fixed, yet elevated, threshold. Perhaps a 36% decrease from peak activity brings SC activity to a level below the threshold to drive saccades. If this were the case, the SC activity at the end of saccades should have low variability. However, our data do not provide evidence supporting this hypothesis: attenuation of peak activity ranged from 10 to 86% and discharge rates were even more variable between neurons (38–571 Hz), making a fixed, elevated threshold unlikely.

Saccade termination, signaled solely by the temporal activity patterns of SNs, seems even more unlikely when we consider...
Although highly controversial, no study to date has convinc-
ingly falsified this “moving hill” hypothesis using the exact
criteria outlined in the original paper by Munoz et al. (1991).
The main prerequisite is that neurons must have open RFs
(Munoz and Wurtz 1995a,b; Munoz et al. 1991; Port et al.
2000). Almost one third of the output SNs we recorded have
open RFs, but our study did not test this hypothesis. Because of
the broad distribution of the projections from the rostral SC
onto OPNs (Büttner-Ennever et al. 1999; Paré and Guitton
1994), neuronal activity outside the fixation zone (and of
neurons others than FNs) may control OPN reactivation and,
ultimately, saccade termination.

Nevertheless, an additional, stronger termination signal is
most likely required to fully stop the eyes. A likely candidate
to provide this termination signal is the cerebellum (LeFèvre
et al. 1998). The oculomotor region within the fastigial nucleus
has been implicated in previous models of saccade control
(Dean 1995; Quaia et al. 1999). This region receives saccade
signals from the SC by neurons in the NRTP (see Scudder et
al. 1996b), and its neurons have activity time locked to saccade
end (Fuchs et al. 1993; Ohtsuka and Noda 1991). Reversible
lesions cause saccades to become hypermetric (Robinson et
al. 1993), whereas subjects with permanent deep cerebellar
lesions have impaired saccade accuracy that does not recover
with time (Barash et al. 1999; Takagi et al. 1998). Finally,
projections from the oculomotor region of the fastigial nucleus
to the brain stem saccade generator circuit have been shown
anatomically (Gonzalo-Ruiz et al. 1988; Langer and Kaneko
1984). Quaia and colleagues (LeFèvre et al. 1998; Quaia et
al. 1999) reasoned that this connection could provide a “choke”
signal to terminate a saccade. Although our study did not
address this hypothesis, our data add to it by indicating that
oculomotor structures other than the SC are necessary to
terminate a saccade.

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