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Discharge Properties of Monkey Tectoreticular Neurons

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[1]*Canadian Institutes of Health Research Group in Sensory-Motor Systems and Centre for Neuroscience Studies, and Departments of* [2]*Physiology* [3]*Psychology, and* [4]*Anatomy and Cell Biology, Queen's University, Kingston, Ontario, Canada*

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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) determine the end of saccades. First, TRNs had discharge properties similar to those of nonidentified 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 postsaccadic 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 carry 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

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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 postsurgical 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/m^2) 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 (γ) was calculated from each neuron's spike density function

$$\gamma = \frac{SD_p - SD_f}{SD_p}$$

where SD_P is the peak saccade activity and SD_f 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 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 1*A* 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 1*B* 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 2*A* 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. 2*B*, *top histogram*). In comparison, 127 of the 210 (60%) nonidentified SNs had visual activity, a proportion not statistically

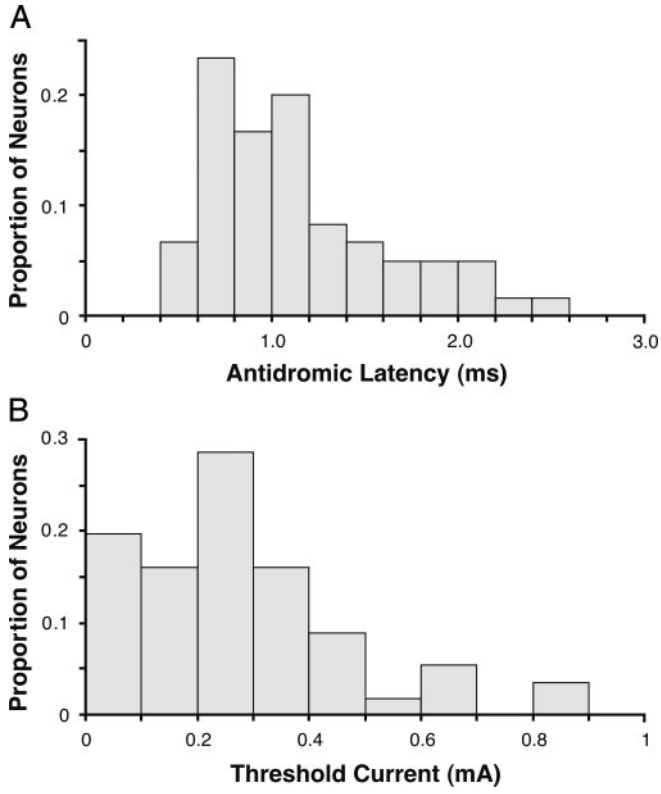

FIG. 1. Summary of the antidromic responses of tectoreticular neurons (TRNs). *A*: distribution of the latencies of the antidromic responses. Latency was calculated as the time delay between stimulation and the first recorded spike in the superior colliculus (SC). *B*: distribution of the (threshold) current to evoke an antidromic spike on 50% of stimulation trials.

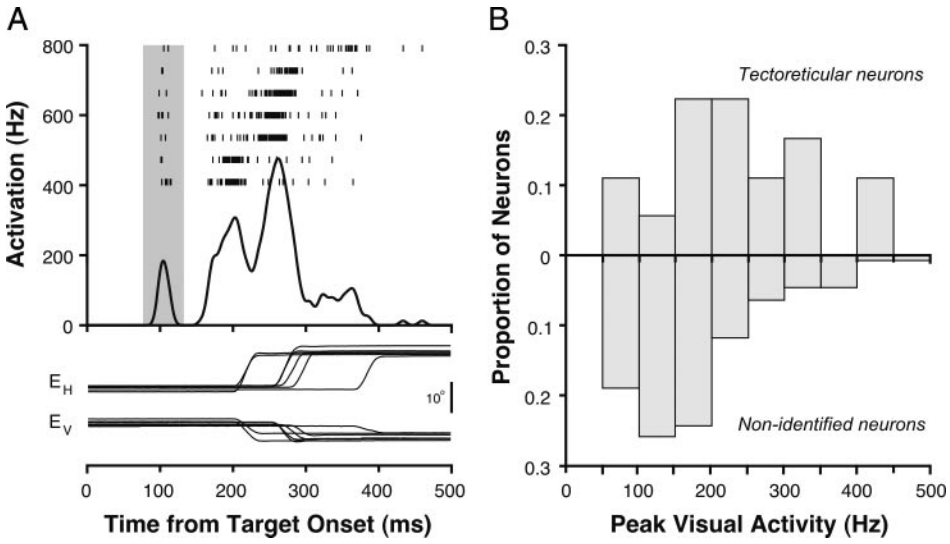

FIG. 2. *A*: example of visually evoked responses (gray area) of an antidromically identified SC neuron when a visual target was presented in the neuron's response field. Raster and spike density function are aligned on target onset together with the horizontal (E_H) and vertical (E_V) eye position traces. *B*: distribution of peak visual activity for all visually responsive neurons from the samples of antidromically identified ($n = 18$, *top histogram*) and nonidentified SC neurons ($n = 127$, *bottom histogram*). Data from 2 nonidentified saccade neurons (SNs) with visual activity >500 Hz are not shown.

different from that of TRNs (χ^2, $P > 0.05$). 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. 2*B*, *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. 4*A*, *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. 4*A*, *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. 4*B*, *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 of 111 ± 81 Hz (Fig. 4*B*, *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 4*C* 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

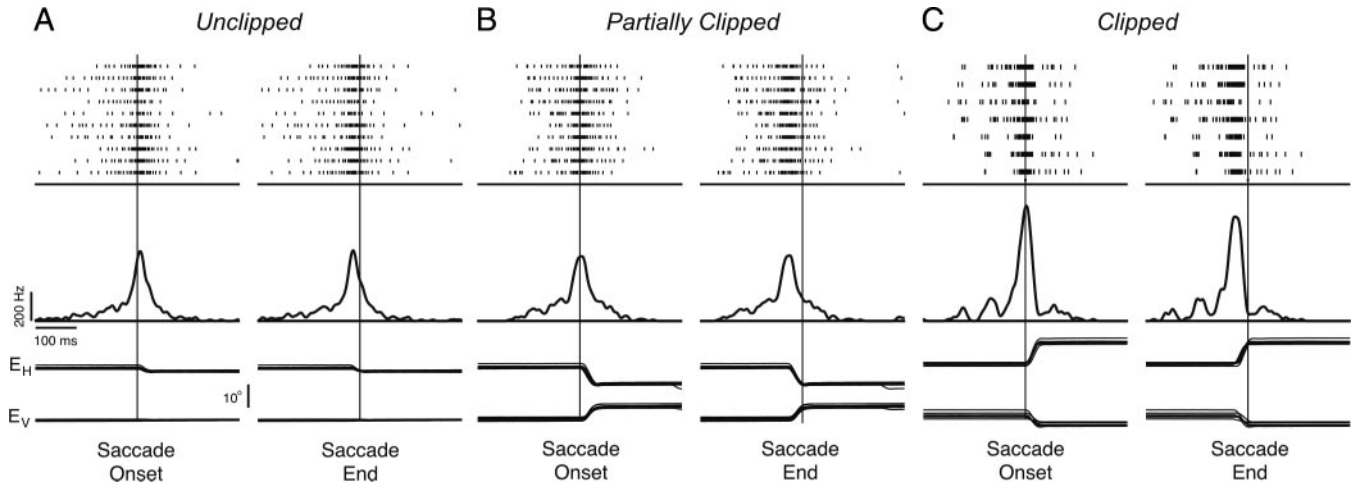

FIG. 3. Examples of neurons with "unclipped," "partially clipped," and "clipped" activity patterns. Raster and spike density functions are aligned on both saccade onset and end, together with the horizontal (E_H) and vertical (E_V) eye position traces. Data are shown for trials in which the monkey made saccades whose vectors were optimal for the neuron being recorded.

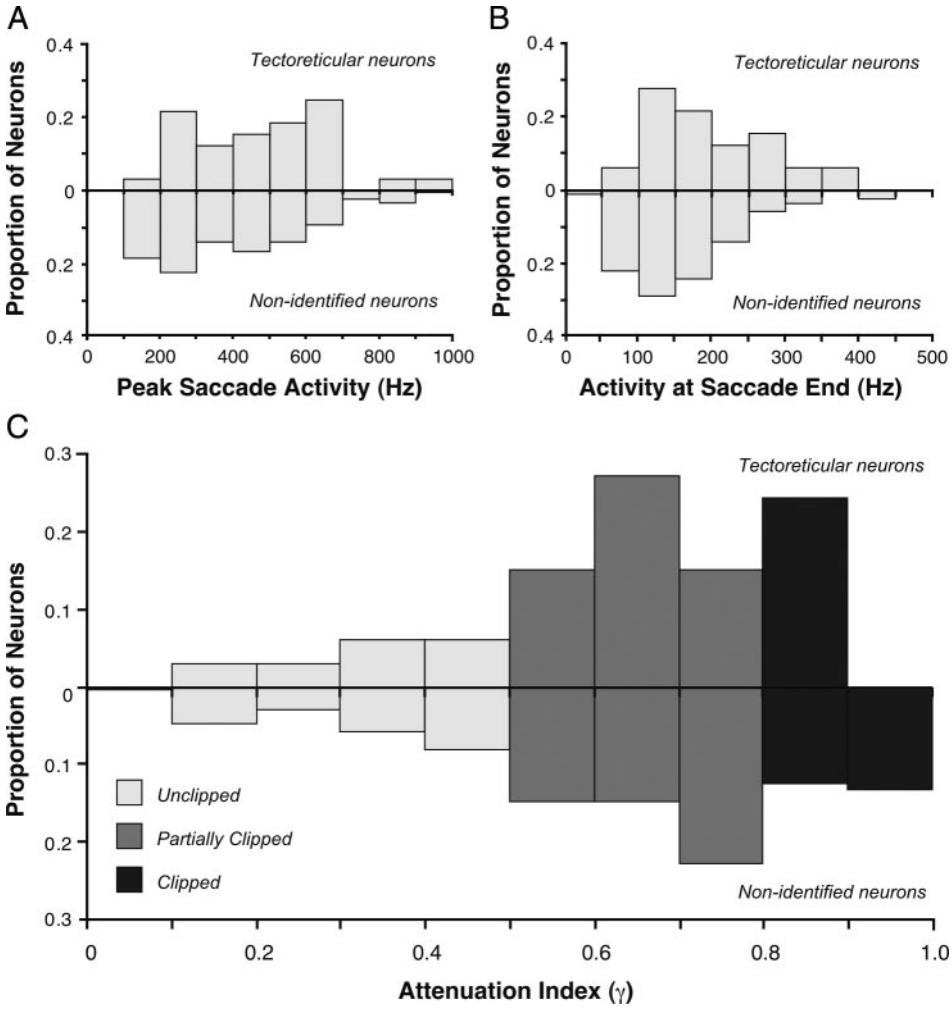

FIG. 4. Summary of the saccade activity exhibited by output and nonidentified SNs. Distribution of peak saccade activity (*A*) and activity at saccade end (*B*) from the samples of antidromically identified ($n = 33$, *top histogram*) and nonidentified SC neurons ($n = 210$, *bottom histogram*). Data from two output SNs with activity at saccade end >500 Hz are not shown. *C*: distribution of the attenuation index (γ) calculated from the activity levels shown in *A* and *B*. Compared with their peak saccade activity, "unclipped" neurons (light gray) had relatively high activity at the time of saccade end ($\gamma < 0.5$), "partially clipped" neurons (dark gray) had moderate activity ($\gamma = 0.5$–0.8), and "clipped" neurons (black) had negligible activity ($\gamma > 0.8$).

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 5*C* 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 γ 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. 6*A*. 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. 6*B*. Figure 6*C* 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 ouput 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

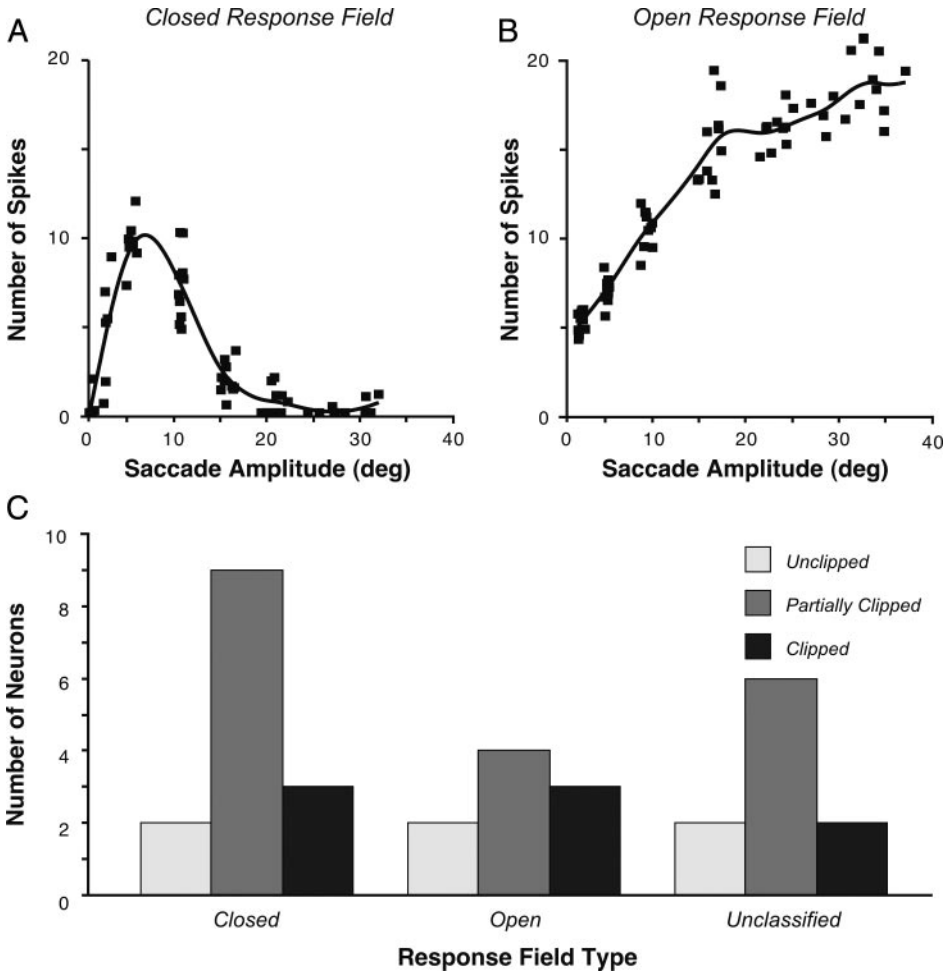

FIG. 5. Examples of output neurons with closed (*A*) and open (*B*) response fields. Number of spikes obtained in the interval spanning 8 ms before saccade onset to 8 ms before saccade end is plotted against the amplitude of saccades in the optimal direction, along with best-fit cubic spline functions. Optimal saccade amplitude for the neurons shown in *A* and *B* was 7 and 17°, respectively. Neurons with open response fields (RFs) discharge for all saccades greater than their optimal amplitude (their number of spikes can increase, but not their peak discharge rate), whereas the neurons with closed RFs did not. *C*: distribution of γ values for neurons with closed (*left*), open (*middle*), and unclassified (*right*) response fields. Neurons are grouped according to unclipped (light gray), partially clipped (dark gray), and clipped (black) activity.

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 (Paré 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; Matsuzaki 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)

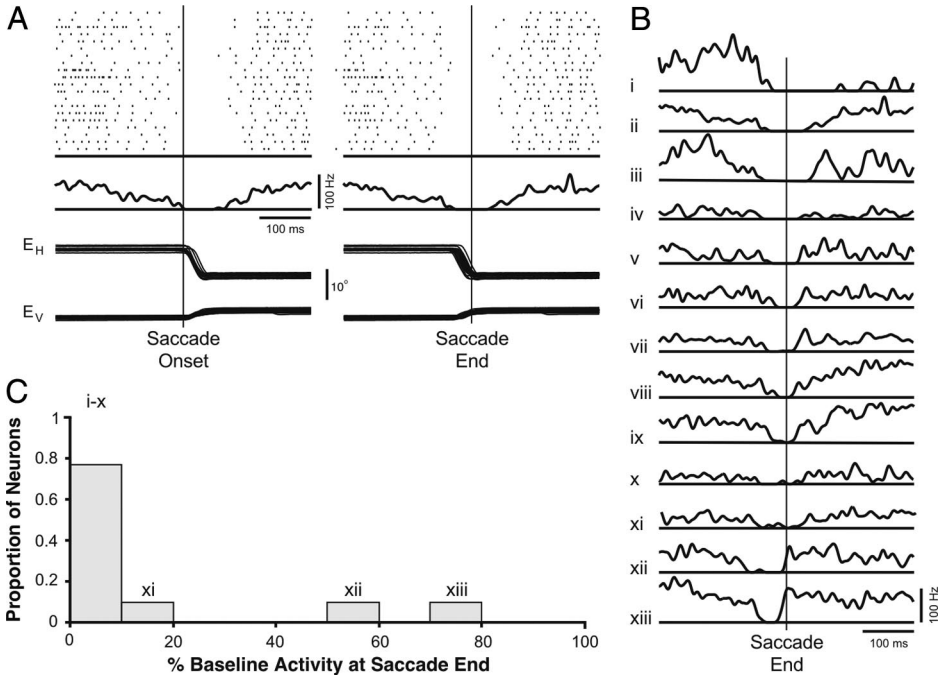

FIG. 6. *A*: example of an output fixation neuron (FN). Raster plot and spike density function aligned on both saccade onset and end during contraversive horizontal 10° saccades, together with the horizontal (E_H) and vertical (E_V) eye position traces. Notice that this neuron's tonic activity did not resume until well after the saccades ended. This trend can be observed throughout the spike density functions aligned on the sample of 13 output FNs shown in *B*. With the exception of neuron *xiii*, FNs did not resume activity until after the end of saccades. Apparent activity at saccade end for neuron *xii* is a function of smoothing; the exact timing of its first spike after the saccade-related pause actually occurred after saccade termination. *C*: distribution of the resumed tonic activity at the time of saccade end for all output FNs. Roman numerals *i–xiii* reference the spike density functions in *B*.

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 γ 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 γ 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

studies of reversible SC lesions. SC inactivation produces only modest deficits in saccade metrics and results in decreased velocity, increased latency, and increased variability of saccade endpoint and trajectory (Hikosaka and Wurtz 1985, 1986; Lee et al. 1988; Quaia et al. 1998). If the SC produced the main saccade termination signal, its inactivation would be expected to produce major deficits in saccade metrics.

Role of FN activity in saccade termination

We demonstrated that nearly all output FNs do not resume their activity at the time of saccade end. These results are consistent with the previous observations of Everling et al. (1998) that the majority of nonidentified FNs resume their activity only after the end of saccades. Their study compared the discharges of FNs and OPNs and noted that, despite a similar overall pattern of activity, there were significant differences in the discharge timing of FNs and OPNs. FNs had a gradual reduction and delayed resumption of activity during saccade onset and end, respectively. Conversely, the pause in OPNs activity was better synchronized to saccade onset and activity resumption was time-locked to saccade end. Despite evidence indicating extensive connections between the rostral SC and the OPN region (Büttner-Ennever et al. 1999; Gandhi and Keller 1997; Paré and Guitton 1994), these observations led Everling et al. (1998) to suggest that OPN activity does not simply reflect an excitatory input from FNs. Our results are in agreement with this hypothesis: Output FNs cannot cause the resumption of OPN activity at the time of saccade end.

Although our results validate those of Everling et al. (1998), they remain difficult to reconcile with the hypothesis of Guitton and colleagues that FNs code gaze position error and that this error signal, represented in progressive reactivation of FNs during gaze shifts, contributes to terminating movements (Bergeron and Guitton 2000, 2002; Bergeron et al. 2003; Munoz and Guitton 1991; Munoz et al. 1991). Perhaps the difference in FN activity patterns reflects a difference in the control of saccades versus combined eye–head gaze shifts. Because Guitton and colleagues recorded FNs from head-free cats and monkeys during gaze shifts, the termination signal to end combined eye–head gaze shifts may need to be stronger to counteract the inertia of the head. Accordingly, FNs may be recruited earlier during gaze shifts than during ocular saccades, to provide an additional stop signal. A comparison of FN activity from head-free and head-fixed monkey is required to test this hypothesis.

How are saccades terminated?

Our findings suggest that the temporal activity patterns of SNs and FNs cannot effectively terminate saccades, and alternative mechanisms must be considered. First, it must be clear that our results cannot rule out that saccades are terminated by a change in the spatial distribution of activity across the SC. Munoz and colleagues (Munoz and Wurtz 1995b; Munoz et al. 1991) hypothesized that a rostral spread of activity across the SC controls the actual metrics of the movement. According to this hypothesis, a saccade is terminated when activity reaches the rostral pole and reactivates FNs. In their view, the SC outputs a dynamic motor error signal that is coded spatially. Although highly controversial, no study to date has convincingly 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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