Central Versus Peripheral Determinants of Patterned Spike Activity in Rat Vibrissa Cortex During Whisking

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Fee, Michale S., Partha P. Mitra, and David Kleinfeld. Central versus peripheral determinants of patterned spike activity in rat vibrissa cortex during whisking. *J. Neurophysiol.* 78: 1144–1149, 1997. We report on the relationship between single-unit activity in primary somatosensory vibrissa cortex of rat and the rhythmic movement of vibrissae. Animals were trained to whisk freely in air in search of food. Electromyographic (EMG) recordings from the mystatial pads served as a reference for the position of the vibrissae. A fast, oscillatory component in single-unit spike trains is correlated with vibrissa position within the whisk cycle. The phase of the correlation for different units is broadly distributed. A second, slowly varying component of spike activity correlates with the amplitude of the whisk cycle. For some units, the phase and amplitude correlations were of sufficient strength to allow the position of the whiskers to be accurately predicted from a single spike train. To determine whether the observed patterned spike activity was driven by motion of the vibrissae, as opposed to central pathways, we reversibly blocked the contralateral facial motor nerve during the behavioral task so that the rat whisked only on the ipsilateral side. The ipsilateral EMG served as a reliable reference signal. The fast, oscillatory component of the spike-EMG correlation disappears when the facial motor nerve is blocked. This implies that the position of vibrissae within a cycle is encoded through direct sensory activation. The slowly varying component of the spike-EMG correlation is unaffected by the block. This implies that the amplitude of whisking is likely to be mediated by corollary discharge. Our results suggest that motor cortex does not relay a reference signal to sensory cortex for positional information of the vibrissae during whisking.

INTRODUCTION

The computational process of extracting a stable picture of the world with actively moving sensors is poorly understood. A prevalent hypothesis of sensory-motor integration is that a copy of motor commands, known as corollary discharge, is used by the sensory system to afford a representation of the environment free of the effects of sensor movement (Evarts 1971; McCloskey 1981). An alternate hypothesis is that direct sensory activation from the movement, either by peripheral reafference or proprioception, provides the required motional information. Evidence exists for both of these possibilities in a number of neural systems (Guthrie et al. 1983; Hopkins 1976; Richmond and Wurtz 1980). However, at present there is no evidence that perception is influenced by corollary discharge at the cortical level (Judge et al. 1980). For the case of somatosensory cortex, the anatomic substrate for corollary discharge exists in terms of the massive projection of motor cortical collaterals to primary somatosensory (S1) cortex (Fabri and Burton 1991; Izraeli and Porter 1995; Miyashita et al. 1994; White and Deamicis 1977) as well as central pathways at the level of brain stem nuclei (Erzurumlu and Killackey 1979).

Here we address the question of peripheral versus central sources of input into rat S1 vibrissa cortex. The whisker system is an inherently active sensory system: the rat rhythmically sweeps its vibrissae through the space around its head to find and characterize objects of interest (Vincent 1912; Wineski 1983). Our experimental paradigm involves recording from single units in S1 vibrissa cortex as trained animals whisk freely in air in search of a food tube. We record single-unit spike trains from multiple electrodes along penetrations through vibrissa cortex along with the contralateral and ipsilateral mystatial electromyograms (EMGs) as an index of whisker position; the vibrissae move largely in concert, so that their motion may be adequately described by a single degree of freedom (Carvell et al. 1991).

The EMG has a fast oscillatory component at the whisking frequency and a slow component that corresponds to changes in the amplitude of a whisk cycle. We consider correlations between spike trains in sensory cortex and each of the EMG components. To distinguish between central versus peripheral origins of both fast and slow EMG signals, we ask whether the correlations between whisking and the spike train persist in the absence of contralateral whisker motion. These latter experiments take advantage of the high degree of the coherence of the ipsilateral and contralateral EMG signals (Wineski 1983) and the persistence of whisking in the absence of sensory feedback (Welker 1964).

Preliminary accounts of this work have appeared (Fee et al. 1995; Kleinfeld et al. 1997).

METHODS

Animals

Three female Long-Evans rats (Charles River, ME), initial weight 270–300 gm, were trained (Carvell and Simons 1990; Guic-Robles et al. 1989; Hutson and Masterton 1986) to perch on the edge of a platform while blindfolded as a means to gain access to a food tube through which they received liquid food (0.2 ml per trial; LD-100; PMI Feeds, St. Louis, MO). Each trial was initiated when the rat began to search for the tube, and after ~5 s the tube was placed within reach of the rat. Up to 100 trials were completed before the rat was sated. Once training was completed, a small chamber containing an array of four stereotrodes (McNaughton et al. 1983) was fit over the right vibrissa cortex and

secured to the skull with screws and dental acrylic. The stereotrodes were individually advanced through the dura into cortex with a vacuum insertion technique that prevented damage to the upper layers (Fee and Kleinfeld, unpublished data). Fine multistranded wires were threaded into the left and right mystacial pads to record the EMG. The care and experimental manipulation of our animals were in strict accord with guidelines from the National Institutes of Health (1985) and have been reviewed and approved by the local Institutional Animal Care and Use Committee.

Reversible nerve block

The sensory (trigeminal) and motor (facial) pathway are isolated in separate nerves in the periphery (Dorfl 1982; Wineski 1985). To provide a means to reversibly and rapidly anesthetize the facial nerve in the awake rat by perfusion with lidocaine (2%), the buccolabialis inferior and buccolabialis superior branches were placed together in a nerve cuff in a separate surgical procedure. A 25-gauge needle was threaded subdermally from the top of the skull and inserted into the nerve cuff as a means to deliver lidocaine. Before a set of trials with nerve block, a bolus of 25 μl of lidocaine was injected into the cuff; after ~10 min, whisker motion and the EMG were abolished and did not recover for ≥60 min. Approximately 30 trials were run over a period of 10 min during the complete blockade. After recovery was established by the return of full whisking, additional trials were run.

Recording

Extracellular cortical potentials were recorded and single units were isolated off-line as described previously (Fee et al. 1996a,b). The extramuscular potential from the mystacial pad was rectified and then low-pass filtered (200 Hz) to form the EMG signal (Kamen and Caldwell 1996). Whisker motion was verified by videotaping the animal during behavior. All data analysis was performed on a 4-s segment of data that preceded the end of the whisking epoch during each trial. We used the multitaper methods of Thomson (1982) for spectral estimation and filtering as previously described (Fee et al. 1996b; Prechtl et al. 1997).

RESULTS

Basic cortical response

We first consider whether a strong relationship exists during whisking between vibrissa position, as determined by the contralateral EMG, and the spiking pattern of neurons in S1 vibrissa cortex. An example of the EMG signal recorded during a 4-s epoch of free whisking, together with three simultaneously recorded single units in the infragranular layers of cortex, is shown in Fig. 1*a*. Note the rhythmic activity of the EMG signal near 8 Hz. The peak of the EMG signal corresponds to the most protracted position of the whisker and the valleys correspond to the retracted state. A quantitative relation between the spike trains and the EMG was found from the cross-correlation of the spike arrival times with the times of the peaks of the EMG during the 4-s epochs of whisking during each trial and summing over all trials (Fig. 1*b*).

We observed a correlation between the spike arrival times and the peaks of the EMG for 57% of the single units ($n = 115$). The modulation depth of the correlation varied between units and ranged from 0.05 to 1.3 times baseline (Fig. 1*c*). The phase of the correlation ranged over the entire whisking cycle, with a bias toward a phase just greater than π, i.e., the initial part of protraction. On average, the most strongly modulated spiking occurs in the retracted part of the cycle (Fig. 1*c*).

The above results show that cortical spike timing is correlated with fast changes in vibrissa position within a whisk cycle. We now ask whether spiking is also correlated with the amplitude of the whisks, i.e., the envelope of the EMG at the whisking frequency (*V;* Fig. 2*a, inset*). We extracted the envelope at every sample time[1] and calculated the number of spikes that occurred at each value of the envelope [$s(V)$; Fig. 2*a*] as well as the overall distribution of values of the envelope [$p(V)$; Fig. 2*a*]. For the particular example of Fig. 2, there is a relative enhancement of spike occurrence at large values of the EMG envelope, i.e., at large amplitude of whisking (Fig. 2*b*). Overall, there is a significant correlation between spike occurrence and the value of the EMG envelope for 43% of the single units ($n = 115$) as quantified by the Kolmogorov-Smirnov test;[2] the statistic exceeds the 95% significance level for all data judged significant. Relative enhancement or suppression of the spike rate at large whisking amplitude occurred with essentially equal probability; the magnitude of the peak fractional change in rate ranged from 0.1 to 0.9 and averaged 0.35. Last, there was no obvious relationship between the extent of the fast (Fig. 1) and slow (Fig. 2) modulation among the single units in our sample.

How well can the whisker position, as inferred from the EMG signal, be predicted from the spike train of a single unit? A linear filter that serves this purpose can be derived from the spike train and EMG signals.[3] We focus on the data for *unit 2* (Fig. 1, *a* and *b*); the linear filter is derived from all but one trial in a data set and tested on the data of the omitted trial. When applied to the spike train of the excluded trial, the predicted EMG signal is seen to coincide well with the measured signal (Fig. 1*a*, overlay). The ability to predict the EMG from spike trains implies that the output of some neurons has both a sufficiently high spike rate and sufficiently deep modulation so as to represent whisker posi-

[1] The slowly varying envelope of the EMG signal was determined by demodulation at the whisking frequency (Black 1953). In brief, we *1*) Fourier transformed the EMG signal; *2*) band-pass filtered the peak near positive values of the whisking frequency, e.g., 9 ± 3 Hz (center ± half bandwidth) for the data of Fig. 1*a*, values at negative frequencies discarded; *3*) transformed back to the time domain; and *4*) computed the magnitude of the resultant complex function.

[2] The statistical significance of the difference between $s(V)$ and $p(V)$ was established by the K-S test (Sokal and Rohlf 1981). The probability distribution function for the EMG envelope values is calculated at the times that spikes occurred, $S(V)$, and at all sample times, $P(V)$, where $S(V) \equiv \int_0^V dx s(x) / \int_0^\infty dx s(x)$ and $P(V)$ is similarly defined. The K-S statistic is given by $\sqrt{N}[P(V) - S(V)]$, where N is the number of samples in the distribution for the spike count.

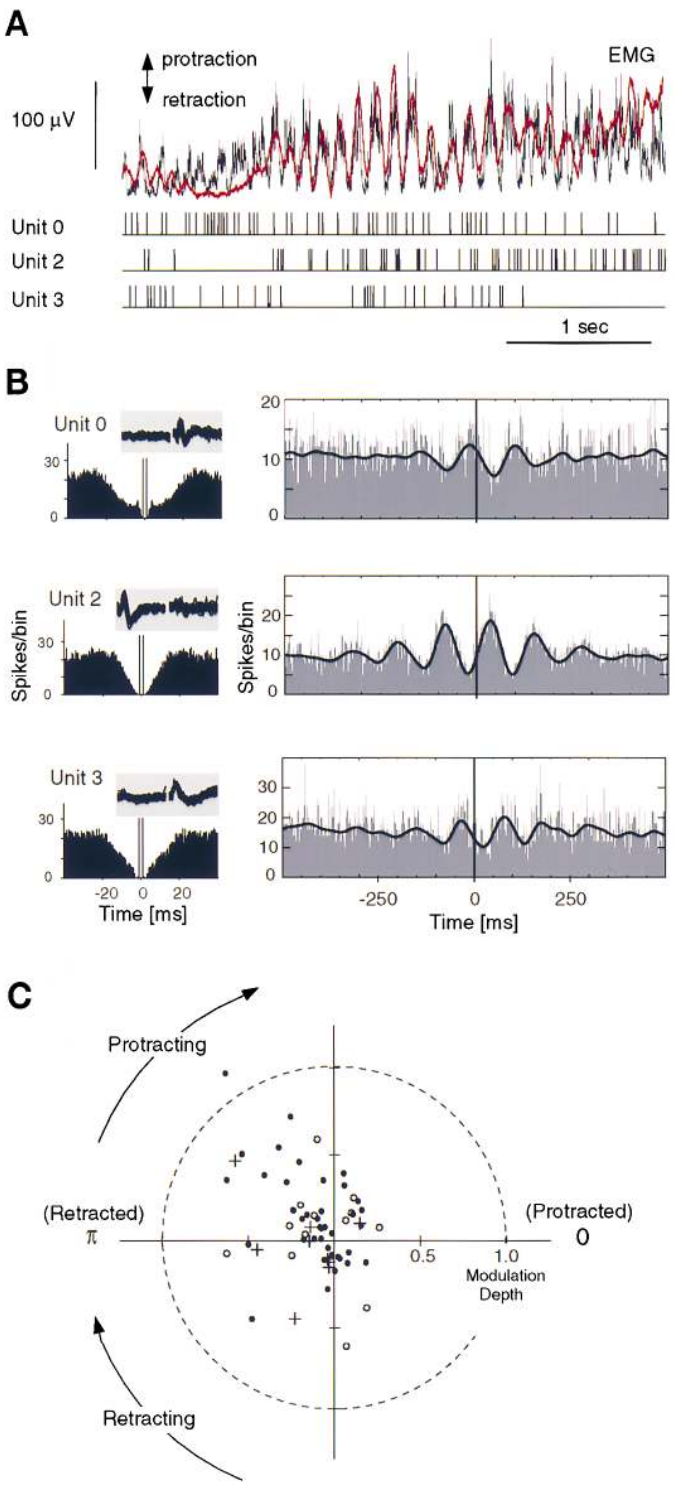
[3] The linear predictor relates the EMG signal predicted from a spike train to the measured spike train (Rieke et al. 1997). We denote $E(f)$, $S(f)$, and $F(f)$ as the Fourier transforms of the EMG signal, the time series that represents the spike train, and the desired filter function, respectively, where $E_{\text{pred}}(f) \equiv F(f)S_{\text{meas}}(f)$. We determine $F(f)$ in the linear least-squares sense by minimizing $\langle |E_{\text{pred}}(f) - E_{\text{meas}}(f)|^2 \rangle_{\text{trial}} = \langle |F(f)S_{\text{meas}}(f) - E_{\text{meas}}(f)|^2 \rangle_{\text{trial}}$. This yields $F(f) = \langle S_{\text{meas}}(f)E^*_{\text{meas}}(f) \rangle_{\text{trial}} / \langle |S_{\text{meas}}(f)|^2 \rangle_{\text{trial}}$. The temporal representation of the EMG predicted from a single spike train is the Fourier transform of $F(f)S_{\text{meas}}(f)$.

tion on a real-time basis. This criterion applied to ~10% of our units across all animals.

Origins of the correlations

We now address the origin of the correlations between the cortical spike trains and the fast and slow signals described above. The free whisking task was split into three sets of trials: the first set was performed with the facial nerves intact, the second set with the contralateral facial motor nerve temporarily blocked and whisking abolished on the contralateral side, and the third set after full recovery. The ipsilateral EMG served as the phase and envelope reference for all trials; the coherence of contra- and ipsilateral sides was observed to be 0.8 at the whisking frequency and >0.9 at the low frequencies of the whisking envelope. As a control, we severed the trigeminal nerve in two additional untrained animals and observed no loss in whisking on the cut side and, further, no loss in bilateral coherence of the whisking. This implies that the whisking motor program operates in the absence of sensory feedback.

We first consider correlations at the whisking frequency. For the example of Fig. 3, we observed a strong modulation of spike output with the ipsilateral (right) EMG during the first set of trials (Fig. 3, Intact), consistent with the fast correlations observed with a contralateral reference (Fig. 1). During the second set of trials, with facial nerve block, we observed no significant correlation between EMG activity and spike arrival times (Fig. 3, Blocked). After recovery from the blockade, the spike activity as well as the modulation depth and phase relation of the correlation with the ipsilateral EMG recovered to their initial value (cf. Fig. 3, Intact and Recovered). In toto, nerve block that abolished motion of the contralateral vibrissae



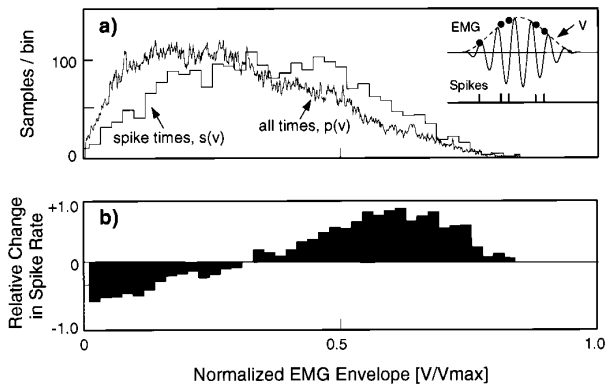


FIG. 2. Relation between amplitude of whisking and spike probability. Amplitude of EMG envelope (V) is sampled at each time point (*a, inset;* dots: amplitudes that coincide with spike). *a*: histogram of number of spikes that occurred at each value of EMG envelope [$s(V)$] and histogram of total number of occurrences at each value [$p(V)$]; scale for $p(V)$ has been compressed. Kolmogorov-Smirnov (K-S) statistic exceeds 95% significance level. *b*: relative change in spike rate as function of EMG amplitude; this function is proportional to $p(V) - s(V)$.

FIG. 1. Relation between phase of whisking cycle and spike arrival times. *a*: electromyographic (EMG) and spike records from 3 simultaneously recorded units during single trial (black). Overlay (red): EMG predicted by filtering spike train for *unit 2* with linear predictor; region of poor prediction at beginning of trial coincides with neuron not firing. *b, left*: stereotrode waveforms and spike autocorrelation functions consistent with single-unit spike trains. *b, right*: cross-correlation function between peaks of EMG signal and spike arrival times; peaks were determined by band-pass filtering data at whisking frequency. Curve: smoothed correlation. Each bin is 1 ms and data are sum over 544 EMG events (21 trials). *c*: scatter plot of modulation depth vs. phase of correlation function for 67 single units that showed significant modulation. Modulation depth is peak-to-trough height of correlation relative to average height. Different symbols: data from different animals.

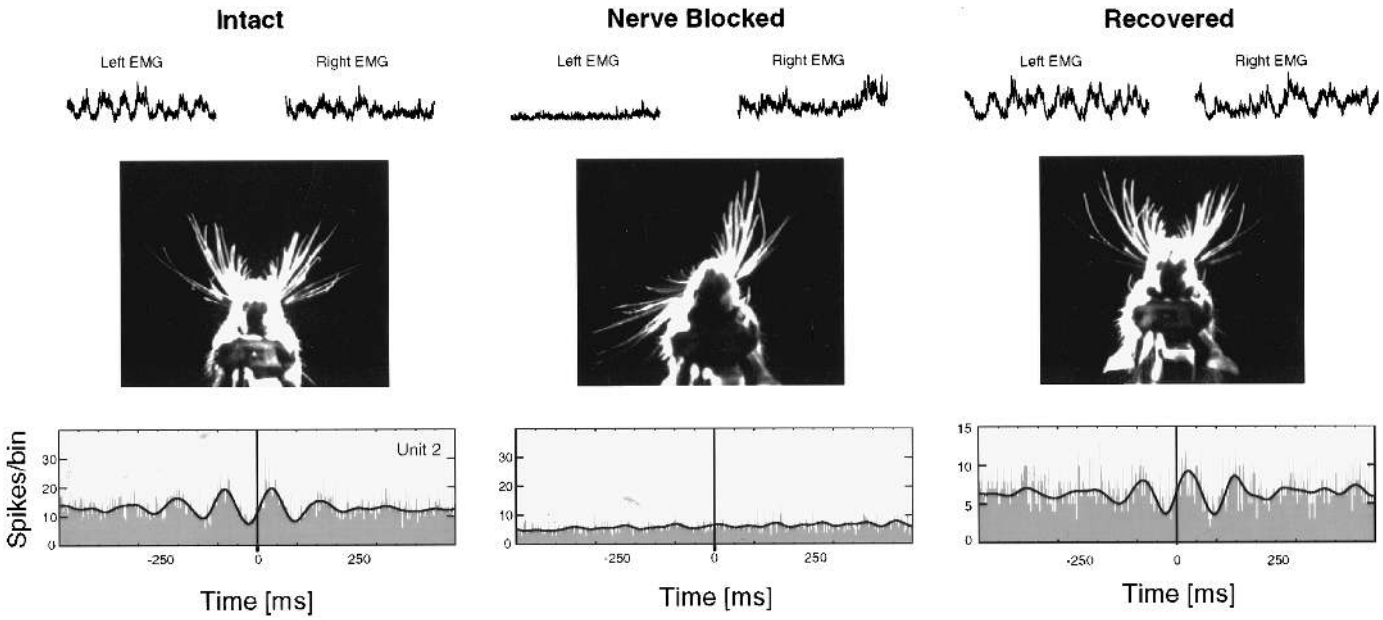


FIG. 3. Effect of blockage of contralateral (left) facial motor nerve on correlation between ipsilateral (right) EMG signal and spike arrival times during whisking in search of food tube. *Top*: 1-s records of EMG signals. *Middle*: video frame of fully protracted vibrissae during whisking. *Bottom*: cross-correlation function. *a*: before paralysis (30 trials): note bilaterally symmetric protraction and strongly modulated spiking. *b*: during paralysis (30 trials): note unilateral protraction and unmodulated spiking; loss of modulation results from lack of coherent spiking. *c*: after recovery from paralysis (10 trials): note return of bilateral protractions and strongly modulated spiking.

led to a loss of correlation between rhythmic EMG activity and spike activity for 85% of those single units ($n = 20$) that showed significant coherence before the block; two weakly modulated units showed no loss and one showed an increase. Critically, all five of the strongly modulated (depth >0.3; e.g., Fig. 1*c*) units showed a complete loss of correlation; the magnitude of the loss in coherence is limited by the background level and ranged between a factor of 7 and 14.

As a control to test whether vibrissa cortex was capable of rhythmic activity during the facial nerve block, a small nozzle was temporarily secured to the skull and was used to passively deflect the whiskers with periodic air puffs at 8 Hz while the nerve was blocked. We observed single-unit responses that were synchronous with the puffs ($n = 10$; data not shown) whose form was similar to that reported previously with awake animals (Chapin et al. 1981; Simons 1978). This shows that S1 cortex is capable of normal sensory responses during the block.

We now consider correlations of the spike train with the envelope of the ipsilateral (right) EMG. We observe that blockage of the facial nerve has essentially no effect on this correlation (Fig. 4). In general, the magnitude of the fractional change in spike rate for units that showed significant correlations during both the intact and blocked states ($n = 7$) was essentially unchanged by the block, although in one case the sign of the correlation changed between the intact and blocked state.

DISCUSSION

We have examined the influence of whisking on the spike activity of single units in S1 vibrissa. The cortical signals we observe are modulated by whisker motion on a cycle-by-cycle basis (Fig. 1) and on the longer time scale of changes in the amplitude of whisking (Fig. 2). The prediction of the EMG from the spike train of some units (Fig. 1*a*) suggests that the output of a single neuron may accurately represent mystacial EMG activity, and, by inference, whisker position.

In the course of this study we observed a second form of activity in two of the three animals during nonexploratory rest periods (unpublished data). This state was characterized by episodes of rhythmic (10-Hz) local field potential (Semba and Komisaruk 1984) activity, synchronous spike activity (Buzsaki 1991), and small-amplitude EMG signals and concomitant whisker motion that were synchronous with

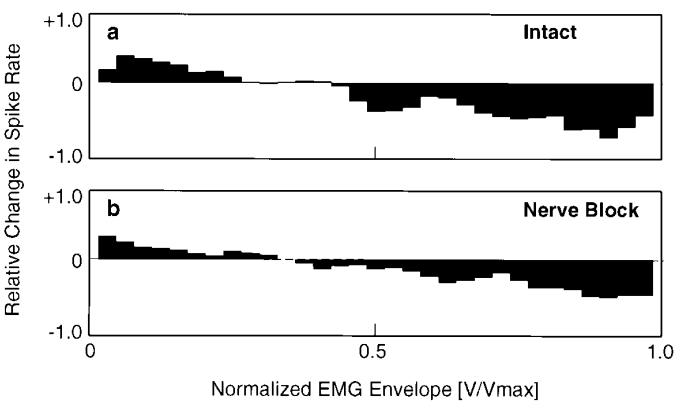


FIG. 4. Relation between amplitude of whisking and spike probability before and during facial nerve block. K-S statistic for both curves exceeds 95% significance level. Change in rate is plotted as function of EMG amplitude (Fig. 2).

neuronal activity. These episodes of large-scale synchronous activity lasted as long as minutes. It has been postulated that such synchrony is requisite to motor planning (Nicolelis et al. 1995). However, we never observed such synchronous activity during our experimental runs, even during the transitions from rest to whisking at the onset of each trial. During epochs of large-scale synchrony all unit activity occurs at the peak of the local field potential and at a fixed phase with respect to the EMG, in sharp contrast to the broad range of phases that we observe during large-amplitude whisking (Fig. 1*c*).

Our data show that the rhythmic pattern of spiking in vibrissa cortex originates from motion of the vibrissa and not from corollary discharge (Fig. 3). It is likely that the motion is transmitted via the vibrissae themselves as opposed to muscle afferents. First, the application of periodic air puffs to drive the whiskers in animals in which the facial nerve was reversibly blocked led to periodic activation of units in S1. Second, anatomic results point to a lack of spindles in the follicle musculature (F. L. Rice, personal communication), which suggests the lack of an anatomic basis for proprioception.

The fast modulation of the spike train was on the order of 1 spike per whisk for the strongly modulated units.[4] An average response of similar magnitude, ~1 spike per cycle, is observed for units in S1 cortex when single vibrissae in awake animals are mechanically moved at frequencies between 5 and 20 Hz (Simons 1978). Further, preliminary data suggest that, on average, units spike only once when animals perform a tactile discrimination task solely with their vibrissae (Fig. 2.11 in Rieke et al. 1997). Thus the fast signal we describe as a possible reference for vibrissa position is of frequency comparable with that of the sensory responses evoked during vibrissal stimulation.

Our results show that corollary discharge may be responsible for modulation of single-unit activity on the relatively long time scale of the envelope of whisking. This discharge may originate in cortical motor areas (Carvell et al. 1996) and may be transmitted via motor-sensory projections. Alternately, the modulation may originate from other central pathways involved in sensory-motor control. Independent of its origin, the role of such slow feedback in sensory processing remains to be understood.

We thank J. K. Chapin and M.A.L. Nicolelus for sharing expertise of recording from awake rats and W. S. Bialek, B. Friedman, and an anonymous referee for comments on a earlier version of the manuscript.

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Received 30 December 1996; accepted in final form 16 May 1997.

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[4] For example, the average number of spikes per protraction for the data of Fig. 1 (*unit 2*) is 1/(544 events) × 10 spike events/bin × 1 bin/ms × 55 ms/protraction ≈ 1 spike/protraction.

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