Effects of Ventrobasal Lesion and Cortical Cooling on Fast Oscillations (>200 Hz) in Rat Somatosensory Cortex

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Staba, Richard J., Barbara Brett-Green, Marcy Paulsen, and Daniel S. Barth. Effects of ventrobasal lesion and cortical cooling on fast oscillations (>200 Hz) in rat somatosensory cortex. J Neurophysiol 89: 2380–2388, 2003. First published January 15, 2003; 10.1152/jn.01098.2002. High-frequency oscillatory activity (>200 Hz) termed “fast oscillations” (FO) have been recorded in the rodent somatosensory cortex and may reflect very rapid integration of vibrissal information in sensory cortex. Yet, while electrophysiological correlates suggest that FO is generated within intracortical networks, contributions of subcortical structures along the trigeminal pathway remain uncertain. Using surface and laminar electrode arrays, in vivo recordings of vibrissal and electrically evoked FO were made within somatosensory cortex of anesthetized rodents before and after ablation of the ventrobasal thalamus (VB) or during reversible cortical cooling. In VB-lesioned animals, vibrissal stimulation failed to evoke FO, while epicortical stimulation in lesioned animals remained effective in generating FO. In nonlesioned animals, cortical cooling eliminated vibrissal-evoked FO despite the persistence of thalamocortical input. Vibrissal-evoked FO returned with the return to physiological temperatures. Results from this study indicate that somatosensory cortex alone is able to initiate and sustain FO. Moreover, these data suggest that cortical network interactions are solely responsible for the generation of FO, while synchronized thalamocortical input serves as the afferent trigger.

INTRODUCTION

The detection of high-frequency oscillations, termed “sigma-bursts,” within the human somatosensory evoked potential (SEP) complex during median nerve stimulation has drawn the attention of researchers concerned with how these brief bursts of highly synchronized activity may relate to sensory processing (Curio 2000). Studies have shown sigma-bursts are sensitive to changes in stimulus rate (Klostermann et al. 1999) as well as to changes associated with states of vigilance or awareness, i.e., during sleep/wake states (Gobbele et al. 2000; Hashimoto et al. 1996; Yamada et al. 1988). Similar to the human sigma-burst, recent studies in the nonprimate somatosensory cortex have identified high-frequency oscillatory activity, termed “fast oscillations” (FO, >200 Hz), associated with large amplitude SEP (>1 mV) evoked by transient stimulation of the rodent’s vibrissa (Jones and Barth 1999, 2002; Jones et al. 2000). Current evidence indicates that smaller amplitude (approximately 100 μV) vibrissal-evoked FO are modality specific, coincide with the short-latency components of the slow-wave SEP, discharge with largest amplitude over the cortical region corresponding to the vibrissa stimulated and rapidly propagate to adjacent barrels, and are associated with an increase in burst firing of fast-spiking cortical neurons (Jones and Barth 1999; Jones et al. 2000). Based on these data, it has been proposed that the discharge of FO may reflect the timing of stimulus onset and rapid spreading of this information within the vibrissa/barrel field (Barth 2002; Jones and Barth 1999; Jones et al. 2000). However, questions remain about the cortical generation of FO and whether high-frequency repetitive afferent input from subcortical structures may initiate and/or sustain this cortically recorded phenomena.

Studies of lower frequency oscillations, such as sleep spindles, have found that their generation derives from interactions between thalamic nucleus reticularis and thalamocortical cells (Bal et al. 1995; Steriade et al. 1993; von Krosigk et al. 1993). Data on the higher frequency sigma-burst in humans (Gobbele et al. 1998, 1999), and more recently in piglets (Ikeda et al. 2002), suggest that one source of the sigma-burst resides within the thalamus. Furthermore, in vitro studies have observed that thalamic neurons are capable of intraburst firing frequencies > 200 Hz (Bal et al. 1995; Deschenes et al. 1984; Jahnson and Llinas 1984; Kim and McCormick 1998), suggesting that thalamic burst discharge may be associated with the generation of somatosensory FO. In contrast, laminar analysis and cross-correlation of unit responses with local field potentials suggest that the generation of FO derives from intracortical network interactions (Jones et al. 2000; Kandel and Buzsaki 1997). These data are consistent with the observation that FO can occur spontaneously in isolated slabs of cat neocortex (Grenier et al. 2001).

In view of the current evidence suggesting thalamic and/or cortical sites of FO generation, we sought to extend the characterization of FO in relation to its site of generation through the use of in vivo surface and laminar electrode array recordings to determine whether the ventrobasal nucleus of the thalamus (VB), the major relay nucleus to somatosensory cortex, is required for the generation of FO, and whether inactivation of cortical cellular activity eliminates the occurrence of FO. To this end, mechanical vibrissal and epicortical electrical stimulation was used to evoke FO before and after electrolytic lesion of the VB and during reversible cortical cooling in ketamine-
anesthetized rodents. Measurements of evoked potential amplitude and spectral power were used to quantitatively evaluate the presence or absence of FO under lesion and cortical cooling conditions.

METHODS

Surgical procedures

All procedures were conducted within University of Colorado Institutional Animal Care and Use Committee guidelines. Adult male Sprague–Dawley rats (300–400 g, n = 10) were anesthetized using subcutaneous injections of a mixture of ketamine HCl (64.2 mg/kg), xylazine (12.8 mg/kg), and acetylpromazine (2.1 mg/kg). Animals were placed in a stereotaxic frame and body temperature was maintained with a regulated heating pad. A unilateral craniotomy was performed over the right hemisphere extending from bregma to lambda and from the midsagittal suture to the lateral aspect of the temporal bone, exposing a large area of the parietotemporal cortex. The dura was reflected and saline was applied regularly to the exposed cortex. At the conclusion of the recordings, animals were killed with an overdose of anesthesia without regaining consciousness.

Recording and signal processing

During the recording sessions, animals were placed on a regulated heating pad and anesthetic levels were maintained such that the eye-blink reflex could barely be elicited. Surface recordings were performed with a 64-contact electrode array (silver wire electrodes, 100 µm diam., interelectrode spacing 500 µm) arranged in a 8 × 8 grid covering 3.5 mm² of parietotemporal cortex. During stimulation of the major vibrissae located on the contralateral mystacial pad, the surface recorded SEP was used to align the array over the postero-medial barrel subfield or vibrissa/barrel field (Fig. 1A). Laminar recordings were made using an electrode consisting of a linear array of 23 platinum contacts (array diameter 0.5 mm, interelectrode spacing 100 µm; EEG KFT, Budapest, Hungary), inserted perpendicular to the cortical surface after removal of the surface array, at the approximate location of the maximum amplitude surface-recorded response. Surface and laminar recordings were referenced to a silver ball placed over the contralateral frontal bone. Evoked potentials were recorded with wideband analog filters (1–3,000 Hz) and digitized at 10 kHz. Digital band-pass filtering of wideband evoked potentials (1–2,000 Hz) and FO (200–500 Hz) was subsequently performed using a second-order Butterworth filter.

Stimulation, lesioning, and cortical cooling

Vibrissae were stimulated using a laboratory-built apparatus previously described (Jones and Barth 1999). Vibrissae from the left mystacial pad were tied together, clipped to 2 cm, and collectively applied to the contralateral mystacial pad. Proper placement of the array was based on the anatomical location of the major vibrissae located on the contralateral mystacial pad (Fig. 1A). Proper placement of the array was based on the anatomical location of the major vibrissae located on the contralateral mystacial pad used in Fig. 1A. During cortical stimulation, a bipolar stainless steel electrode (100 µm diam., 0.5 mm intertip distance) was positioned stereotaxically within the ventrobasal nucleus of the thalamus (AP: 3.6, ML: 2.7, DV: 6.0) (Paxinos and Watson 1986). Biphasic square-wave current pulses (50 to 80 mA, 0.1 ms duration, 0.5 s interevent interval) were delivered to electrically elicit an evoked response (Fig. 2A). During cortical stimulation, biphasic current pulses (1.0 mA, 0.1 ms duration, 0.5 s interevent interval) were delivered through a bipolar stainless steel electrode placed on the cortical surface anterior to the vibrissa/barrel field to generate a direct cortical response (Fig. 2C).

Electrolytic lesion of the VB was made using constant current (30–50 µA for 10 s; Ugo Basile, Comerio-Varese, Italy). Functional confirmation of VB lesion was determined by the absence of surface recorded evoked potential during vibrissal stimulation. At the end of the electrophysiological recordings, histological analysis of VB lesion was performed in three of four animals to determine the size and extent of lesion. Animals were deeply anesthetized and perfused with 0.1 M phosphate buffer (50–100 ml; room temperature). This was followed with a 6% paraformaldehyde buffered fixative (500 ml; 4–10°C) and 10% sucrose in 0.1 M phosphate buffer (4–10°C). Brains were removed, cryoprotected in 30% sucrose in 0.1 M phosphate buffer (4–10°C), sectioned coronally at 40 µm on a cryostat, and mounted on gelatinized slides. Sections were stained with cresyl violet, cover-slipped, and examined using a microscope under bright-field conditions. The location and extent of lesion was transposed onto coronal plates derived from a stereotaxic rodent brain atlas (Swanson 1992).

A laboratory-built aluminum cooling plate (9.0 × 5.0 mm) was lowered onto the cortical surface and aligned such that the centrally located access hole within the plate (3.0 mm diam.), through which the laminar electrode was inserted, was positioned over the vibrissa/barrel field. A thermocouple device attached to the plate monitored plate temperature and controlled a variable-speed pump that circulated ethanol through surgical tubing that connected the plate with a cooling column containing dry ice. During cortical cooling, the plate temperature was lowered to approximately 5–10°C, as measured by the thermocouple device located 3 mm away from the laminar electrode, until the SEP could no longer be evoked during vibrissal stimulation.

Data analysis

Power spectra were calculated on wideband-recorded evoked potentials using a 256-point fast Fourier transform (Bartlett window, 128 point overlap). Amplitude of evoked potential slow wave was measured between maximum of P1 and minimum of N1 components. FO parameters (peak frequency and duration) were calculated from a subset of FO each having an amplitude (sum of rectified band-pass signal) that exceeded the grand mean amplitude derived from the entire 64- or 23-contact array. FO peak frequency was determined by the primary peak in power located between 200 and 500 Hz of the power spectra. FO duration was measured as the total time during which the amplitude of the rectified band-pass signal exceeded the mean amplitude of the signal calculated over 100 ms. Evoked potential P1-N1 power was calculated as the sum of power between 1 and 40 Hz. FO power was calculated as the sum of power between 200 and 500 Hz. Comparisons of mean P1-N1 amplitude and mean FO power, frequency, and duration before and after lesion of the VB were made using paired t-tests that were corrected for multiple comparisons. Comparisons of P1-N1 power and FO power before, during, and following cortical cooling were performed using a repeated-measures ANOVA. Posthoc analyses were carried out using the Bonferroni t-test. Significance was set at P ≤ 0.05.

RESULTS

Wide bandwidth field potential recordings were obtained using a 64-contact surface electrode array positioned over the posteromedial barrel subfield or vibrissa/barrel field of the rodent somatosensory cortex (Fig. 1A). The template of the vibrissa/barrel field used in Fig. 1A was derived from a previous study using cytochrome oxidase histology (Jones and Barth 1997) and is used here for illustrative purposes only to show the approximate position of the surface array in relation to the rodent barrel field. Proper placement of the array was based on the vibrissa-evoked surface recording of the SEP (Fig. 1B). Simultaneous displacement of the major vibrissae located on the contralateral mystacial pad generated a SEP consisting of an initial large amplitude slow wave, labeled P1 and N1 to indi-
Figure 1: Alignment of surface array and recording of sensory evoked potentials. 

A: approximate position of the surface electrode array in relation to the right posterior medial barrel subfield or vibrissa/barrel field of the rat somatosensory cortex. Black dots indicate individual electrode contacts in the 8 x 8 array, while the surface representation of the rodent vibrissa/barrel field was derived from previous study using cytochrome oxidase histology (Jones and Barth 1997) and used here for illustrative purposes only. B: representative example of averaged (n = 32) somatosensory evoked potential (SEP) recorded from each contact of the 64-contact array during stimulation of the contralateral vibrissa from one animal. Wideband (1–2,000 Hz) traces, illustrating biphasic slow wave potential, were band-pass filtered (200–500 Hz), revealing small amplitude fast oscillation (FO). Each trace represents 50 ms of the SEP recorded from stimulus onset. C: wideband SEP, taken from traces within the box of B, shown at higher magnification. Note the inflections associated with the P1-N1 complex align with the high-frequency FO (dashed lines). Power spectra (inset) of the SEP reveals peak in power at 312 Hz reflecting FO activity. Scale bars = 400 μV for P1-N1 complex and 40 μV for FO, negative–up.

cate polarity and sequence of occurrence of the slow wave components (Fig. 1C). Closer inspection of the wideband recorded SEP indicated the presence of a high-frequency oscillatory component associated with the P1-N1 complex (Fig. 1C). Power spectral analysis of the SEP showed a peak in power centered at approximately 300 Hz (Fig. 1C, inset), while the band-pass filtered trace, shown superimposed on the SEP, more clearly revealed the high-frequency FO (Fig. 1C). Across all animals used in this study, physiological stimulation consistently evoked FO that were associated with the P1-N1 complex of the SEP. Analysis of surface-recorded FO revealed a frequency of 288 ± 23 Hz (mean ± SE) and duration of 20.0 ± 1.0 ms.

Single-pulse stimulation of the VB generated an evoked response within the vibrissa/barrel field that approximated the response elicited during physiological stimulation (Fig. 2A). Similar to the SEP recorded during vibrissal stimulation, the evoked potential recorded during VB stimulation had a comparable spatial distribution and stereotypical waveform morphology that included FO associated with the P1-N1 complex (Fig. 2B). In the example shown in Fig. 2B, power spectral analysis of the wideband-recorded evoked potential again revealed a peak in power between 300 and 400 Hz corresponding to the FO. FO had a mean frequency of 271 ± 12 Hz and duration of 19.3 ± 1.0 ms, comparable to the frequency and duration of vibrissal-evoked FO.

The direct cortical response demonstrated a similar spatial organization and waveform morphology as the evoked SEP during vibrissal and VB stimulation but was concentrated more rostrally in the vibrissa/barrel field due to the location of the stimulating electrode (Fig. 2C). Power spectral analysis of the evoked potential recorded during epicortical stimulation indicated a peak in power centered on 300 Hz (Fig. 2D). Overall, the mean frequency of FO produced by epicortical stimulation was 301 ± 14 Hz and duration was 20.8 ± 0.9 ms, characteristics that were similar to vibrissal-evoked FO (frequency: t = −0.37, df = 3, P = 0.7; duration: t = −1.29, df = 3, P = 0.3).

To determine the possible role of VB input for the generation of FO, we examined both vibrissal and electrically evoked potentials before and after VB ablation. Postlesion results from three animals, shown on coronal templates of the rodent brain in Fig. 3B, revealed that the majority of damage was within the area of the VB (ventral posteromedial and ventral posterolateral nuclei), although portions of the lesion were found to extend into the ventral anterior-lateral complex, posterior complex, and lateral posterior nucleus. As previously illustrated in Figs. 1 and 2, before the VB lesion, both vibrissal (Fig. 3A) and epicortical stimulation (Fig. 3D) generated an evoked potential comprised of the slow wave P1-N1 complex and FO. However, after the lesion, vibrissal stimulation predictably failed to evoke any response in somatosensory cortex (Fig. 3C). In contrast, postlesion epicortical stimulation had no effect on the slow or fast components of the evoked response (Fig. 3E). Analysis of FO after lesion revealed a frequency of 303 ± 19 Hz, similar to that observed prelesion (before lesion, 20.8 ± 0.9 ms; t = 4.89, df = 3, P = 0.01).

Despite differences in the size and extent of lesions across animals (Fig. 3B), surface recordings confirmed that, after

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lesioning, vibrissal-evoked FO was abolished in all animals, while epicortical stimulation remained equally effective in generating FO. Figure 4 summarizes these results obtained from four animals. During vibrissal stimulation, the mean (± SE) P1-N1 amplitude was significantly smaller after the lesion compared with the amplitude before the lesion (Fig. 4A; 0.08 ± 0.01 versus 2.65 ± 0.34 mV; t = 13.51, df = 3, P = 0.0008). In contrast, during epicortical stimulation, no difference was observed in P1-N1 amplitude between postlesion and prelesion conditions (2.97 ± 0.22 versus 2.79 ± 0.09 mV, P = 0.5). Similar to the differences observed in P1-N1 amplitude, during vibrissal stimulation, FO power was significantly reduced after lesioning compared with FO power before lesioning (Fig. 4B). Normalizing FO power to the “before lesion” condition, during vibrissal stimulation, FO power declined 98% after lesioning compared with FO power before lesioning (0.02 ± 0.01 versus 1.0 ± 0.19; t = 7.39, df = 3, P = 0.005). During epicortical stimulation, no difference was observed in FO power between post- and prelesion conditions (0.93 ± 0.11 versus 0.82 ± 0.10, P = 0.3).

While electrically evoked FO before and after VB lesion were not significantly different, the VB could still have an influence on FO initiation when intact. To explore this possibility, we examined the effects of cortical cooling on the generation of FO. During vibrissal stimulation and prior to cooling, the familiar P1-N1 components of the SEP were observed in wideband traces recorded from the 23-contact laminar electrode that was oriented perpendicular to the cortical surface (Fig. 5A, left column). In the deeper cortical layers, the P1-N1 complex reversed in polarity (Fig. 5A, between trace 4 and 5) and decreased in amplitude. Similar to the surface recorded SEP, high-frequency FO was associated with the P1-N1 complex throughout the cortical depth. The band-pass-filtered traces in Fig. 5B more clearly show FO activity. Spectral analysis of the evoked response across all contacts revealed a peak in power centered at 280 Hz of the averaged power spectra that corresponded to the FO activity observed across the cortical layers (Fig. 5C).

Lowering of cooling plate temperature was associated with an increase in latency and decrease in amplitude of vibrissal-evoked FO. While electrically evoked FO before and after VB lesion were not significantly different, the VB could still have an influence on FO initiation when intact. To explore this possibility, we examined the effects of cortical cooling on the generation of FO. During vibrissal stimulation and prior to cooling, the familiar P1-N1 components of the SEP were observed in wideband traces recorded from the 23-contact laminar electrode that was oriented perpendicular to the cortical surface (Fig. 5A, left column). In the deeper cortical layers, the P1-N1 complex reversed in polarity (Fig. 5A, between trace 4 and 5) and decreased in amplitude. Similar to the surface recorded SEP, high-frequency FO was associated with the P1-N1 complex throughout the cortical depth. The band-pass-filtered traces in Fig. 5B more clearly show FO activity. Spectral analysis of the evoked response across all contacts revealed a peak in power centered at 280 Hz of the averaged power spectra that corresponded to the FO activity observed across the cortical layers (Fig. 5C).

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**FIG. 2.** Evoked potentials during thalamic or cortical stimulation. A: averaged (n = 32) evoked potential recorded from surface array placed over rat vibrissa/barrel field during electrical stimulation of the ventrobasal thalamus (VB). B: evoked potential, taken from traces within box of A, shown at higher magnification illustrating P1-N1 complex and FO. To the right of the traces is the power spectra of the evoked potential revealing a peak in power at approximately 320 Hz corresponding to FO activity. C: averaged evoked potential during cortical stimulation. Stimulating electrode was positioned anterior to the rat vibrissa/barrel field (right side between 4th and 5th row of surface array). D: evoked potential (taken from traces within box of C) showing P1-N1 complex and FO. Note the similarity in spatial distribution of evoked potential and presence of FO during VB and epicortical stimulation. Evoked potentials shown in this figure were recorded from the same rat as shown in Fig. 1.
evoked SEP. Only when plate temperature fell below 10°C did vibrissal stimulation fail to generate FO within the vibrissa/barrel field (Fig. 5, A and B, middle column). Power spectral analysis showed that, during cortical cooling to <10°C, power across all frequencies was greatly reduced, including power within the FO frequency band (Fig. 5C). However, during cooling, a small amplitude response beginning approximately 6–8 ms after vibrissal stimulation was observed in the wide-band traces (Fig. 5A, middle column). Figure 5B shows the band-pass–filtered representation of this short latency potential (see also Fig. 5A, inset). The stability of this initial response throughout the cooling process suggested that this nonrhythmic potential most likely reflected the arrival of the thalamic volley during vibrissal stimulation. Within 30 min following the end of cortical cooling, a full recovery of FO was observed within somatosensory cortex (Fig. 5, A–C, right columns). Both the amplitude and spatial distribution of the FO indicate that surface cooling had a reversible, suppressive effect on cortical cellular activity. Figure 6 summarizes the results of the cortical cooling experiments conducted on six animals. Given the
P1-N1 complex reverses in polarity across cortical layers, which makes it difficult to accurately measure the peak amplitude of both the P1 and N1 components, we quantified the power (1–40 Hz) that corresponded with the P1-N1 complex, as well as FO power (200–500 Hz), before, during, and following cortical cooling. Normalizing P1-N1 power to the “before” condition, during vibrissal stimulation, P1-N1 power was significantly reduced during cooling compared with P1-N1 power before cooling and following cooling (Fig. 6A; \(F(2,12) = 70.6, P < 0.0001\)). During cooling, P1-N1 power declined >99% compared with P1-N1 power during both before and following cooling (0.003 ± 0.001 vs. 1.00 ± 0.29 and 1.11 ± 0.31, both \(P < 0.0001\)). No difference was observed in P1-N1 power before between and following cooling conditions (\(P = 0.8\)). Similar to the differences observed in P1-N1 power, FO power was significantly reduced during cortical cooling (Fig. 6B; \(F(2,12) = 110.6, P < 0.0001\)). During cooling, FO power declined 99% compared with FO power before cooling (0.009 ± 0.001 vs. 1.00 ± 0.43, \(P < 0.0001\)) and declined 93% compared with FO power following cooling (0.009 ± 0.001 vs. 0.94 ± 0.22, \(P < 0.0001\)). No difference was observed in FO power between before and following cooling conditions (\(P = 0.5\)).

**Discussion**

There are three results of this study. First, physiological stimulation and electrical stimulation applied to either the VB nucleus of the thalamus or directly to somatosensory cortex evoke a cortical P1-N1 complex with superimposed FO that are comparable in frequency and amplitude across conditions. Second, complete ablation of the VB nucleus, sufficient to eliminate all physiologically evoked activity in the vibrissa/barrel field, has no significant effect on responses evoked by direct cortical stimulation, indicating that the cortex is capable of producing both slow wave and FO independent of thalamic influence. Finally, cortical cooling effectively blocks the physiologically evoked response, leaving only a brief, nonrhythmic, thalamocortical volley that precedes the P1-N1 and FO, further suggesting that the neural generators for these responses are exclusively cortical and that thalamic input serves only as an afferent trigger.

Rapid, transient displacement of multiple facial vibrissae repeatedly elicits a SEP possessing waveform characteristics similar to those previously described in surface recordings within the rodent vibrissa/barrel field (Di and Barth 1991; Jones and Barth 1999). The large amplitude slow-wave P1-N1 complex is accompanied by a smaller amplitude high-frequency FO that is apparent in the band-pass–filtered trace of the SEP, producing a peak in spectral power at approximately 300 Hz that is consistent with findings of Jones and colleagues (1999, 2000). The possibility that cortical FO may result from high-frequency vibration of the stimulating apparatus or vibrissa-sae may be ruled out for several reasons. It has been demonstrated that cortical FO may be evoked with vibrissa stimulation using a piezoelectric translator that delivers a rapid displacement of the vibrissae in a dorsal–ventral direction (10 μm at 5 mm/s) with negligible afteroscillations, as verified by an infrared emitter-detector photodiode pair (Barth 2002; Jones and Barth 2002). In addition, recordings performed directly from the infraorbital nerve during transient vibrissal displacement indicate that only a single compound action potential is produced, with no repetitive discharges (Barth 2002). This conclusion is further supported by the present observation that stimulation of the VB with single current pulses is equally effective in eliciting cortical FO. FO evoked with VB stimulation are of similar frequency to physiologically evoked FO but are of earlier poststimulus latency, probably due in large part to elimination of transmission delays imposed by the peripheral pathway. Finally, epicortical stimulation reliably evokes FO within rodent vibrissa/barrel field that is similar to, and spatially overlaps, physiologically evoked FO. Previous studies using epicortical stimulation have demonstrated that it is an effective method to activate cortical neuronal circuits and study physiological, as well as pathological, evoked responses (Barth and Di 1991; Barth et al. 1989; Harding 1992; Lesnick et al. 1986; Momma et al. 1988). In the present study, epicortical stimulation provides an essential means for evaluating FO before and after thalamic ablation.

Surface recordings reveal that vibrissal-evoked FO is abolished after VB ablation. Given that the VB is the major thalamic relay nucleus mediating whisker information, our results are consistent with a complete blockade of the afferent pathway to the vibrissa/barrel field. However, in the absence of an intact and functional VB, epicortical stimulation remains effective in eliciting FO, suggesting that rhythmic thalamocortical input is not required for cortical FO generation. The presence of electrically evoked FO in the surface recordings indicate that activation of barrel circuitry can be achieved through antidromic and orthodromic activation of intracortical neuronal networks. These data are consistent with the findings of Morin and Steriade (1981) demonstrating that stimulation of the white matter underlying somatosensory cortex in the VB-lesioned cat elicits a series of “fast” postsynaptic potentials, which are believed to reflect activation of local neocortical neuronal networks.

Our results suggest that somatosensory cortex is capable of...
producing FO in the absence of thalamic input, a conclusion that is supported by the recent observations that spontaneous FO persist in the isolated cortical slab (Grenier et al. 2001). Yet, they do not rule out possible contributions of fast rhythmic thalamocortical input to FO generation in the intact preparation. What does cast doubt on putative thalamic participation are results from thalamic stimulation and from cortical cooling. Similar to FO evoked by VB stimulation in the present study, previous studies report the occurrence of high-frequency oscillations in somatosensory cortex during transient thalamic stimulation. Kandel and Buzsaki (1997) describe the presence of fast “ripple” oscillations in somatosensory cortex using single-pulse stimulation in the rodent ventral posterolateral thalamus. Similarly, a single shock within the VB evokes a series of small amplitude potentials associated with the primary slow wave of the surface-recorded somatosensory evoked response in the cat (Morin and Steriade 1981). The first of these potentials is thought to reflect the presynaptic thalamocortical volley, while subsequent potentials are believed to reflect the postsynaptic activation.

Yet, in none of these studies was the effect of transient thalamic stimulation monitored within the thalamus. It is possible that transient stimulation could evoke fast rhythmic thalamic discharge that, when imposed on the cortex, could drive or sustain FO in the vibrissa/barrel field. However, results from cortical cooling suggest that physiological stimulation does not produce fast rhythmic thalamic input to the cortex. Reversible cortical cooling dissociates thalamocortical activity with FO genesis and effectively eliminates vibrissal-evoked FO across the cortical lamina (Fig. 5A). Previous studies using cooling pulses applied to the cortical surface demonstrate that, as the cooling wave traverses cortical lamina, the principal components of the SEP are suppressed in an ordered sequence, reflecting the inactivation of supragranular activity followed by infragranular activity (Kublik et al. 2001). Strong cooling pulses eliminate almost all of the vibrissal-evoked SEP with the exception of a short-latency, small-amplitude potential thought to reflect the incoming thalamocortical volley. In agreement with these results, in the present study, cooling plate temperatures below 10°C eliminate vibrissal-evoked FO and
leave only a small-amplitude early latency response (Fig. 5B). While cooling has been shown to alter the temporal properties of synaptic activity (Volgushev et al. 2000), the latency and amplitude of the initial evoked potential remains stable throughout the cooling process. The absence of a polarity reversal in the depth (Fig. 5B) may in part reflect volume-conducted currents generated subcortically (Givre et al. 1994; Morin and Steriade 1981; Schroeder et al. 1990). However, the amplitude was highest in the middle cortical layers and did not increase in the cortical depth, suggesting sources within middle cortical layers may be oriented parallel to the cortical surface and also contributing to this early latency potential.

These data, and the recent evidence identifying a presynaptic component to the high-frequency oscillation recorded in the piglet somatosensory cortex (Ikeda et al. 2002), suggest that short latency potentials reflect the coincident activity of thalamocortical projections. The apparent absence of a series of FO-frequency potentials during cortical cooling, which might be interpreted to reflect sustained rhythmic thalamocortical activity, argues against the hypothesis that high-frequency activity within subcortical structures drives cortical FO. Instead, these data suggest that synchronized thalamocortical input provides an impulse that activates cortical network interactions exclusively responsible for the generation of FO within the rodent somatosensory cortex. While the nature of these interactions are unresolved, evidence indicates that the genesis of FO may involve the discharge of local cortical neuronal networks (Barth 2002; Jones and Barth 2002; Jones et al. 2000), synchronized through electronic coupling, i.e., gap junctions, which are believed to be present between cortical neurons (Galarreta and Hestrin 1999; Gutnick and Prince 1981). Similar mechanisms have been proposed to be involved in the generation of the sharp wave–associated high-frequency ripple oscillation observed in the hippocampus (Buzsaki et al. 1992; Draguhn et al. 1998; Schmitz et al. 2001; Traub et al. 1994, 1999; Ylinen et al. 1995).

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