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Sensorimotor integration in human primary and secondary somatosensory cortices

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Abstract

We measured somatosensory evoked fields (SEFs) to electric median nerve stimuli from eight healthy subjects with a whole-scalp 122-channel neuromagnetometer in two different conditions: (i) 'rest', with stimuli producing clear tactile sensation without any motor movement, and (ii) 'contraction' with exactly the same stimuli as in 'rest', but with the subjects maintaining sub-maximal isometric contraction in thenar muscles of the stimulated hand. The aim was to study the role of the primary (SI) and secondary somatosensory (SII) cortices in sensorimotor integration. The amplitude of the SI response N20m did not change with coincident isometric contraction, whereas P35m was significantly reduced. On the contrary, activation of contra- and ipsilateral SII cortices was significantly enhanced during the contraction. We suggest that isometric contraction facilitates activation of SII cortices to tactile stimuli, possibly by decreasing inhibition from the SI cortex. The enhanced SII activation may be related to tuning of SII neurons towards relevant tactile input arising from the region of the body where the muscle activation occurs. © 1998 Elsevier Science B.V.

Keywords: Magnetoencephalography; Somatosensory cortex; Interaction; Muscle contraction

1. Introduction

Somatosensory and motor functions are tightly bound together: the primary motor (MI) cortex receives somatosensory input directly via thalamus and indirectly from various somatosensory cortical areas, and conversely, the primary somatosensory (SI) cortex forms part of descending pyramidal tract to muscles. Accurate function of both systems and integration of the sensorimotor information are required to perform precise and purposeful movements.

Motor activity may affect cortical responsiveness to coinciding tactile input from the same region of the body. Somatosensory gating studies have indicated either attenuation or facilitation of different SI responses during active exploratory finger movements [5,19], but the role of SII cortices in sensorimotor integration have remained obscure.

To further elucidate this issue we employed a whole-scalp 122-channel neuromagnetometer to study the effect of isometric contraction on SI and SII cortices during electric median nerve stimuli.

2. Materials and methods

Somatosensory evoked fields (SEFs) were recorded from eight healthy subjects (five males, three females; age range 30–45 years; seven right-handed) to 0.3 ms constant current pulses delivered to left median nerve (MN) at the wrist. During the recording, the subject was sitting comfortably in a magnetically shielded room with the head supported against the helmet-shaped sensor array of the magnetometer. All subjects were experienced in SEF measurements, and they were instructed to ignore the stimuli by reading a self-chosen book. The interstimulus interval (ISI) was constantly 3 s. SEFs were first recorded to 'strong' stimuli clearly exceeding the motor threshold (mean intensity 6 mA); the stimuli elicited cortical responses with excellent signal-to-noise ratio. After this, SEFs were recorded during two different conditions: (i) 'rest', with the stimuli (mean 2 mA) producing clear tactile sensation without any motor movement, and (ii) 'contraction' with exactly the same stimuli as in 'rest', but the subjects were asked to maintain sub-maximal isometric

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contraction in left thenar muscles without changing the position of the hand. Care was taken to avoid any changes in the location and contact of the stimulator with the skin. The stimulated hand was monitored throughout the measurements with video camera. The order of different sessions was randomized over subjects.

Although electric stimuli activate different afferent fiber types, they were applied because they produce highly synchronized action potential volleys, resulting in excellent signal-to-noise ratio of the cortical responses. With the low intensities used, mainly large diameter cutaneous afferents were probably activated.

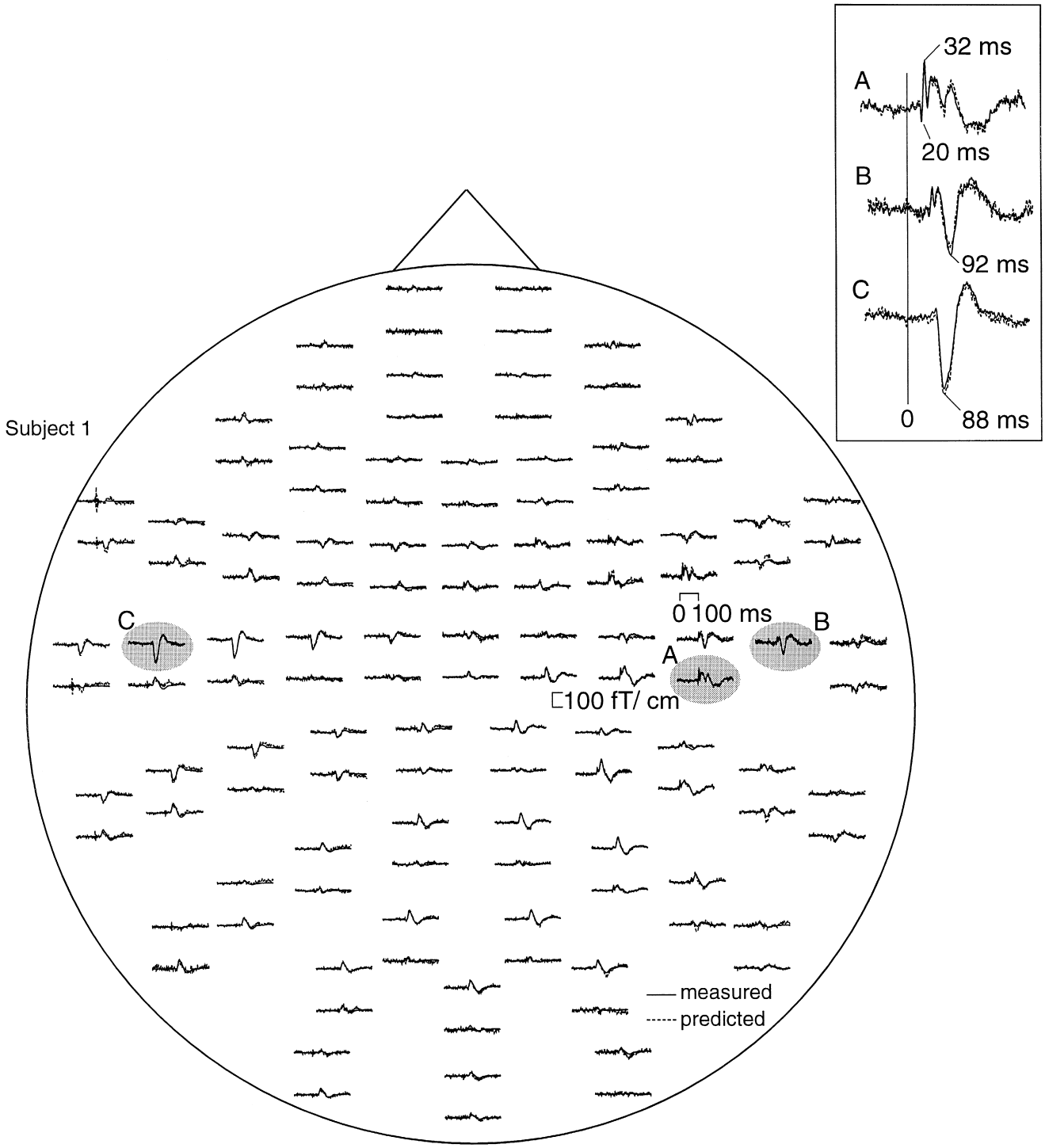

Fig. 1. SEFs of Subject 1 to left 'strong' median nerve stimuli (solid line) and predicted data from 3-dipole model (dotted line). The head is viewed from the top, and in each response pair, the upper trace illustrates the field derivate along the latitude and the lower trace along the longitude. The insert shows enlarged responses from shaded areas. ISI is 3 s, passband 0.03–320 Hz.

In a control experiment, the left radial nerve at the distal forearm was stimulated while the subject kept contraction in the left thenar to test the effect of muscle contraction on purely sensory nerve SEFs. In one subject, SEFs to median nerve stimuli were recorded during self-paced thumb movements at a rate of about 1/s.

SEFs were recorded with a helmet-shaped magnetometer array which has 122 planar first-order SQUID gradiometers, placed in 61 measurement sites (Neuromag-122™; [1]). The planar gradiometers detect the largest signal just above the local source area, where the field gradient is at its maximum. Each sensor unit contains a pair of gradiometers that measures two orthogonal tangential derivatives of the magnetic field component normal to the helmet surface at the sensor location. The exact location of the head with respect to the sensors was found by measuring the magnetic signals produced by currents led to three head position indicator coils placed at known sites on the scalp. The locations of the coils with respect to anatomical landmarks on the head were determined with a 3-D digitizer to allow the alignment of the MEG and magnetic resonance (MR) image coordinate systems.

The signals were bandpass filtered (0.03–320 Hz) and digitized at 1 kHz. The analysis period of 600 ms included a pre-stimulus baseline of 200 ms, and about 100–120 averages were collected. Sub-averages (of 30 averages) and 2–3 min of spontaneous activity were measured in each condition. Responses coinciding with signals exceeding 150 μV in the simultaneously recorded vertical electro-oculogram (EOG) were automatically rejected from the analysis.

To identify the sources of measured signals, deflections exceeding the pre-stimulus noise level (about 5 fT/cm) were first visually searched in order to select the time windows and cortical areas of interest for further analysis. During these time windows (from the beginning of the deflection to its return to the baseline level), the magnetic field patterns were first visually studied in 2 ms steps to create the initial forecast for the number of active sources within that time period and to estimate the stability of the dipolar magnetic field pattern. Then the equivalent current dipole (ECD), which best explains the measured data, was found by a least-squares search using a subset of 16–18 channels over the response area. These calculations resulted in 3-dimensional location, orientation, and strength of the ECD in a spherical conductor model, which was based on subject's MR images. Goodness-of-fit (*g*-value) of the model was also calculated to tell in percentage how much the dipole accounts for the measured field pattern; only ECDs explaining more than 85% of the field variance at selected periods of time over a subset of channels were used for further analysis.

After identifying the single dipoles (3–6 in total for each subject), the analysis period was extended to the entire measurement epoch and all channels were taken into account in computing a time-varying multi-dipole model; now the strengths of the previously found ECDs were allowed to change as a function of time while their locations and orientations were kept fixed. The validity of the multi-dipole model was evaluated by comparing the measured signals with responses predicted by the model. If signals of any brain region were left inadequately explained by the model, the data were re-evaluated for more accurate estimation of the generator areas. The *g*-values were calculated over all 122-channels and over the entire time period, and compared between different models to

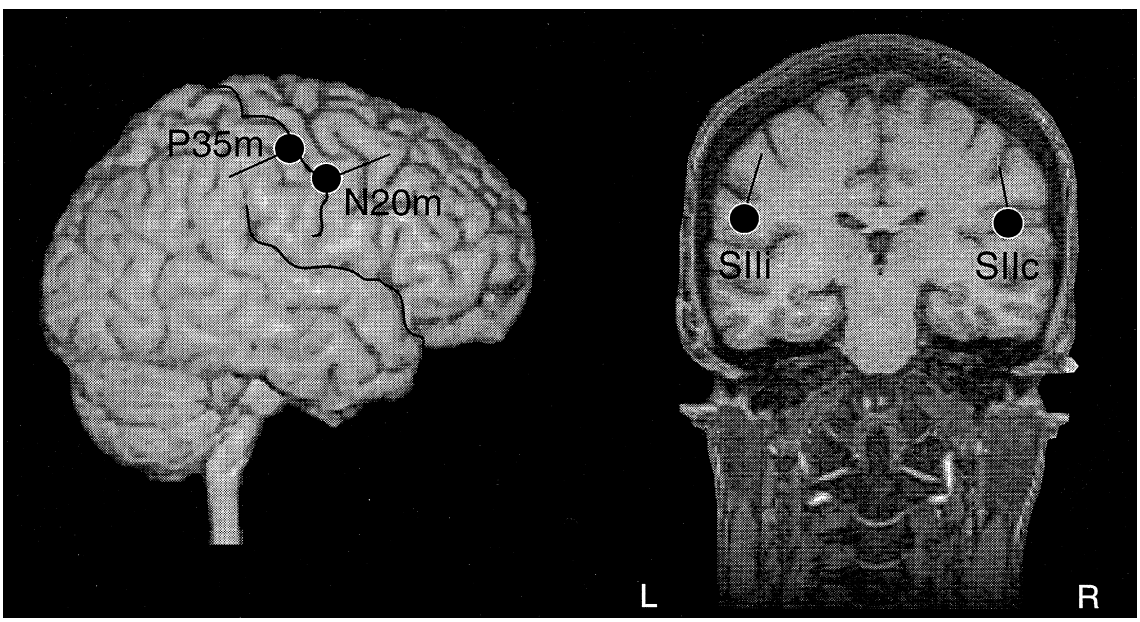

Fig. 2. Source locations and orientations of responses of Subject 1 superimposed on the 3D rendering (on the left) and on sagittal plane of MR images (right). SIIc = contralateral SII, SIIi = ipsilateral SII, L = left, R = right.

find best possible solution. This approach has been explained in detail by Hämäläinen et al. [10] and has been successfully used in several previous reports (e.g., Refs. [13,6]).

The spontaneous data were first visually inspected to reject events with excessive noise or artefacts. After that, the amplitude spectra of spontaneous activity was calculated by fast Fourier transformation; epochs of 1 s were calculated separately for both conditions.

MR images of subjects were acquired with a 1.5-T

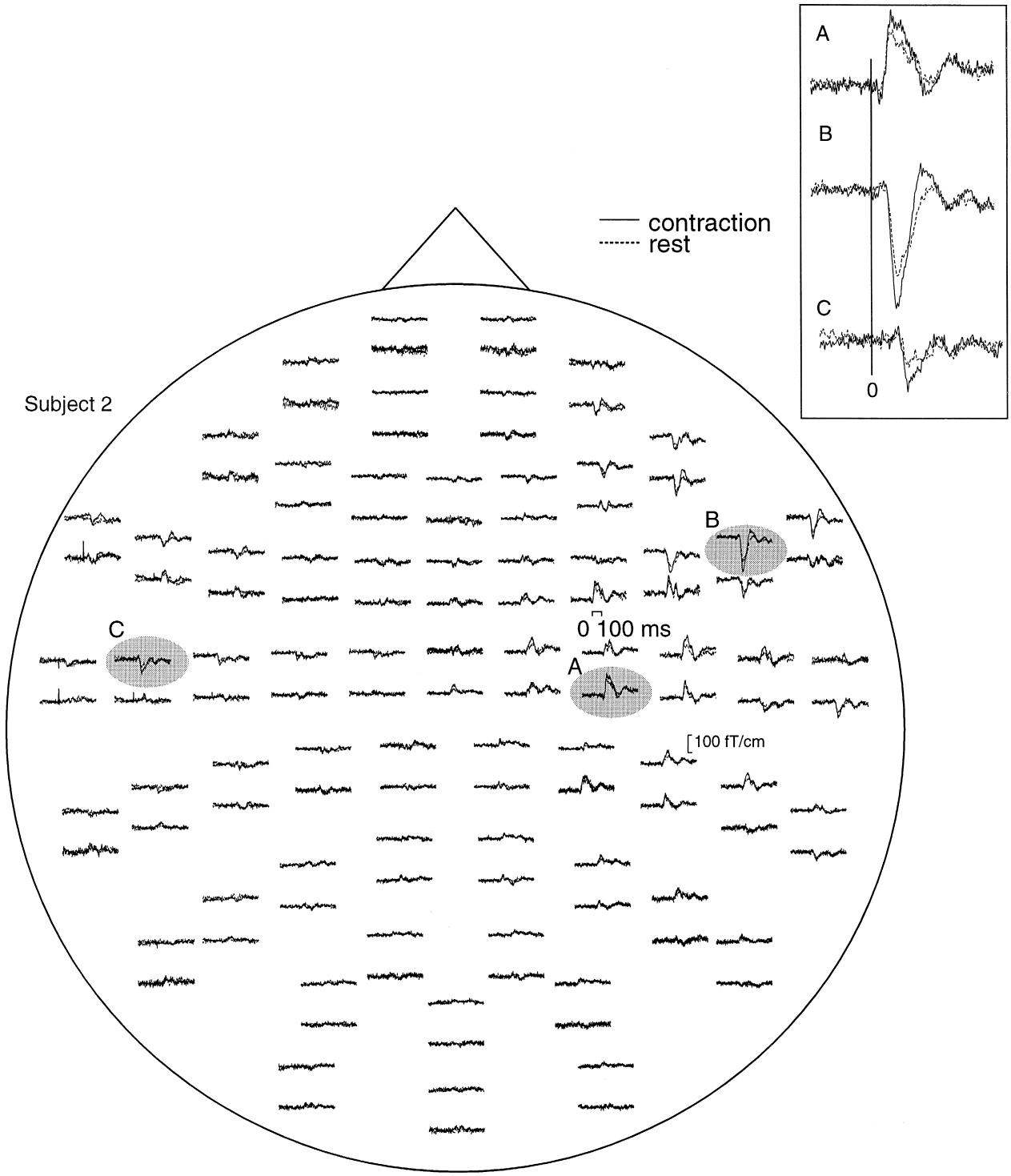

Fig. 3. SEFs of Subject 2 in the 'contraction' (solid line) and 'rest' (dotted line) conditions. The insert shows enlarged responses from the shaded areas (passband 0.03–120 Hz); The vertical line indicates the stimulus onset.

Siemens Magnetom™ scanner. A set of 128 coronal slices (thickness, 1.3 mm) was used for rendering the 3-D reconstruction of the brain's surface.

The statistical significance of results was validated by Student's paired two-tailed *t*-test.

3. Results

3.1. Generators of SEFs

Fig. 1 shows the distribution of SEFs to 'strong' left median nerve stimulation of Subject 1. Several local maxima suggest that multiple generator areas are active during the first 100 ms. The earliest deflection N20m corresponding to N20 in electric measurements, peaks at 20 ms over the contralateral anterior parietal cortex, followed by another deflection P35m of opposite polarity at 32 ms. Longer-latency responses peak over the lateral temporoparietal regions bilaterally at 88 and 92 ms. Dipole modelling resulted in three source areas, one in the anterior parietal lobe and two bilaterally in the lateral parietal lobes. Fig. 1 shows the predicted responses from the time-varying 3-dipole model superimposed on the measured responses. All responses are adequately explained, and the *g*-values exceed 80%, implicating good accuracy of the model. The insert shows enlarged responses from the three areas.

Fig. 2 shows the locations of generator areas superimposed on the same subject's MR images. In agreement with the earlier studies, N20m and P35m are generated in the post-central wall of the central fissure, i.e., in the SI cortex [28,2]. The longer-latency responses originate bilaterally from areas in the upper lip of the Sylvian fissure in parietal operculum. Although we cannot separate between the multiple somatosensory representations in the parietal

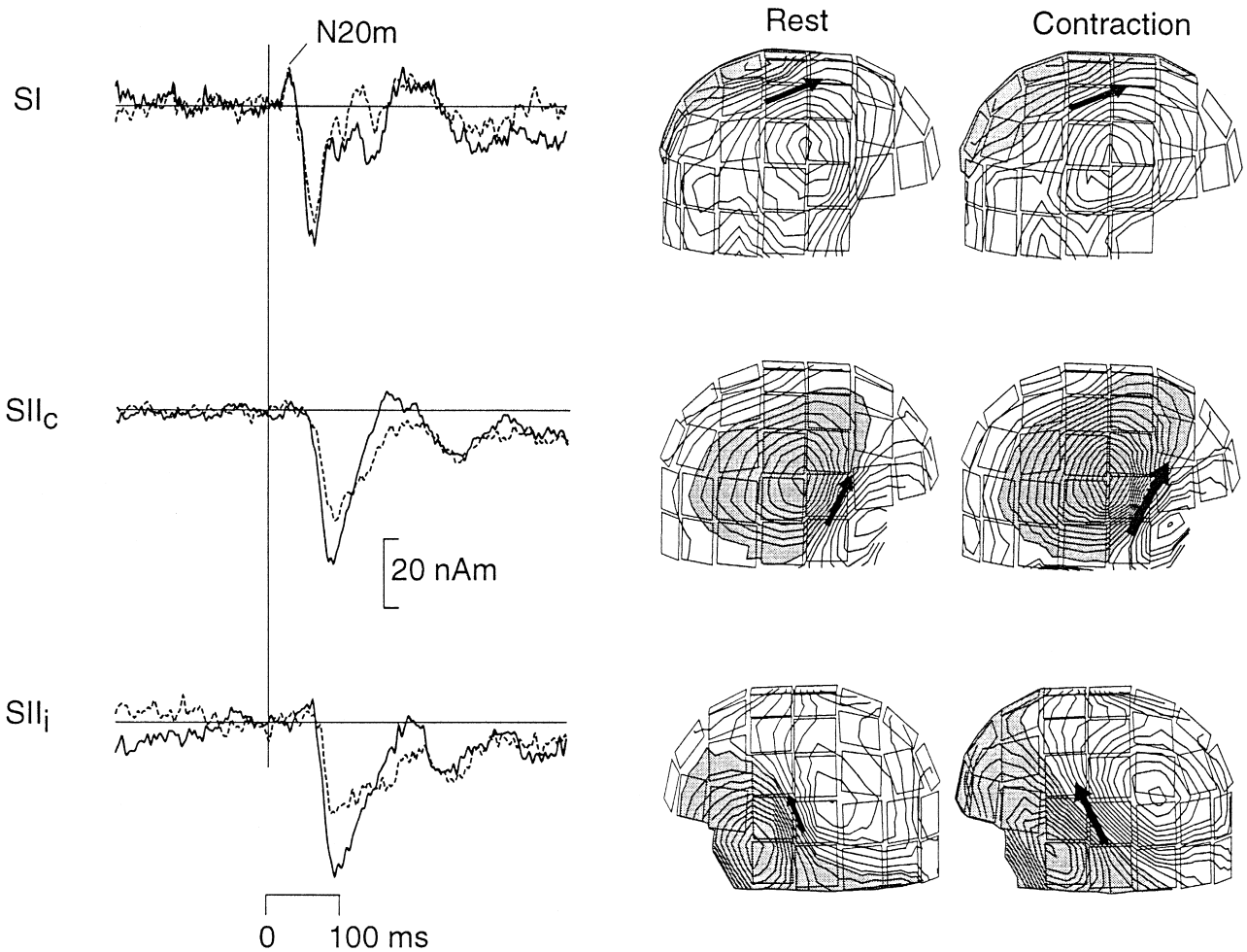

Fig. 4. Left: Strength of the SI, contra- and ipsilateral SII sources of Subject 2 as a function of time in 'rest' (dotted line) and 'contraction' (solid line) conditions. Right: Magnetic field patterns at the peak of the responses (N20m in SI) in the both conditions displayed over the helmet shaped sensor array. The sensor array is viewed from the right (upper and middle rows) and left (bottom row). The squares show the locations of the sensor units, and the arrows indicate the dipoles. The isocontours are separated by 20 fT for SI response (top row) and 40 fT for SII responses. The shaded areas indicate magnetic flux emerging from the head.

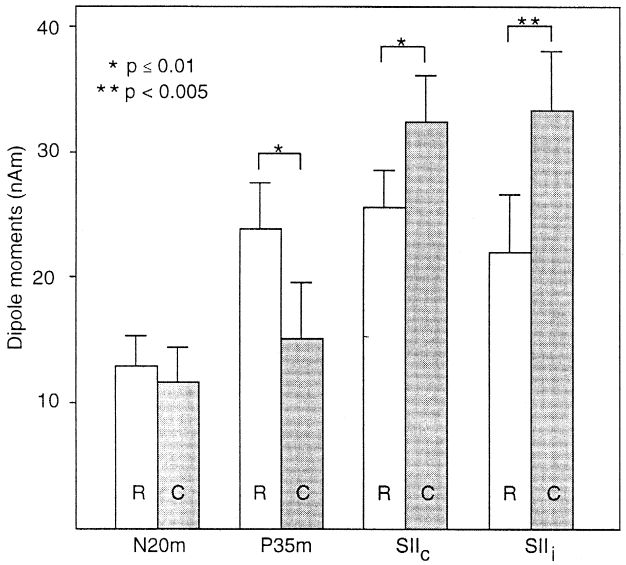

Fig. 5. Mean (±SEM; seven subjects) amplitudes of the N20m, P35m and SII responses in 'rest' (R; white bars) and 'contraction' (C; grey bars) conditions. Statistical significance $^{*}p < 0.01$; $^{**}p < 0.005$.

operculum [21], these generator areas agree best with the activation of SII cortices. All these sources have been described earlier in several magnetoencephalographic (MEG) studies [11,12,6].

The average of the first 30 responses had the same source configuration, but the SI and SII responses were slightly larger in amplitude both in 'rest' and 'contraction' conditions compared with the whole data set (15.8 vs. 12.8 nAm for SI; 30.0 vs. 25.6 nAm for contra- and 32.4 vs. 22.0 nAm for ipsilateral SII in 'rest' condition).

Responses from the contralateral SI and from the bilateral SII cortices were detected in all subjects. The activation of the posterior parietal cortex (PPC) was observed in four subjects, in agreement with an earlier MEG study [6]. In addition, activity in the mesial paracentral lobule, previously reported during the detection task of infrequent somatosensory stimuli [8], was observed in three subjects in the average of the first 30 responses. Signals in the ipsilateral sensorimotor cortex at 70–80 ms were seen in two subjects, in line with an earlier MEG study [20].

All the generator areas found in each subject were taken into account in the individual multi-dipole model, resulting in 3–6 sources per subject. However, in the present report, the effect of motor activity on SEFs was quantified only in the SI and SII cortices.

To evaluate the validity of the source model found to suprathreshold 'strong' stimuli, the responses were separately analyzed also in 'rest' and 'contraction' conditions. The source locations and orientations remained stable through the conditions; no additional source areas emerged, neither did any of the source areas 'drop out' during any condition. On average, source locations of the SI responses were less than 3 mm and those of the SII responses less than 7 mm apart from each other in horizontal plane in different conditions. Therefore, the dipole model based on the high signal-to-noise responses to 'strong' stimuli was applied to the two other conditions.

3.2. Effect of isometric contraction on activation of SI and SII cortices

Fig. 3 shows SEFs of Subject 2 in the 'rest' and 'contraction' conditions. The experimental setup and the stimuli are identical in the two conditions, except of the isometric contraction of thenar during the 'contraction' condition. The earliest response N20m does not change, but both SII responses are clearly enhanced with muscle contraction.

Fig. 4 shows the source strengths as a function of time and magnetic field patterns at peak latencies of the SI and SII responses of the same subject in both conditions. The amplitude of N20m remains the same, whereas the later SI component P35m, small in amplitude in this subject, is further decreased during contraction. Strengths of both contra- and ipsilateral SII responses are clearly increased during contraction. The similar magnetic field patterns during rest and contraction show that the source locations and orientations do not change although the strength of source vary between the conditions.

Fig. 5 shows the mean (±SEM; seven right-handed subjects) strengths of the SI and SII responses in the two conditions. N20m is not changed, whereas P35m is statisti-

Table 1
Mean (±SEM) strengths and latencies in two conditions

	All responses				First 30 responses			
	Strength (nAm)		Latency (ms)		Strength (nAm)		Latency (ms)	
	R	C	R	C	R	C	R	C
Si N20m	12.9 ± 2.3	11.7 ± 2.8 *	21.8 ± 1.0	21.5 ± 1.0	15.8 ± 4.9	15.7 ± 4.0	21.3 ± 0.3	21.4 ± 0.3
P35m	23.9 ± 3.7	15.1 ± 4.5 *	35.1 ± 1.9	33.5 ± 1.1	30.2 ± 4.5	16.8 ± 3.8	32.8 ± 0.3	32.2 ± 0.7
SIIc	25.6 ± 2.8	32.4 ± 3.7 *	87.8 ± 6.5	88.7 ± 6.1	30.0 ± 8.0	36.1 ± 6.2	89.5 ± 12	87.2 ± 11
SIIi	22.0 ± 4.6	33.4 ± 4.6 * *	94.7 ± 5.5	93.2 ± 5.7	32.0 ± 6.1	44.0 ± 5.9	94.9 ± 13	94.7 ± 9.5

Statistical significance between 'rest' (R) and 'contraction' (C) conditions (all responses).
$^{*}p \le 0.01$; $^{**}p \le 0.005$.

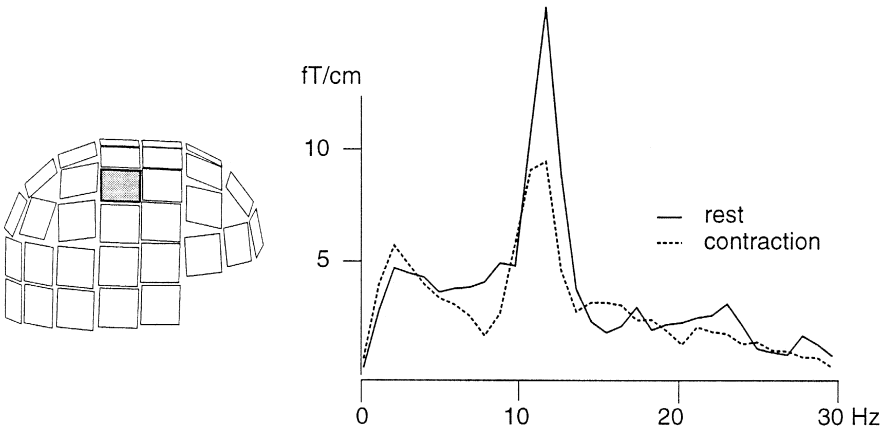

Fig. 6. Pre- and poststimulus spectra of Subject 3 in 'rest' (R) and 'contraction' (C) conditions from one chosen channel over the right SI cortex. Shaded area on the sensor array shows the location of the chosen channel.

cally significantly reduced during contraction ($p \leq 0.01$). Contra- and ipsilateral SII responses are increased during contraction by 26% ($p < 0.01$) and 52% ($p < 0.005$), respectively. The response latencies did not differ between conditions. Table 1 summarizes the results showing mean ($\pm$SEM) amplitudes and latencies in all subjects, both for the whole data set and for the averages of the first 30 responses.

The analysis of the average of the first 30 responses paralleled the overall results; N20m did not show any changes in strength, whereas P35m was significantly attenuated during contraction. The amplitude increase of the SII responses during contraction was evident already during the first 30 responses, and the effect was twice as strong for the ipsilateral (right) SII than for the contralateral (left) SII.

Stimulation of the purely sensory radial nerve stimuli elicited responses with similar field patterns as the median nerve stimuli, with dominant sources in contralateral SI and bilateral SII cortices. However, contraction of the left thenar did not affect any of these responses. In the other control study with one subject, self-paced thumb movements during the MN stimulation enhanced the SII responses but to a lesser extent than isometric contraction (24 vs. 61% for contra- and 22 vs. 85% for ipsilateral SII).

3.3. Spontaneous data

To monitor the effect of contraction on cortical reactiveness, spontaneous data were recorded during the MN stimulation. The amplitude spectra revealed frequency peaks around 10 Hz in six and additional 20 Hz in two subjects over the contralateral SI, in line with earlier studies [4,27]. Fig. 6 shows the amplitude spectra of one subject over the right (contralateral) SI cortex. The isometric contraction clearly dampens the 10 Hz peak compared with the one in 'rest' condition.

Across subjects, the 10 Hz peak was diminished on average by 31% in contra- and 26% in ipsilateral somatomotor areas during contraction. The 20 Hz peak in two subjects behaved in a similar manner, but the diminution was slightly smaller than for 10 Hz peak.

4. Discussion

The present study showed that responses of contra- and ipsilateral SII cortices to MN stimuli were enhanced during contraction of muscles innervated by the same nerve. Of the SI responses, N20m did not change with coincident isometric contraction, whereas P35m was clearly reduced. Isometric thenar contraction did not affect the SI or SII responses to purely sensory radial nerve stimuli. These findings agree with earlier studies showing enhancement of SII responses as electric stimuli exceeded motor threshold [7] and increase of SII responses during voluntary finger movements [16]. However, in the present study, voluntary self-paced movement of the thumb enhanced the SII responses less than isometric contraction. This may be due to the different timing of the stimuli and movements; the thumb was moved approximately 1/s, and therefore, some of the stimuli were delivered while the thumb was at rest.

The effect of muscle contraction on cortical reactiveness was evaluated with the help of reactivity of cortical mu-rhythm, which typically is suppressed during voluntary and electrically stimulated movements; the effect is usually more pronounced during voluntary movements [4,27,14]. The 10 Hz peak was clearly diminished or abolished bilaterally in all subjects during isometric contraction, which can be considered as an objective sign of sufficient contraction of the muscles to have an impact on cortical reactiveness.

Since N20m (the first SI component) was not enhanced during contraction, it is unlikely that the enhancement of the SII responses would be due to facilitation at peripheral or spinal level. Rather, the interaction is likely to take place at cortical level. This is in line with direct recordings of somatosensory evoked potentials which have shown that

somatosensory gating occurs mainly in the cerebral cortex and only weakly at thalamic or brainstem level [15].

The neural generators underlying P35m have been previously suggested to represent inhibitory postsynaptic potentials in 3b [30]. If this were the case, the observed attenuation of P35m could represent diminished inhibition in SI. This would, in turn, result in subsequent enhancement of SII responses, if serial processing in SI and SII cortices is favored [24]. Accordingly, in patients with myoclonus epilepsy, P30m (corresponding to P35m in this study) was increased five-fold compared with the controls, whereas only negligible SII responses were observed [17], possibly reflecting increased inhibition in SI.

The validity of our results largely depends on the accuracy of the source model. Separate analysis of all conditions revealed the same generator areas, justifying the use of the same model, based on the responses with best signal-to-noise ratio, across all conditions. MEG can easily separate sources which are about 2 cm apart and even less if the source orientations differ. However, since MEG measures cerebral magnetic fields from a distance, sources very close to each other cannot be separated. Therefore, the observed increased activation of SII could also be due to an increased number of nearby sources seen as a single entity in MEG.

Numerous electric and magnetic studies have shown that N20(m) increases almost linearly with the increasing stimulus intensity up to a certain level (e.g., Refs. [29,18,7]. Since the amplitude of N20m in the present study remained unchanged or even slightly decreased with contraction, changes in the stimulus intensity due to different contact of the stimulator pads with the skin cannot explain the observed differences between the two conditions.

Variation of vigilance and attention are known to effect the activation of second and other 'higher order' somatosensory areas more than the SI cortex [12,22]. Although we used only experienced subjects who were familiar with electric stimuli, and the subjects were reading a book to keep the vigilance stable, possible modification of the responses due to attentional factors is difficult to exclude. However, the differences in responses between rest and contraction were very replicable even between measurements performed in different days. Further, the mesial activation, previously observed in all subjects during voluntary attention to tactile stimuli [8], was observed only in three subjects during the first 30 responses after which it gradually diminished to pre-stimulus baseline level; in all other subjects, the mesial response was neglible even in the first sub-average. The behavior of the mesial response is in line with earlier observations that linked activation of mesial cortex to voluntary attention to stimuli; the three subjects may have unintentionally observed the first stimuli, after which they have become more used to experimental situation. In the control study with purely tactile stimulation, the SII responses were not altered between the two conditions, in contrast to what one would expect if the changes were due to attentional factors coupled with voluntary contractions. Therefore, it is likely that the attentional factors can not solely explain the present findings.

The functional role of the human SII areas has remained obscure. Lesion studies in monkeys have shown that even a unilateral lesion of SII area severely impairs tasks of tactile learning and retention [26,9]. Only in seldom cases do lesions in humans damage selectively the SII cortex because of its location and small size, and profound disabilities comparable to monkey data have not been reported. However, the early neurophysiological studies by Penfield and Jasper [23] have implied the potential role of SII areas in sensorimotor integration. On the basis of connectional evidence, SII has also been suggested to provide a cortical connection for cutaneous inputs to motor cortex [3].

On the basis of the present results, we suggest that motor activity enhances cortical effects of tactile impulses from the same region either by increasing synchronicity or by adding number of activated neurons in SII. This may be due to decreased inhibitory activation of SI, reflected in diminished amplitude of P35m. Enhanced SII activation may reflect tuning of the SII neurons towards relevant tactile impulses from the region of contracting muscles; this could help in monitoring and correcting the movements and in guiding exploratory movements. This interpretation agrees with earlier studies in monkeys, which showed increased responsiveness of SII neurons during behaviorally important input [25]; such a sensorimotor integrative function of SII would also explain impaired tactile learning in monkeys with SII lesion.

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