Difference in Somatosensory Evoked Fields Elicited by Mechanical and Electrical Stimulations: Elucidation of the Human Homunculus by a Noninvasive Method

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Abstract: We recently recorded somatosensory evoked fields (SEFs) elicited by compressing the glabrous skin of the finger and decompressing it by using a photosensor trigger. In that study, the equivalent current dipoles (ECDs) for these evoked fields appeared to be physiologically similar to the ECDs of P30m in median nerve stimulation. We sought to determine the relations of evoked fields elicited by mechanically stimulating the glabrous skin of the great toe and those of electrically produced P40m. We studied SEFs elicited by mechanical and electrical stimulations from the median and tibial nerves. The orientations of dipoles from the mechanical stimulations were from anterior-to-posterior, similar to the orientations of dipoles for P30m. The direction of the dipole around the peak of N20m from median nerve electrical stimulation was opposite to these directions. The orientations of dipoles around the peak of P40m by tibial nerve stimulation were transverse, whereas those by the compression and decompression stimulation of the toe were directed from anterior-to-posterior. The concordance of the orientations in ECDs for evoked fields elicited by mechanical and electrical stimulations suggests that the ECDs of P40m are physiologically similar to those of P30m but not to those of N20m. The discrepancy in orientations in ECDs for evoked field elicited by these stimulations in the lower extremity suggests that electrical and compression stimulations elicit evoked fields responding to fast surface rubbing stimuli and/or stimuli to the muscle and joint. *Hum Brain Mapp* 24:274–283, 2005. © 2005 Wiley-Liss, Inc.

Key words: magnetoencephalography; human; homunculus; somatosensory evoked fields; SEFs; primary somatosensory cortex; N20m; P40m

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INTRODUCTION

Somatosensory cortical functions in animals have been elucidated by using microelectrode maps, but for humans, this invasive approach is limited to patients who need neurosurgery. Magnetoencephalography (MEG), however, enables evaluation of somatotopic functioning of the cortex in humans.

N20/N20m primary responses of human somatosensory evoked potentials/fields (SEPs/SEFs) to median nerve are generated from the posterior bank of the central sulcus (area 3b) and can be modeled by a fixed single tangential equivalent current dipole (ECD) [Allison et al., 1991a; Desmedt et al., 1987]. On the other hand, P25/N35 potentials (human) are thought to be generated from the crown of the central gyrus (area 1), because a reduction of P12-N25 potentials was observed after excising the crown of this area in the monkey [Allison et al., 1991b]. Furthermore, separate activities in area 1 and area 3b have been demonstrated after electrical stimulation of the median nerve using a multi-dipole model [Inui et al., 2003, 2004].

Although there is a good understanding of the generator of upper SEPs/SEFs, the generator of P40/P40m primary responses of SEPs/SEFs to tibial nerve still is not clear. It is known that there is a large interindividual variability of tibial nerve SEPs in terms of recorded waveforms, dipole orientation, and topography of each component [Ozaki, 1996]. This variability might be due to anatomical variations in the primary foot somatosensory cortex (SI).

The source locations in the mesial wall of the hemisphere were confirmed by recording SEFs elicited by lower limb stimulation [Hari et al., 1984; Okada et al., 1984]. Later, they were characterized in more detail by recordings of tibial and sural nerve SEFs [Huttunen et al., 1987; Yamada et al., 1996], and the origin in the mesial wall of the hemisphere was suggested by signal patterns, with temporally changing orientations of the source currents. Similar changes in orientation were also found by observation of the field patterns in response to tibial nerve stimulation rotating as a function of time that agreed with the field patterns in response to successive activation of different cytoarchitectonic areas in the foot SI cortex [Kakigi et al., 1995]. Thus, it is thought that the large interindividual difference in them is due to the two cortical components that generate P40/P40m primary responses with the same latency [Hari et al., 1996; Shimojo et al., 1996].

We recently studied evoked fields elicited by compression and decompression of the glabrous skin of the finger [Shirai et al., 2004]. The results of that study revealed that ECDs are

directed from anterior-to-posterior and are located around the central sulcus, apparently similar functionally to ECDs of P30m in median nerve electrical stimulation.

Because P30m is known to originate from area 3b and area 1 of the SI cortex, ECDs of the lower extremity elicited by mechanical stimuli should also originate from those areas. By comparing evoked fields elicited by electrical stimulation and those elicited by mechanical stimulation, we hope to obtain a better understanding of the physioanatomical origin of lower extremity SEPs/SEFs. In this study, we examined evoked fields elicited by mechanical stimuli to the lower extremity as well as to the upper extremity.

SUBJECTS AND METHODS

Subjects

Ten healthy, right-handed subjects (9 men, 1 woman, age range 25–40 years) participated in this study. The evoked fields in the upper extremity were examined in seven of the subjects and the evoked fields in the lower extremity were examined in six of them. The instrument used in this study was approved by the Japanese Ministry of Health, Labor and Welfare (No. 20800BZY00275000), and informed consent was obtained from each subject after a full explanation of the experiment. During the experiment, the subject and the examiner looked at an image of a clock projected on a screen in front of them in a magnetically shielded room. This setting prevented subjects from looking at their hands or moving their eyes.

Stimuli and Device

Compression and decompression stimuli

Glabrous skin contact was made by the examiner compressing the left second finger or the first toe of the subject with his right index finger at ~1-s intervals. One set of the examiner's action consisted of touch, sustained contact, and removal of his finger as shown in Figure 1. The examiner tried to make the interval from decompression of the finger or toe to compression as short as possible.

Two recording sessions with different trigger timings were carried out for the task. In one recording session, the trigger was elicited by light reflection through an optic fiber placed on the subject's second finger or first toe when the examiner compressed the subject's second finger or first toe (compression trigger session). In the other recording session (decompression trigger session), the trigger was elicited by decompression of the examiner's finger from the subject's second finger or first toe (Fig. 1).

The evoked fields in each recording session were averaged 100 times for second finger stimulation and 400 times for first toe stimulation in all subjects. The performance was continuously monitored by means of a video camera, and the operator confirmed that there was no contact with the subject other than the second finger or first toe.

Neuromagnetic recordings were made using a whole-head 306-channel planar gradiometer system. A 204-chan-

Abbreviations

ECDs	equivalent current dipoles
MEG	magnetoencephalography
MR	magnetic resonance
SEFs	somatosensory evoked fields
SEPs	somatosensory evoked potentials
SI	primary somatosensory cortex

Figure 1.

The head, viewed from the top, is flattened onto a plane. The upper and lower curves in each location represent the longitudinal and latitudinal gradients of the radial component of the magnetic field, respectively. For lower extremity stimulation, the middle curves in each location represent the gradients recorded by the magnetometer. Somatosensory evoked field (SEF) waveforms in a representative subject (Subject 1) were obtained by the following stimulations: electrical stimulation of the median nerve (a), electrical stimulation of the tibial nerve (b), mechanical stimulation of the second finger (c), and mechanical stimulation of the first toe (d). c: The amplitude of evoked fields for the compression trigger sensor is higher than that of evoked fields for the decompression

trigger sensor. The evoked fields for decompression starts before the triggering, and evoked fields for compression starts after the triggering. c,d: Illustrations showing the examiner's action on the finger or toe of the subject. The optic fiber device is located on the subject's index finger or first toe on glabrous skin. Note that these evoked fields arise from the primary somatosensory area. d: In the lower extremity stimulation, the upper curves representing the longitudinal gradients of the radial component of the magnetic field were more distinct than the lower curves representing the latitudinal gradients of that by the electrical stimulation, whereas the opposite phenomenon was observed in the case of the mechanical stimulation. C, compression; D, decompression.

nel planar gradiometer system was used for upper extremity examination, and a 306-channel planar gradiometer, including a 102-channel magnetometer sys-

tem was used for lower extremity examination. Mutually orthogonal, tangential magnetic field gradients were obtained simultaneously at 102 recording sites. The record-

Figure 1. Continued

ing bandpass was 0.1–200, and the signals were digitized at 600 Hz.

The analysis period of 1,000 ms started 500 ms before triggering. One hundred artifact-free responses and 400 responses were averaged online separately for each task for the second finger stimulation and for the first toe stimulation, respectively. When triggering failure (i.e., no triggering or triggering twice) occurred

in more than 10% of a recording, that recording was discarded.

Electrical stimuli

Electrical stimuli were delivered to the left median nerve at the wrist and left tibial nerve at the ankle. The analysis period of 300 ms started 50 ms before the stimulation. Con-

TABLE I. Localization of ECDs

		x / mm		y / mm		z / mm	
Upper extremity	N20m	51.6 (0.6)	**	11.9 (2.1)	**	85.2 (1.9)	*
	P30m	49.2 (1.5)		11.9 (2.1)		85.9 (1.2)	
	C	45.3 (1.6)		15.9 (1.7)		89.1 (1.0)	
	D	47.7 (1.0)		18.6 (1.1)		86.0 (0.6)	
Lower extremity	P40m	9.9 (0.6)	**	19.5 (1.7)	**	116.2 (1.0)	*
	C	11.8 (0.9)		14.4 (2.4)		121.4 (1.2)	
	D	14.1 (1.6)		12.7 (1.9)		119.2 (2.9)	

Values are expressed as mean ($\pm$ SEM). The location of each dipole at the peak ECD strength latency was calculated.

* $P < 0.05$; ** $P < 0.005$.

ECDs, equivalent current dipoles; C, compression; D, decompression.

stant-current square-wave pulses of 0.2 ms in duration and of motor threshold / three-times larger than sensory threshold intensities on the median or tibial nerve were delivered at 400-ms interstimulus intervals. One hundred evoked fields for the median nerve and 400 evoked fields for the tibial nerve were averaged and bandpass-filtered at 0.1 to 200 Hz, and they could be examined during the course of the experiment.

For source identification, the head was assumed to be a sphere, the dimensions of which were determined on the basis of individual magnetic resonance (MR) images obtained by using a GE Yokogawa SIGNA 1.5 Tesla device (slice thickness of 2 mm; 3D-SPGR). The two coordinate systems (MEG and MR imaging) were aligned by applying markers in MR imaging and by identifying these landmarks with a three-dimensional (3-D) digitizer (Isotrack; Polhemus Navigation Sciences, Colchester, VT) before MEG recordings. All source analysis was based on signals high-pass filtered at 4 Hz to eliminate baseline fluctuation and low-pass filtered at 100 Hz.

Data Analysis

Two main components of evoked fields appeared in each session. Sources for the evoked responses were modeled as single-current dipoles. The magnetic field patterns were first visually examined in 1-ms steps to identify all local and stable "dipolar field patterns," i.e., all field distributions resembling those produced by a single-current dipole. Then the equivalent current dipole (ECD) best describing the most dominant source during the strongest signals of each dipolar field pattern was identified by a least-squares search using a subset of 20 to 30 channels over the source area. Thereafter, the ECD position and direction were fixed and the strengths of ECDs were calculated over the time. The origin of the head-based coordinate system was defined as the midpoint between the preauricular points. The x -axis pointed from the origin to the right preauricular point, the y -axis to the nasion, and the z -axis to the vertex in a direction perpendicular to the x - y plane. Analysis was focused on the evoked fields elicited by compression stimuli in the compression trigger

session and those elicited by decompression stimuli in the decompression trigger session. Because human manipulation has a variation of a few milliseconds in the timing to trigger the photosensor in latencies, we analyzed the ECDs from 3 ms pre-peak to 3 ms post-peak of evoked fields for the electrical and mechanical stimulations. The signal-to-noise ratio was sufficiently good during this period. The ECDs were superimposed on a high-resolution T1-weighted MR image of the brain for each subject and were depicted in the coordination system on the MR image.

Statistical Tests

A two-way analysis of variance (ANOVA) was used for statistical comparisons of the orientations and strengths of ECDs and response latencies of each component of SEFs from the SI cortex for each stimulation. Fisher's protected least significant difference test was used for post hoc comparisons ($P < 0.05$).

RESULTS

Response Distributions

Figure 1a,b shows SEF waveforms obtained by electrical stimulation of the median and tibial nerves. The main responses such as N20m and P30m in median nerve stimulation peaked over the contralateral anterior parietal cortex. N20m peaked at 22 ± 0 ms and was followed by P30m, with opposite polarity at 30 ± 1 ms. The main responses such as P40m and N60m in tibial nerve stimulation peaked over the anterior parietal cortex at a mesial site of the hemisphere. P40m peaked at 39 ± 1 ms and was followed by a response, N60m, with opposite polarity. Only two subjects showed responses in tibial nerve stimulation before the responses of P40m, and those of N35m peaked at 35 ± 1 ms.

Figure 1c,d shows SEF waveforms obtained by compression and decompression stimulations on the finger and toe. The locations of main responses in these stimulations appeared close to those of electrical stimulations. When the trigger was elicited by compression, the signals for compres-

Figure 2.

Mean ($\pm$ SEM) source strengths for each stimulation, shown separately for the upper extremity and lower extremity stimuli. Single asterisks indicate $P < 0.05$; double asterisks indicate $P < 0.005$. C, compression; D, decompression.

sion responses were much larger than those for decompression responses. (Decompression responses were barely detectable.) When the trigger was elicited by decompression, the signals for decompression responses were much larger than those for compression responses.

In the compression trigger, the signals of the compression response peaked at 57 ± 1 ms and 82 ± 2 ms for the second finger and first toe stimulations, respectively. In the decompression trigger, the signals of the decompression response peaked at 22 ± 2 ms and 38 ± 4 ms for the second finger and first toe stimulations, respectively.

Source Modeling

Table I shows the average localizations of ECDs for each stimulation. Results of ANOVA showed a significant differ-

ence in the x values, y values, and z values of the coordinates for the upper extremity (x: $F = 3.49$, $P = 0.02$; y: $F = 3.46$, $P = 0.02$; z: $F = 2.83$, $P = 0.04$). The results of ANOVA also showed a significant difference in the x values and z values of the coordinates for the lower extremity (x: $F = 3.74$, $P = 0.03$; z: $F = 4.22$, $P = 0.02$). Post hoc analysis indicated that the ECDs for electrical stimuli (N20m and P30m) were located more laterally (x axis), posteriorly (y axis), and deeply (z axis) than those for the mechanical stimuli in the upper extremity and that those for electrical stimuli (P40m) in the lower extremity were located more medially (x-axis) and superficially (z axis) than those for the mechanical stimuli ($P < 0.05$).

Figure 2 shows the average strengths of ECDs for each stimulation. Results of ANOVA showed a significant difference in the strengths of ECDs for each stimulation in the upper and lower extremities (upper extremity: $F = 3.33$, $P = 0.02$; lower extremity: $F = 3.73$, $P = 0.03$). Post hoc analysis indicated larger signals for decompression responses than those for compression responses in the second finger stimulation and much smaller signals for those in the first toe stimulation ($P = 0.05$).

Figure 3a (left) shows the source areas for the same subject superimposed on MR images. The ECDs for N20m, P30m, and finger compression and decompression responses arise in the posterior wall of the central fissure, in the hand area of the SI cortex. Figure 3a (right) shows the strength of ECDs in each latency. The strength curve of ECDs for N20m is similar but not identical to that for P30m. Figure 3b (left) shows the source areas of ECDs for P40m, toe compression and decompression responses in the foot area of the SI cortex. Figure 3b (right) shows the strength of ECDs in each latency. The mean latencies of each largest signal for each modality are shown in Table II.

Figure 4 shows the field distributions and isofield contour maps for each stimulation in Subject 1. Note the orthogonal directions of compression and decompression responses to

Figure 3.

a: The source areas for Subject 1, superimposed on magnetic resonance images. The equivalent current dipoles (ECDs) for N20m, P30m, and finger compression and decompression responses arise in the posterior wall of the central fissure, in the hand area of the primary somatosensory cortex (SI) cortex. The right figures show the strength of ECDs in each latency. The strength curve of ECDs for N20m is similar but not identical to that for P30m. **b:** The source areas of ECDs for P40m, toe compression, and decompression responses in the foot area of the SI cortex. The curves show the strengths of ECDs in each latency. C, compression; D, decompression.

TABLE II. Peak latencies of ECDs in each stimulation

		Peak latencies of ECDs (msec)
Upper extremity	N20m	22 (0)
	P30m	30 (1)
	C	57 (1)
	D	22 (2)
Lower extremity	P40m	39 (1)
	C	82 (2)
	D	38 (4)

Values are expressed as mean ($\pm$ SEM). The latency of each dipole at the peak ECD strength was measured.

ECDs, equivalent current dipoles; C, compression; D, decompression.

the ECDs for P30m. The directions of ECDs for the compression responses are closer to those for P30m than those for the decompression responses. In the lower extremity, the directions of ECDs for the electrical compression and decompression responses were different. Those for decompression responses were directed anterior-to-posterior, and those for the electrical responses were directed transversely. All subjects showed ECDs of P40m in the transverse direction. Only two of the subjects showed ECDs of N35m in the posterior-to-anterior direction. However, for compression and decompression responses, the orientations of ECDs were anterior-to-posterior in all six subjects. Results of ANOVA showed a significant difference in the azimuth absolute values for the upper and lower extremities (upper extremity: $F = 6.97$, $P = 0.002$; lower extremity: $F = 31.7$, $P < 0.0001$). Post hoc analysis indicated that the azimuth absolute values for N20m were larger than those for the compression responses and that those for the decompression responses were larger

Figure 4.

Field distributions and isofield contour maps for each stimulation in Subject I. Note the orthogonal directions of compression and decompression responses to the equivalent current dipoles (ECDs) for P30m. The directions of ECDs for the compression responses are closer to those for P30m than those for the de-

compression responses. In the lower extremity, the directions of ECDs for the electrical, compression, and decompression responses were different. Those for decompression responses were directed anterior-to-posterior, and those for electrical responses were directed transversely. C, compression; D, decompression.

TABLE III. Orientations of ECDs and GOFs

		Orientations of ECDs (azimuth, degree)		GOFs (%)
Upper extremity	N20m	76 (1)	**]*	91 (1)
	P30m	70 (5)		87 (1)
	C	60 (1)		88 (0)
	D	66 (1)		91 (1)
Lower extremity	P40m	33 (5)	] **	91 (1)
	C	68 (3)		90 (1)
	D	78 (4)		80 (0)

Azimuth is defined as an absolute value of an angular distance (degree) from anterior (y-axis) toward right lateral (x-axis) direction. Values are expressed as mean ($\pm$ SEM).

* $P < 0.05$; ** $P < 0.005$.

ECDs, equivalent current dipoles; GOFs, goodness of fit; C, compression; D, decompression.

than those for the compression responses ($P < 0.05$). Those for P40m were smaller than those for the compression and decompression responses ($P < 0.05$; Table III). The signals with a GOF less than 80% or which showed confidence volumes more than 1.0 cm^3 were eliminated as candidates for statistical analysis (Table III).

DISCUSSION

Our results demonstrated stronger average signals of ECDs by compression stimuli to the first toe with high GOFs and low confidence volume than those of ECDs by decompression stimuli. This strength difference between compression and decompression stimuli in the cortex is in agreement with results of previous studies on recording of action potentials from a nerve in the upper arm [Knibestöl and Vallbo, 1970; Knibestöl, 1973]. On the other hand, the action potentials recorded from the nerve terminals close to Pacinian corpuscles showed almost the same amplitudes in compression and decompression responses [Talbot et al., 1968]. Only a small portion of the stimuli by compression is relayed through the peripheral nerve to the brain by decompression. Therefore, the decompression stimuli elicit smaller evoked potentials in the upper arm than do compression stimuli [Knibestöl and Vallbo, 1970]. Thus, the responses recorded by MEG were much smaller in the case of decompression stimuli than in the case of compression stimuli to the toe.

In contrast to the first toe stimuli, the average signal of ECDs by compression on the second finger was weaker than that by decompression from the second finger. This finding was a surprising result, because it showed a remarkable difference between the second finger and first toe stimulations, which seemed to be essentially equal. It can be explained by two neurophysiological phenomena called "central amplification" and "gating" [Eisen and Elleker, 1980; Jones et al., 1989; Fujii et al., 1994]. It was thought that the more condensed distributions of Pacinian corpuscles on the finger were stimulated so as to saturate the neural activities of the receptive field in area 3b. After amplification of

evoked fields at the central level, the suppression mechanism from the joint movement or muscle contraction works to decrease the strengths of ECDs by compression and decompression responses in area 3b and area 1. A previous study showed that area 3b is less involved than area 1 by gating [Nelson et al., 1991]. Because the average responses by compression were weaker than those by decompression from the second finger, compression activates more neurons in area 1 than does the decompression. However, the inputs to the brain of responses from the first toe by decompression were much smaller than those by compression. As a result, although there is some suppression by passive movement, the signals of ECDs by compression were much stronger than those by decompression in the first toe stimuli.

The peak latency of ECDs by decompression was shorter than that by compression. This finding can be explained partially by the delay of trigger timing for removing the finger, that is, the examiner's finger moves over the skin indentation before terminating the light reflection to trigger the sensor. However, with much slower peak latency of them in first toe stimulations than in second finger stimulations, we should also consider a central mechanism regarding offset responses. For examples, in auditory evoked fields, the latencies were 10–20 ms longer for offset responses than for onset responses, with longest latencies at the shortest stimulus durations [Joutsiniemi et al., 1989]. It has been suggested that there are independent generators of on and off responses [Hari et al., 1987; Pantev et al., 1996].

There have been few reports of the existence of on/off responses to stimuli in the SI recorded by MEG [Shirai et al., 2004]. Although long onset latencies have been revealed in studies using diaphragms driven by compressed air for mechanical stimulation [Hamalainen et al., 1990; Simoes et al., 2001], offset latencies were not examined in those studies. To our knowledge, both onset and offset latencies have only been described in one study [Tuunanen et al., 2003]. Tuunanen et al. used a device to apply vibration to the subject's palm and fingers, and the results of their study showed that there was no difference between onset and

TABLE IV. Activated area by each modal stimuli (area 3b and area 1)

		area 3b	area 1
Upper extremity	N20m	(+)	(-)
	P30m	(+)	(+)
	C	(+)	(+)
	D	(+)	(-)
Lower extremity	N35m	(+)	(-)
	P40m	(+)	(+)
	C	(+)	(+)
	D	(+)	(-)

(+) indicates the activated area; (-) indicates the inactivated area. C, compression; D, decompression.

offset latencies using vibrotactile stimulation. The discrepancy between their results and our results that showed much shorter offset latencies might be due to the inherently different activations such as central effects on the SI dominated by top-down instead of afferent influences.

N20m and P30m can be recorded more easily in median nerve stimuli than can N35/N35m and P40/P40m in tibial nerve stimuli. The homunculus of the primary somatosensory cortex for the lower limb is located deep in the inhemisphere fissure [Nakamura et al., 1998]. An increase in the distance between the sensors and this area attenuates the strength of magnetic fields. Therefore, in tibial nerve stimuli, only two subjects demonstrated N35m and strengths of ECDs for P40m were much smaller than those for N20m or P30m in our study. The orientations of ECDs for N35m were posterior-to-anterior and the orientations of ECDs for P40m were transverse. A significant difference between orientations of ECDs in electrical and mechanical stimuli suggests that those for mechanical compression have a stronger vector to orient the ECDs transversely. It was also observed that the ECDs for P30m are directed more closely to those for compression stimuli in the upper extremity study. Our P30m, which was analyzed by single dipole fitting, should contain the factor of ECDs for P25m originating from area 1 in Kakigi's study [Kakigi, 1994]. It was thought that the evoked fields are more closely related to area 1 in the order of P40m, compression stimuli and decompression stimuli (Table IV). Thus, the posterior-to-anterior directions of ECDs for N35m suggest that lower limb N35m is equivalent to upper limb N20m and that lower limb P40m is equivalent to upper limb P25m/N35m [Kakigi et al., 1995; Yamada et al., 1996].

In animal studies, experimentally induced lesions of various somatic areas of the cortex proved that area 3b projects to both areas 1 and 2. The projection to area 1 is concerned primarily with texture, whereas the projection to area 2 is concerned with size and shape. Neurons in area 3b receive inputs from rapidly adapting and slowly adapting receptors in the superficial layers of skin [Sur et al., 1984]. Studies on SEFs suggest that N20m is evoked by electrical stimulation

of nerves from rapidly adapting receptors such as Pacinian and Meissner corpuscles [Forss et al., 1994].

The dipoles of the evoked fields elicited by compression and decompression were not identical in terms of their orientations. It was demonstrated that the opposite orientation of ECDs for N20m is close to the orientation of ECDs for decompression stimuli and that the orientation of ECDs for P30m or P40m of the lower extremity is close to that for compression stimuli. These differences in dipole orientations are probably due to the difference in stimulated corpuscles and nerves relayed from different corpuscles. At the peripheral levels, action potentials by decompression have only been recorded by decompression of Pacinian corpuscles [Knibestöl, 1973; Knibestöl and Vallbo, 1970]. Thus, decompression stimulation mainly elicits stimulation of Pacinian corpuscles. The dipole orientations of P40m were significantly different from the localizations of first toe stimulation, and these were directed transversely and from anterior to posterior, respectively. This discrepancy regarding dipole orientation seems to be due to the contribution by the activation of area 1, which was stimulated less in the order of decompression, compression, and electrical stimulations (Table IV).

Such a current flow would agree with activation either of area 3a at the depth of the Rolandic fissure or of area 1. Because lower limb nerves contain more muscle afferents than the median nerve does, muscle afferents might, in principle, contribute to cortical responses after tibial nerve stimulation. It was demonstrated that stimulation of the Achilles tendon, i.e., activation of muscular afferents, can produce prominent evoked responses [Cohen and Starr, 1985]. However, very similar current orientations after stimulation of cutaneous fibers from the sural nerve should exclude the possibility of activations from area 3a [Hari et al., 1996]. Thus, the transverse orientation was considered to be result of gyrus activities of area 1.

In conclusion, we demonstrated that the main signals of ECDs from mechanical stimulations originated from area 3b and that the directions of those ECDs were anterior-to-posterior. The results suggest that electrical stimuli elicit more activation of area 1 than do mechanical stimuli and that these mechanical stimuli elicit more activation of area 3b. Furthermore, because the compression stimuli by human manipulation elicited fast skin surface rubbing sensations, they could activate more neurons in area 1 than could decompression stimuli.

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