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"DOMINANCY" IN THE SECOND SOMATOSENSORY AREA REVEALED BY MAGNETOENCEPHALOGRAPHY

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ABSTRACT

The second somatosensory area (SII) has been studied both by electrical evoked potentials[24] and magnetoencephalography (MEG).[5] Magnetic evoked fields of contralateral primary somatosensory and ipsilateral second somatosensory cortices of 12 normal subjects were recorded in response to median nerve electrical stimulation by means of a single magnetometer. We detected, in addition to the usual contralateral SI responses, ipsilateral second somatosensory evoked fields (sSEF's) in 4 subjects at a latency of 35-45 ms over the dominant hemisphere for handedness, i.e., right hemisphere in left handed subjects and vice versa. On no occasion were ipsilateral somatosensory fields recorded from the non-dominant hemisphere whichever median nerve was stimulated. There were no consistent responses at 90-150 ms. We concluded that the ipsilateral responses were from the SII area.

Our observations indicate the following: a) despite the known bilateral representation of SII, there is hemispheric dominance, b) we could not confirm the long latency (>95 ms) signals reported by Hari et al. in any of our subjects.[5,6]

Keywords: Second somatosensory area, Magnetoencephalography, Evoked magnetic fields.

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INTRODUCTION

The pioneering work of Adrian described the existence of two sensory areas in the cat brain. Experiments revealing the presence of the second sensory area in other mammalian species soon followed.[11,12,17,20,22] However, it was not until 1954 that Penfield and Jasper first described the second somatosensory area (SII) in human beings in their studies of electrical stimulation of perirolandic cortex.[16] In the late 1970's, Woolsey and colleagues, in a detailed study of somatosensory evoked potentials of human cortex, discovered a second sensory area as a result of stimulation of the hand.[24]

Anatomically, in men and monkeys, SII is located in the superior bank of the posterior limb of the lateral fissure adjoining the insula. Evoked potential studies indicate a somatotopic organization of SII with the representation of the face area in the anterior and that of the leg area in the posterior aspects. Animal studies have indicated that both SI and SII are organized somatotopically but the main feature distinguishing them is the activation of a significant proportion of SII neurons by both ipsilateral and contralateral stimuli.[4,19,21,23] The existence of a well-defined, relatively small SII area (less than 2 cm^2) in human cortex

has also been demonstrated using cortical evoked potential and electrical stimulation techniques in an epileptic right-handed woman.[13] Recordings and stimulation were by means of chronically implanted electrodes. The authors reported evoked potentials in the right hemisphere of as early as 28 ms latency in SII on left median nerve stimulation having similar waveforms and delayed by only 2.4 ms with respect to the evoked potentials from SI. They failed to record any ipsilaterally evoked potentials in SII.

It is not ethically possible to implant subdural electrodes in normal human subjects and therefore magnetoencephalography (MEG) has been particularly useful to detect cerebral activity with accurate temporal and spatial localization. MEG detects the evoked magnetic fields arising from the intracellular currents in the apical dendrites of pyramidal cells, especially from those oriented tangentially. Thus it is capable of revealing activation of neuronal populations lying within the sylvian fissure. The first MEG study of SII in man demonstrated responses with latencies between 95 and 125 ms following either ipsilateral or contralateral stimulation of the median nerve.[5] The responses had a mean amplitude of 0.2-0.3 pT (picotesla). A subsequent study confirmed the separation of sources related to different parts of the body.[6]

MATERIALS AND METHODS

Four female and eight male healthy volunteers (Table I) were studied in our eddy-current shielded room.[3] Square wave constant current pulses of 0.3 ms duration were applied to both contra- and ipsilateral median nerves with a stimulus intensity just enough to produce a visible thumb twitch. Stimuli were delivered pseudorandomly with a mean interstimulus interval of 1 second.

MEG signals were recorded over the temporal and parietal areas with a single channel second order gradiometer (BTi 601-10, San Diego). The measurement locations are shown in Fig. 1 and relate to a line drawn at 45° to a line connecting the vertex and the periauricular point intersecting at the C3-C4 positions of the international 10-20 system. Individual recording positions were on a matrix separated by 2 cm. Bandpass filters were set at 0.3 Hz and 100 Hz (-3 dB). The digitizing rate was 10 kHz. One hundred stimuli were averaged for each location on the head and each average was replicated twice to check for reproducibility.

Table I. Sex and handedness of our twelve subjects.

	Handedness	
	Right	Left
Female	3	1
Male	7	1

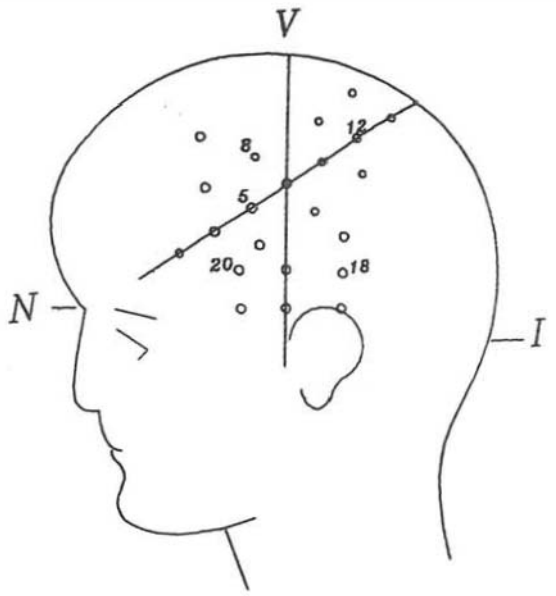

Fig. 1. Measurement locations of left hemisphere. V, vertex or Cz; N, nasion; I, inion.

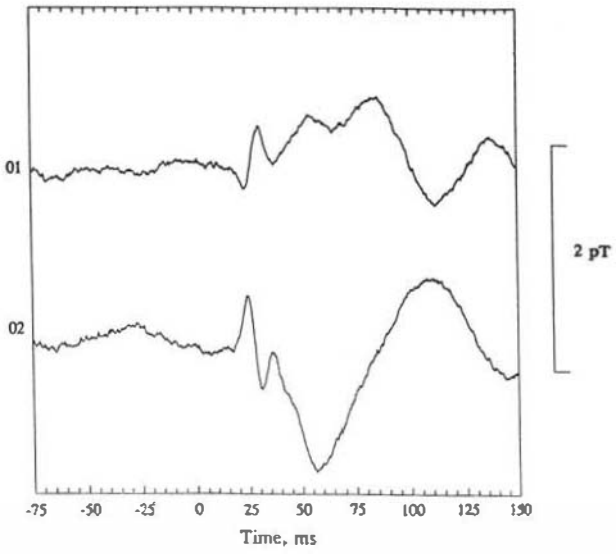

Fig. 2. Contralateral SEF's recorded from points 5 (upper trace) and 12 (lower trace), right hemisphere. Stimulus at time zero. pT, pico Tesla.

Recording of both contralateral primary somatosensory cortices were performed in one session. A mean of 8 locations were recorded from each hemisphere. Contralateral SI evoked fields were recorded from two positions close to the expected extremata for comparison with those from SII (Fig. 2).

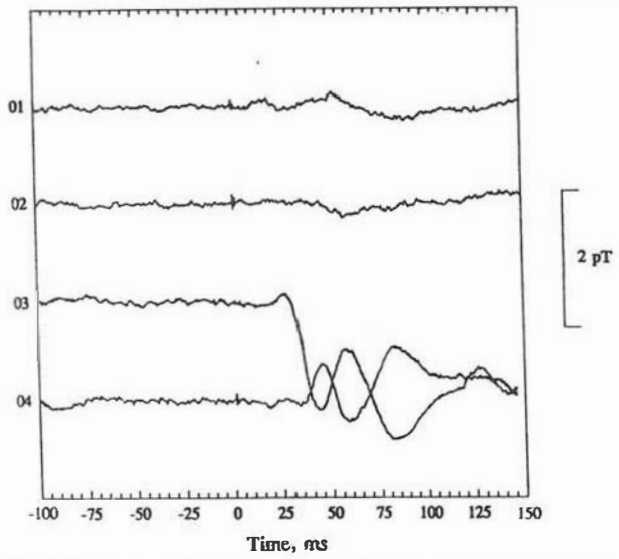

Fig. 3. Ipsilateral sSEF's from subject 01. Trace 1: point 5, left hemisphere (LH); trace 2: point 12, LH; trace 3: point 5, right hemisphere (RH); trace 4: point 12, RH.

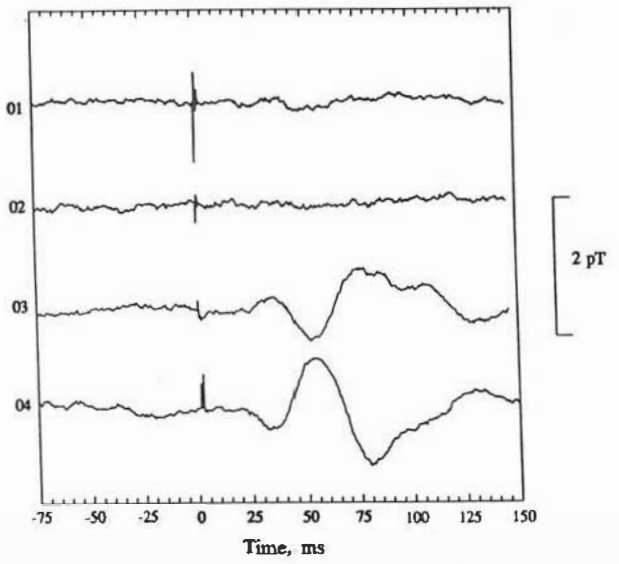

Fig. 4. Ipsilateral sSEF's from subject 03. Trace 1: point 12, right hemisphere (RH); trace 2: point 18, RH; trace 3: point 12, left hemisphere (LH); trace 4: point 18, LH.

Table II. Latencies (in milliseconds) of responses recorded from SII on ipsilateral median stimulations of subjects 1 to 4.

	Subjects			
	Female		Male	
sSEF's	No1	No2	No3	No4
First	44	40	38	45
Second	57	55	53	-
Third	-	69	-	-
Fourth	85	87	83	-

RESULTS

Only four of twelve subjects studied showed reproducible waveforms over the ipsilateral hemisphere following stimulation of the median nerve. Although responses from contralateral SII were expected to be larger in amplitude, we did not attempt to record from that location, as evoked magnetic fields from SI contaminate those from SII.[8] SII ipsilateral neuromagnetic fields were recognizable at four latencies: wave 1, 38-45 ms; wave 2, 53-57 ms; wave 3 (one subject), 69 ms and wave 4, 83-87 ms (Table II). Figure 3 demonstrates typical ipsilateral responses from the right hemisphere of subject 1, a left handed female, peaking at 44 ms, 57 ms and 85 ms latencies. The extremata were near points 5 (negative) and 12 (positive) (Fig. 1) reversing across the line from Cz to the periauricular point. No ipsilateral second somatosensory evoked fields (sSEF's) were detected from her left hemisphere. The other three subjects, all right handed, showed responses of similar latencies, signs of which reverse across the 45° angle line rather than this vertical line. No sSEF's were detected from their right hemispheres. Figure 4 shows responses from subject 3. The extremata of responses from subjects 2 were near points 8 and 20 and for subjects 3 and 4 were near 12 and 18.

DISCUSSION

There was considerable inter-subject variability of responses. In 8 subjects the responses were absent. The highly localized nature of the source may be responsible for the latter, compounded by the limitations of recording with a single channel neuromagnetometer and the locating of a source by a matrix related to external fiduciary points.[13] Individual variability of source location and orientation when responses were detected may also be dependent on this procedure. Lüder and co-workers demonstrated that contralateral SII evoked potentials from the right hemisphere disappeared completely in all electrodes located over the proposed SII area (electrode centers separated by 1 cm) indicating the smallness of the source.[13] However, they failed to record ipsilateral SII evoked potentials. One reason might be the fact that low amplitude ipsilateral somatosensory evoked potentials are easily abolished with relatively light anesthesia.[21]

Since there is no anatomical evidence for projection of sensory afferent fibers to ipsilateral SI in mammals, the latencies and orientation of dipoles in our 4 subjects were suggestive of activation of SII. The latencies of the first detectable SII responses are some 10 ms later than the arrival of the thalamocortical volley in the contralateral SI. This raises several alternative explanations. First, the ipsilateral activation may follow the contralateral SI with

the additional time taken by the passage of the volley across the corpus callosum. There is however anatomical evidence in cats and primates of direct projection from thalamus to SII.[2,9,10,14,15] Thus the pathway could be direct, but delayed by intranuclear synaptic transmission in the ventral thalamus, or conduction of impulses to SII in a small fiber (and low conduction velocity) afferent system as suggested by Jones and Powell in the Rhesus monkey.[10] Thirdly, with the evidence from Lüder and colleagues of a response in SII recorded electrically 2.4 ms after SI, we must consider that the source from which he was recording was either too small and deep to be detected by our neuromagnetometer, or with a radial orientation which would produce no net magnetic flux in the plane of our gradiometer.[13]

Hari and colleagues reported no early waveforms of the type and latency reported by ourselves, but consistent high amplitude waves at 100 ms after stimulation.[5,6] We have never seen similar waveforms in any of our studies, although at 100 ms a small amount of magnetic flux is detectable, it did not have a dipolar distribution.

Knowledge of the functions of SII is limited at present. If our observations of the correlation of ipsilateral responses in SII with cerebral dominance are confirmed by studies of a larger group of subjects, a new line of research into factors influencing cerebral dominance can begin with a relatively simple neuromagnetometer.

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