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Research report

Interaction between afferent input from fingers in human somatosensory cortex ☆

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Abstract

We recorded somatosensory evoked magnetic fields from eight healthy subjects with a 122-channel whole-scalp SQUID magnetometer. The stimulus sequence consisted of 'standard' stimuli (85%) delivered to palmar side of the left thumb with an interstimulus interval of 0.6 s and of 'deviants' (15%), randomly interspersed among the standards, to little finger, and vice versa. Both stimuli activated four source areas: the contralateral primary somatosensory cortex (SI), the contra-and ipsilateral secondary somatosensory cortices (SII), and the contralateral posterior parietal cortex (PPC). The short-latency (20–40 ms) responses originated in the SI cortex, whereas long-latency responses arose from all 4 areas. At SII and PPC, the deviant stimuli elicited larger responses when presented alone, without intervening standards, than among standards. This implies interaction between afferent impulses from the two fingers and/or partly intermingled cortical representations. Our findings show, in agreement with animal data, different excitatory/inhibitory balance in the various somatosensory areas.

Keywords: Somatosensory cortex; Magnetoencephalography; Evoked response; Median nerve; Ulnar nerve; Human

1. Introduction

Several areas of the human cerebral cortex process somatosensory information. The primary somatosensory cortex (SI) is located in the central sulcus and postcentral gyrus. All Brodmann's areas in SI (3a, 3b, 1, and 2) have distinct somatotopic maps, in which different body parts are represented in proportion to the density of their innervation [24]. The bilaterally activated SII cortex in the parietal operculum is considerably smaller than SI which, together with its location, makes it a difficult target to investigate. Animal studies have suggested existence of additional somatosensory areas in the vicinity of SII, but boundaries between them are difficult to determine. The SII cortex shows signs of somatotopical organization as well [16,18].

Neuromagnetic signals from the SI cortex and the SII region in the parietal operculum can be easily differentiated [19,20]. Recently an additional source area to tactile stimulation was identified in the human posterior parietal cortex (PPC), in the wall of the postcentral fissure, probably reflecting activation of cytoarchitectonic areas 5 or 7 [10]. In monkey, this region is best activated by complex stimuli, and it combines proprioceptive and tactile information from different body parts [23].

Different functional roles of the somatosensory areas propose different degrees of convergence of afferent signals from various body parts. Animal studies have shown that receptive fields of neurons in SII area and PPC are usually considerably larger than in SI, and the receptive field of a single neuron in PPC may include entire limb [7,23]. The aim of the present study was to investigate convergence of impulses from median and ulnar nerves at the human SI and SII cortices, and at the PPC, to obtain information about functional organisation of these somatosensory areas.

2. Materials and methods

Somatosensory evoked fields (SEFs) were recorded from eight healthy laboratory members (5 females, 3 males, ages 22–34 years, 1 left-handed). During the recording,

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the subject was sitting comfortably in a magnetically shielded room with the head supported against the helmet-shaped sensor array of the magnetometer. Subjects were instructed to relax the stimulated hand, to support it on the elbow rest of a chair, and to ignore the stimuli; the subjects were asked to read a self-chosen book. The palmar sides of the distal left thumb and little finger were stimulated with 0.3-ms constant current pulses, delivered with bipolar electrodes (pad separation 25 mm) to glabrous skin of the fingertips. Stimulus intensity varied from 5 to 10 mA among subjects, and it was adjusted to produce subjectively equally strong sensation in thumb and little finger. The intensity was kept fixed after initial adjustment throughout the measurement session. The stimulus sequence consisted of 'standard' stimuli delivered to the thumb (85% probability) and 'deviant' stimuli (15%), randomly interspersed among standards, to the little finger, or vice versa. The interstimulus interval (ISI) was 0.6 s for standards and on average 4 s for deviants (range 1.8–12 s). In another stimulus sequence (referred in the text as 'deviants alone'), the standards to thumb were omitted and only the deviants to the little finger were delivered. This means that 'deviants' and 'deviants alone' were exactly the same in site and timing; the only difference between the conditions was that standard stimuli were delivered to thumb between the 'deviants' but not between the 'deviants alone'. This kind of stimulus set-up allowed to observe if the intervening standards had any effect on responses to deviant stimuli; any difference between responses to 'deviants' and 'deviants alone' would indicate interaction between impulses from the two fingers.

In control experiments, the effect of the stimulation side (left vs. right hand) was studied in one subject, and for another subject, deviants were presented to the middle finger instead of the little finger to replicate an earlier experimental setup [20]. Moreover, to clarify the effect of ISI on SEFs, responses were recorded from one subject to thumb stimulation with constant ISIs of 0.6, 1, 2, 3, and 4

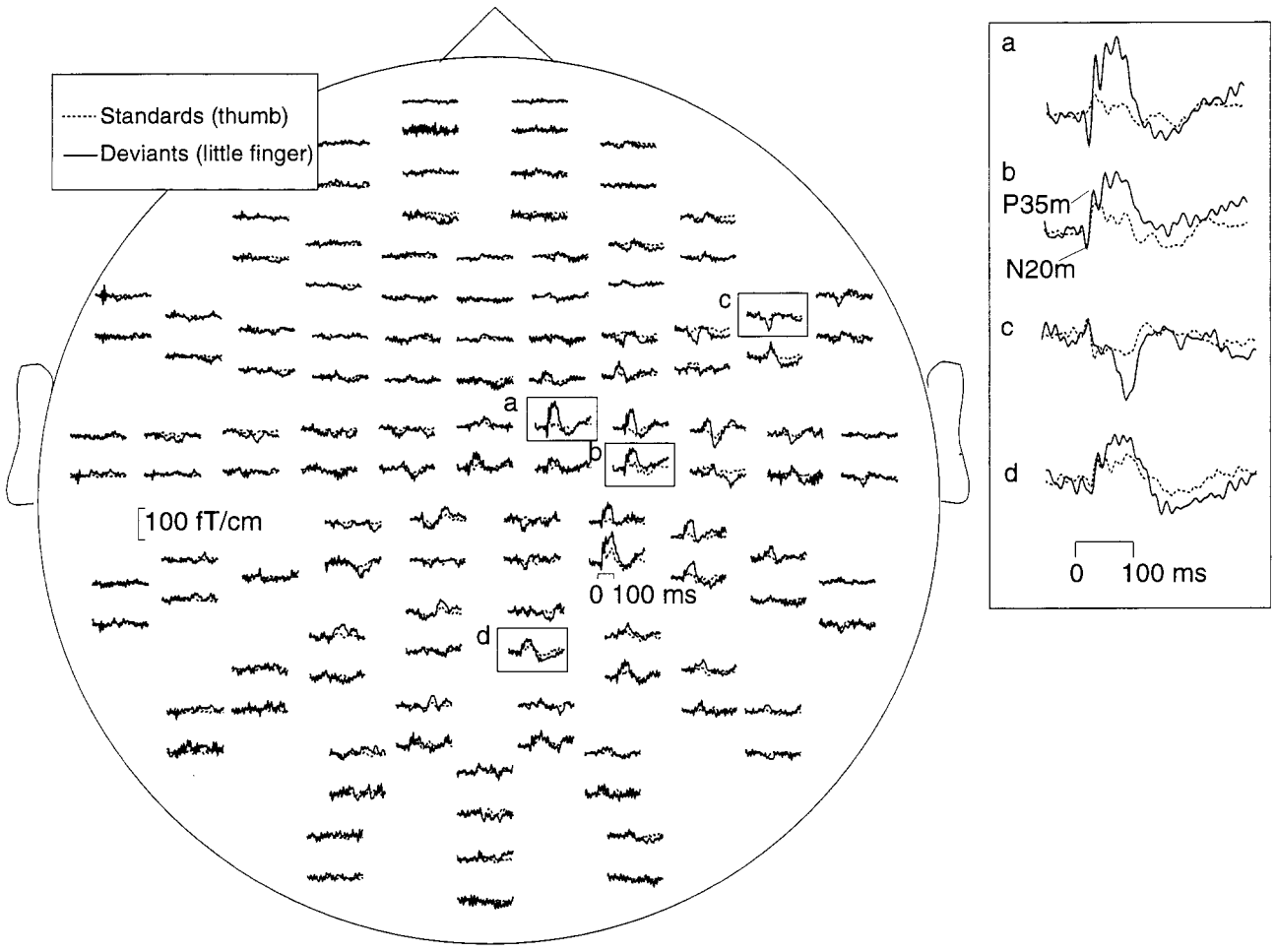

Fig. 1. SEFs of Subject 5 to standards delivered to thumb and to deviants delivered to little finger. The head is viewed from the top, and in each response pair, the upper trace illustrates the field derivative along the longitude (positive signal from vertex to decreasing latitude) and the lower trace along the latitude (positive signal counterclockwise). The insert shows enlarged responses from two channels over the contralateral SI hand area (a,b), contralateral SII area (c), and posterior parietal cortex (d). Note that responses to thumb and little finger stimulation have maximum in different channels.

s. To monitor possible changes in the subject's state, responses to same stimuli were recorded in the beginning and in the end of the recording session.

SEFs were recorded with a helmet-shaped Neuromag-122™ magnetometer array which has 122 planar first-order SQUID gradiometers, placed at 61 measurement sites [1]. The planar gradiometers detect the largest signal just above the local source area, where the field gradient has its maximum. Each sensor unit contains a pair of gradiometers that measure 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 magnetic signals produced by currents in 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 alignment of the MEG and magnetic resonance (MR) image coordinate systems.

The whole-head MEG allows differentation between several simultaneously active cortical areas if the distance between the areas exceeds 2–3 cm; two nearby sources can be separated even better if orientation of the sources is different, as is the case with SI and SII cortices. For further technical details of the MEG method and MEG/MR integration, see [17]. MR images of 4 subjects were acquired with a 1-T Siemens Magnetom™ system with MPR3D sequences. A set of 128 coronal slices (thickness 1.3 mm) was used for rendering a 3D-reconstruction of the brain's surface [31].

The signals were bandpass filtered (0.03–190 Hz), digitized at 0.6 kHz, and about 650 single responses to standards and 150 to deviants were averaged on-line. The analysis period of 400 ms included a prestimulus baseline of 50 ms. Responses with amplitudes exceeding 150 μV in the simultaneously recorded vertical electro-oculogram (EOG) were automatically rejected from the analysis.

To identify sources of the evoked responses, the signals were divided into several time periods, during each of which one equivalent current dipole (ECD), best describing the most dominant source, was first found by a least-squares search using a subset of channels over the response area. These calculations resulted in the 3-dimensional location, orientation and strength of the ECD in a spherical conductor model. The ECDs were then superimposed on subjects' MRI to show the source locations with respect to anatomical structures.

Goodness-of-fit (g) of the model tells in percentage how much the dipole accounts for the measured field variance. Only ECDs explaining more than 80% of the

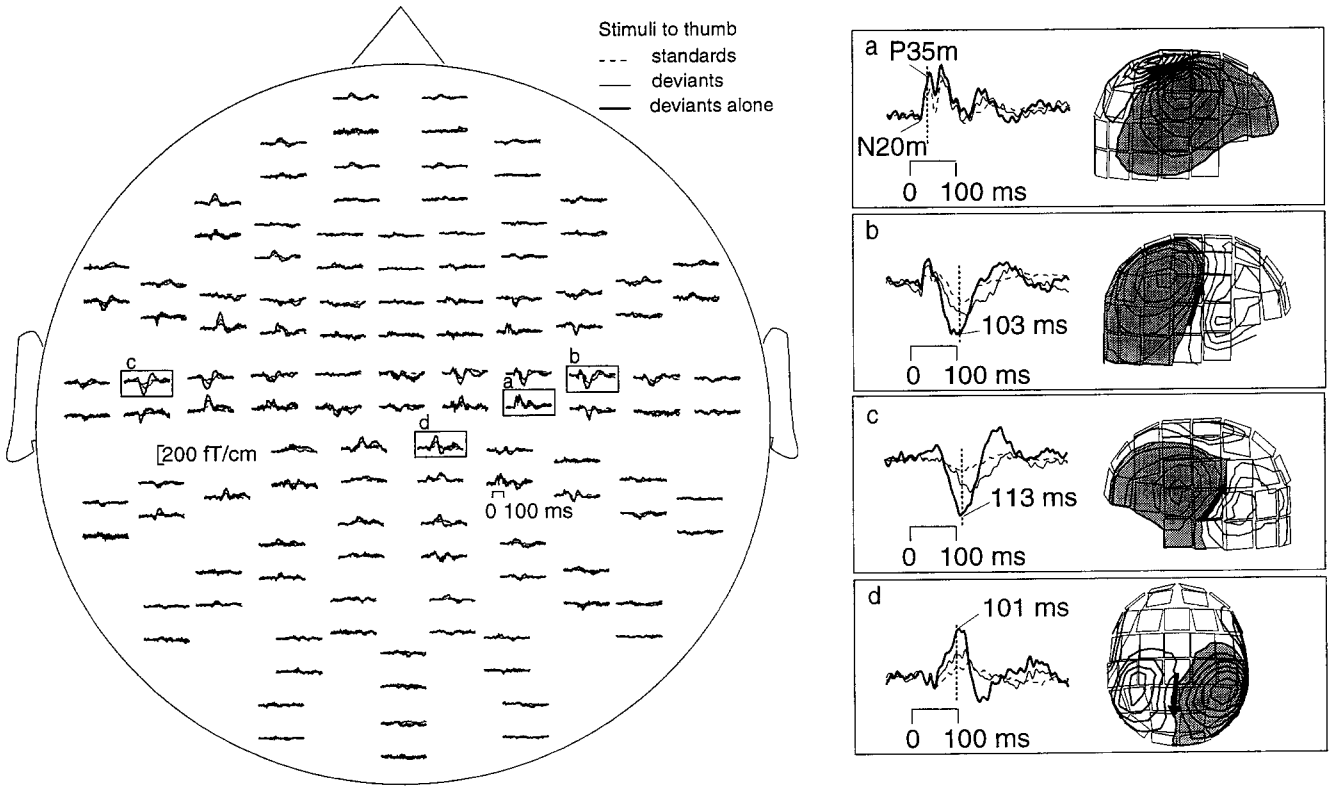

Fig. 2. SEFs of Subject 4 to thumb stimuli presented as standards, deviants and deviants alone. The inserts show enlarged responses at different moments of time, and field patterns to standards. The sensor array is viewed from right (a,b), left (c), and top (d). The inserts show the field patterns at the peaks of the responses (indicated with dashed vertical lines). The squares show the locations of the sensor units, and the arrows indicate the ECDs. The isocontours are separated by 40 fT, and the shaded areas indicate the magnetic field emerging from the head.

field variance at selected periods of time in the subset of 16–18 channels were used for the further analysis. Thereafter, the entire time period and all channels were taken into account in computing a time-varying multi-dipole model. For this, the strengths of the previously found ECDs were allowed to change while their locations and orientations were kept fixed.

3. Results

Fig. 1 shows SEFs of Subject 5 to left-sided standards (thumb) and deviants (little finger). The responses are stronger to deviants than to standards, but the morphology is quite similar. The earliest deflections, N20m and P35m (enlarged in insert a and b), peak over the anterior parietal cortex at 22 and 39 ms to standards, and at 23 and 38 ms to deviants, respectively. The largest N20m occurs more medial to little finger than to thumb stimuli. Long-latency responses peak over the right temporal lobe at about 100 ms (insert c); on the homologous ipsilateral side the activation is neglible in this subject. An additional deflection is seen over the right posterior parietal cortex at about 90 ms (insert d), medial and posterior to the approximated SI hand area. Responses in the beginning and in the end of the session were the same, indicating that the subject's state did not affect them.

Fig. 2 shows SEFs of Subject 1 to thumb stimuli when they were presented in different sessions as standards, deviants, and deviants alone. The waveforms are rather similar in all three conditions, but the amplitudes vary according to the condition; all deflections after N20m are

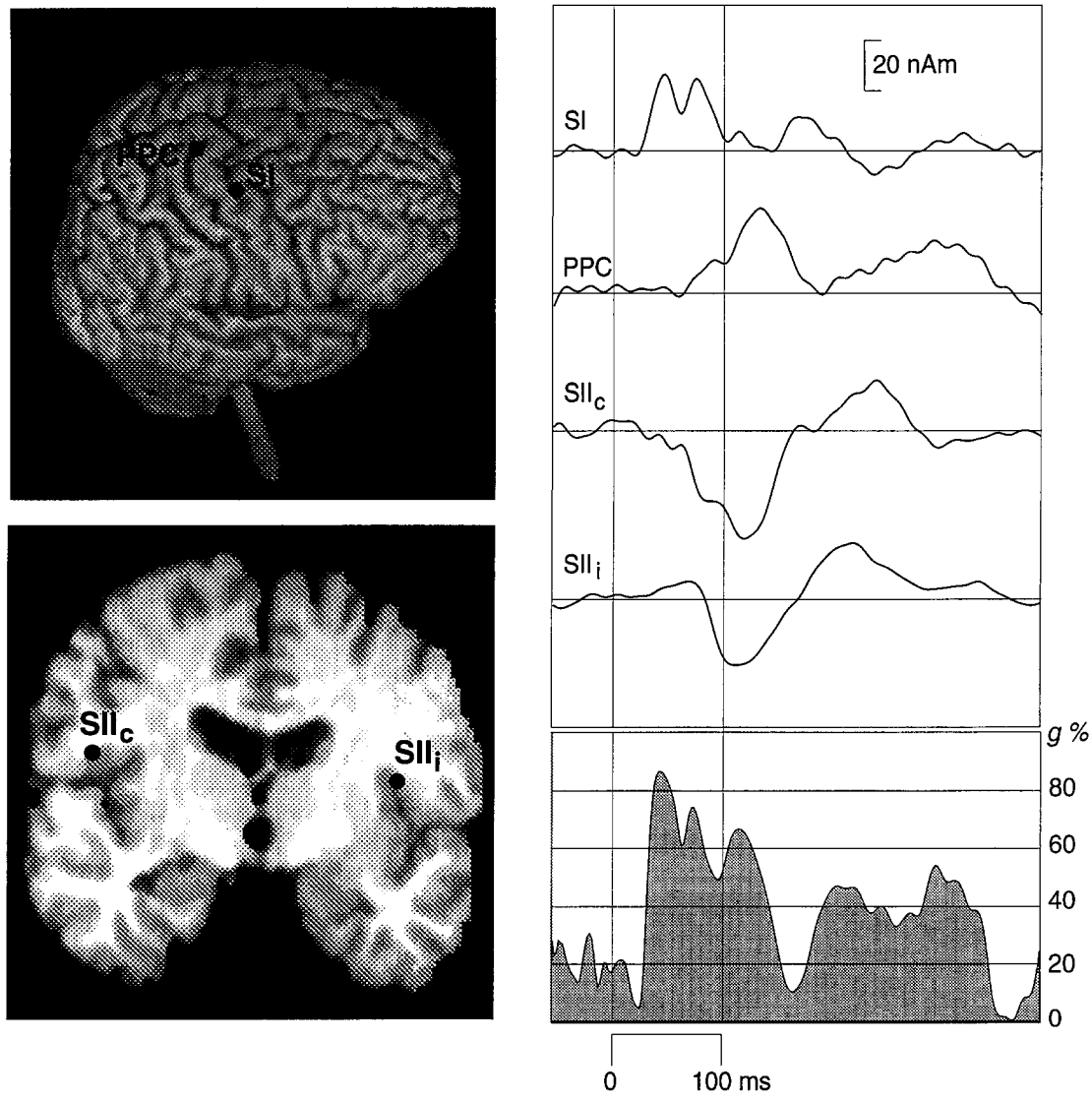

Fig. 3. Left: ECDs of Subject 1 superimposed on his MR images. ECD locations (indicated with black spheres) in SI (P35m) and PPC areas are projected to the surface of the brain in viewing direction. Coronal section shows locations of ECDs in SII area. Right: strengths of the sources as a function of time in the 4-dipole model. The lowest part indicates the goodness-of-fit of the model (SII_c = contralateral SII area, SII_i = ipsilateral SII).

larger in amplitude to deviants than to standards. Further, the long-latency responses (after P35m), peaking at 103–132 ms over the temporal regions and at about 100 ms over the posterior parietal cortex, are stronger to deviants alone than to deviants.

The insert of Fig. 2 shows enlarged responses and the field patterns at different latencies. During the earliest deflections the patterns agree with activation of the contralateral SI hand cortex (insert a). The ECDs for longer latency responses over temporal lobes suggested activation of the contra-and ipsilateral SII areas (inserts b and c). The fourth ECD agreed with activation of the PPC (insert d). Every subject had somewhat different response distribution (cf. Figs. 1 and 2), because of both interindividual variation in source sites and slightly different head positions inside the sensor helmet.

Fig. 3 (left) shows the four source areas superimposed on the subject's MR images. ECD for the early response (P35m in the figure) is located in the SI cortex in the bank of the central sulcus. The posterior dipole (at 101 ms) in the wall of the postcentral fissure suggests activation of the PPC. The two temporal ECDs originate in the depths of the Sylvian fissures implying activation of the SII region in the parietal operculum.

Fig. 3 (right) shows the strengths of these four ECDs as a function of time to standard thumb stimuli. The strengths of the dipoles do not interact, implicating proper modelling of the active brain areas. The four ECDs explain the whole-scalp data (all 122 channels included) best during the first 150 ms.

Fig. 4 shows responses of all subjects in the three stimulus conditions, both to thumb and little finger stimulation. Waveform configurations and, to a lesser extent, the temporal patterns of the responses are variable between different individuals, probably reflecting both the anatomic and functional differences. N20m is of same amplitude in different conditions, whereas P35m is larger to deviants than to standards. The long-latency responses over SII and

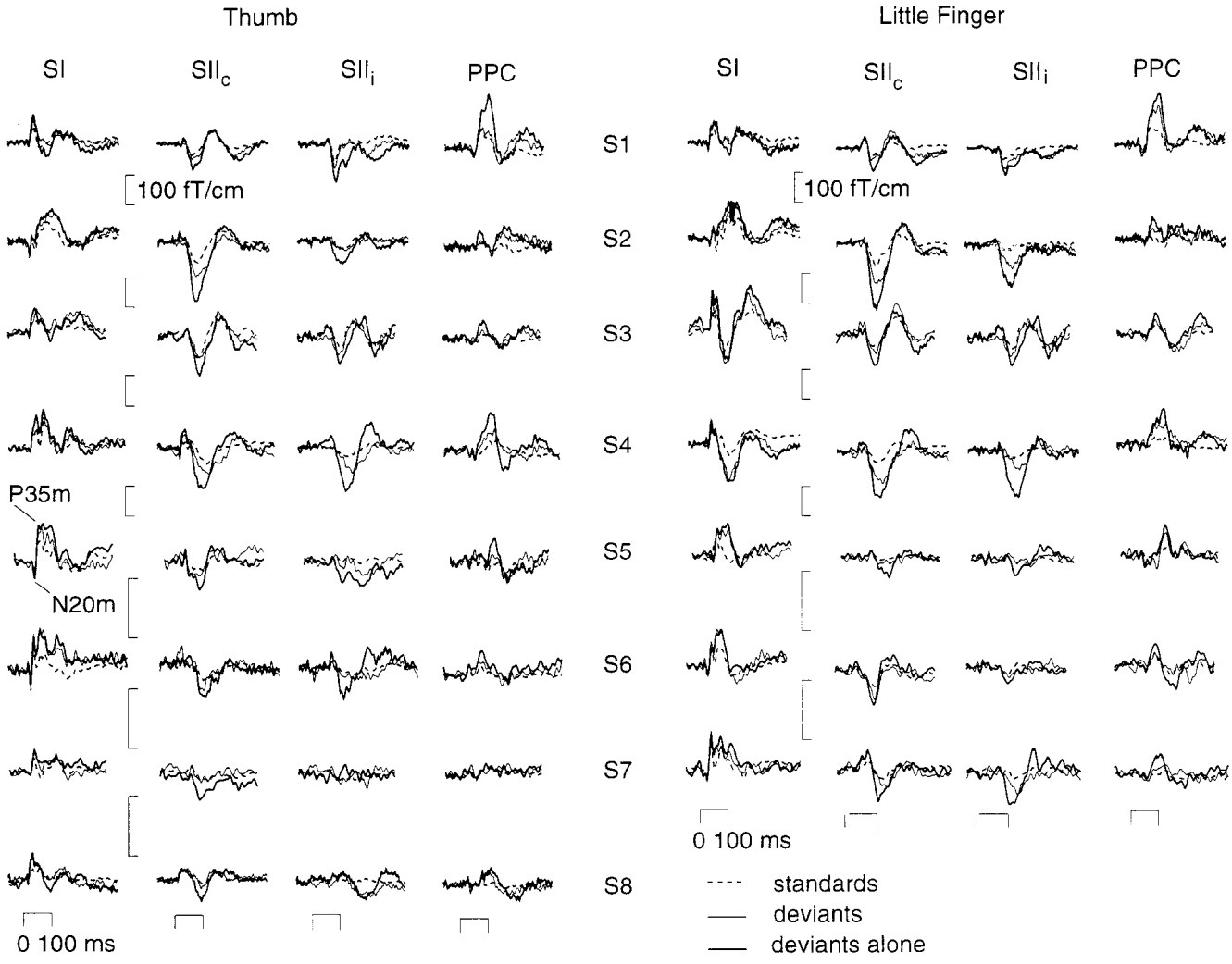

Fig. 4. Responses of all subjects (S1–S8) to thumb and little finger stimuli when they were presented as standards, deviants, and deviants alone. Signals are shown from the channel with the largest response over the (right) SI cortex, both SII cortices and PPC.

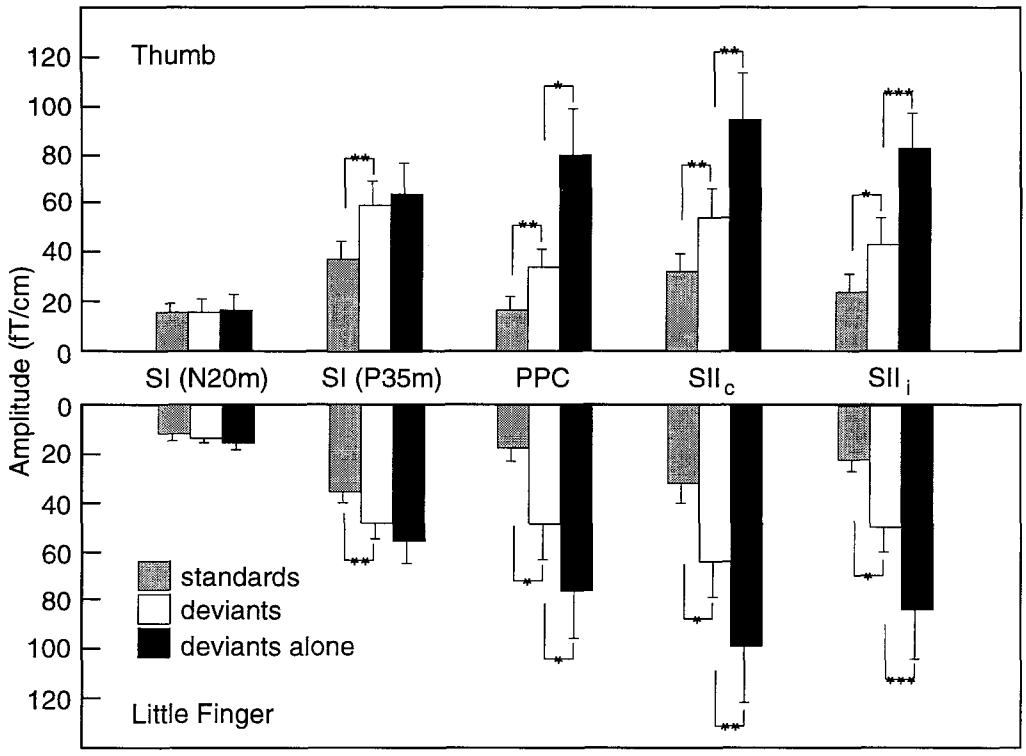

Fig. 5. The mean (±S.E.M.; 8 subjects) amplitudes of responses from all source areas to thumb (upper part) and little finger (lower part) stimuli in the three conditions. Statistical significance in paired two-tailed *t*-tests is indicated (*** $P < 0.0005$; ** $P < 0.005$; * $P < 0.05$).

PPC are largest for deviants alone and smallest for standards.

Fig. 5 shows the mean (±S.E.M.) response amplitudes over the four source areas in all stimulation conditions. The responses to thumb and little finger stimuli behaved in a similar fashion in the three conditions. N20m was of about equal amplitude to standards and deviants, whereas P35m and the long-latency responses from areas outside SI were clearly stronger to deviants than to standards. The long-latency responses from SII and PPC were significantly larger to deviants alone than to deviants among standards; no such difference was seen for N20m nor P35m. The statistical significance of the differences is indicated in the figure. The response latencies were not significantly different between the three conditions in any areas.

The ECD locations did not depend on the stimulation condition. Fig. 6 shows the mean locations of ECDs for all

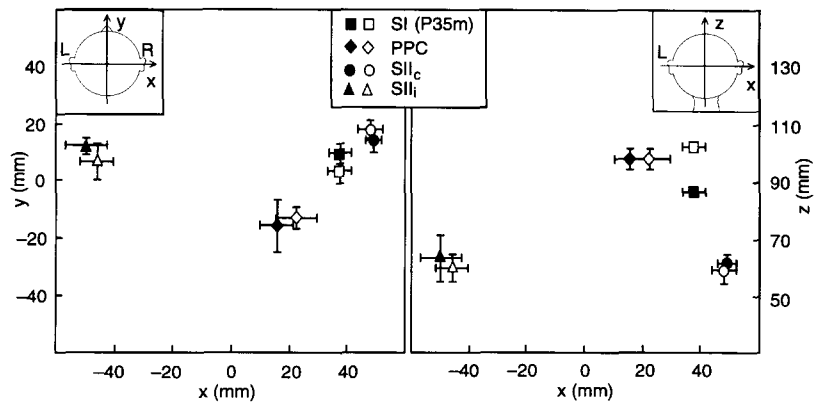

Fig. 6. The mean (±S.E.M.) locations of ECDs to standards delivered to thumb and little finger. Responses from SI (P35m), SII and PPC are indicated by squares, triangles, and spheres, respectively. Black symbols represent responses to thumb and white to little finger stimulation. The coordinate system is based on external landmarks of the skull; the *x*-axis goes through preauricular points from left to right, *y*-axis from the back of the head to nasion, and *z*-axis points to the vertex (see the inserts).

subjects to standard stimuli. N20m could not be reliably located in two subjects because of the low signal-to-noise ratio, and therefore N20m locations are not included in the figure. At the contralateral SI, the ECD for P35m was on average 1.5 cm more superior to little finger than thumb stimuli; the difference was statistically significant ($P < 0.001$). At SII and PPC, the ECD locations did not differ between thumb vs. little finger stimuli. The PPC source was always contralateral to stimulation, about 2 cm medial and 2 cm posterior to the P35m source.

In the control measurement, the increase of ISI from 0.6 s to 4 s did not affect N20m but P35m was significantly enhanced, as were also the SII and PPC responses. In a further control measurement in one subject the responses behaved in a very similar manner, independently whether the deviants were presented to the right or left hand, or to the little or middle finger.

4. Discussion

In agreement with our recent studies on median nerve SEFs [10], electric stimulation of the thumb and little finger elicited clear and replicable SEFs at four brain areas: the contralateral SI cortex, the left and right SII cortices, and the contralateral posterior parietal cortex.

Due to the close vicinity of the sensory and motor cortices in the opposite walls of the central sulcus, contribution from motor cortex activity to early SEFs is difficult to rule out. However, the present and previous MEG recordings from our laboratory strongly suggest that N20m and P35m have their origin in the SI cortex. Moreover, recent comparison of early median-nerve evoked responses recorded with electrocorticography, scalp-EEG and MEG showed that one tangential source in the posterior wall of the central sulcus and one radial source in the the anterior crown of the postcentral gyrus were sufficient to explain a large majority ($> 85\%$) of the responses [6]. The present study showed that long-latency responses arise from areas outside SI, as well.

In the auditory modality, infrequent deviant stimuli among frequent standard sounds elicit a specific deflection called the mismatch field. In the present study, the response waveform and location was very much the same to standards and to deviants, and in agreement with a previous MEG study [22], no extra deflections to deviants were observed. Therefore, the difference between responses to deviants and standards is probably due to refractoriness of the neural generator.

Somatosensory evoked responses are known to increase in amplitude as a function of ISI [2,29]. In the auditory modality, such a behavior has been interpreted to reflect varying lifetimes of sensory memory traces at different brain areas [25]. At very short ISIs, the recovery cycle of the afferent pathways, or of cortical cells, determines the response amplitude of short-latency responses [27]; the long-latency responses are conveyed through polysynaptic pathways, and their recovery cycles are considerably longer, up to several seconds in SII and PPC [10,20]. In paired stimulus presentation, the N20m response to median nerve stimulation is suppressed at ISIs shorter than 120–150 ms [21], probably due to infield inhibition [12], which seems to have a very similar time course as lateral inhibition, both of the order of the duration of one inhibitory postsynaptic potential.

The ISI of 0.6 s, used in the present study, clearly exceed the recovery time of N20m and, consequently, no differences were detected in N20m between the 3 conditions. In line with the different ISIs for standards (0.6 s) and deviants (on average 4 s), all responses after N20m were significantly larger in amplitude to deviants than to standards. However, P35m was larger to deviants than to standards, but was not increased further when deviants were presented without intervening standards. Since any difference between responses to deviants and to deviants alone (both presented with identical ISIs) would indicate interaction between the impulses coming from the two fingers, these results imply no such interaction for N20m nor for P35m at the ISI used.

The long-latency responses after P35m were stronger to deviants alone than to deviants, implying interaction between afferent signals from the stimulated fingers or intermingled cortical representations. The differences in amplitudes between deviants and deviants alone in various areas possibly reflect different degrees of interaction of afferent signals in these areas.

The conclusion about partly intermingled representations of different fingers in SII and PPC areas contradicts an earlier comparable study of Hari et al. [20], which revealed no significant differences in responses to deviants and deviants alone. However, the conclusion was based on the data of one subject only; thus individual differences in degree of interaction between afferent impulses, evident also in the present study, may explain the variation in results. Attention may have also affected the amplitude of the responses; the subject of Hari et al. [20] was highly attentive, whereas in the present study the subjects were reading a book to ignore the stimuli.

Serial vs. parallel cortical processing of sensory information has been recently under extensive debate. Deactivation of SI in cats or prosimian primates had no notable effect on the responsiveness of SII neurons to cutaneous stimuli [9,14,26]. Nevertheless, ablations of SI, and even areas 3a and 3b alone, totally deactivated the SII area in monkeys, implying serial processing of the somatic information [15,28]. However, recent studies by Turman et al. [32] and Rowe et al. (1994, personal communication) suggest that tactile input can reach SII directly from thalamus both in cats and in monkeys, and that SI may have a backround facilitatory influence on the function of SII.

In the present study, no early activation was detected in

SII area although short-latency activity has been observed in cat SII (e.g. [5]). Neither have short-latency SEPs been observed from human parietal operculum in scalp or intracranial recordings, although longer-latency potentials attributed to SII are well discernible [3,4]. These findings support serial flow of tactile information in human SI and SII areas.

Varying duration of interaction in different somatosensory areas is reasonable for the functional role of these regions. SI as a primary cortical station of tactile input has a distinct somatotopic arragement which was seen also in the present study as clearly different ECD locations for thumb and little finger. SI is likely to code in detail the stimulus type and location, further improved by lateral inhibition if impulses from nearby body parts occur within a short interval. The bilaterally activated SII areas are probably involved in tactile learning and memory [13], functions which necessitate intermingled representation of digits. PPC as a part of the association cortex probably integrates information from different body parts, and accordingly had signs of intermingled finger representation. Convergence of afferent impulses from different fingers and even from larger body parts to single neurons in SII and PPC has been observed previously in monkeys [8,23]. The present results support these findings and suggest similar convergence of tactile information in humans. Whisker stimulation in rat elicites both excitatory and inhibitory optical signals in SI, but only exitation in SII [30]. Accordingly, our results suggest different excitatory/inhibitory balance in the various somatosensory areas [18].

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