Timing and Spatial Distribution of Somatosensory Responses Recorded in the Upper Bank of the Sylvian Fissure (SII Area) in Humans

We studied responses of the parieto-frontal opercular cortex to electric stimuli, as recorded by intra-cortical electrodes during stereotactic EEG presurgical assessment of patients with drug-resistant temporal lobe epilepsy. After electrical stimulation of the median nerve at the wrist, we consistently recorded a negative–positive biphasic response peaking at 60 ms (N60) and 90 ms (P90) post-stimulus in the upper bank of the sylvian fissure contralateral to stimulation. Talairach stereotactic coordinates of the electrode contacts recording these responses covered the pre- and post-rolandic part of the upper bank of the sylvian fissure (25 < x < 55 mm; −27 < y < +13 mm; 0 < z < 21 mm), corresponding to the accepted localization of SII area in man. The sources of these responses were deeply situated in the cortex of the upper bank of the sylvian fissure at ∼40 mm from the midline sagittal plane, so that some of them could be located in the insular cortex. Moreover this study suggests the existence of dipolar SII sources radial to the scalp surface, which are overlooked in magnetic recordings. Somatosensory evoked potentials (SEPs) recorded in SII are delayed by ∼40 ms as compared with SEPs generated in the primary somatosensory cortex. This long delay between SI and SII responses is not fully explained though it is coherent with the timing of activation issued from MEG source modeling data.

Introduction

Based on numerous anatomical and microelectrode studies in monkeys (Burton, 1986; Burton et al., 1995; Krubitzer et al., 1995), it is now well accepted that the second somatosensory area plays a major role in the processing of somatosensory inputs as part of a distributed cortical network which includes the post-central gyrus (SI), the parietal operculum (SII), the posterior parietal and granular prefrontal cortices, and the limbic areas (Jones and Powell, 1970; Hyvärinen, 1982; Kaas and Pons, 1988; Preuss and Goldman-Rakic, 1989). Corresponding knowledge of the functional anatomy of the SII area in humans is limited by the difficulty in assessing, with adequate temporal and spatial resolution, the sequential activation of cortical areas during processing of afferent somatosensory inputs. Somatosensory evoked potential (SEP) studies have brought consistent and converging information on location and inputs. Somatosensory evoked potentials (SEPs) recorded in SII are delayed by ∼40 ms as compared with SEPs generated in the primary somatosensory cortex. This long delay between SI and SII responses is not fully explained though it is coherent with the timing of activation issued from MEG source modeling data.

Materials and Methods

Patients

We recorded median SEPs from 16 patients (22–47 years, mean age 33 years; 10 females, 6 males). All patients included in this study presented with refractory temporal lobe epilepsy and were investigated using stereotactically implanted intracerebral electrodes before surgical surgery. All patients had electrodes chronically implanted in the parieto-rolandic opercular cortex (one or two electrodes per patient with 10 or 15 contacts per electrode). The decision to explore this area resulted from the observation of ictal symptoms suggesting the possibility of a suprasylvian and/or insular spreading of seizures (lip and face paresthesiae or tonic–clonic movements, laryngeal contraction, gustatory illusions, hypersalivation). Patients were fully informed of the stimulation paradigm and of the aim of SEP recording which is part of the routine functional mapping performed prior to surgical cortectomy in our department. Auditory EPs were recorded in most patients and stimulations of functionally eloquent areas were performed (hippocampus, language areas, motor strip, etc.) depending upon the localization of implanted electrodes. No clinical somatosensory testing was performed with intracortical electrodes in place, but all patients had normal sensation before and after stereotactic EEG (SEEG) exploration (touch, two-point determination, pain and temperature, tactile recognition of objects).

Patients included in this study were those for whom, after the SEEG investigation, we did not observe early spreading of low-voltage fast ictal activity in the parieto-rolandic operculum during ictal discharges. Moreover, no sustained after-discharges was elicited by electrical stimulation of this area. Only late clonic spike wave activity was observed in the operculum during the spread of ictal discharges in some of these patients. Therefore, one can assume that the opercular area explored did not show abnormal hyperexcitability and was not involved in the seizure triggering. This is confirmed by the fact that the cortectomy involved the amygdala, hippocampus and temporal pole but spared the suprasylvian cortex in all operated patients (15) and was followed by disappearance of
seizures (Engel's class Ia) in the 12 patients with a post-operative follow-up of >1 year.

Implantation of SEEG Electrodes

Intracerebral electrodes were implanted using Talairach’s stereotactic frame. As a first step, a cerebral angiography was performed in stereotactic conditions using a X-ray source located 4.85 m from the patient’s head, thus eliminating the linear enlargement due to X-ray divergence, so that the films could be used for measurements without any correction. In a second step, the pertinent targets were identified on the patient’s magnetic resonance (MR) image, previously enlarged at scale 1. As MR and angiographic images were at the same scale of 1, they could easily be superimposed, thus minimizing the risk of any damage to cerebral veins or arteries during implantation. The electrodes were then orthogonally implanted using Talairach’s stereotactic grid. Each electrode had 10–15 contacts, each 2 mm long, separated by 1.5 mm; they could be left in place chronically up to 15 days. Because of the physical characteristics of the contacts (stainless steel), it was impossible to perform MRI with electrodes in place. The stereotactic coordinates of electrode positions were those preoperatively calculated on MRI for each target using scale 1 skull radiography superimposed to scale 1 angiography. It was thus possible to check that the depth coordinates (z) of each contact were those preoperatively calculated on MRI for each target using scale 1 skull radiography superimposed to scale 1 angiography. In each individual patient. Apart from the pre- and/or post-rolandic opercular cortices, the most commonly investigated targets were the hippocampus, the amygdala, the temporal pole, the temporal neocortex, the cingulate gyrus and the orbitofrontal cortex. Since there was no patient in whom the epileptic discharges were suspected to spread to the perirolandic cortex, the primary somatosensory area (SI area) was not explored. Electrodes were implanted in the parieto-occipital region caudal and rostral to the VAC plane (p = 0), which in most subjects corresponds to the end of the rolandic fissure on the outer aspect of the hemisphere. Eight patients were implanted by a single opercular electrode exploring either the frontal (six cases) or the parietal (two cases) suprasylvian cortex; in eight patients both of these regions were explored. Therefore the electrode tracks and the contacts of each electrode were plotted onto the appropriate MRI slices of each patient, and we could localize each contact on MRI by calculating the distance between the contacts and the median sagittal plane, the AC–PC (anterior commissure–posterior commissure (horizontal plane); VCP,vertical posterior commissure plane (frontal plane); AC–PC, anterior commissure–posterior commissure (horizontal plane).

Stimulation Paradigm

During the recording, the patients lay relaxed on a couch in a semi-dark room. Electrical stimuli of 100 μs were delivered by skin electrodes (cathode proximal) to the median nerve at the wrist. The stimulus intensity was set at motor threshold eliciting a twitch in thenar muscles at a constant intensity of 15–25 mA. Responses were averaged using two analysis times: (i) 128 ms with an interstimulus interval varying randomly between 640 and 780 ms, a sampling frequency of 2000 Hz and an analog filter bandpass of 1–500 Hz (6dB down per octave); (ii) 512 ms with an interstimulus interval varying randomly between 4500 and 5500 ms, a sampling frequency of 500 Hz and an analog filter bandpass of 1–250 Hz (6dB down per octave). The reference electrode was on the earlobe ipsilateral to the stimulated hand, and the ground was a circular wrapped electrode on the forearm ipsilateral to stimulation. For 12 of the 16 patients, responses were recorded contralateral and ipsilateral to stimulation. For the four remaining patients responses were only recorded contralateral to the stimulus. Two runs of 100 responses were averaged in each recording condition.

Intracerebral Recording Sites

Electrodes were implanted in sites chosen according the clinical and EEG presentation of seizures, as assessed by video EEG recordings, and to MRI, FDG PET (fluorodeoxyglucose positron emission tomography) and ictal blood-flow SPECT (single photon emission computerized tomography) in each individual patient. Apart from the pre- and/or post-rolandic opercular cortices, the most commonly investigated targets were the hippocampus, the amygdala, the temporal pole, the temporal neocortex, the cingulate gyrus and the orbitofrontal cortex. Since there was no patient in whom the epileptic discharges were suspected to spread to the perirolandic cortex, the primary somatosensory area (SI area) was not explored. Electrodes were implanted in the parieto-occipital region caudal and rostral to the VAC plane (p = 0), which in most subjects corresponds to the end of the rolandic fissure on the outer aspect of the hemisphere. Eight patients were implanted by a single opercular electrode exploring either the frontal (six cases) or the parietal (two cases) suprasylvian cortex; in eight patients both of these regions were explored.
explored. Thus our data were collected using a total of 24 electrodes (171 contacts). The y coordinates of implantation sites were +13 mm and –27 mm for the most rostral and the most caudal electrode respectively. The rostro-caudal extent of the explored area was thus 40 mm; 10 electrodes were located in the post-rolandic operculum (y = –9.6 ± 7.7 mm) and 14 in the pre-rolandic operculum (y = 4.9 ± 3.9 mm). For safety reasons, simultaneous scalp recordings were not possible; hence scalp SEPs were not investigated in this study.

Nomenclature of SEP Components
Responses were labeled according to the polarity-latency nomenclature in which the letters N and P, referring to the polarity of the potential in the contacts close to scalp surface, is followed by the mean latency in milliseconds. In all figures, negative potentials at the intracortical recording site are represented upward.

Results

Polarity, Latency and Voltage of SII Responses in the 50–150 ms Latency Range

Responses Contralateral to Stimulation
Two potentials contralateral to stimulation were recorded along all of the 24 electrode tracks in the cortex of the upper bank of the sylvian fissure, rostral and caudal to the rolandic fissure (Fig. 1). They consisted of a negative response (N60) followed by a positivity (P90), of which latencies and voltages are given in Table 1. No early response peaking earlier than 50 ms after stimulus was observed along the electrode tracks where the N60–P90 potentials were recorded. Figure 2 shows the coordinates of all contacts where a N60–P90 response, contralateral to the stimulation, was obtained in the Talairach and Tournoux stereotactic system. This pooling of individual data shows that the contralateral N60–P90 response was recorded along the trajectory of all electrodes penetrating the operculum and upper bank of the suprasylvian cortex between vertical planes at 13 mm rostral and 27 mm caudal to the anterior commissure vertical plane (VAC), and between horizontal planes at 0 and 21 mm above the horizontal AC–PC plane. The N60–P90 potentials were not recorded at any other recording sites outside this area, including hippocampus, amygdala, and temporal gyri T1 and T2. Figure 3 illustrates the fact that, in one individual, the N60–P90 potentials were not recorded along the electrode situated 7 mm above (P electrode) the volume delineated before.

In the eight patients in whom both the pre- and post-rolandic regions of the suprasylvian cortex were explored there was no significant difference between the latencies of the N60 (P = 0.33) and P90 (P = 0.66) potentials recorded by the two electrode tracks.

Responses Ipsilateral to Stimulation
Negative–positive responses ipsilateral to stimulus were also recorded along all of the 19 electrode tracks in the cortex of the upper bank of the sylvian fissure, rostral and caudal to the rolandic fissure. Latencies and voltages of these two potentials are given in Table 1. SEPs in the cortex of the upper bank of the sylvian fissure ipsilateral to stimulation had a similar waveform as that recorded in the homologous cortex contralateral to stimulation and peaked 12.4 ± 5.2 and 16.1 ± 9.7 ms later than contralateral N60 and P90 respectively. This latency difference between ipsi- and contralateral responses was not different for N60 and P90 potentials (P = 0.1).

In the seven patients whose pre- and post-rolandic responses ipsilateral to stimulation were available there was no significant...
latency difference between the N60 ($P = 0.32$) and P90 ($P = 0.74$) potentials recorded along the two electrode tracks.

Stereotactic Coordinates of the N60–P90 Sources

The depth coordinates ($x$) of the SEP sources were assessed by the depth of the contacts where a polarity reversal occurred in bipolar (Fig. 1) or earlobe recordings (Figs 4, 5a). When no polarity reversal (Fig. 5b) was observed along the whole electrode track it was considered that the contact where the response reached its maximal amplitude was the closest to the source.

In the eight patients in whom both the pre- and post-central regions of the suprasylvian cortex were explored, we considered that the two electrode tracks recorded responses originating from the same source for the following reasons: (i) there was no difference in latency or waveform between responses recorded along the two electrode tracks; (ii) we never observed in earlobe reference recordings polarity reversals of the responses at different depths along the two electrode tracks in the same individual; (iii) the behavior of responses to variations of stimulus rates were similar along the two electrode tracks. Therefore for assessing the source coordinates in these eight patients we selected the electrode track according the following hierarchical criteria: (i) polarity reversal in earlobe reference recording (see Fig. 6); (ii) polarity reversal in bipolar recording; (iii) maximal peak amplitude in the absence of polarity reversal along any of the two electrode tracks. Thus, all of our data were analyzed in terms of stereotactic coordinates of sources using a total number of 16 electrode tracks (one per patient).

Cortical stimulations (1 or 50 Hz, 2–3 mA) were performed at the contacts in the suprasylvian cortex close to the maximum, or polarity reversal, of the N60–P90 potentials in eight patients. Five of them reported a somatic sensation which was elaborated illusions of movement (two patients), or unpleasant paresthesiae (three patients). These latter sensations were located in contra-lateral forearm and hand in two patients and scalp in one. Of the three remaining patients, two did not experience any sensation during stimulation and one reported a taste, suggesting a response from the neighboring insular cortex (Hausser-Hauw and Bancaud, 1987).

Sources of Contralateral Responses

A clear-cut maximum with a polarity reversal of the N60–P90 response contralateral to stimulation was observed along 14 of

Table 1

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<th>Pre-rolandic operculum</th>
<th>Post-rolandic operculum</th>
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<td>Contralateral to the stimulation</td>
<td>Ipsilateral to the stimulation</td>
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<td>Mean</td>
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<td>Peak latencies (ms)</td>
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<tr>
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<td>58</td>
<td>6</td>
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<tr>
<td>P90</td>
<td>88</td>
<td>9</td>
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<tr>
<td>Peak amplitudes (µV)</td>
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<td></td>
</tr>
<tr>
<td>N60</td>
<td>25.2</td>
<td>12.9</td>
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<td>P90</td>
<td>34.7</td>
<td>24.3</td>
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the 16 electrode tracks, in bipolar (7) or earlobe (7) recordings (see Fig. 7). Along the two remaining electrode trajectories there was no observable phase reversal of the N60–P90 response in bipolar or earlobe recordings. The depth and vertical coordinates of SEP sources along the $x$ and $z$ axes showed small interindividual variations and were located at $x = 41 \pm 8$ mm from midline and $z = 12 \pm 5$ mm above the horizontal AC–PC plane for both the N60 and P90 potentials (Table 2). Conversely, as expected from the low rostro-caudal electrode sampling in each patient, the $y$ source coordinates showed more interindividual variations and were located at $x = 41 \pm 8$ mm from midline and $z = 12 \pm 5$ mm above the horizontal AC–PC plane for both the N60 and P90 potentials (Table 2). Conversely, as expected from the low rostro-caudal electrode sampling in each patient, the $y$ source coordinates showed more interindividual variations and were located at $x = 41 \pm 8$ mm from midline and $z = 12 \pm 5$ mm above the horizontal AC–PC plane for both the N60 and P90 potentials (Table 2).

Figure 4. Earlobe reference contralateral recording in the post-rolandic operculum of one patient. A polarity reversal is observed for the N60–P90 potentials at a depth of 35–39 mm from vertical midline plane ($x$ coordinate). Abbreviations as in Figure 1 legend.

Figure 5. Each graph represents the voltage profile of the N60 potential recorded in earlobe reference recordings along the depth electrodes for each patient. The negative values of the voltages ($\mu$V) correspond to the amplitudes of negative potentials oriented upwards in all the figures representing evoked potentials recordings. Each point (●) corresponds to one contact, localized on the graph according to its depth ($x$) stereotactic coordinate, i.e. the distance between the contact and the sagittal interhemispheric plane (median line). (a) Polarity reversals of the N60 potentials were recorded along seven electrode tracks of seven different patients. The voltage gradients are very sharp, suggesting that the $x$ coordinates of the N60 sources correspond to the intersection points of the curves (voltage profiles) and the dashed lines. (b) No polarity reversals of the N60 potentials were recorded in earlobe reference recordings along nine electrode tracks of nine different patients. In the majority of cases the N60 potential reached a maximum of amplitude, suggesting that these electrodes were implanted close to the sources.
obtained in the 16 patients. This volume was of 3.2 cm$^3$ using a cortical surface (Allison et al. in a single epileptic patient, Lüders the upper wall of the sylvian fissure. Using subdural electrodes range following stimulation of the median nerve are located in These previous studies converged on the conclusion that the electrode matches that of the long-latency SEPs or SEFs recorded on the scalp (Forss lateral SII response recorded in the cortex with implanted SEP sources were distributed was assessed using the mean and SD values of x, y and z coordinates of the N60–P90 sources obtained in the 16 patients. This volume was of 3.2 cm$^3$ using a confidence interval of ±1 SD and of 25.6 cm$^3$ with a confidence interval of ±2 SD. This evaluation is approximate due to the low spatial sampling of SEEG recording in the antero-posterior (y) and vertical (z) axis. Sources of Ipsilateral Responses
The depth coordinates of ipsilateral sources along the x axis were ±7 mm from midline for both N60 and P90 potentials. The cortical volume where ipsilateral SEPs were recorded was of 2.5 cm$^3$ using a confidence interval of ±1 SD and of 19.7 cm$^3$ with a confidence interval of ±2 SD. There was no difference between the stereotactic coordinates of the contra- and ipsilateral N60–P90 potentials in the 12 patients in whom both responses were recorded.

Discussion
SEPs recorded with intracerebral electrodes identified a well-defined somatic responsive area in the cortex of the superior bank of the sylvian fissure, corresponding to the accepted localization of area SII in the human brain (Penfield and Jasper, 1954; Woolsey et al., 1979; Van Buren, 1983; Burton et al., 1993). In this cortical area, a negative potential at a latency of 60 ms (N60) followed by a positivity at 90 ms (P90), has been consistently recorded after stimulation of the contralateral median nerve at the wrist; these responses also occurred in the same area ipsilateral to the stimulus and peaked 12–16 ms later than in contralateral responses. This latency range of contralateral SII response recorded in the cortex with implanted electrodes matches that of the long latency SEPs or SEFs recorded on the scalp (Forss et al., 1994; Forss and Joussmäki, 1998; Hari et al., 1983, 1993; Mauguière et al., 1997) or directly from the cortical surface (Allison et al., 1989b, 1992; Mima et al., 1997). These previous studies converged on the conclusion that the generators of SEPs or SEFs recorded in the 70–140 ms latency range following stimulation of the median nerve are located in the upper wall of the sylvian fissure. Using subdural electrodes in a single epileptic patient, Lüders et al. recorded early responses to contralateral median nerve and finger stimulation in the inferior frontal gyrus (Lüders et al., 1985). These responses peaked only 2.4 ms later than SI responses (20–25 ms), and were considered to originate in the SII area. These authors failed to record such early responses ipsilateral to stimulation. Moreover their measurement area was clearly anterior to the cortical zone producing perisylvian responses peaking at 100–120 ms in human corticograms (Allison et al., 1989b). The early SII responses reported by Lüders et al. culminated at the anterior border of our suprasylvian recording sites (see figure 2 of this study and figure 1 in Lüders et al., 1985). Therefore differences in electrode locations between the two studies is unlikely to account for this discrepancy between subdural and intracortical recordings, which remains unexplained.

Activation Timing of SEP Sources
In the absence of SEP recordings along electrode tracks implanted in SI (see Materials and Methods), we cannot bring any direct argument on the activation timing of SEPs sources in the primary and secondary somatosensory cortex. However, no earlier responses were observed along the electrode tracks in the upper bank of the sylvian fissure where the N60–P90 potentials were recorded, in agreement with conclusions from surface recordings and SII source modeling of SEPs and SEFs (Hari et al., 1984, 1990, 1993; Allison et al., 1989a, b; Mauguière et al., 1997). The human SI area is known to be activated by electrical stimulation of the median nerve in the 20–35 ms latency range [recently reviewed (Allison et al., 1989a; Mauguière et al., 1995)]. The delay of ~40 ms between SI and parietal operculum responses is compatible with the hypothesis that SII might depend on cortical inputs from SI cortex for its activation by non-noxious stimuli. This hypothesis is supported by the observation in monkeys that postcentral somatosensory cortex ablations render SII area somatically unresponsive (Pons et al., 1987; Garraghty et al., 1990) and that the SII area is connected with areas 3b, 1 and 2 of the primary somatosensory cortex (Friedman et al., 1986; Cusick et al., 1989; Burton et al., 1995). However, the delay of 40 ms seems to be quite a long conduction time between two cortical areas directly interconnected and is difficult to explain. The hypothesis of a multisynaptic processing occurring in SI before input transmission to SII could explain the long latency of the SII responses. Source modeling of SI activity in humans (Hari et al., 1984; Forss et al., 1994; Mauguière et al., 1997) using MEG or direct SI recordings in the awake monkey (Kulics and Cauller, 1986; Nicholson Peterson et al., 1995) suggest that SI remains active for 70–150 and 40–110 ms.

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<th>Coordinates of sources (mm)</th>
<th>Contralateral to the stimulation</th>
<th>Ipsilateral to the stimulation</th>
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<tr>
<td>x</td>
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<td>Mean</td>
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<td>N60</td>
<td>41 8 42 7</td>
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<tr>
<td>P90</td>
<td>41 8 42 7</td>
<td>10 –3 11</td>
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Table 2
Mean coordinates (mm) of the contacts calculated according to the stereotactic system of Talairach and Tournois where N60–P90 were recorded: x for the lateral medial axis, y for the anterior posterior axis and z for the vertical axis.

Figure 6. Earlobe reference contralateral recordings in the pre- and post-rolandic operculum of one patient. A clear polarity reversal is observed for the N60–P90 potential along the electrode track implanted in the post-rolandic operculum, at a depth of 25–38 mm from vertical midline plane, whereas no polarity reversal is observed along the other electrode track. For assessing the source coordinate of the N60–P90 potential in this patient we selected the post-rolandic electrode track.

Table 2
Mean coordinates (mm) of the contacts calculated according to the stereotactic system of Talairach and Tournois where N60–P90 were recorded: x for the lateral medial axis, y for the anterior posterior axis and z for the vertical axis.
Figure 7. Distribution of the 16 contacts with y values between -28 mm and +12 mm (Talairach and Tournoux) where a N60—P90 response either showed: a polarity reversal in earlobe recordings (seven contacts), white crosses; a polarity reversal in bipolar derivations (seven contacts), white circles; or no polarity reversal (two contacts), white triangles. The plotted spots correspond to contacts showing maximal amplitudes of the N60–P90 potential. MRI have been chosen in the pool of the MRI of all patients included in this study, in accordance with the corresponding slice of the Talairach atlas.
respectively after the response onset. However, the hypothesis of slow input processing in SI before transmission to SII does not explain the fast interhemispheric delay of 12–16 ms that we observed between the two SII areas. This latter observation would then suggest that the processing for transmission from the SII area contralateral to the stimulus to the opposite SII area via transcallosal fibers would be faster than that occurring in SI before input transmission to ipsilateral SII area.

An alternative hypothesis to explain this delay between SI and SII responses would be that the SII response is triggered via thalamo-cortical fibers (Friedman et al., 1980; Friedman and Murray, 1986) with a slower conduction velocity than that of thalamic afferents to the primary somatosensory cortex. However, this assumption of a direct thalamo-cortical pathway is in contradiction with the hypothesis that the SII area relays somatic information from SI to limbic structures of the medial temporal lobe (amygdala and hippocampus) via the insula (Friedman et al., 1986) [reviewed by Augustine (Augustine, 1996)].

Transcallosal Input Transmission between SII Areas

The existence of responses ipsilateral to stimulation in SII fits well with the anatomical observation in monkeys that a small but consistent proportion of neurons (~20%) in the hand representation of SII were activated from bilateral symmetrical or predominantly ipsilateral receptive fields (Robinson and Burton, 1980). To the question whether SII responses ipsilateral to stimulation are triggered via direct ipsilateral thalamic inputs, or via transcallosal fibers coming from the opposite SII area, our study suggests that the delays of 12 and 16 ms measured between ipsilateral and contralateral SII responses, for N60 and P90 potentials respectively, are compatible with the latter mode of transmission. The shortest callosal transmission time between the two SII areas has been estimated at 6–7 ms (Noatchar, 1998) and 8–9 ms (Cracco et al., 1989) on the basis of direct cortical recordings and transcranial brain stimulation studies respectively. These delays of 12–16 ms are in the same range as those measured between ipsi- and contralateral SII magnetic fields evoked by electrical stimulation of the median nerve [20 ms (Hari et al., 1993), 10 ms (Mauguière et al. 1997)]. However, a callosal transfer from contralateral to ipsilateral SII cannot be concluded only on the basis of this time difference. The possibility remains that responses ipsilateral to the stimulus could be triggered via ipsilateral thalamic fibers with slower conduction velocity. Only intracortical recordings of SII SEPs to ipsilateral stimuli in patients with a SII lesion in the opposite hemisphere could address directly this question.

Location of SEP Sources in the Suprasylvian Cortex

The depth coordinates (x) of the SEP maximum can be assessed with a spatial sampling of 1.5 mm in each individual. Conversely SEEG recordings do not allow precise evaluation of the distribution of responses in the rostro-caudal (y) and vertical (z) axis of the suprasylvian parieto-opercular cortex in a single subject because of their restricted spatial sampling (one or two electrode tracks per subject). However, one can assess the cortical volume where SEPs are recorded by pooling the stereotactic y and z coordinates of the N60–P90 responses in all subjects, a method that is used in PET studies based on statistical probability mapping (SPM). As shown in Table 3 the mean coordinates of the N60–P90 sources contralateral to stimulation are very similar those of the maximal blood flow responses observed during vibratory stimulation but more anterior than the blood flow response for active tactile exploration in PET studies (Burton et al., 1993; Ledberg et al., 1995; Roland et al., 1998). The best spatial correlations between intracortical SEPs and PET data are observed for the depth (x) coordinates. During active touch the maximal activation lies more superficially in the upper bank of the sylvian fissure than the area responsive to passive vibratory stimuli (Burton et al., 1993) and the sources of our N60–P90 response. However, the volume of the PET-activated cortical area during active touch is included in that explored by our electrode sampling probably because responses to median nerve stimulation are likely to involve a larger area than that activated by natural stimulation or active touch.

In addition, on the basis of anatomical data in monkeys, Burton et al. reported that a vibratory stimulus is likely to activate also the insular cortex (Burton et al., 1993). It is noteworthy that in four of our subjects the N60 potential maximum was deeply situated with x coordinates of 25, 29, 34 and 35 mm from midline (see Fig. 7). These sites are located at the edge between the suprasylvian and insular cortices. Moreover the x coordinates of the maximal depth of the N60 maximum confidence interval is at 33 mm (±1 SD) and 25 mm (±2 SD) from midline and thus likely to include the upper part of the insular cortex. Therefore, even if the precise stereotactic coordinates of the border between SII and the insular granular cortex are unknown in the human brain, there is a possibility that the insular cortex contributes to the generation of SEPs. Moreover, this assumption agrees with some observations in monkeys showing that the SII area is reciprocally connected with the granular and dysgranular insular fields and the retroinsular area (Friedman et al., 1986; Mufson and Mesulam, 1984) and some neurons within the retroinsular field and the granular insula are activated by tactile stimuli (Robinson and Burton, 1980).

Table 3

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<th>SEEG study</th>
<th>PET studies</th>
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<td>Passive electrical stimuli (Frot and Mauguière, 1989)</td>
<td>Passive vibratory stimuli (Burton et al., 1993)</td>
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<tr>
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Dipolar Source Modeling versus Intracortical Recordings

Most of the available data on the sources of SII responses in humans have been acquired from dipolar modeling of surface recordings. This modeling is based on the hypothesis that an equivalent electric dipole perpendicular to the cortical surface is adequate to model the source of the postsynaptic potentials fields produced by the afferent volley in the cortical gray matter (Henderson et al., 1975; Smith et al. 1985; De Munck et al., 1988; Lehman and Michel, 1989; Scherg, 1990). Concerning SEPs peaking at ~100 ms after stimulus, which show a field reversal across the sylvian fissure, both modeling studies (Hari et al. 1996; 1998a) have attempted to identify the location of their sources.
al., 1983, 1984, 1990, 1993; Mauguëre et al., 1997; Forss and Jousmäki, 1998) and direct recordings on the cortical surface (Allison et al., 1989b) converged on the conclusion that their dipolar source is likely to be tangential to the scalp surface and located in the upper wall of the sylvian fissure. However, as illustrated in Figures 7 and 5a, we observed such a potential reversal in earlobe reference recordings along seven of our electrodes tracks perpendicular to the midline sagittal plane. This observation is consistent with the hypothesis of dipolar sources oriented parallel to the electrode tracks and thus radial to the scalp surface. Allison et al. suggested that some of the perisylvian SEPs could be generated by dipolar sources in the portion of SII area located in surface cortex above the sylvian fissure (Allison et al., 1989b). However, the depth of sources as assessed in this study is not compatible with this hypothesis. A more likely explanation would be that, due to the several folds of the suprasylvian cortex, deep dipolar sources radial to the cortical surface, as expected from the orientation of functional cortical columns, are oriented perpendicular to the scalp surface.

Notes
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