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Evidence for a frontal cortex role in both auditory and somatosensory habituation: A MEG study

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Abstract

Auditory and somatosensory responses to paired stimuli were investigated for commonality of frontal activation that may be associated with gating using magnetoencephalography (MEG). A paired stimulus paradigm for each sensory evoked study tested right and left hemispheres independently in ten normal controls. MR-FOCUSS, a current density technique, imaged simultaneously active cortical sources. Each subject showed source localization, in the primary auditory or somatosensory cortex, for the respective stimuli following both the first (S1) and second (S2) impulses. Gating ratios for the auditory M50 response, equivalent to the P50 in EEG, were 0.54 ± 0.24 and 0.63 ± 0.52 for the right and left hemispheres. Somatosensory gating ratios were evaluated for early and late latencies as the pulse duration elicits extended response. Early gating ratios for right and left hemispheres were 0.69 ± 0.21 and 0.69 ± 0.41 while late ratios were 0.81 ± 0.41 and 0.80 ± 0.48 . Regions of activation in the frontal cortex, beyond the primary auditory or somatosensory cortex, were mapped within 25 ms of peak S1 latencies in 9/10 subjects during auditory stimulus and in 10/10 subjects for somatosensory stimulus. Similar frontal activations were mapped within 25 ms of peak S2 latencies for 75% of auditory responses and for 100% of somatosensory responses. Comparison between modalities showed similar frontal region activations for 17/20 S1 responses and for 13/20 S2 responses. MEG offers a technique for evaluating cross modality gating. The results suggest similar frontal sources are simultaneously active during auditory and somatosensory habituation.

Introduction

Habituation to redundant sensory input, or sensory gating, has been studied utilizing electroencephalography (EEG) recordings of a paired click auditory stimulus. These studies have largely focused on habituation of the primary auditory response approximately 50 ms after presentation of the stimulus (P50). Gating is the reduction in amplitude of response to the second click relative to the first click measured as the event-related brain potential (ERP). The mechanisms responsible for habituation are hypothesized to protect higher cortical centers from flooding with irrelevant information (Boutros et al., 2004; Boutros et al., 1991; Freedman et

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al., 1998; Venables, 1964) and to protect processing of the first response by filtering redundant sensory inputs (Edgar et al., 2005; Thoma et al., 2007). These studies are of importance for consistently showing gating deficit in schizophrenia populations.

Localization of the P50 generators has not yielded consensus largely due to limitations with the spatial resolution of scalp-recorded EEG/ERP reconstructions (Barkley, 2004). EEG/ERP measurements result from the electric potential of all simultaneously active neuronal generators making separation and localization of such generators at a given latency difficult. Complicating the localization problem are the varied conductances and resistivities of cortical tissues and fluids between the generators and measurement on the scalp (Korzyukov et al., 2007; Tepley, 2005).

Magnetoencephalography (MEG) can overcome the conductivity and resistivity variations with both high spatial and temporal resolution (Hamalainen et al., 1993; Hari, 1998; Tepley, 2005). MEG has been used to evaluate auditory gating with the magnetic equivalent of the P50, designated the M50 response (Huotilainen et al., 1998). Many MEG auditory studies have modeled brain response as equivalent current dipoles (ECDs) and calculated gating ratios from the source strength of ECDs following the S1 and S2 stimuli. Gating ratios for the M50 calculated using MEG ECDs have proven similar to EEG results for normal subjects (Blumenfeld and Clementz, 1999; Clementz et al., 1997; Hanlon et al., 2005; Huang et al., 2003; Thoma et al., 2003; Weisser et al., 2001). Garcia-Rill et al. used current density reconstruction of MEG recordings to localize the M50 response to multiple sources, usually including the frontal lobe, suggesting diffuse arousal-related projections in the cortex (Garcia-Rill et al., 2008). The present study also utilizes a current density source reconstruction evaluating M50 generators proposed to exist beyond the auditory cortex.

Somatosensory gating has been examined using a mixed modality paradigm consisting of paired auditory clicks and paired median nerve stimulation using EEG (Arnfred and Chen, 2004; Arnfred et al., 2001a; Arnfred et al., 2001b; Kisley and Cornwell, 2006), though no specific correlation between the auditory and somatosensory gating ratios has been determined. Our preliminary work has shown somatosensory gating of early latency response in the primary somatosensory cortex to paired finger taps accompanied by simultaneous frontal activation (Bowyer et al., 2006). We performed an auditory evoked study of normal subjects complimented with a somatosensory evoked study. This study aimed to utilize current density MEG reconstruction to define magnetic auditory and somatosensory gating in normal subjects and to evaluate common extended sources during gating in the two modalities.

Methods

Subjects

Ten right-handed normal controls (five females, 20–60 years, mean age 38.7 ± 12.4) were recruited to participate in this study evaluating both auditory and somatosensory evoked fields. All subjects were known personally to members of the research team; none of them had history of drug use, psychiatric or neurological (including head injury leading to loss of consciousness) problems and were not on any active central nervous system medications. Edinburgh Handedness Inventory laterality quotients ranged from 45 to 100, indicating strong right-handed preference for all subjects (Oldfield, 1971). All subjects gave informed consent prior to participation. All studies were approved by the Henry Ford Hospital Human Research Internal Review Board.

MEG Data Collection

Studies were performed using a 148-channel Neuromagnetometer (4D Neuroimaging, WH2500, San Diego, CA), a helmet shaped device covering the entire adult head, except the face. The individual sensors were SQUID (super conducting quantum interference device) magnetometers. After signing a consent form, each subject changed into a hospital gown and removed all metal articles from his/her body. Three small electrode coils, used to transmit subject location information to the neuromagnetometer probe, were taped to the forehead with two-sided tape. Two electrode coils were taped in front of the right and left pre-auricular point. A commercial videotape eraser was used to demagnetize dental work. Next, the subject lay on the positioning bed inside the magnetic shielded room and the digitization system (Fastrack, 4-D Neuroimaging) was used to establish head coordinate reference points and the location of the electrode coils relative to the head x, y, z coordinates. Activation of these electrode coils before and after each study allowed the localization of the MEG measurement array with respect to the subject's head. The shape of the head was also digitized for help with later co-registration to a standard MRI scan. The neuromagnetometer helmet containing the detector array was placed around the subject's head in close proximity to the scalp surface. The subject was asked to avoid both eye and body movements during data collection.

Cortical Model of Brain Electric Sources

Imaging results across subjects were related to brain structure by using a single realistic model of cortical gray matter was created based on a volumetric MRI scan (GE 1.5 Tesla scanner) of a normal head. This scan consisted of 124 coronal T1 images with 256x256 pixels per slice. For each subject, the model was coregistered to the MEG coordinates of the sensor array and row, column, and slice pixel dimensions which were rescaled such that MRI head surface matched the head surface digitized points describing the subject's skull surface. On average a localization error of 5 mm is expected, due to differences in brain geometry between the subject and the model (Holliday et al., 2003).

Auditory Evoked Fields

Auditory evoked fields (AEFs) were measured during of an auditory paired-stimulus paradigm widely used in sensory gating studies. The auditory S1-S2 paired-stimulus design consists of pairs of short tones separated by a 500-ms interstimulus interval (ISI) with an intertrial interval (ITI) of 8 sec at 60 dB above threshold. These ISI and ITI parameters have been shown to provide maximum differentiation between the gating rations of normal and schizophrenia groups (Nagamoto et al., 1996; Zouridakis and Boutros, 1992). Stimuli were monaural 3 ms-clicks, created and delivered using NeuroStim software (Compumedics, El Paso, TX) through Etymotic earphones (Etymotic Research, Inc., Elk Grove Village, IL) placed bilaterally in the participant's ear canal. Right and left side stimulations were presented as separate trials, each series lasting approximately 13 minutes. MEG responses were time-locked to stimuli for averaging using NeuroStim and 4-D data acquisition software (1998); 100 trials were collected per stimulus side. Each epoch consisted of 100 ms of pre-stimulus data and 2 seconds of post-stimulus data. Data were digitized at 508 samples per second from 0.01 Hz to 100 Hz and forward and backward filtered using a 3–50 Hz bandpass with a 60 Hz notch filter. Data was visually inspected and epochs containing movement artifact were discarded and the remaining epochs were averaged. Finally, MR-FOCUSS, a current density imaging technique which utilizes a gradient search algorithm, was used to determine source amplitudes for all cortical model locations (Moran et al., 2005). The statistical significance for cortical source amplitudes was based on pre-stimulus baseline data (Sekihara et al., 2005).

Auditory M50 Localization

The amplitude and locations of S1 and S2 cortical responses are based on cortical activity within a spherical region of interest (ROI) 1.6 cm (~ 20 pixels) in diameter that was centered on the location of the S1 response maximum. A source localization algorithm that identified local maxima of cortical amplitude and merged sources based on a nearness threshold was used to establish the ROI diameter of 1.6 cm, such that only S1 auditory cortical activity was included. ROIs contained 40 – 50 cortical model source locations. The amplitude of all sources within this ROI was summed and the S1 latency was determined by the peak source amplitude between 40 and 80 ms post-stimulus. This window for S1 latency has been established by numerous EEG studies of auditory gating which measured waveform peaks (Boutros et al., 1999; Boutros et al., 2004; Boutros et al., 1991; Patterson et al., 2008). For each subject, the amplitude of activation and location of S2 was based on the ROI established for the activity of S1 as in previous MEG studies (Edgar et al., 2005). Therefore the same ROI defined by the S1 response was analyzed to obtain peak S2 source amplitude. The S2 latency was limited to a time window within 30 ms of the S1 latency. An auditory gating ratio, S2/S1, was calculated by dividing the peak source amplitude of the S2 response by the peak source amplitude of the S1 response. Auditory M50 localizations were identified for both the S1 and S2 responses for all subjects in the respective hemispheres. These individual cortical response results were normalized to a common total power of activation then combined to obtain an across subject average response.

Somatosensory Evoked Fields

Somatosensory evoked fields (SEFs) were measured in the same subjects with a somatosensory paired-stimulus paradigm that mimicked the S1-S2 auditory paradigm. The somatosensory stimuli were delivered via pneumatically driven tactile stimulators (4-D Neuroimaging). Finger taps (15–17 psi) were applied to the middle finger in pairs separated by a 500-ms interstimulus interval (ISI) with an intertrial interval (ITI) of 8 sec. Right and left side stimulations were presented as separate trials, each series lasting approximately 15 minutes. MEG responses were time-locked to stimuli for averaging; 100 trials were collected per stimulus side. Data were collected and processed as described in the AEF section above.

Somatosensory M50 Localization

SEF peak responses were observed in all subjects and were localized to the contralateral somatosensory cortex where sensory processing for the middle finger is known to occur as shown in previous work by our group (Bowyer et al., 2006). An early peak response to the stimulus was associated with the initial sensory inputs from the finger; a secondary, or late, peak was observed ~45 ms later in this extended response. This extended response occurred for both the S1 and S2 inputs. Again a source localization algorithm that identified local maxima of cortical amplitude and merged sources based on a nearness threshold was used to establish the ROI as a 1.6 cm diameter sphere which included 40 – 50 magnetic sources. The amplitude of all sources within this ROI was summed and the early and late S1 latencies were determined by the peak source amplitudes between 30 and 130 ms post-stimulus. Using the identical ROI, early and late S2 peak source amplitudes and latencies were determined in the same manner. Somatosensory gating ratios, S2/S1, were calculated by dividing the peak source amplitude of the S2 response by the peak source amplitude of the S1 response for both the early and late response. These individual cortical response results were normalized to a common total power of activation then combined to obtain an across subject average response.

Results

Auditory Response

AEF peak responses for both S1 and S2 were observed in all subjects. MR-FOCUSS localized the response to the primary auditory cortex at expected latencies (range: 44–79ms for S1 and 44 – 85 ms for S2). MR-FOCUSS showed the underlying source for the right hemisphere response to the first click, for a 20 pixel ROI as defined above, to have an average amplitude of 6.27 ± 4.83 nAm at 67 ± 8 ms. For the second click, responses averaged 3.61 ± 3.33 nAm at 69 ± 10 ms. The average gating ratio (S2/S1) for the right hemisphere response was 0.54 ± 0.24 . The left hemisphere response to the first click had an average amplitude of 3.03 ± 3.44 nAm at 66 ± 10 ms. For the second click, responses averaged 1.32 ± 1.41 nAm at 65 ± 10 ms. The average gating ratio for the left hemisphere response was 0.63 ± 0.52 . Group average gating ratios and source amplitudes are displayed in Figure 1. Latency and source amplitudes for individual subjects are presented in Table 1. The normalized group average solution for both the S1 and S2 response for the right hemisphere response to left auditory stimulus, showed habituated reduction in peak source amplitude, presented in Figure 2.

MR-FOCUSS localized extended sources, active at a significance levels above 90%, within 25 ms of peak S1 response time points in the frontal lobe in 19/20 subjects (both hemispheres) and in 15/20 subjects for the S2 response. Right and left frontal locations, designated superior frontal, medial frontal or inferior frontal, were identified when simultaneously active in both the S1 and S2 latency windows and are listed in Table 1. The S1 response shared at least one frontal region of activation with the S2 response in the right hemisphere response to the left auditory stimulus for 9/10 subjects. The left hemisphere response to right auditory stimulus showed shared S1 and S2 frontal region activation in only 3/10 subjects. Figure 3A displays the extended cortical sources active in a typical subject. At 77 ms post S1 auditory stimulus, the auditory cortex as well as frontal areas was significantly active. The frontal activation appeared in the superior frontal region simultaneous to the auditory response indicated by the red line on the waveform. The reduction in the second peak response clearly shows the gating effect.

Somatosensory Response

For the right hemisphere, averaged MEG data results from all subjects showed an early S1 activation arising from the first pulse at 44 ± 8 ms. The locations of cortical sources for the early response were found to be very focal in the somatosensory cortex on the post central gyrus in the location normally active for finger tapping responses. MR-FOCUSS showed the underlying source for this response had average activity amplitude of 6.1 ± 3.7 nAm for the ROI defined by a 20 pixel sphere. This response was followed by a second peak at 93 ± 16 ms with average amplitude of 5.4 ± 3.3 nAm for the same ROI. For the second pulse, again for the same ROI, peak responses occurred at 41 ± 7 ms with average amplitude of 3.7 ± 1.8 nAm, followed by second peak response at 82 ± 14 ms with average amplitude of 3.8 ± 2.4 nAm. The average gating ratio for the right hemisphere, S2/S1 amplitude, was 0.69 ± 0.21 for the early somatosensory response and 0.81 ± 0.41 for the late somatosensory response.

The left hemisphere, showed peak S1 responses at 47 ± 8 ms of 1.9 ± 1.6 nAm and at 93 ± 18 ms of 4.3 ± 6.6 nAm with peak S2 response at 44 ± 6 ms of 1.2 ± 1.4 nAm and 94 ± 17 ms of 2.7 ± 4.9 nAm. The average gating ratio for the left hemisphere was 0.69 ± 0.41 for the early somatosensory response and 0.60 ± 0.48 for the late somatosensory response. Group average gating ratios and source amplitudes are displayed in Figure 1. Latency and source amplitudes for individual subjects are presented in Table 2. The normalized group average solution for both the S1 and S2 response for the right hemisphere response to left somatosensory stimulus, showing a habituated reduction in peak source amplitude, is presented in Figure 2.

For the somatosensory response, frontal activity was evaluated in the time window between the first and second peak responses. Extended sources localized to the frontal cortex, active at a significance levels above 90%, between the early and late response latency to both the S1 stimulus and the S2 stimulus in all subjects in both hemispheres. These active locations were identified as either right or left superior frontal, medial frontal or inferior frontal and are listed in Table 2. At least one of the same regions was active during both the S1 and S2 response window for all subjects regardless of the stimulus side and responding hemisphere. Figure 3B displays the extended cortical sources active in the same subject discussed above. At 51 ms post S1 somatosensory stimulus, the somatosensory cortex as well as frontal areas was again significantly active. The frontal activation appeared in the superior frontal region simultaneous to the sensory response indicated by the red line on the waveform. The reduction in the second peak response clearly shows the gating effect similar to that in the auditory study.

Frontal Activation in Auditory and Somatosensory Responses

Frontal activation, significant above 90%, was evident in all subjects in response to either the S1 or S2 stimulus with both modalities. Our results show consistent activation, of the same frontal areas, for the S1 response in both the auditory and somatosensory tests for 10/10 subjects in the right hemisphere and 7/10 for the left hemisphere. The S2 response, had lower agreement between modalities, with similar frontal active areas in 8/10 subjects in the right hemisphere and 5/10 in the left hemisphere. Active frontal areas for each subject are shown in Table 2.

Discussion

The most important finding of this work relates to the nature of frontal activation during a sensory habituation paradigm. Our results show frontal activation following either auditory or somatosensory stimulation, simultaneous to the activation in the expected sensory cortex for both hemispheres. The areas of activation are consistent across modalities in 17/20 responses to S1 stimulus and 13/20 to the S2 stimulus. This is the first study to provide evidence of cross modal activation of the frontal cortex during gating and suggests that the frontal lobe has an important role in the neural mechanisms of habituation.

A large number of studies have been dedicated to comparing the gating responses of various clinical disorders, particularly schizophrenia to normal controls (Adler et al., 1982; Boutros et al., 1999; Boutros et al., 1991; Bramon et al., 2004; Clementz et al., 1997; Freedman et al., 1987; Nagamoto et al., 1996; Nagamoto et al., 1989; Thoma et al., 2003). These studies consistently show P50 gating deficits with repetitive auditory stimulation in the schizophrenia population. In a recent review, Grill-Spector and colleagues suggested that at least four mechanisms might play a role in sensory inhibition with stimulus repetition: firing rate adaptation, synaptic depression, long-term depression, and long-term potentiation (Grill-Spector et al., 2006). Our work does not attempt to elucidate the possible role of these mechanisms, but does provide essential preliminary data for normal controls setting the stage for further study of abnormal gating in psychiatric disorders.

This study does not attempt to answer the continuing difference between EEG and MEG source localization as the two imaging techniques may reflect activation of sources not well visualized by the other. Despite those differences, MEG auditory gating ratios for the M50 modeled as ECD source strengths have proven similar to EEG ratios for normal controls while yielding inconsistent results in schizophrenics (Blumenfeld and Clementz, 1999; Clementz et al., 1997; Hanlon et al., 2005; Huang et al., 2003; Thoma et al., 2003; Weisser et al., 2001). However, the dipole model yields considerable intragroup variability in source strength and orientation (Edgar et al., 2003). Yvert et al. using a minimum current estimates distributed source method for MEG analysis, reported that a single dipole model was a good approximation of auditory activity only at the early stages (before 25 ms) of evoked response and mapped

several simultaneous sources for longer latencies (Yvert et al., 2005). The Garcia-Rill et al. MEG study used current density reconstruction for localization of auditory gating. The M50 response, to both S1 and S2, localized to multiple sources usually including the frontal lobe and suggested diffuse arousal-related projections in the cortex (Garcia-Rill et al., 2008). In our study, the ECD solution was only valid when applied to the peak of the primary evoked response. Our current density technique, MR-FOCUSS, localized extended cortical sources for the M50 generators proposed to exist beyond the auditory cortex.

The auditory gating ratios from this study are in agreement with previous literature (see (Patterson et al., 2008) for a comprehensive review). Specifically our study compares well with the current density technique employed by Garcia-Rill. That study presented a paired pulse binaural stimulation and calculated gating ratios from the amplitudes of intertrial Coherence (ITC) for S1 and S2. ITC is a phase-locking factor indicating synchronization of oscillations of selected independent components (Garcia-Rill et al., 2008). Our results also indicate a source strength difference between hemispheres with right S1 peak response amplitudes of 6.27 ± 4.83 nAm as compared to left S1 peak amplitude 3.03 ± 3.44 nAm despite nearly identical latencies. These results are consistent with previous MEG ECD studies showing auditory source asynchrony (Blumenfeld and Clementz, 2001; Huang et al., 2003) and further validates the current density technique presented here.

In addition, our results showed significant activation in the frontal cortex simultaneous with auditory activation following both the S1 and S2 stimuli. Weisser reported frontal activation from ECDs seeded in the frontal cortex within 10 ms of P50 activation (Weisser et al., 2001) though this activation was not simultaneous to the peak auditory response, presumably due to the limitation of simultaneous source localization with the ECD analysis. Previous work, using subdural electrodes, also demonstrated substantial P50 generators localized to the temporal lobe in half of presurgical epilepsy patients studied (Korzyukov et al., 2007). These results are in agreement with general EEG and animal studies that have demonstrated P50 frontal generators (Grunwald et al., 2003; Mears et al., 2006; Weisser et al., 2001). Existence of frontal generators are consistent with auditory gating deficits shown in patients with prefrontal damage (Knight et al., 1999). In our study, localization of frontal activation was constrained to areas of activation with significance levels higher than 90% relative to pre-stimulus activity, regardless of source amplitude. Regions of statistically significant activation in the frontal cortex for one subject during the auditory (as well as the somatosensory) protocol are illustrated in Figure 3.

A recent EEG study suggested P50 gating suppression may be modality nonspecific based on visual and auditory studies sharing a frontal dipole source (Oranje et al., 2006). This activation may be a higher level of processing that collects and stores the outcome of S1 processing (Korzyukov et al., 2007). In agreement with that study, which showed the localization of auditory gating related activation consistent with S1 frontal P50 generators, we have now demonstrated that the M50 frontal generators may be represented in the same cortical areas for both auditory and somatosensory gating.

The somatosensory response to repetitive sensory stimuli has been proposed as short-term plasticity in the somatosensory cortex (McLaughlin and Kelly, 1993). Calculation of somatosensory gating ratios provides a complimentary technique for comparison with auditory ratios, however as large a body of literature for somatosensory gating ratios does not exist. MEG dipole analysis was used by Kakigi to identify the somatosensory evoked field (SEF) waveform components arising from the electrical stimulation of the index finger. The short and mid latency dipoles labeled 1M, 2M, 3M, 4M (essentially the magnetic equivalents of the N30, P50, N65 and P80 responses shown by Arnfred and colleagues) localized to the primary somatosensory cortex (SI) (Hashimoto, 1988; Kakigi et al., 2000). In another MEG dipole

study, peak responses localized to the contralateral SI at 20–40 ms latencies but to the secondary somatosensory cortex (SII) at later (100–140 ms) latencies. They also noted considerable variability across subjects (Hari et al., 1993). Most recently, a MEG study by Thoma and colleagues calculated SI and SII gating ratios for the M20 (or P20m) somatosensory response using a similar electrical paired pulse paradigm evaluated using ECD analysis. That study showed SI gating in both normals and schizophrenic patients. The patients, however, showed a gating deficit compared with the normals in M20 SII gating suggesting a cross-modal gating deficit in schizophrenic patients (Thoma et al., 2007).

We propose that our early and late somatosensory latencies correspond to the P50 and N80 responses elicited by electrical stimulation (Arnfred and Chen, 2004; Kakigi et al., 2000). Direct comparison with work by Edgar and Thoma is more difficult as an early study evaluated M20 responses in the primary somatosensory cortex (Edgar et al., 2005) while later work evaluated M20 responses in the secondary somatosensory cortex (Thoma et al., 2007). However, their work does suggest frontal activation involved in habituation to paired pulse stimuli. Studies of somatosensory stimulation, which localized differently during high and low cognitive tasks using ECDs, also suggest a prefrontal-cortical sensory gating system (Schaefer et al., 2005). Our results showed significant (>90%) activation of the frontal cortex in all subjects within the window between the early and late response to both the S1 and S2 somatosensory stimuli in both hemispheres.

In conclusion, the data provided from this study represent evidence for a crucial frontal lobe role in sensory gating in multiple modalities. This mechanism, consistent across modalities for normal controls, provides groundwork for comparing habituation deficits in patient populations.

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Fig. 1.

(A). Group average gating ratios ($n = 10$) for right and left hemisphere responses to auditory and somatosensory stimuli. Error bars indicate standard error for each calculated ratio. (B). Average source amplitude ($n = 10$) for S1 and S2 stimuli for auditory and somatosensory data.

A. Normalized Group Average Auditory Response

B. Normalized Group Average Somatosensory Response

Fig. 2.

(A). Group average (n=10) MR-FOCUSS solution displaying amplitudes in NanoAmpMeters for the right hemisphere response to the left auditory stimulus showing S1 and S2 localizations. Arrows highlight auditory cortex. (B). Group average (n=10) MR-FOCUSS solution amplitudes for the right hemisphere response to the left somatosensory stimulus showing S1 and S2 localizations. Arrows highlight somotosensory cortex

Fig 3.

(A). Auditory response following S1 for one subject showing extended cortical sources. Also displayed are MEG waveforms with red line indicating latency of 77 ms. (B). Somatosensory response following S1 for same subject showing similar frontal sources. MEG waveforms are shown with red line indicating latency of 51 ms.

Table 1
Individual Results for Auditory Paired Stimulus

				RIGHT HEMISPHERE RESPONSE TO LEFT AUDITORY STIMULUS									LEFT HEMISPHERE RESPONSE TO RIGHT AUDITORY STIMULUS								
				ACTIVE				ACTIVE				AUD. GATING	ACTIVE				ACTIVE				AUD. GATING
				S1		FRONTAL AREA		S2		FRONTAL AREA			S1		FRONTAL AREA		S2		FRONTAL AREA		
Subject	H	G	Age Yrs	Lat (ms)	Ampl (nAm)	Right	Left	Lat (ms)	Ampl (nAm)	Right	Left	S2/S1 Ratio	Lat (ms)	Ampl (nAm)	Right	Left	Lat (ms)	Ampl (nAm)	Right	Left	S2/S1 Ratio
1	R	F	43	63	6.76	I,M	I,M,S	61	3.45	I,M		0.51	44	0.62	I		44	0.35	I	I,M,S	0.56
2	R	F	60	73	6.45	M,S	I,M,S	85	4.81	S	M	0.75	73	2.14	I,S	I,M	61	0.60	I		0.28
3	R	F	45	69	1.48	M,S	I,M,S	61	0.22			0.15	71	0.92	S	I,S	77	0.36			0.39
4	R	M	26	55	5.03	M,S	S	49	0.96	S	I,M,S	0.19	67	6.78	M,S	I,M,S	59	1.65	I		0.24
5	R	F	31	71	2.41	M,S		75	1.41	I,S	M	0.59	67	0.51			69	0.92		I,M	1.80
6	R	F	40	71	10.45	M,S	I,S	75	6.69	M	M,S	0.64	71	9.94	M	M	71	4.63		I	0.47
7	R	F	20	73	1.25	I,M,S	I,M,S	77	1.19	I,M,S	M,S	0.95	60	1.93	I,M,S	I,M,S	65	0.74	M,S	M,S	0.38
8	R	M	27	69	9.34	M,S	M,S	69	5.41	M	I,M,S	0.58	79	1.05		I,M	77	0.74	M	I	0.70
9	R	M	47	75	16.62	M	S	67	10.71	I,M,S		0.64	73	1.41		S	77	1.30			0.92
10	R	M	48	51	2.88	I,M		71	1.21	I,M,S	S	0.42	59	0.52	I		67	0.40			0.77
Ave			38.7	67.0	6.27			69.0	3.61			0.54	65.8	3.03			65.4	1.32			0.63
SD			12.4	8.1	4.83			10.2	3.33			0.24	9.8	3.44			10.9	1.41			0.52

Key to Frontal Areas: I - Inferior, M - Medial, S - Superior

Table 2
Individual Results for Somatosensory Paired Stimulus

Subject	RIGHT HEMISPHERE RESPONSE TO LEFT SOMATOSENSORY STIMULUS														LEFT HEMISPHERE RESPONSE TO RIGHT SOMATOSENSORY STIMULUS													
	S1 Early							S2 Early							S1 Early							S2 Early						
	ACTIVE				ACTIVE				ACTIVE				ACTIVE				ACTIVE				ACTIVE				ACTIVE			
	Lat	Ampl	Lat	Ampl	Lat	Ampl	Lat	Ampl	Lat	Ampl	Lat	Ampl	Lat	Ampl	Lat	Ampl	Lat	Ampl	Lat	Ampl	Lat	Ampl	Lat	Ampl	Lat	Ampl	Lat	Ampl
	(ms)	(nAm)	(ms)	(nAm)	(ms)	(nAm)	(ms)	(nAm)	(ms)	(nAm)	(ms)	(nAm)	(ms)	(nAm)	(ms)	(nAm)	(ms)	(nAm)	(ms)	(nAm)	(ms)	(nAm)	(ms)	(nAm)	(ms)	(nAm)	(ms)	(nAm)
	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left
	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1	S2/S1
	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio	Ratio
1	40	3.34	81	7.22	I,M,S	I,M,S	40	2.38	71	4.42	I,M,S	M,S	0.71	0.61	44	0.53	77	21.13	I,M,S	I,M,S	40	0.23	77	15.36	I,M,S	M	0.43	0.73
2	36	3.57	105	2.60	I,M,S	I,M,S	40	2.8	99	2.94	M	S	0.78	1.13	45	1.63	85	1.86	I,M,S	M,S	48	1.65	83	0.70	I,S	M,S	1.01	0.38
3	42	7.18	103	3.46	I,M,S	S	48	5.65	81	3.23	I,S		0.79	0.93	59	1.25	112	2.54	I,M,S	I,M,S	52	1.01	119	0.55	I,M		0.81	0.22
4	42	3.82	61	3.41	I,M,S	I,M,S	54	2.73	73	2.19	I,M,S	I,M	0.71	0.64	40	1.55	79	0.40	I,M,S	I,M,S	48	0.28	87	0.75	I	I,M	0.18	1.88
5	48	2.46	106	2.87	I,M,S	I,M,S	42	2.07	61	1.92	I,M,S	I,M,S	0.84	0.67	55	0.87	95	0.98	I,M	M	48	0.55	107	0.41	I,M		0.63	0.42
6	44	6.36	112	4.76	I,M,S	I,M,S	38	3.47	95	4.08	I,M,S	I,M,S	0.55	0.86	42	4.17	97	1.63	I,M,S	I,M,S	40	1.30	105	0.56	I,M,S	I,S	0.31	0.34
7	32	10.40	99	11.83	I,M,S	M,S	26	6.82	85	1.62	I,M,S	I,M,S	0.66	0.14	36	1.26	97	2.12	M,S	I,M,S	34	0.73	81	0.81	I,M,S	S	0.58	0.38
8	42	1.56	99	2.94	I,M,S	I,M,S	40	1.68	103.00	4.95	I,M,S	M	1.08	1.68	46	0.88	94	0.88	I,M,S	I,M,S	40	1.39	97	0.24	M		1.58	0.27
9	61	10.08	91	10.48	I,M,S	S	36	3.00	85	9.73	I,M,S	I,M,S	0.30	0.93	51	0.83	126	0.98	I,M,S	I,M	48	0.39	115	0.72	I,M,S	M,S	0.47	0.73
10	48	11.76	77	4.71	I,M,S	I,M,S	40	5.91	73	2.38	I,M,S		0.50	0.51	51	5.10	71	6.58	M,S	I,M	38	4.81	75	4.09	I,M,S	I,M,S	0.90	0.62
Average	43.5	6.05	93.4	5.43			40.4	3.65	82.6	3.75			0.69	0.81	46.9	1.81	93.3	3.91			43.6	1.21	94.6	2.42			0.69	0.60
SD	7.8	3.66	15.9	3.32			7.3	1.80	13.5	2.38			0.21	0.41	7.1	1.54	16.6	6.30			5.9	1.29	16.2	4.68			0.41	0.48

Key to Frontal Areas: S - Superior, M - Medial, I - Inferior