Auditory sensory gating to the human voice: A preliminary MEG study

Yoji Hirano\textsuperscript{a}, Toshiaki Onitsuka\textsuperscript{a,⁎}, Toshihide Kuroki\textsuperscript{a}, Yuji Matsuki\textsuperscript{b}, Shogo Hirano\textsuperscript{a}, Toshihiko Maekawa\textsuperscript{a}, Shigenobu Kanba\textsuperscript{a}

\textsuperscript{a}Department of Neuropsychiatry, Graduate School of Medical Sciences, Kyushu University, Fukuoka, Japan
\textsuperscript{b}Department of Intelligent Systems, Faculty of Information Science and Electrical Engineering, Kyushu University, Fukuoka, Japan

Received 4 July 2006; received in revised form 25 June 2007; accepted 16 July 2007

Abstract

The ability of the brain to suppress incoming irrelevant sensory input is termed ‘sensory gating,’ and auditory sensory gating is often indexed by the auditory evoked response. We recorded the auditory evoked magnetic fields to the human voice, using the conditioning–testing paradigm, to investigate whether or not healthy subjects show less activation to the second voice stimulus. Seventeen healthy adults (mean age 27.9 ± 4.8 years, 9 males and 8 females) participated in the experiment. The auditory stimuli were presented monaurally as a series of 120 paired voices, with 500-ms interstimulus intervals and 6-s interpaired stimulus intervals. The P50m and the N100m responses were investigated, and dipole source localization was performed. Root mean squares of both P50m and N100m were significantly suppressed to the second stimulus bilaterally, and the suppression was more significant in N100m. The N100m was located significantly more laterally than the P50m for both hemispheres. These results therefore demonstrate the presence of sensory gating for auditory inputs of the human voice in the primary auditory cortex and the auditory association area.

© 2007 Elsevier Ireland Ltd. All rights reserved.

Keywords: P50m; Human voice; Sensory gating; Magnetoencephalography

1. Introduction

Sensory gating is often indexed by evoked potentials such as P50, which is a positive potential at around 50 ms elicited by auditory stimuli. The P50 component has recently become the focus of clinical interest in patients with various mental diseases (Adler et al., 1982; Freedman et al., 1983; Buchwald et al., 1989, 1992; Neylan et al., 1999; Jessen et al., 2001). Using a conditioning–testing paradigm, Adler et al. (1982) reported that patients with schizophrenia showed deficits in sensory gating indexed by decreased P50 inhibition to the second auditory stimulus. However, the published reports of P50 generation are mixed, and generators of P50 in the human brain still remain unclear. For example, Lee et al. (1984) have recorded P50 from subdural electrodes and have reported that human P50 is a near-field potential in the primary auditory cortex. On the other hand, P50 is sensitive to the level of arousal (Erwin and Buchwald, 1986), thus suggesting that P50 is from the ascending reticular activating system. It has been reported that human scalp P50 may be an overlapping
potential (Cacace et al., 1990; Ninomiya et al., 1997; Onitsuka et al., 2000). Huang et al. (2003) reported that multiple generators contributed to the scalp P50 in patients with schizophrenia.

For other regions involved in sensory gating, the hippocampus has been reported to play a crucial role in the auditory sensory gating system (Krause et al., 2003; Grunwald et al., 2003; Boutros et al., 2005). Of note, Grunwald et al. (2003) reported that habituating auditory evoked responses in the neocortex and the hippocampus peaked around 50 ms and 250 ms, respectively. They conclude that such sensory gating may therefore be a multistep process subserved by different brain areas.

Magnetoencephalography (MEG) offers high spatial resolution to accurately locate the position of neuronal activity to auditory stimuli compared with electroencephalography (EEG) (Yamamoto et al., 1988). EEG can record activity in the subcortex as well as in the auditory cortex (Cacace et al., 1990; Onitsuka et al., 2000) whereas MEG can predominantly detect auditory cortical response, since MEG is sensitive to the current components flowing tangentially with respect to the head surface (Huizinga et al., 2001). In the MEG literature, P50m (the magnetic counterpart of P50) has been reported to be generated in or near the primary auditory cortex (Mäkelä et al., 1994; Yoshiura et al., 1995; Kanno et al., 2000; Onitsuka et al., 2000; Godey et al., 2001). In addition, Godey et al. (2001) reported that the P50m source was localized in the lateral part of Heschl’s gyrus (HG). It may therefore be important to investigate auditory sensory gating in the human auditory cortex using MEG with a conditioning–testing paradigm, since the source of auditory evoked responses has been clear in MEG.

Auditory stimuli also elicit a negative potential termed N100, which is one of the best-investigated auditory evoked responses with a negative peak at approximately 100 ms after auditory stimuli. N100 response arises from the primary and auditory association areas (Jacobson et al., 1997). In MEG studies, it has been reported that N100m is located in the auditory association area (planum temporale [PT]) (Pantev et al., 1995; Lütkenhöner and Steinsträter, 1998).

Recently, functional magnetic resonance imaging (fMRI) studies have revealed that human voices are perceived, at least in part, by a processing stream separate from that used in processing other non-vocal environmental sounds (Belin et al., 2000). More specifically, the bilateral superior temporal sulcus (STS) regions have been reported to selectively respond to the human voice (Belin et al., 2000; Fecteau et al., 2004). As noted before, patients with schizophrenia may have deficits for auditory sensory gating. Schizophrenic patients often show auditory hallucinations of the human voice, which may be related to a reduced gray matter volume of left superior temporal gyrus (STG) including the HG and the PT (Onitsuka et al., 2004). It will therefore be important to investigate sensory gating in the human voice. The present study was designed to investigate whether or not healthy subjects show less activation to the second voice stimulus with a conditioning–testing paradigm, which supports sensory gating to the human voice in the auditory cortex.

2. Methods

2.1. Subjects

Seventeen healthy adults (mean age 27.9 ± 4.8 years, range 21 to 38; 9 males and 8 females) participated in the experiment. Handedness was assessed using the Edinburgh inventory, and all subjects were right-handed (the score of handedness: 94.6 ± 11.6) (Oldfield, 1971). The subjects were screened using the Structured Clinical Interview (SCID) — non-patient edition (Spitzer et al., 1990). To rule out otolaryngologic disorders, the subjects were also screened with a questionnaire assessing physical condition and past history, and a brief physical examination for hearing was performed. None of the subjects had either an Axis-I psychiatric disorder or otolaryngologic disorders. After a complete description of the study, all participants signed an informed consent form in accordance with the regulations of the Ethic commission of the Graduate School of Medical Sciences, Kyushu University.

2.2. Stimulation and procedures

During the experiment, all subjects were in the lateral recumbent position on a bed in a magnetically shielded room (Sumitomo Metal Ind., Ltd.) at Kyushu university Hospital. Auditory evoked responses in both hemispheres were recorded alternately. The subjects were instructed to keep their eyes open and not to sleep. The Japanese vowel sound /a/ was used as an auditory voice stimulus. The stimulus was the voice spoken by an actor who was a native Japanese speaker, and was digitized and edited with 200-ms duration (rise/fall 10 ms). The frequencies for the formants (F) of the vowel /a/ were as follows: FO = 140 Hz, F1 = 760, F2 = 1250, F3 = 2750, F4 = 3600. At the earpiece, the intensity of the stimulus was 60 dB sound pressure level.

The software for stimulus generation was run on a mini-computer (NEC PC-9801FA). The software...
controlled interstimulus intervals (between conditioning–testing stimuli) (ISI) and inter paired-stimulus interval (IPI), and provided a trigger signal at every stimulus onset. Stimuli were delivered to the ear contralateral to the hemisphere being recorded through a 2.3-m plastic tube with a plastic insert earpiece at the tip. The stimuli were presented consecutively as a series of 120 paired voice stimuli with 500-ms ISI and 6-s IPI. The order of the recorded hemisphere was counterbalanced between the subjects.

2.3. Data acquisition and analysis

A 37-channel biomagnetometer (Magnes, Biomagnetic Technologies Inc., San Diego, CA) was used for the magnetic measurements. The sensing coils consisted of first-order axial gradiometers, measuring 20 mm in diameter, with a baseline of 50 mm, and the distance of the center between adjacent coils was 22 mm. The coils were arranged uniformly in concentric circles over a spherical surface covering a circle of 14.4 cm in diameter with a 12-cm radius. To define the head shape of each subject in 3-dimension (3D) for dipole source localization, the head shape was traced using 3D digitizer. The sensor was positioned over the left and the right temporal region of the subject, and remained in the same position for each hemisphere. The location of these coils in relation to the preauricular points and nasion were determined with a 3D digitizer before the start of data acquisition.

All signals were digitized at 4167 Hz and stored in a magneto-optical disk. The data were collected and analyzed using a software package (MSI software, WHS version 1.2.4, Biomagnetometer system) on a workstation (SUN, SPARC Station™). We used a single equivalent current dipole analysis based on the non-linear inverse problem with the least squares search (Sarvas, 1987), and the best fit single equivalent dipole was estimated every 0.5 ms using a software program (MSI software, WHS version 1.2.4, Biomagnetometer system) on a workstation (SUN, SPARC Station™). We calculated the correlation coefficient between the theoretical field generated by the estimated equivalent dipole model and the observed field for P50m and N100m. The correlation coefficients indicate how closely the measured values correspond to the theoretical field generated by the model. When the calculated correlation coefficient was less than 0.90, we determined that the single equivalent dipole was not observed. The dipole locations, moments and directions of P50m and N100m were calculated at the latency where the correlation coefficient was a maximum level. A spherical model was fitted to the digitized head shape of each subject and then the dipole locations were expressed by $x$, $y$ and $z$-coordinates. The zero point was the exact mid-point of the line between the bilateral preauricular points. The $x$-axis was the antero-posterior line with a positive $x$-axis extended from the zero point toward the nasion. The $y$-axis was the line from the right preauricular point to the left with positive $y$-axis extended from the zero point toward the left preauricular point. The (vertical) $z$-axis was the line orthogonal to both the $x$-axis and $y$-axis with positive $z$-axis extended from the zero point toward the upper side.

T1-weighted coronal MR images of eight subjects were acquired with contiguous (no-gap) 5-mm slice thickness using a Toshiba (MRT-50A) 0.5 T system. In order to overlay the calculated dipoles onto the MRI of the subject’s brain, the nasion and bilateral preauricular points were identified on the MRI images by registering the fiduciary points and the traced points over the scalp to MRI imaging.
2.4. Statistical analysis

The peak RMS or the peak latency values of the P50m and N100m were submitted to repeated measures ANOVA with stimulus type (S1 or S2), hemisphere (left or right) and component (P50m or N100m) as within-subjects factors. To assess the difference in the source location between P50m and N100m, we investigated the locations of the statistically significant P50m and N100m dipoles to S1. A repeated measures MANOVA with axis (x, y or z) and component (P50m or N100m) as within-subjects factors was performed for each hemisphere. Finally, to assess any differences in the source location between the S1 and the S1–S2 responses, we investigated the locations of the statistically significant P50m and N100m dipoles. A repeated measures MANOVA with axis (x, y or z) and response (S1 or S1–S2) as within-subjects factors was performed for each hemisphere and each component.

3. Results

The numbers of epochs for averaging were $117 \pm 7$ (mean±S.D.) for S1 and $116 \pm 7$ for S2 in the left hemisphere, and $116 \pm 10$ for S1 and $116 \pm 11$ for S2 in the right hemisphere, with no significant difference...
(one-way ANOVA, $F_{[3,64]}=0.08, P=0.97$). All of the subjects had P50m and N100m peaks to S1 and S2. For the left hemisphere, the number of peaks between 20 and 70 ms was as follows: a single peak (11 out of 17 subjects for S1 and 4 for S2), two peaks (5, 10), three peaks (1, 3). For the right hemisphere, the number of peaks was as follows: a single peak (11 for S1 and 9 for S2), two peaks (6, 7), three peaks (0, 1). The largest RMS amplitude with the reversed polarity to the N100m at the range was defined as the P50m RMS. Fig. 1 shows the auditory evoked magnetic responses to S1 and S2 from one subject.

3.1. RMS of P50m and N100m

The latency and RMS of P50m and N100m are shown in Table 1. Fig. 2 shows scattergrams of RMS to S1 and S2. A repeated measures ANOVA demonstrated significant main effects of stimulus ($F_{[1,16]}=86.2, P<0.001$) and component ($F_{[1,16]}=124.6, P<0.001$), but no main effect of hemisphere ($F_{[1,16]}=0.2, P=0.69$). No significant stimulus-by-hemisphere-by-component ($F_{[1,16]}=2.2, P=0.16$), stimulus-by-hemisphere ($F_{[1,16]}=0.8, P=0.38$), hemisphere-by-component ($F_{[1,16]}=0.9, P=0.36$) interactions were observed. However, there was a significant stimulus-by-component interaction ($F_{[1,16]}=59.3, P<0.001$). To further delineate the source of the stimulus-by-component interaction, we performed post hoc repeated measures ANOVAs with stimulus and hemisphere as within-subject factors for each component.

For P50m, repeated measures ANOVA demonstrated a significant main effect of the stimulus ($F_{[1,16]}=7.7, P=0.01$), but no main effect of the hemisphere ($F_{[1,16]}=0.8, P=0.40$) and no significant stimulus-by-hemisphere interaction ($F_{[1,16]}=0.3, P=0.57$), thus indicating that P50m to the second stimulus was significantly suppressed in both hemispheres. For N100m, a repeated measures ANOVA demonstrated a significant main effect of stimulus ($F_{[1,16]}=85.6, P<0.001$), but no main effect of hemisphere ($F_{[1,16]}=0.5, P=0.50$) and no significant stimulus-by-hemisphere interaction ($F_{[1,16]}=1.7, P=0.21$), thus indicating that the second stimulus was suppressed significantly in both hemispheres. In summary, both P50m and N100m showed significant suppression to the second stimulus, and the suppression was more significant in N100m.

3.2. Latency of P50m and N100m

Repeated measures ANOVA demonstrated a trend-level main effect of stimulus ($F_{[1,16]}=4.1, P=0.06$) and no main effect of hemisphere ($F_{[1,16]}=1.2, P=0.29$). No significant stimulus-by-hemisphere-by-component ($F_{[1,16]}=0.4, P=0.51$), stimulus-by-hemisphere ($F_{[1,16]}=1.6, P=0.23$), hemisphere-by-component ($F_{[1,16]}=0.1, P=0.79$) interactions. However, there was a significant stimulus-by-component interaction ($F_{[1,16]}=9.7, P=0.007$). To further delineate the source of the stimulus-by-component interaction, we performed post hoc repeated measures ANOVAs with stimulus and hemisphere as within-subjects factors for each component.

For P50m, there were no main effects of stimulus ($F_{[1,16]}=0.004, P=0.95$), hemisphere ($F_{[1,16]}=1.0, P=0.33$), and no stimulus-by-hemisphere interaction ($F_{[1,16]}=0.6, P=0.46$), thus indicating no stimulus effect to P50m latency. For N100m, a repeated measures ANOVA demonstrated a significant main effect of stimulus ($F_{[1,16]}=8.4, P=0.01$), but no main effect of hemisphere ($F_{[1,16]}=0.5, P=0.51$) and no significant stimulus-by-hemisphere interaction ($F_{[1,16]}=1.3, P=0.26$), thus indicating that the N100m latency of the second stimulus was earlier than the first one.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Latency and RMS of P50m and N100m to the first (S1) and second (S2) stimuli for each hemisphere</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>S1</td>
</tr>
<tr>
<td>P50m</td>
<td></td>
</tr>
<tr>
<td>Left</td>
<td></td>
</tr>
<tr>
<td>Latency (ms)</td>
<td>48.5±11.2</td>
</tr>
<tr>
<td>RMS (fT)</td>
<td>38.1±13.4</td>
</tr>
<tr>
<td>Right</td>
<td></td>
</tr>
<tr>
<td>Latency (ms)</td>
<td>44.7±8.3</td>
</tr>
<tr>
<td>RMS (fT)</td>
<td>34.8±15.2</td>
</tr>
<tr>
<td>N100m</td>
<td></td>
</tr>
<tr>
<td>Left</td>
<td></td>
</tr>
<tr>
<td>Latency (ms)</td>
<td>97.7±9.9</td>
</tr>
<tr>
<td>RMS (fT)</td>
<td>132.8±37.1</td>
</tr>
<tr>
<td>Right</td>
<td></td>
</tr>
<tr>
<td>Latency (ms)</td>
<td>93.0±8.1</td>
</tr>
<tr>
<td>RMS (fT)</td>
<td>144.5±47.3</td>
</tr>
</tbody>
</table>
3.3. The dipole sources of P50m and N100m

Thirteen subjects had statistically significant P50m and N100m dipoles to the first stimulus. The x, y, and z-coordinates of P50m and N100m are shown in Table 2. For the left hemisphere, a repeated measures MANOVA demonstrated a significant component-by-axis interaction ($F_{[2,11]}=10.1$, $P=0.003$) but no main effect of the component ($F_{[1,12]}=2.6$, $P=0.13$). To further delineate this interaction, we performed paired t-tests for each axis. There was a significant difference in the y-coordinate ($t_{[12]}=-3.9$, $P=0.002$), thus indicating that N100m was located significantly more laterally than P50m. There was no difference in the x-coordinate ($t_{[12]}=1.1$, $P=0.31$) or the z-coordinate ($t_{[12]}=0.4$, $P=0.67$). For the right hemisphere, a repeated measures MANOVA demonstrated

<table>
<thead>
<tr>
<th></th>
<th>P50m</th>
<th>N100m</th>
<th>df</th>
<th>t</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>x (cm)</td>
<td>0.13±0.93</td>
<td>-0.62±0.77</td>
<td>12</td>
<td>1.05</td>
<td>0.31</td>
</tr>
<tr>
<td>y (cm)</td>
<td>4.26±1.25</td>
<td>5.66±0.47</td>
<td>12</td>
<td>-3.86</td>
<td>0.01</td>
</tr>
<tr>
<td>z (cm)</td>
<td>5.27±1.82</td>
<td>5.11±0.64</td>
<td>12</td>
<td>0.43</td>
<td>0.67</td>
</tr>
<tr>
<td>Right</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>x (cm)</td>
<td>0.42±1.34</td>
<td>0.32±0.98</td>
<td>12</td>
<td>0.25</td>
<td>0.81</td>
</tr>
<tr>
<td>y (cm)</td>
<td>-4.58±0.76</td>
<td>-5.42±0.57</td>
<td>12</td>
<td>-4.81</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>z (cm)</td>
<td>5.24±1.43</td>
<td>4.89±0.67</td>
<td>12</td>
<td>0.86</td>
<td>0.41</td>
</tr>
</tbody>
</table>
a significant component-by-axis interaction ($F[2,11] = 5.5$, $P = 0.02$) but no main effect of the component ($F[1,12] = 0.4$, $P = 0.54$). To further delineate this interaction, we performed paired $t$-tests for each axis. There was a significant difference in the $y$-coordinate ($t[12] = -4.8$, $P < 0.001$), thus indicating that N100m was located significantly more laterally than P50m. There was no difference in the $x$-coordinate ($t[12] = 0.3$, $P = 0.81$) or the $z$-coordinate ($t[12] = 0.9$, $P = 0.41$). For the dipole strength, the dipole moments ($|Q|$) and directions ($Q_x$, $Q_y$, $Q_z$) of P50m and N100m are shown in Table 3. There were significant $Q_x$ and $Q_z$ differences between P50m and N100m, thus indicating that the P50m direction is opposite to the N100m. In summary, N100m was located more laterally than P50m for both hemispheres. Fig. 3 shows the source locations of P50m and N100m to the first stimulus of one subject, which are projected onto the appropriate coronal MRI sections. The P50m was located in the HG and the N100m was located in the PT for both hemispheres.

3.4. The dipole source differences between S1 and S1–S2 responses

Nine of the 17 subjects had statistically significant dipoles of P50m, and 11 of the 17 subjects had significant dipoles of N100m in both hemispheres. The $x$, $y$, and $z$-coordinates of the S1 and the S1–S2 responses are shown in Supplement 2. For the left hemisphere, a repeated measures MANOVA demonstrated no main effect of the response (P50m: $F[1,8] = 0.1$, $P = 0.82$, N100m: $F[1,10] = 1.5$, $P = 0.24$) with no significant response-by-axis interaction (P50m: $F[2,7] = 0.4$, $P = 0.68$, N100m: $F[2,9] = 1.7$, $P = 0.23$). For the right hemisphere, a repeated measures MANOVA demonstrated no main effect of the response (P50m: $F[1,8] = 3.5$, $P = 0.1$, N100m: $F[1,10] = 2.3$, $P = 0.16$) with no significant response-by-axis interaction (P50m: $F[2,7] = 2.2$, $P = 0.18$, N100m: $F[2,9] = 0.9$, $P = 0.43$).

4. Discussion

This study investigated the auditory evoked magnetic fields to voice stimuli using a conditioning–testing paradigm. The major findings of this study were: (1) for RMS both P50m and N100m showed a significant suppression to the second stimulus bilaterally, and the suppression was more significant in N100m; (2) The N100m was located significantly more laterally than the P50m for both hemispheres.

The ability of the brain to suppress incoming irrelevant sensory input is called sensory gating (Freedman et al., 1991). The sensory auditory gating has been found as the P50 suppression in normal subjects (Edgar et al., 2003; Hanlon et al., 2005). Voice recognition represents an evolutionary significant element of social communication. To suppress incoming irrelevant vocal sensory input seems important to solve adaptive problems critical for survival, such as distinguishing friend from foe, familiar from unfamiliar, related from unrelated. It has been reported that deficits of sensory gating cause sensory overload in patients with schizophrenia (Adler et al., 1982; Braff, 1993). Moreover, auditory verbal hallucinations are the most
common symptoms, being reported by approximately 70% of patients with schizophrenia (Sartorius et al., 1986). The present study indicated a significant suppression of P50m and N100m to the second voice stimulus in the bilateral HG and PT, suggesting that there is the gating effect to vocal sounds as well as to non-vocal sounds in the human brain. The future study is thus called for the investigation on the sensory gating to vocal sounds in patients with schizophrenia. This study also showed that the N100m suppression was more significant than that for P50m. The larger effects of ISI may indicate that the number of synaptic chains is larger in the more affected component. Functionally, the primary auditory area is mainly located in the HG, and the auditory association area is located in the PT (Hall et al., 2003); thus it may be reasonable to assume that the N100m suppression was more significant than the P50m.

The N100m peak latency in the current study was shorter in comparison to several reports on the N100m to vocal sounds (e.g., Obleser et al., 2003; Tiitinen et al., 2005). One possible reason for shorter latencies of N100m in the current study may be methodological differences. For example, Obleser et al. (2003) and Tiitinen et al. (2005) delivered auditory stimuli binaurally and recorded N100m. Yoshiura et al. (1994) pointed out that the ipsilateral N100m latency was delayed since the ipsilateral N100m was activated through corpus callosum. Binaurally stimulated N100m may be an overlapping activity of the ipsilateral and the contralateral N100m, and thus the N100m latency is longer compared to the contralateral N100m latency with monaural stimulation.

In terms of dipole locations, it was reported that the P50m located anterior to the N100m (Mäkelä et al., 1994; Huotilainen et al., 1998), although statistical significances were unclear in these studies. In the present study, the N100m source was localized significantly more laterally than the P50m source. The P50m located anterior and inferior to the N100m, but these differences did not reach statistical significance. Differences in the stimuli (i.e., click, tone burst or human voice) used across studies may account for differences in the findings between those studies. In the present study, both P50m and N100m to voice stimulus were located in STG, while recent fMRI studies reported that bilateral STS regions responded to human voice selectively (Belin et al., 2000; Fecteau et al., 2004). In MEG, the single equivalent current dipole indicates the center location of auditory evoked responses. The difference in the time resolution between MEG and fMRI should be noted.

The suppression effect of P50 to the second stimulus is referred to as a gating ratio (S2/S1). The gating ratio of the present study seems smaller compared to previous studies on P50 evoked by click stimuli in normal subjects (Edgar et al., 2003; Hanlon et al., 2005). Chen et al. (1997) reported that the P50 to the human voice stimuli was significantly larger than the P50 to tone burst stimuli and there was no significant difference between stimuli in N100, using a repetitive stimulus paradigm with 2-s ISI. The ISI effect to the P50 elicited by the vocal sound might be smaller compared to the effect to the P50 evoked by non-vocal sounds, perhaps related to a smaller gating effect to vocal sounds.

There are several limitations in this study. First, it should be noted that the present study used shorter IPI compared to previous EEG studies. For EEG, the recovery time of P50 was reported to be around 8 s (Zouridakis and Boutros, 1992), and that of the N100 was reported to be nearly 10 s (Davis et al., 1966). For MEG, Lu et al. (1992) reported that the N100m response was recovered more than 90% with 6-s ISI and the recovery was asymptotic. Second, it is still unclear how methodological differences (e.g. click or voice, durations of stimuli, the number of epochs for averaging) affect MEG waveforms in the conditioning–testing P50 paradigm. Third, we cannot rule out the possibility that all artifacts arose from the head movement of subjects since we used a 37-channel biomagnetometer with a spherical surface covering a circle of 14.4 cm. To reduce artifacts, we registered the locations of the preauricular points and nasion in relation of the biomagnetometer for each session in the present study.

In summary, both P50m and N100m showed a significant suppression to the second stimulus bilaterally, and the N100m was located significantly more laterally than the P50m in both hemispheres. These results are thus considered to demonstrate the presence of sensory gating for the auditory inputs of the human voice in the primary auditory cortex and the auditory association area.

Acknowledgments

This work was supported in part by a grant-in-aid for Scientific Research (C17591218) from the Ministry of Education, Culture, Sports, Science and Technology, Japan (to Dr. Onitsuka). The authors gratefully acknowledge the valuable technical assistance of Mr. Kazumitsu Takahashi and Miss Yuko Somehara.

Appendix A. Supplementary data

References


