Cortical Response to Auditory Motion Suggests an Asymmetry in the Reliance on Inter-Hemispheric Connections Between the Left and Right Auditory Cortices

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Krumbholz K, Hewson-Stoate N, Schönwiesner M. Cortical response to auditory motion suggests an asymmetry in the reliance on inter-hemispheric connections between the left and right auditory cortices. J Neurophysiol 97: 1649–1655, 2007. First published November 15, 2006; doi:10.1152/jn.00560.2006. The aim of the current study was to measure the brain’s response to auditory motion using electroencephalography (EEG) to gain insight into the mechanisms by which hemispheric lateralization for auditory spatial processing is established in the human brain. The onset of left- or rightward motion in an otherwise continuous sound was found to elicit a large response, which appeared to arise from higher-level nonprimary auditory areas. This motion onset response was strongly lateralized to the hemisphere contralateral to the direction of motion. The response latencies suggest that the ipsilateral response to the leftward motion was produced by indirect callosal projections from the opposite hemisphere, whereas the ipsilateral response to the rightward motion seemed to receive contributions from direct thalamocortical projections. These results suggest an asymmetry in the reliance on inter-hemispheric projections between the left and right auditory cortices for auditory spatial processing.

INTRODUCTION

Motion processing is an important aspect of spatial hearing and has attracted considerable interest in the auditory neuroimaging literature (Baumgart et al. 1999; Hart et al. 2004; Krumbholz et al. 2005a,b; Seifritz et al. 2002b; Warren et al. 2002). In humans, the perception of sound location and motion is mainly based on binaural cues, i.e., interaural level differences (ILDs), produced by the head shadow effect, and differences in sound arrival time at the two ears (interaural time differences, ITDs), produced by path length differences between the ears and the sound source. While the initial processing of ILDs and ITDs is known to begin as early as the brain stem (see, e.g., Palmer and Kuwada 2005), the neuroimaging data suggest that sensitivity to auditory motion does not emerge before the level of nonprimary auditory cortex (Krumbholz et al. 2005a). Moreover, the neuroimaging data also indicate that auditory motion processing engages the right hemisphere more strongly than the left (Baumgart et al. 1999; Krumbholz et al. 2005b; Seifritz et al. 2002b). However, inferences about the hierarchy and hemispheric lateralization of processing from neuroimaging data are bound to be indirect, and to a certain extent reliant on assumptions, because these data convey no information about the number, the latencies, or the lateralization of the subprocesses that might be contributing to the activation. Electrophysiological (electro- and magnetoencephalography, EEG/MEG) measures of the brain’s response to sounds have been shown to consist of a series of deflections, which are likely to be composed of functionally independent subcomponents of the response (for a review, see Näätänen and Winkler 1999). Some of these deflections have been related to specific anatomical areas (Lütkenhöner and Steinrücker 1998; Lütkenhöner et al. 2003; Yvert et al. 2001), and it has been shown that different deflections may exhibit different patterns of hemispheric lateralization (Chait et al. 2004).

The current study uses EEG to investigate the processing of sound motion in the human auditory cortex. The response to the onset of motion in an otherwise continuous sound was measured when the response to sound onset had already subsided to isolate any motion-sensitive response from the unspecific response to the onset of sound energy. The perception of motion was mediated purely by ITDs to ensure that any motion-sensitive response would not be confounded with spectral or energetic changes in the sound. The aim of the study was to test whether the pattern of hemispheric lateralization found in the neuroimaging data would also be reflected in the electrophysiological response to motion onset. Specifically, we hoped that the latencies of the electrophysiological response would reveal whether the hemispheric asymmetry in the cortical response to auditory motion is due to an asymmetry in the indirect, inter-hemispheric transfer of auditory spatial information or in the direct projections from subcortical structures. Moreover, we wanted to investigate whether the temporal dynamics of the initial response to the onset of sound motion would support the conclusion from the neuroimaging data that sensitivity to auditory motion first emerges at the level of nonprimary auditory cortex.

METHODS

Stimuli

The stimuli consisted of a 1,000-ms “control” portion and a 150-ms “test” portion and were presented with an interstimulus interval (ISI) of 1,000 ms. Both the control and the test portion contained a bandpass-filtered noise. The ITD of the noise was zero throughout the control portion and was changed linearly from zero to ±1,000 μs (a

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positive ITD means the right ear is leading) during the test portion to create the perception of a sound that was first stationary in the center of the head and, after 1,000 ms, moved swiftly from the center to the left or right ear. As the band-pass-filtered noise also served as control stimuli in a companion study investigating the neural correlates of phonological processing, its spectral and temporal properties were designed to match the properties of vowels. The noise was filtered into three frequency bands (equivalent to the formant frequencies in vowels, each with a bandwidth of \( \pm 30\% \) around the respective filter center frequency. The center frequencies of the three bands were selected randomly so that the bands would fall within the ranges set by the lowest and highest frequency values of the first three formants of the vowels \( /a/, /e/, /i/, \) and \( /o/ \) (0.22–0.86 kHz for the lowest band, 0.87–2.25 kHz for the middle band, and 1.96–3.3 kHz for the highest band). The three frequency bands were weighted according to a spectral profile with a slope of \(-6\) dB per octave. The noise was multiplied with a periodic envelope mimicking the sequence of glottal pulses. The shape of each pulse was a gamma function featuring a fast attack and an exponential decay with a half-life of 2.5 ms; the pulses were repeated at a rate of 100 Hz. The overall level of the noise amounted to \(-65\) dB SPL. The noise was generated afresh, with different filter frequencies, for each stimulus presentation.

**Data acquisition**

Participants were seated in a comfortable chair and auditory-evoked potentials were recorded using a 61-channel "subtemporal" EEG cap with equidistantly arranged Ag/AgCl sintered ring electrodes (Easy Cap, Falk Minow Services, Munich, Germany) and the BrainAmp DC and BrainAmp MR EEG amplifiers (Brain Products, Munich, Germany). Data were recorded continuously at a sampling rate of 500 Hz and high-pass-filtered on-line at 0.1 Hz. To maintain vigilance, participants watched a self-chosen silent movie during the recording. Three additional electrodes were positioned around the left eye to record horizontal and vertical eye movements. The ground electrode was placed on the center of the forehead, just above the Nasion, and for on-line monitoring, data were referenced to an additional central electrode in the occipital region, a little superior to the Inion.

**Data analysis**

The raw data were corrected for ocular artifacts using the Gratton et al. (1983) procedure as implemented in the BrainVision Analyser software (Brain Products) and re-referenced to the average of all 61 channels. Data exceeding a maximum-minimum difference of 150 \( \mu \text{V} \) within any 100-ms window were considered artifactual, and a 500-ms window surrounding the artifact was removed from subsequent analysis. The data were low-pass-filtered at 35 Hz (with a slope of 48 dB per octave) and divided into 2,500-ms stimulus-locked epochs covering the period from \(-500\) to 2,000 ms relative to stimulus onset. The epochs for each condition were averaged and baseline-corrected to the 200-ms period of silence just before stimulus onset. The response to the control portion shows that the stimuli produced a relatively large sustained response (SR), on which the transient response to the onset of motion at the beginning of the test portion was superposed (see Fig. 1B). The sustained response appeared to decay back to baseline between about 500 and 675 ms after the onset of the test portion (1,500–1,675 ms rel. stimulus onset). Thus, to analyze the transient motion onset response (MOR), the data were corrected for a baseline that was constant and equal to the average of the 200-ms period just before the onset of the test portion (800–1,000 ms) for times \( \pm 1,500\) ms, and then decreased linearly to zero between 1,500 and 1,675 ms.

The sources of the responses to the control and test portions of the stimuli were analyzed with a single fixed dipole in each hemisphere and a four-shell ellipsoidal volume conductor as head model using version 5.1 of the Brain Electrical Source Analysis software (BESA, Gräfelfing, Germany).

**Participants**

A total of 15 participants (7 female, 8 male, age range: 23–40 yr) took part in the current experiment after having given written informed consent. All participants were right handed [as assessed by a modified version of the Edinburgh inventory (Oldfield 1971)] and had no history of audiological or neurological disease. The experimental procedures conformed with the Code of Ethics of the World Medical Association (Declaration of Helsinki) and were approved by the Ethics Committee of the University of Nottingham Medical School.

**RESULTS**

**Cortical response to the onset of motion in a continuous sound**

Although the perception of motion during the test portion of the stimuli was based purely on changes in the sounds’ interaural temporal properties and was not associated with any spectral or energetic changes (the change was inaudible when listening monaurally), the onset of motion in an otherwise continuous sound produced a prominent transient response, the morphology of which differed markedly from that of the transient response to sound energy onset. Figure 1B shows the grand-average response to the entire stimulus (see Fig. 1A), averaged across both the left- and rightward motion conditions and baseline-corrected to the 200-ms period prior to stimulus onset. The response has a transient onset, followed by a sustained phase. The transient onset response (TOS) is characterized by an initial positive deflection, followed by a negative peak, and then a return to baseline. The sustained phase is characterized by a positive deflection that decays back to baseline.
onset. As expected from previous data (Chait et al. 2004) the energy onset response (EOR) to the noise was dominated by two vertex-positive deflections. In the vertex channel (Cz; highlighted by bold black line in Fig. 1B and shown separately by the gray line in Fig. 2), the first positive deflection exhibited two peaks, one at ~38 and one at 62 ms, suggesting that this deflection represents a superposition of two responses, the P30 of the middle-latency responses and the P1 (Fig. 2) (Yvert et al. 2001). The first positive deflection was followed by a small negative deflection (N1) (Na¨a ¨ta¨nen and Picton 1987), peaking at ~94 ms (in Cz; same for the rest of the latencies given in this paragraph), and another large positive deflection (P2) (Lütkenhönner and Steinsträter 1998) at 168 ms. In contrast, the motion onset response (see MOR in Fig. 1B and black line in Fig. 2) was dominated by a large negative deflection, henceforth referred to as the “change”-N1 (cN1), peaking at ~140 ms after motion onset (1,140 ms rel. stimulus onset). Baseline-correcting to the 200-ms period prior to motion onset (see METHODS) revealed that the cN1 was preceded by a small positive deflection, referred to as the cP1, peaking ~76 ms post motion onset, and followed by two positive deflections, referred to as the cP2a and cP2b, at 228 and 322 ms post motion onset (Fig. 2).

Overall, the motion onset response started much later than the energy onset response. In the vertex channel, the energy onset response first exceeded the 99% quantile of the distribution of root mean square (rms) potentials during the 200-ms prestimulus baseline as early as 14 ms after stimulus onset (see solid gray vertical line in Fig. 2). In contrast, the motion onset response did not exceed this criterion before 54 ms after motion onset (solid black vertical line).

Averaging the motion onset responses separately for the left- and rightward motion conditions (Fig. 3A) revealed that the onset of motion produced a larger response in the hemisphere contralateral to the motion direction compared with the ipsilateral hemisphere. Moreover, the scalp distributions of the most prominent deflection of the responses (cN1; taken at the maximum of the rms amplitude across all channels, see Fig. 3B) suggest that the response to the leftward motion onset exhibited a stronger contralateral bias than the response to the rightward motion onset (see postero-temporal pattern of distributions, shown in red).

**Hemispheric asymmetry of the motion onset response**

It is difficult and often misleading to infer the hemispheric lateralization of a response from the channel data directly because a dipolar source in one hemisphere may well project maximally to the opposite side of the scalp, depending on the orientation of the source. To obtain a more reliable estimate of response lateralization, we analyzed the sources of the responses using equivalent current dipole modeling (BESA v5.1). The locations and orientations of two dipoles, one in each hemisphere, were fit to the grand-average motion onset response, averaged across the left- and rightward motion conditions and baseline-corrected to the 200-ms period prior to motion onset (see METHODS). The locations of the two dipoles were constrained to be symmetric about the mid-sagittal plane, and the fitting was performed within a 40-ms time window around the rms peak of the average cN1 response (120–160 ms post motion onset). For comparison, a similar two-dipole model was fit to the sustained response during the control portion of the stimuli within the time window from 400 to 1,000 ms post stimulus onset. Previous findings indicate that the sustained response to a noise represents an unsppecific response to the presence of sound energy and is generated by a source centered on the planum temporale (PT) (Gutschalk et al. 2002, 2004). The two-dipole model for the motion onset response was then used as a spatial filter to derive the activation time-course of each dipole (source waveform) for the two stimulus conditions (left- and rightward motion) and for each individual separately.

Within the fit window, the residual variance of the dipole model for the motion onset response amounted to only 1.37% for the leftward and 0.99% for rightward motion condition; the residual variance of the dipole model for the sustained response
was 2.32%. The dipole locations were consistent with the responses being generated on the supratemporal plane in the region of the auditory cortex. The dipole for the motion onset response (red dipoles in Fig. 4) was located slightly anterior (2 mm), inferior (4.4 mm), and medial (3.4 mm) to the dipole for the sustained response (blue dipoles). However, given that dipole locations can exhibit a considerable inter- and intra-individual variability (Lütkenhöner and Steinsträter 1998), these differences would probably be considered insignificant.

The source waveforms indicate that the responses to both the left- and rightward motion onset were biased toward the respective contralateral hemisphere (Fig. 5); however, consistent with the scalp distributions of the responses, the contralateral bias appeared to be larger for the response to the leftward (Fig. 5A) than the rightward motion condition (Fig. 5B). The four deflections cP1, cN1, and cP2a/b, identified from the channel data (Fig. 2), also appear to be clearly recognizable in the source waveforms, suggesting that all four deflections receive major contributions from sources within or close to auditory cortex. Moreover, the pattern of lateralization appeared to be reasonably consistent across the four deflections. The source waveforms also revealed a sizable latency difference in the cN1 deflection between the ipsi- and contralateral dipole. For the leftward motion condition (Fig. 5A), the cN1 deflection in the source waveform of the right (contralateral) dipole (dashed line) peaked by as much as 18.7 ms earlier than in the source waveform for the left (ipsilateral) dipole (solid line). The cN1 deflection in the source waveforms for the response to the rightward motion onset (Fig. 5B) exhibited a corresponding latency difference, but the difference was considerably smaller in this case (6.1 ms).

To test the statistical significance of these effects, the peak amplitude and latency of the cN1 deflection in the source waveform for each of the two dipoles and for each of the two motion conditions were assessed separately for each individual. The mean peak amplitudes and latencies as well as the mean latency differences between the ipsi- and contralateral responses are summarized in Fig. 6. The amplitudes and latencies were each submitted to a two-way, repeated-measures ANOVA with motion direction (left and right) and hemisphere (ipsi- and contralateral) as independent within-subjects factors. The analyses revealed that the main effect of hemisphere was highly significant for both the amplitudes \[ F(1,14) = 20.370, P < 0.0005 \] and the latencies \[ F(1,14) = 23.996, P < 0.0002 \] and that the main effect of motion direction was insignificant in both cases. However, the interaction between motion direction and hemisphere reached significance only for the latencies \[ F(1,14) = 9.557, P = 0.008 \]. The interaction was due to the difference between the ipsi- and contralateral latencies being significantly larger for the response to the leftward than the rightward motion onset (18.7 > 6.1 ms; \[ t(14) = 3.091, P = 0.008 \]). In fact, although the difference between the ipsi- and contralateral latencies was highly significant for the leftward motion condition \[ t(14) = 6.217, P < 0.0001 \], the difference was insignificant for the rightward motion condition when...
using a two-tailed test [$t(14) = 1.769, P = 0.0986$] and only became significant when using a one-tailed test (ipsi > contra; $P = 0.0493$).

**DISCUSSION**

The current results show that the onset of motion in an otherwise continuous sound produces a prominent transient response, the morphology of which differs markedly from that of the transient response to sound energy onset and the topography of which exhibits a striking sensitivity to the direction of the motion. In the current experiment, the perception of motion was mediated purely by ITDs; this means that the motion onset response must have been generated by neural elements sensitive to the interaural temporal properties of sounds.

The motion onset response started much later (by ~40 ms, see **RESULTS**) than the energy onset response and its first positive deflection (cP1) did not seem to be double-peaked as in the energy onset response. The rising flank of the first peak in the first positive deflection of the energy onset response (P30) is thought to represent the initial primary auditory cortical response to the sound generated when the neural volley reaches the cortex from subcortical structures (Borgmann et al. 2001; Eggermont and Ponton 2002; Lütkenhöner et al. 2003; Yvert et al. 2001). The absence of any P30 deflection in the motion onset response was not due to the energy onset producing an overall larger response than the motion onset; on the contrary, the later deflections in the motion onset response were even larger than in the energy onset response. Moreover, it seems unlikely that the absence of any P30 deflection in the motion onset response was due to the deflection being superposed by another deflection with opposite polarity and a similar latency. It may be the case that the motion-onset response contains earlier induced components, i.e., components that are not time-locked to the stimulus and would thus not be reflected in the average response. However, this possibility also seems unlikely as any motion-specific induced response in the primary auditory cortex would be expected to be detectable through fMRI, and fMRI has so far failed to show any significant motion-specific activity in primary auditory areas (e.g., Krumbholz et al. 2005a). Thus although the absence of activation is generally difficult to interpret, the absence of any significant response to motion onset for latencies up to ~50 ms suggests that the predominant contributions to the motion onset response arise from higher-order nonprimary auditory areas with comparatively little contribution from primary auditory cortex. Recent fMRI results have shown that activity in nonprimary auditory areas shows a greater degree of adaptation to prolonged, unchanging sounds, such as the control portion of the stimuli used in the current experiment, than activity in the primary auditory cortex (Seifritz et al. 2002a; see also Giraud et al. 2000; and Harms and Melcher 2002). The current results suggest that the motion onset response was caused by ITD-sensitive neural elements in which the swift change in ITD at the onset of the test portion of the stimuli triggered a release from the adaptation caused by the stimuli’s prolonged control portion; this conclusion is also consistent with recent fMRI results on motion processing (Krumbholz et al. 2005).

The finding that motion-sensitive responses measured with fMRI and EEG seem to be generated in auditory areas does not mean that ITD sensitivity does not emerge until after the primary auditory cortex. ITD processing requires extreme temporal precision (Louage et al. 2005), which auditory cortical neurons would be unable to provide (Eggermont 2002; Lu et al. 2001). This means that the basic processing of ITDs must be completed by the time the neural volley reaches the auditory cortex and thus that the primary auditory cortex must contain neurons that exhibit ITD sensitivity of some form. The fact that these neurons do not seem to appreciably contribute to the auditory cortex’s response to ITD-mediated sound motion is intriguing and may suggest a change in the format of the ITD code from the primary to nonprimary auditory areas with a possible role of the change in the neurons’ temporal response characteristics (response adaptation) (Seifritz et al. 2002a). A similar situation seems to apply to other sound qualities, such as interaural correlation or temporal pitch, the processing of which would be expected, or has been shown, to start at subcortical levels (Griffiths et al. 2001), but the cortical response of which nevertheless seems to be confined to nonprimary areas (Budd et al. 2003; Hall et al. 2005).

Previously, it has been shown that late cortical responses can also be elicited by other kinds of changes in the interaural temporal properties of otherwise continuous sounds, such as an abrupt change in interaural coherence (Chait et al. 2005; Jones et al. 1991) or a singular change in ITD (Jones et al. 1991; McEvoy et al. 1990; Sams et al. 1993; Ungan et al. 2001). The generators of the motion onset response observed in the current study would be expected to be closely related to the generators of these other kinds of responses. Particularly, given that the current stimuli involved a swift change in ITD over time and, conversely, a singular ITD change evokes a strong sense of motion, the motion onset response would be expected to involve similar processes as the response to a singular ITD change. It is thus surprising that neither of the two previous studies that have investigated the response to a singular ITD change separately within the left and right hemifields (Jones et al. 1991; Ungan et al. 2001) have observed any hemispheric lateralization with respect to the hemifield within which the ITD change occurred. Given the unequivocal response lateralization in the current as well as previous fMRI data (Krumbholz et al. 2005b) and given previous findings that even the energy onset response (N1) to sounds with ITDs exhibits contralateralization (McEvoy et al. 1993, 1994; Soeta and Nakagawa 2006), we assume that the absence of lateralization in these two studies was due to procedural reasons. Jones et al. for instance, used only a few electrodes, which may have made it difficult to detect any response lateralisation. In the case of Ungan et al., the absence of lateralization may have been due to the use of broadband clicks as stimuli in combination with a fairly large ITD change of 1,000 μs. Theoretical considerations suggest that if the brain codes interaural phase rather than interaural delay, which physiological studies indicate is the case, the pattern of lateralisation in the response to sounds with ITDs might change from favoring the contralateral to favoring the ipsilateral hemisphere when the ITD is large and the sound contains a large proportion of higher-frequency energy (McAlpine 2005; McAlpine and Grothe 2003; Thompson et al. 2006; Trahiotis and Stern 1989). However, as these explanations remain tentative, more research into the relationship between the motion onset response and the response to a
singular ITD change will be needed to resolve the discrepancy between the present and previous results.

The dipole analysis suggests that the location of the source of the motion onset response is similar to that of the sustained response to the noise stimulus. According to previous research (Gutschalk et al. 2002, 2004), the sustained response to a noise may be assumed to represent an unspecific response to the presence of sound energy and is generated by a source centered on the planum temporale (PT), similar to the N1 response to sound energy onset (Gutschalk et al. 2004). In contrast to the motion onset response, different kinds of pitch-related responses (i.e., the pitch-related sustained response and the response to the onset of pitch in an otherwise continuous sound) have consistently been shown to arise from a source located at a considerable distance (~10–15 mm) anterior to the energy-related responses (Gutschalk et al. 2004; Krumhholz et al. 2003). Although the relative locations of the motion onset and pitch-related responses would have to be confirmed in a dedicated experiment measuring pitch and motion onset responses in the same set of participants, the current data, together with previous results, seem to be consistent with the finding from fMRI data that pitch and motion processing activate different nonprimary auditory areas with the pitch-related area being located anterior to the motion-related area (Barrett and Hall 2006; Warren and Griffiths 2003).

Comparing the motion onset response for the left- and rightward motion conditions revealed that the response was considerably stronger and earlier in the hemisphere contralateral than ipsilateral to the motion direction. Note that in the current experiment, the direction of the motion was identical to the hemifield within which the sound was moving, and so it cannot be excluded (it is indeed likely) that the lateralization of the motion onset response was determined by the range of locations covered by the sounds’ motion rather than the direction of the motion as such. Superimposed on the pattern of contralateralization appeared to be a rightward asymmetry in that the contralateral latency advantage (i.e., the difference in latency between the ipsilateral and contralateral response) in the cN1 deflection of the response was about three times larger for the leftward than the rightward motion onset. Moreover there was a tendency (albeit insignificant) for the contralateral advantage in the size of the response to also be larger for the leftward than the rightward motion condition. This pattern of hemispheric asymmetry is consistently consistent with certain results from the electrophysiological and neuroimaging as well as the neuropsychological literature. A recent fMRI study on auditory motion processing, for instance, also found that sounds moving within the right hemifield activate both the contra- and ipsilateral auditory cortices, whereas sounds moving within the left hemifield predominantly activate the contralateral (right) hemisphere (Fig. 7). Depending on stimulus context, a similar pattern of lateralisation may also be exhibited by monaural sounds (Schönwiesner et al. 2006) and by dichotically presented sounds (Deoull et al. 1998). The large discrepancy in contralateral latency advantage between the leftward (18.7 ms) and rightward motion conditions (6.1 ms) suggests that the ipsilateral responses in the two conditions were produced by different mechanisms. In view of the large contralateral latency advantage for the leftward motion response (18.7 ms), it seems likely that a major part of the ipsilateral response to the leftward motion onset was relayed from the contralateral hemisphere through callosal connections. Although psychophysical estimates of the interhemispheric transfer time range from a few milliseconds to a few tens of milliseconds depending on task and sensory modality (Bjorklund and Lian 1993; Cherbuin and Brinkman 2006; Fendrich et al. 2004; Jäncke 2002; Jäncke and Steinmetz 1994), theoretical estimates based on physiological considerations usually range around a few tens of milliseconds (see Hutsler et al. 2002 for a review). In contrast, the ipsilateral response to the rightward motion onset probably also contained direct contributions from subcortical structures. The hypothesis that there is a fundamental asymmetry in the role of interhemispheric processes in auditory spatial processing between the left and right hemispheres is broadly consistent with recent findings of specific sound lateralization deficits in patients with callosotomy (Hausmann et al. 2005), which also indicate a stronger reliance of the left hemisphere on the callosal transfer of auditory spatial information form the right hemisphere than vice versa. Moreover, it would also appear to be related to the finding that the degree of plastic reorganization of the adult human auditory cortex consequent to late-onset profound unilateral deafness is ear dependent (Khosla et al. 2003), such that a reduction in the contralaterality of the late auditory evoked potentials in response to monaural sounds occurs only in the case of left-ear but not right-ear unilateral deafness.

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**References**


