Cortical evoked potentials to an auditory illusion: Binaural beats

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A B S T R A C T

Objective: To define brain activity corresponding to an auditory illusion of 3 and 6 Hz binaural beats in 250 Hz or 1000 Hz base frequencies, and compare it to the sound onset response.

Methods: Event-Related Potentials (ERPs) were recorded in response to unmodulated tones of 250 or 1000 Hz to one ear and 3 or 6 Hz higher to the other, creating an illusion of amplitude modulations (beats) of 3 Hz and 6 Hz, in base frequencies of 250 Hz and 1000 Hz. Tones were 2000 ms in duration and presented with approximately 1 s intervals. Latency, amplitude and source current density estimates of ERP components to tone onset and subsequent beats-evoked oscillations were determined and compared across beat frequencies with both base frequencies.

Results: All stimuli evoked tone-onset P50, N100 and P200 components followed by oscillations corresponding to the beat frequency, and a subsequent tone-offset complex. Beats-evoked oscillations were higher in amplitude with the low base frequency and to the low beat frequency. Sources of the beats-evoked oscillations across all stimulus conditions located mostly to left lateral and inferior temporal lobe areas in all stimulus conditions. Onset-evoked components were not different across stimulus conditions; P50 had significantly different sources than the beats-evoked oscillations; and N100 and P200 sources located to the same temporal lobe regions as beats-evoked oscillations, but were bilateral and also included frontal and parietal contributions.

Conclusions: Neural activity with slightly different volley frequencies from left and right ear converges and interacts in the central auditory brainstem pathways to generate beats of neural activity to modulate activities in the left temporal lobe, giving rise to the illusion of binaural beats. Cortical potentials recorded to binaural beats are distinct from onset responses.

Significance: Brain activity corresponding to an auditory illusion of low frequency beats can be recorded from the scalp.

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1. Introduction

1.1. Binaural beats

When two sinusoids with slightly different frequencies are superimposed, their interference results in periodic amplitude fluctuations whose frequency corresponds to the frequency difference between the sinusoids. When sound with slightly different frequencies is mixed in the same ear, the fluctuations in the intensity of the sound are perceived as beats, reflecting the acoustic properties of the stimulus and are thus called acoustic beats. When a tone of one frequency and steady intensity is presented to one ear and a slightly different frequency is presented to the other ear, a perception of beats is experienced, with a beat frequency corresponding to the frequency difference between the two ears. These beats do not reflect a physical property of the sound, but probably the convergence of neural activity from the two ears in the central binaural auditory pathways of the brain. The intensity changes in the perceived sound are therefore called “binaural beats”. Binaural beats are present when the disparate frequencies to the two ears are low, and beats are difficult to hear at frequencies above 1000 Hz (Licklider et al., 1950).

The most widely accepted physiological explanation for binaural beats suggests that discharges of neurons that preserve phase information of the sound in each ear according to the volley principle (Rose et al., 1968; Palmer and Russel, 1986; Goldberg and Brownell, 1973) converge on binaurally-activated neurons in the ascending auditory pathway (Kuwada et al., 1979; McAlpine et al., 1996, 1998; Spitzer and Semple, 1998) that, in turn, generate...
the neurophysiological correlate of binaural beats (Wernick and Starr, 1968) in the brainstem.

Normative human studies on binaural beats showed differences between the subjective rhythm perceived and the difference between the two frequencies, indicating a central effect (Fritze, 1985). Clinical studies of binaural beats in humans varied in their sensitivity to cortical damage. Some reported that binaural beats were not sensitive to cortical damage (Noffsinger, 1982) but others showed that binaural beats could not be perceived by patients with cortical lesions associated with severe aphasia (Barr et al., 1977). A detailed study of a single patient with Auditory Neuropathy reported that a marked impairment of binaural beats was observed in conjunction with impaired auditory perceptions dependent on temporal cues, such as lateralization of binaural clicks, change of binaural masked threshold with changes in signal phase, detection of paired monaural clicks, monaural detection of a silent gap in a sound, and monaural threshold elevation for short duration tones (Starr et al., 1991). Effects of binaural beats at the Beta EEG frequency on psychomotor performance, mood and arousal (Lane et al., 1998) have also been reported.

Thus, animal experiments indicate the auditory brainstem as the level of binaural beat formation, human studies agree that binaural beats are a central effect which is sensitive to temporal acoustic cues and which may interact with cortical function.

1.2. ERPs to binaural beats

Although the psychoacoustic effect of binaural beats has been well-studied in normals and patients, only very few studies reported ERP correlates of binaural beats. Two studies recorded auditory steady-state responses to a binaural beat frequency of 40 Hz. In the first (Schwarz and Taylor, 2005), the right and left ears were stimulated with tones differing in frequency by 40 Hz, and binaural beat steady-state potentials were recorded. A 40-Hz binaural beat potential was evoked with a low base stimulus frequency (400 Hz) but became undetectable when base frequency was above 3 kHz. In another study (Karino et al., 2006), magnetic fields evoked by beats of 4.00 or 6.66 Hz in base frequencies of 240 and 480 Hz were described. The fields showed small but sufficient amplitudes to be distinguished from the noise in the recordings. Spectral analyses of the magnetic fields revealed that the responses contained a specific spectral component of the beat frequency. Source estimates suggested multiple sources in the left and right auditory cortices and in parietal and frontal cortices. The phase of the beat-evoked waveforms showed great variability, suggesting that the responses did not represent changing interaural phase differences but a cognitive process corresponding to subjective fluctuations of binaural beats. More recently, 40 Hz binaural beats of a 500 Hz base frequency were used to evoke steady-state magnetic fields which were also compared with the transient-evoked N1m (Draganova et al., 2008). The auditory steady-state sources were lateralized to the right hemisphere and were anterior, inferior, and medial compared with onset N1m sources.

Two of the three previous reports on electrophysiological correlates of binaural beats used a beat frequency of 40 Hz. This frequency is optimal for thalamic and primary cortical steady-state activity, but later cortical contributions associated with perception are markedly attenuated at this rate. Moreover, the filter properties of scalp and skull attenuate 40 Hz potentials relative to lower frequencies and this attenuation of a beat frequency of 40 Hz may impede the detection and characterization of the slower brain activity associated with the perception of binaural beats. There is disagreement among previous studies on the sources of the beat-evoked oscillations: One study found bilateral sources in temporal lobe (Karino et al., 2006) while in the other – sources were lateralized to the right (Draganova et al., 2008).

1.3. Purpose of this study

The purpose of this study was to define cortical auditory evoked potentials to binaural beats of 3 or 6 Hz in high- (1000 Hz) and low- (250 Hz) base frequency tones, and to estimate their intracranial sources. Potentials to the onset of the tones were also recorded for comparison.

2. Methods

2.1. Subjects

Eighteen (16 men and 2 women) 18 to 25 years old right handed normal hearing subjects participated in the study. Subjects were paid for their participation and all procedures were approved by the institutional review board for experiments involving human subjects (Helsinki Committee).

2.2. Stimuli

Binaural tone bursts of 2000 ms duration and 70 dBnHL intensity were presented through earphones (Sony MDR-CD770) with a flat frequency response (within 10 dB across the frequency range 100–10,000 Hz). Tones were presented at interstimulus intervals that randomly varied between 950 and 1050 ms. Low frequency tone bursts included synchronous presentation of 250 Hz to the left or right ear and 253 or 256 Hz to the opposite ear, resulting in binaural beats of 3 or 6 Hz, respectively (Fig. 1, top). Similarly, 1000 Hz tone bursts were presented to one ear and 1003 or 1006 Hz to the other, resulting in binaural beats of 3 or 6 Hz, respectively. In all, there were four stimulus conditions: 250 and 1000 Hz base frequencies with 3 and 6 Hz binaural beats, which were randomly presented during the recording session, with equal probability for each stimulus condition (25%) and right (50%) or left (50%) ear receiving the sound with the higher base frequency. Each stimulus condition was randomly presented 400 times.

These stimulus parameters were selected following preliminary experiments in which subjects reported detection of beats in a series of binaural tones some of which had no beats while others had beats of different frequencies and intensities. In line with earlier psychoacoustic reports, perception of binaural beats was found sensitive to base frequency, intensity and beat rate. The stimulus parameters of this study were chosen to optimize psychoacoustic and electrophysiological detection of beats.

2.3. Procedure

Twenty-two 9 mm silver disc electrodes were placed according to the 10–20 system at: Fp1, F7, F3, Fz, F4, F8, Fp2, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, O1, O2, 1.5 cm above the left and right mastoids (M1 and M2), all referenced to the center of the chin, to record the electroencephalogram (EEG). The mastoidal electrodes were placed above their standard positions to avoid distortion in the source estimation procedures. In addition, an electrode below the left eye, referenced to Fz, was used to control for eye movements (EOG). In total, EEG was recorded from 21 electrodes and EOG was recorded from one diagonal differential recording below the left eye referenced to Fz. An electrode over the 7th cervical spinous process served as ground. Impedance across each electrode pair was maintained below 5 kΩ.

Subjects were subsequently seated in a comfortable reclining armchair in a sound-proof chamber and instructed to read a complicated text, on which they were to be examined, while stimuli were presented (subjects were actively distracted from the stimuli). The diverted attention of subjects from the auditory stimuli,
the equal probabilities of the four stimulus conditions, as well as the randomness of beats and their timing were designed to reduce endogenous contributions to the brain potentials. In preliminary tests on four subjects, the beats-evoked potentials recorded while subjects indicated the detection of beats or their absence (attending to sound), were indistinguishable from the respective potentials when subjects were reading and not responding to the beats (actively distracted from sounds). This absence of attention-related effects indicated an exogenous nature of the beats-evoked potentials. Subjects were allowed breaks as needed.

2.4. Data acquisition

Potentials from the EEG (X100,000) and EOG (X20,000) channels were amplified, digitized with a 12 bit A/D converter at a rate of 256 samples/s, filtered (0.1–100 Hz, 6 dB/octave slopes) and stored for off-line analysis. EEG processing began with segmentation of the continuous EEG to epochs beginning 100 ms before until 2300 ms after each tone burst onset. Eye movement correction (Attias et al., 1993) and artifact rejection (+150 μV) followed segmentation. Average waveforms were then computed for the 3 Hz and 6 Hz beats in the 250 Hz and 1000 Hz base frequencies. Consequently, between 300 and 350 repetitions were averaged to obtain the potentials evoked by each stimulus condition. These 4 separate averages (2 beat frequencies × 2 base frequencies) were computed for each subject, as well as across subjects to obtain grand mean waveforms. After averaging, the data were band-pass filtered (FIR rectangular filter with a cutoff at 2–10 Hz) and baseline (average amplitude across the 100 ms before stimulus onset) corrected. This filter was chosen to enhance the detection of the beats-evoked oscillations and was found to have only a minor effect on the amplitudes of the onset-evoked potentials.

2.5. ERP waveform analysis

ERP analysis included peak latency and amplitude comparisons among stimulus types as well as comparisons of the respective source current density estimates. $P_{50}$ was defined as the positive peak in the range of 20–85 ms, preceding $N_{100}$ which was the most negative peak between 60 and 150 ms. $P_{200}$ was defined between 130 and 220 ms and was usually followed by a positive peak between 230 and 370 ms from tone onset which we called $P'_{200}$ (Fig. 1, bottom schematic waveform). Periodic peaks and troughs following $P'_{200}$, corresponding to the 3 and 6 Hz beats, were also defined and measured. Group grand averaged ERPs for each condition determined the latency ranges for peak identification.

ERP peak amplitudes and latencies were subjected to a repeated measures analysis of variance (ANOVA) with Greenhouse–Geisser correction for violation of sphericity and Bonferroni corrections for multiple comparisons. Potentials to binaural beats were analyzed using repeated measures analysis of variance, for the effects of the following factors: Base frequency (250 Hz, 1000 Hz), beat frequency (3 Hz, 6 Hz) and 9 of the Electrodes (F3, Fz, F4, C3, Cz, C4, P3, Pz, P4) representing left, right and midline locations in the frontal, central, and parietal scalp areas. Probabilities below 0.05, after Greenhouse–Geisser corrections for violations of sphericity (when deemed necessary), were considered significant.

2.6. ERP functional imaging

Standardized Low Resolution Electromagnetic Tomographic Analysis (sLORETA, Pascual-Marqui et al., 1994; Pascual-Marqui, 2002) was applied on the 21-channel ERP records to image the estimated source current density throughout the duration of the onset-evoked $P_{50}$–$N_{100}$–$P_{200}$ and the subsequent beats-evoked oscillations, in response to the four stimulus conditions (2 base frequencies × 2 beat frequencies) and to compare the current density distributions among stimulus conditions.

sLORETA is a functional brain imaging method that estimates the distribution of current density in the brain as suggested by the minimum norm solution. Localization inference is based on standardized values of the current density estimates. The solution space is restricted to cortical gray matter and hippocampus. A total of 6430 voxels at 5 mm spatial resolution are registered to the Stereotaxic Atlas of the Human Brain (Talairach and Tournoux, 1988). In this study, differences in current density distributions between the onset response and the beats-evoked oscillations, across stimulus conditions, were assessed using Statistical Non-Parametric Mapping (SnPM). The SnPM method estimates the probability distribution by using a randomization procedure, corrects for multiple comparisons and has the highest possible statistical power (Nichols and Holmes, 2002). The SnPM method in the context of ERP source estimation was validated in our previous studies by comparing its results with more conventional ANOVA results (Lauffer and Pratt, 2003a; Sinai and Pratt, 2003). Specifically, in this study we used the ‘pseudo-t’ statistic which reduced noise in the data.
by averaging over adjacent voxels (Nichols and Holmes, 2002). In order to compare source current distributions between onset-evoked components and beats-evoked components we compared them across 45 ms (11 time frames) from 5 time frames before- until 5 after the peaks of the compared components. Of the repetitive beats-evoked peaks in the recording, the peak used for comparison with the onset components was the one with the source distribution closest to the grand-averaged source distribution of the beats-evoked potentials. Differences were considered significant if the results were significant either using 11 time frames from each comparison or using the average current density values across the 11 time frames.

Average current density values from the most active regions in the brain during the beats-evoked oscillations were analyzed using repeated measures analysis of variance, for the effects of the following factors: stimulus condition (2 base frequencies × 2 beat frequencies), brain region (BA 11, 20, 21, 37 and 38) and hemisphere (left and right). Probabilities below 0.05, after Greenhouse–Geisser corrections for violations of sphericity (when deemed necessary), were considered significant.

2.7. ERP spectral analysis

In addition to time-domain peak measures, the frequency content of the waveform following P200 and preceding the offset response at the end of the waveform (Fig. 1, bottom), with a total duration of about 1400 ms (4 periods of 3 Hz oscillations, 8 periods of 6 Hz oscillations) was determined and the spectral peaks defined and measured. Spectral content was quantified in arbitrary relative units and analyzed similarly to the time-domain waveform peaks.

3. Results

All subjects reported perceiving beats in all four stimulus conditions (3 and 6 Hz beats in 250 and 1000 Hz base frequencies). The beats were described as faint to comfortable and as emanating from the center of the head. At onset, the binaural tones evoked components beginning with a P50, N100, and P200 followed by a positive peak labeled P200′ and a negativity termed N0, followed by repetitive negative/positive oscillations corresponding to the frequency difference between left and right ear. We identify these oscillations at the positive peaks as P1, P2, P3, etc. An offset complex of P50, N100, and P200 occurred at the end of the stimuli (Figs. 2 and 3). Descriptive statistics of the latencies and amplitudes of the peaks evoked by the four stimulus conditions are listed in Table 1. The interpeak latency differences between the oscillatory peaks following the onset response are listed in Table 2. Note that these latency differences correspond to the period of the respective beat frequency: approximately 330 ms for the 3 Hz beats and about 165 ms for the 6 Hz beats. Also note that N0 preceded P1 by approximately half a period of the respective beat frequency and was therefore considered the initial negative peak of the beat oscillations.

The oscillations following the onset response were not the result of interaction of the disparate frequencies of the stimulus artifacts from the left- and right-earphones. This was determined in five subjects by comparing the potentials recorded with normal conditions and with the ears occluded. When ears were occluded sound intensity arriving at the ears was reduced by approximately 30–35 dB, while the electrical signal to the earphones was unaffected. With ears occluded the onset-evoked potentials as well as the beats-evoked oscillations were reduced in amplitude in contrast to artifactual beats that would not have been affected by the occlusion of ears.

3.1. Evoked potentials to sound onset

Tone onset-evoked components were larger and earlier to 1000 Hz than to 250 Hz base frequency and were occasionally marginally affected by beat frequency, as detailed below.

3.1.1. Waveform analysis

P50, N100, P200 and P200′ latencies to tone onset were significantly affected by the tones’ base frequency [F(1, 16) = 104.62; p < 0.001 for P50, F(1, 16) = 135.81; p < 0.001 for N100, F(1, 16) = 287.32; p < 0.001 for P200 and F(1, 16) = 11.14, p < 0.001 for P200′] with consistently longer latencies (by 10–20 ms, on average) with the 250 Hz base frequency. The effects of beat frequency on the latency of these components was marginal (4–8 ms – 1 or 2 dwell-times) albeit, at times statistically significant. P50, N100, P200 and P200′ amplitudes were significantly affected by base frequency [F(1, 16) = 7.82, p < 0.006 for P50, F(1, 16) = 109.04, p < 0.001 for N100, F(1, 16) = 30.64, p < 0.001 for P200 and F(1, 16) = 6.97, p < 0.009 for P200′], with larger amplitudes to 250 Hz than to
1000 Hz base frequency. P50, N100, P200 amplitudes were also affected by beat frequency but in an inconsistent manner \( F(1, 16) = 8.67, p < 0.004 \) for P50 (3 Hz > 6 Hz), \( F(1, 16) = 5.82, p < 0.02 \) for N100 (6 Hz > 3 Hz) and \( F(1, 16) = 17.40 p < 0.001 \) for P200 (6 Hz > 3 Hz). The amplitude of P200 was unaffected by beat frequency. Significant electrode effects indicated largest P50 amplitudes frontally and smallest posteriorly \( F(8, 128) = 40.47, p < 0.001 \), lower N100 amplitudes at central scalp locations \( F(8, 128) = 15.06, p < 0.001 \) and larger P200 amplitudes at central and midline scalp locations \( F(8, 128) = 39.62, p < 0.001 \). P200 amplitude was not significantly affected by electrode location.

3.1.2. Source current density estimates

Source current density estimates for P50 were mostly localized to left lateral posterior and right anterior-temporal lobe areas, with parietal contributions as well, while N100 and P200 sources were estimated to bilateral inferior and lateral-posterior temporal lobe areas (BA 20, 21, 37), with somewhat lower current densities in the right hemisphere (Fig. 4). P200 activity was estimated to originate in bilateral parietal regions and right temporal lobe.

3.2. Evoked potentials to binaural beats

Binaural beats were associated with oscillatory brain activity at the beat frequency that was larger to the low- than to the high-base frequency. For both high- and low-base frequencies 3 Hz beats evoked larger oscillations than 6 Hz beats. The oscillations did not have a clear scalp prominence and their sources were located to the left temporal lobe with a minor frontal contribution across all stimulus conditions, as detailed below.

3.2.1. Waveform analysis

P1, P2, P3, and P4 were the initial four positive peaks of the beat oscillations. Their latencies were not affected by base frequency nor by electrodes. Their amplitudes were all affected by beat frequency \( F(1, 16) = 182.83, p < 0.0001 \) for P1, \( F(1, 16) = 36.89, p < 0.001 \) for P2, \( F(1, 16) = 38.21, p < 0.001 \) for P3 and \( F(1, 16) = 12.00, p < 0.001 \) for P4] with larger amplitudes to 3 Hz beats than to 6 Hz beats. P2 and P3 amplitudes were also affected by significant base frequency effects \( F(1, 16) = 10.32, p < 0.002 \) for P2 and \( F(1, 16) = 29.00 p < 0.001 \) for P3 with larger amplitudes with a base frequency of 250 Hz. The effects of base frequency and beat fre-

### Table 1

| Beats (Hz) | Base (Hz) | P50 | N100 | P100 | P200 | N0 | P1 | P2 | P3 | P4 |
|------------|-----------|-----|------|------|------|----|----|----|----|----|
| Latency (ms) |           |     |      |      |      |    |    |    |    |    |
| Avg 3       | 250       | 45  | 103  | 176  | 344  | 465| 693| 1034| 1364| 1680|
| SD 3        | 250       | 14.4| 19.5 | 19.4 | 63.7 | 73.1| 80.5| 106.1| 147.8| 196.77|
| Avg 3       | 1000      | 39  | 95   | 166  | 324  | 426| 678| 1022| 1354| 1654|
| SD 3        | 1000      | 10.3| 12.2 | 17.4 | 49.7 | 50.8| 62.5| 101.3| 144.4| 187.4|
| Avg 6       | 250       | 45  | 107  | 188  | 287  | 362| 451| 617 | 786 | 954|
| SD 6        | 250       | 16.4| 20.8 | 21.2 | 58.0 | 74.4| 72.0| 75   | 84.2 | 75.6|
| Avg 6       | 1000      | 37  | 95   | 170  | 287  | 368| 473| 641 | 799 | 964|
| SD 6        | 1000      | 10.4| 13.5 | 17.0 | 31.6 | 45.0| 70.7| 85   | 86.4 | 83.7|
| Amplitude (μV) |        |     |      |      |      |    |    |    |    |    |
| Avg 3       | 250       | 1.09| 1.84 | 2.72 | 0.60 | 1.19| 0.88| 0.83 | 0.88 | 0.73|
| SD 3        | 250       | 0.88| 1.39 | 1.46 | 0.71 | 0.66| 0.66| 0.43 | 0.31 | 0.31|
| Avg 3       | 1000      | 1.16| 2.63 | 3.64 | 0.76 | 1.25| 0.89| 0.85 | 0.66 | 0.80|
| SD 3        | 1000      | 0.76| 1.66 | 1.38 | 0.85 | 0.50| 0.32| 0.32 | 0.26 | 0.36|
| Avg 6       | 250       | 0.86| 2.06 | 3.47 | 0.77 | 1.36| 0.43| 0.76 | 0.71 | 0.73|
| SD 6        | 250       | 0.71| 1.64 | 1.59 | 1.18 | 0.90| 0.72| 0.41 | 0.51 | 0.30|
| Avg 6       | 1000      | 0.93| 2.84 | 3.83 | 0.71 | 1.31| 0.30| 0.63 | 0.59 | 0.75|
| SD 6        | 1000      | 0.75| 1.57 | 1.45 | 1.13 | 0.71| 0.54| 0.37 | 0.39 | 0.42|
freqency interacted in their effects on P3 \([1, 16] = 4.03, p < 0.05\) with the larger amplitudes to 3 Hz beats more evident at the 1000 Hz base frequency. P2, P3, and P4 amplitudes were also affected by a significant electrode effect \([F(8, 128)] = 5.58, p < 0.001\) for P2, \([F(8, 128)] = 3.89, p < 0.001\) for P3, and \([F(8, 128)] = 6.17, p < 0.001\) for P4 with no obvious pattern except a tendency toward lower P4 amplitudes in posterior scalp electrodes.

### 3.2.2. Source current density estimates

Source current density distribution of the oscillatory beat potentials were practically identical when estimated from scalp-recorded waveform peaks (e.g., peak P3, Fig. 5, bottom) or from spectral peaks at the beat frequency (e.g., 3 Hz peak, Fig. 5, top). In both cases activity peaked in the left lateral and inferior temporal lobe (in the general locations of BA 20, 21 and 38), in the temporo-parietal area (around BA 37) and to a lesser degree (pink areas in Fig. 5) frontally (approximately BA 11).

Analysis of variance indicated a significant effect of stimulus condition on source current density \([F(3,51)] = 4.13, p < 0.007\) with higher values associated with low base frequency and low beat frequency. Brain region was also found to significantly \([F(4,68)] = 18.57, p < 0.001\) affect source current density with lower values around BA 11 than in the vicinities of BA 20, 21, 37 and 38. Source activation was significantly \([F(1,17)] = 37.97, p < 0.001\) affected by hemisphere and was larger in the left hemisphere regions than in their right hemisphere counterparts. A significant brain region × hemisphere interaction \([F(4,68)] = 3.55, p < 0.008\) showed more pronounced differences among regions in the left hemisphere.

When analysis of variance procedures were applied to each brain region separately, for the effects of base frequency, beat frequency and hemisphere, significant effects of hemisphere were found on BA 20 \([F(1,17)] = 11.09, p < 0.002\), BA 21 \([F(1,17)] = 13.76, p < 0.001\), and BA 37 \([F(1,17)] = 10.26, p < 0.002\), showing more activation in all the left hemisphere areas. BA 11 was not lateralized but showed a significant \([F(1,17)] = 17.21, p < 0.001\) effect of beat frequency, with more activation with 3 Hz beats.

**Table 2**

| Beats | Base | P1–N0 | P2–P1 | P3–P2 | P4–P3 |
|-------|------|-------|-------|-------|-------|
| Avg   | 3    | 250   | 338.29| 333.73| 314.63| 341.54|
| SD    | 3    | 250   | 65.15 | 63.42 | 72.93 | 77.02 |
| Avg   | 3    | 1000  | 341.11| 335.90| 298.14| 346.31|
| SD    | 3    | 1000  | 68.46 | 66.55 | 76.89 | 92.41 |
| Avg   | 6    | 250   | 89.14 | 166.11| 169.10| 167.72|
| SD    | 6    | 250   | 24.67 | 21.93 | 23.10 | 31.11 |
| Avg   | 6    | 1000  | 104.31| 168.18| 157.84| 165.88|
| SD    | 6    | 1000  | 341.11| 335.90| 298.14| 346.31|

**3.3. Comparisons between onset- and binaural beat potentials**

Components to stimulus onset were, in general, unaffected by changes in base frequency and beat frequency, whereas beat oscillations were markedly affected by stimulus conditions. In addition, although beat oscillations and the onset response \(N_{100}\) and \(P_{200}\) involved similar cortical areas, their hemispheric laterality and other source distribution differences were noted, as detailed below.

**3.3.1. Waveform analysis**

\(P_{50}, N_{100}\) and \(P_{200}\) to tone onset were similar to previously reported onset-evoked potentials. As detailed above, the onset-evoked potentials latencies were somewhat affected by base frequency, reflecting the tone onset envelope, and their amplitudes were practically unaffected across stimulus conditions. In contrast, the beat oscillations were markedly affected by base frequency and by beat frequency.

**3.3.2. Source current density estimates**

Onset-evoked \(P_{50}\) had superior parietal source activation that was absent in the beat oscillations and the temporal distribution of its sources (left lateral posterior and right anterior) was also different than that of the beat oscillations in the left lateral and inferior temporal lobe and the temporo-parietal area.

Statistical non-parametric t-value mapping of current density differences between onset-evoked \(N_{100}\) and beat oscillations of 3 Hz with a base frequency of 250 Hz found statistically significant differences during the 45 ms around the respective peaks, with temporo-parietal area (vicinity of BA 40) significantly more active in \(N_{100}\) than in beats-evoked peaks. A similar comparison of \(P_{200}\) indicated frontal areas (the vicinity of BA 11) significantly more active in \(P_{200}\) than in the beats-evoked oscillations. When these comparisons were conducted with beat oscillations of 6 Hz and base frequency of 250 Hz, \(N_{100}\) was found to be associated with significantly more frontal activity than the beats-evoked oscillations.

**3.4. Summary of results**

Overall, all stimulus conditions (3- and 6 Hz beats with 250- and 1000-Hz base frequencies) evoked tone onset and offset components \(P_{50}, N_{100}\) and \(P_{200}\), straddling oscillations corresponding to the beat frequency. Beats-evoked oscillations were higher in amplitude with the 250 Hz compared to the 1000 Hz base frequency and to 3 compared to 6 Hz beat frequency. Sources of the beats-evoked oscillations were located mostly in the vicinity of the left lateral and inferior temporal lobe in all stimulus conditions. Onset components were not markedly different across stimulus conditions. \(P_{50}\) had significantly different sources than the beat-evoked oscillations, with more parietal activation; \(N_{100}\) and \(P_{200}\) sources located to the same temporal lobe regions as the oscillations, but with a more bilateral distribution and significantly larger activation of frontal and temporo-parietal areas.

**4. Discussion**

### 4.1. The results of this study

In this study brain potentials associated with the auditory illusion of binaural beats were recorded and compared across two beat frequencies with tone bursts of two base frequencies. Beat-evoked oscillations were preceded by tone-onset components \(P_{50}, N_{100}\) and \(P_{200}\), and were followed by tone-offset components. Sources of the beats-evoked oscillations across all stimulus conditions were mostly in the left lateral and inferior temporal lobe. This lateralized temporal activity is in line with other studies demon-
The beats-evoked oscillations were not generated by interference of stimulus artifacts from the right- and left-earphones: When ears were occluded in five of the subjects, the onset-evoked potentials as well as the beats-evoked oscillations were significantly reduced, confirming that they depended on acoustic input to the ears as opposed to an artifact of interference of electrical signals from the right- and left-earphones. Moreover, the source estimation results indicated temporal activity which was consistently lateralized to the left, as opposed to a symmetrical and close to midline stimulus artifact from bilateral earphones.
Binaural beats are the perception of rapid acoustic changes in perceived intensity without such changes in the physical intensity or spectral content of the sound. The lateralization of beat-evoked oscillations to the left temporal lobe are therefore in line with behavioral, positron emission tomography evidence and clinical findings on specialization of the left-hemisphere auditory cortex.

Fig. 5. Average source strength estimates associated with the spectral peak at 3 Hz (top) and with the 3 Hz beats-evoked oscillation peak P3 with 250 Hz base frequency (bottom). For the spectral peak, strength is given in arbitrary relative units, while for peak P3, strength is given in current density. Six views of the brain are presented for each display, as indicated on each view.
for rapid acoustic changes and temporal processing (Johnsrude et al., 1997; Zatorre and Belin, 2001), distinct from the spectral processing specialization of the right temporal lobe (Zatorre, 1988; Zatorre and Belin, 2001). Moreover, the localization of activity to left hemisphere is compatible with the report that binaural beats could not be perceived by patients with cortical lesions associated with severe aphasia (Barr et al., 1977).

The electrophysiological correlates of binaural beats in this study are in line with suggestions that neural activity with slightly different volley frequencies from left and right ear converge and interfere in the central auditory pathway to generate beats of neural activity, giving rise to the illusion of amplitude modulation (Moore, 1997). The temporal pattern of firing of some auditory units (particularly those encoding low frequencies) follows the stimulus waveform in a ‘phase locked’ manner up to frequencies of about 4–5 kHz in auditory nerve (Rose et al., 1968; Palmer and Russel, 1986) and anteroventral cochlear nucleus (Goldberg and Brownell, 1973) of experimental animals. In addition, binaurally-activated phase-sensitive neurons are found in the Superior Olivary Complex and Inferior Colliculus (Kuwada et al., 1979; McAlpine et al., 1996, 1998; Spitzer and Semple, 1998) as well as in auditory cortex (Reale and Brugge, 1990). In humans, measures of the frequency following response that reproduce low frequency tones (Moushegian et al., 1973) as well as psychoacoustic studies (Javel and Mott, 1988) suggest a sharp decline in phase locking in the ascending auditory pathway, beginning at about 1 kHz. This decline mirrors the decline in perception of binaural beats above 1000 Hz.

Binaural beats were demonstrated in field potentials of the superior olive (Wernick and Starr, 1968) sensitive to the phase and amplitude differences of the monaural stimuli. The sum of monaurally generated field potentials were suppressed during binaural stimulation at a rate consistent with the frequency differences between the ears. Estimating the source current density of the scalp-recorded potentials to the beats-evoked oscillations revealed activity that peaked in a number of brain structures approximately at the time of scalp-recorded peaks and troughs (Fig. 6). This correspondence indicates that both surface recorded peaks and troughs in the beats-evoked oscillations are associated with increased intracranial activity.

In general, measures of scalp-recorded activity and of intracranial estimated source activity need not necessarily correspond: fields from different intracranial sources summate differently in different scalp locations, depending on distance from each source and the orientation of each source relative to the electrode. The uniform latencies, across electrodes, of the scalp-recorded beats-evoked oscillations and their temporal correspondence to the estimated source activity therefore indicate that they reflect a single major intracranial source. Indeed, observing Figs. 5 and 6, the intracranial sources are mostly in the vicinity of the left lateral and inferior temporal lobe.

4.2. Comparisons with previous binaural beats studies

The involvement of left temporal and temporo-parietal areas in binaural beats is congruent with earlier observations that binaural beats could not be perceived by patients with cortical lesions associated with severe aphasia (Barr et al., 1977).

In two recent studies brain responses to binaural beats were recorded using the steady-state technique with a beat frequency of 40 Hz. In the first study (Schwarz and Taylor, 2005), a 40 Hz binaural
beat potential was recorded when the EEG was band passed accord-
ingly and averaged relative to stimulus onset. It was recorded with a
low base frequency of 400 Hz but became undetectable beyond
3 kHz, its amplitude was maximal at fronto-central leads and it di-
played a fronto-occipital phase delay of several ms. The phase shift
across electrodes suggested more than one origin of the 40 Hz oscil-
lations. In our study, no consistent scalp prominence or significant
effect of electrode on latency was observed for the beats-evoked oscil-
lations except for a tendency toward lower Pa amplitudes in posterior
scalp electrodes. Consistent with the previous study, amplitudes were higher with low (250 Hz) than with the higher
(1000 Hz) base frequency. Thus, the previously reported 40 Hz bin-
aural beats-evoked potential oscillations were similar to our results
in terms of the effects of base frequency, similar to some extent in
their tendency for lower amplitudes posteriorly, but different from
our results in terms of latency differences across the scalp and the
distribution of intracranial sources.

More recently, 40 Hz binaural beats of a 500 Hz base frequency
were used to evoke steady-state magnetic fields which were also
compared with the transient-evoked N1m (Draganova et al., 2008).
These components were modeled with a single spatiotemporal
equivalent current dipoles in each hemisphere, with steady-state
sources lateralized to the right hemisphere and more anterior, more
inferior, and more medial compared with the N1m sources. In our
study, beats-evoked oscillations across all stimulus conditions were
lateralized to the left, suggesting involvement of different brain
processes in the two studies. On the other hand, in our study frontal
(−BA 11) and anterior-temporal (−BA 37) activity, in addition to
lateral–inferior temporal regions, was estimated to contribute to the
difference between the onset- and beats-evoked N100 potential.
In the previous study 40 Hz beats-evoked fields were more anterior
than onset N1m, perhaps because only a single-dipole source model
was used, collapsing the additional, more anterior, beats-evoked
sources into the single dipole, moving it forward.

The two earlier studies on 40 Hz binaural beats differed in the
distribution of their responses. The 40 Hz potentials of the first
study (Schwarz and Taylor, 2005) had maxima at fronto-central
leads and displayed a fronto-occipital phase delay of several ms.
This distribution is similar to onset responses, and the phase shift
across the electrode array was interpreted to suggest more than
one origin of the oscillations (Schwarz and Taylor, 2005). The sec-
ond study (Draganova et al., 2008) reported magnetic field sources
that were lateralized to the right hemisphere and were more ante-
rior, more inferior, and more medial compared with the onset-evoked
N1m. These differences between the earlier studies in distribution
and sources of their beats-evoked oscillations may be reconciled by
differences in the fields recorded: electric potentials (Schwarz
and Taylor, 2005) are affected by the filter properties of tissues,
while magnetic fields (Draganova et al., 2008) are not. Thus the rel-
ative contributions of different intracranial sources to the scalp-
recorded responses were different between these earlier studies.

Another earlier study of magnetic field correlates of low fre-
quency binaural beats (Karino et al., 2006) indicated bilateral tem-
poral sources for the fields, while the present study found the
temporal sources lateralized to the left. Moreover, that previous
study found that the phase of the beat-evoked oscillations showed
great variability, suggesting that the responses were affected by
subjective fluctuations. In contrast, in the present study clear aver-
aged oscillations could be recorded, suggesting a stable phase
throughout the recording. In the earlier study, symmetrical con-
tributions of thalamo-cortical magnetic fields were likely more
prominent than in this study in which their higher frequency elec-
tric waveforms may have been attenuated by the filter properties
of tissues. Moreover, in the previous study subjects watched a si-
 lent video during recording, and their level of vigilance may have
varied during the recording, resulting in fluctuating contributions
of the late cortical potentials and a relative augmentation of thal-
amo-cortical components. In the present study vigilance was main-
tained by subjects reading a challenging text on which they were
tested, and the potentials were therefore predominantly late corti-
cal oscillations with stable vigilance throughout the recording,
resulting in constant phase of the response.

Two of the previous studies on ERP correlates of binaural beats
used a beat frequency of 40 Hz which is optimal for recording steady-
state potentials (Galambos et al., 1981; Kileny and Shea, 1986)
from primary auditory cortex and thalamus (Hashimoto, 1982; Lee
et al., 1984; McGee et al., 1992; Zaaroor et al., 2003). In contrast,
the beat frequencies of 3 and 6 Hz used in the present study and the
4 and 6.66 Hz used in another study (Karino et al., 2006) aug-
ment the contribution of slower cortical activity from secondary
and non-specific cortex, overlapping with the smaller middle laten-
ty activity from specific auditory cortex. However, the contribu-
tion of cortical activity may have been diminished by the unstable
phase of the low frequency oscillations in the previous study (Kar-
ino et al., 2006). These beat-frequency and phase stability differ-
ences may explain the differences in sources across studies. The
earlier studies and this study thus complement each other, show-
ing that binaural beats, most probably arising in the brainstem,
persist through auditory processing at the thalamic and specific
auditory cortex (previous studies) and further involve secondary
and non-specific auditory areas (this study).

In summary, the results of this study are in general agreement
with previous studies that recorded brain potentials to binaural
beats. The differences in findings are attributable to the beat fre-
quency, and hence the cortical structures involved, and to the mod-
els used for source estimation.

4.3. Comparison of potentials to stimulus onset with beats-evoked
oscillations

Onset- and beats-evoked potentials were clearly different, both
in terms of the effects of stimulus conditions on them and in their
estimated intracranial sources. Whereas onset-evoked components
were barely and inconsistently affected across the stimulus condi-
tions of this study, beats-evoked oscillations were appreciably
smaller to the higher frequency beats and with the higher base fre-
quency. Of the onset-evoked components, only the sources of N100
and P200 located to similar regions as the beats-evoked oscillations.
But whereas the beats-evoked oscillations were associated with a
left-lateralized temporal activity, the sources of N1m to tone onset
had a more bilateral temporal distribution and some frontal and
tempo-parietal contributions.

Overall these differences and similarities show that the beats-
evoked oscillations involve the same temporal lobe structures acti-
vated during the onset-evoked vertex potential, but with a left
hemisphere prominence. The difference in lateralizations mirrors
the different processes involved in beats-evoked and in the on-
set-evoked N100. These findings are in line with previous evidence
that the time period of N100 involves a variety of distinct processes
that have all been estimated to arise in auditory cortex (Vaughan
and Ritter, 1970; Scherg and von Cramon, 1985, 1986; Scherg
and Picton, 1991; Ponton et al., 2002; Laufer and Pratt, 2003b,
2005) as well as lateral temporal lobe with some activity possibly
originating in the anterior cingulate cortex or supplementary
motor areas of the prefrontal cortex (Picton et al., 1999).

In general, the temporal lobe and frontal N100 has been sug-
gested to reflect the detection of acoustic change in the environ-
ment (Hyde, 1997). Change can be effected by the transient onset
and offset responses (Michelewski et al., 2005; Pratt et al., 2005,
2007); by the M-process in response to deviation (‘Mismatch’)
from the preceding sequence of auditory events (Jones et al.,
1998; Vaz Pato and Jones, 1999); the C-process in response to
acoustic ‘Change’ within an ongoing stream (Martin and Boothroyd, 2000; Jones, 2003; Jones and Perez, 2001); and the F-process associated with ‘Fusion’ of acoustic elements to form a new auditory object (Laufer and Pratt, 2003b, 2005). The evoked activity to binaural beats is yet another type of change processing taking place at this latency in the same general areas. However, unlike the other constituents, this activity is associated with an auditory illusion and not a physical acoustic change.

5. Conclusions

The results of this study confirm that brain activity corresponding to an auditory illusion can be recorded from the scalp. The oscillations associated with binaural beats were larger to low- than to high frequency sound, congruent with the involvement in this illusion of ‘volley principle’ neural encoding. The results are in line with the suggested mechanism of binaural beats: neural volleyes with slightly different frequencies from left and right ear converge and interfere in the central auditory pathway to generate beats of neural activity. The beats of neural activity recorded in this study originated in the same temporal lobe regions as onset responses, but were lateralized to the left hemisphere. Thus, potentials recorded to binaural beats involve the same brain structures, but are distinct from onset responses.

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