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Research paper

A comparison of auditory evoked potentials to acoustic beats and to binaural beats

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ABSTRACT

The purpose of this study was to compare cortical brain responses evoked by amplitude modulated acoustic beats of 3 and 6 Hz in tones of 250 and 1000 Hz with those evoked by their binaural beats counterparts in unmodulated tones to indicate whether the cortical processes involved differ.

Event-related potentials (ERPs) were recorded to 3- and 6-Hz acoustic and binaural beats in 2000 ms duration 250 and 1000 Hz tones presented with approximately 1 s intervals. Latency, amplitude and source current density estimates of ERP components to beats-evoked oscillations were determined and compared across beat types, beat frequencies and base (carrier) frequencies.

All stimuli evoked tone-onset components followed by oscillations corresponding to the beat frequency, and a subsequent tone-offset complex. Beats-evoked oscillations were higher in amplitude in response to acoustic than to binaural beats, to 250 than to 1000 Hz base frequency and to 3 Hz than to 6 Hz beat frequency. Sources of the beats-evoked oscillations across all stimulus conditions located mostly to left temporal lobe areas. Differences between estimated sources of potentials to acoustic and binaural beats were not significant.

The perceptions of binaural beats involve cortical activity that is not different than acoustic beats in distribution and in the effects of beat- and base frequency, indicating similar cortical processing.

1. Introduction

1.1. Acoustic and binaural beats

When two sinusoids of steady amplitude but with slightly different frequencies are summated, their interference results in periodic amplitude fluctuations whose frequency corresponds to the frequency difference between the sinusoids. When such a mix of tones is presented to the same ear, the percept is of amplitude modulation "beats" occurring at a rate corresponding to the frequency difference between the two tones. The perceived beats correspond to the periodicity of amplitude modulation of the acoustic stimulus and are therefore called "acoustic beats". When a tone of one frequency and steady intensity is presented to one ear and a similar tone of slightly different frequency is presented to the other ear, although sound intensity to either ear is steady, a perception of amplitude modulated "beats" that are localized to the middle of the head is experienced. The perceived beat frequency corresponds to the frequency difference between the tones presented to each ear and these beats are known as "binaural beats". Binaural beats reflect the convergence of neural activity from the two ears in central binaurally sensitive neurons within the auditory pathways of the brain (Moore, 1997). Binaural beats are present when the disparate frequencies to the two ears are low, and the stimulating tones are less than 1000 Hz (Licklider et al., 1950). In contrast, acoustic beats can be appreciated to high rates of acoustic beating and also across the audible frequency range.

Both animal experiments (Wernick and Starr, 1968; Kuwada et al., 1979; McAlpine et al., 1996, 1998; Spitzer and Semple, 1998; Reale and Brugge, 1990) and psychoacoustic and clinical observations in humans (Barr et al., 1977; Starr et al., 1991; Lane et al., 1998) indicate that acoustic and binaural beats involve the auditory brainstem and cortex. It is not clear, however, whether acoustic beats and binaural beats are processed differently and whether the involved cortical brain structures are different, and this study aimed at these questions.

1.2. ERPs to acoustic and to binaural beats

Acoustic beats, i.e., amplitude modulated continuous tones, are one way of evoking auditory steady-state evoked potentials (ASSRs). ASSRs can be recorded from the human scalp in response to stimulus rates between 1 and 200 Hz (Dolphin, 1997; Picton

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et al., 2003) and in awake subjects the responses are most prominent at rates near 40 Hz (Galambos et al., 1981). Potentials evoked by stimuli at higher rates are less affected by changes in arousal and can be evoked by multiple simultaneous stimuli without significant loss of amplitude. ASSRs are generated at the brainstem and cortical levels of the auditory pathway, with bilateral cortical contributions more prominent with lower modulation frequencies (Herdman et al., 2002). ASSRs are widely used for objective determination of auditory thresholds (Picton et al., 2005) as well as for monitoring depth of anesthesia (Plourde, 2006).

In contrast to the widely used and studied ASSRs, only very few studies reported ERP correlates of binaural beats. In the first (Schwarz and Taylor, 2005), a 40 Hz binaural beat potential was evoked with a low base (carrier) 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 showed small amplitudes which were sufficient to be distinguished from the noise in the recordings using spectral analyses of the magnetic fields. Source estimates suggested multiple sources in the left and right auditory cortices and in parietal and frontal cortices. More recently, 40 Hz binaural beats of a 500 Hz base frequency were used to evoke steady-state magnetic fields (Draganova et al., 2008). The auditory steady-state sources were lateralized to the right hemisphere. Recently, we recorded brain potentials in response to binaural beats of 3 and 6 Hz, in base frequencies of 250 and 1000 Hz (Pratt et al., 2009). 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. Thus, there is disagreement among previous studies on the sources of binaural beats-evoked oscillations, and therefore on their comparison with acoustic beats: one study found bilateral sources in temporal lobe (Karino et al., 2006), in the other – sources were lateralized to the right (Draganova et al., 2008) while in our recent study activity peaked in the left temporal lobe.

Two of the four previous reports on electrophysiological correlates of binaural beats and most ASSR work used a beat frequency of 40 Hz or higher. Such frequencies are optimal for thalamic and primary cortical steady-state activity, but later cortical contributions that are more likely to be associated with perception are markedly attenuated at these frequencies. 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 beats. The earlier studies complement each other and together show that binaural beats, most probably arising in the brainstem, persist through auditory processing at the thalamic and specific auditory cortex (previous studies using 40 Hz beats) and further involve secondary and non-specific auditory areas (studies that used slower beat frequencies). However, methodological differences in stimuli (beat frequency) and recording modality (electric or magnetic) among previous studies prevented comparison of brain activity of binaural and acoustic beats. In this study, we used the same subjects and parameters to compare acoustic and binaural beats to allow comparison of binaural beats with their better studied acoustic counterparts.

1.3. Purpose of this study

The purpose of this study was to determine whether cortical activations differed between acoustic beats and binaural beats with beat frequency, base frequency and distribution. To this end auditory evoked potentials and their estimated intracranial sources to binaural- and to acoustic beats of 3 or 6 Hz in high- (1000 Hz) and low (250 Hz) base frequency tones were compared.

2. Methods

2.1. Subjects

Eighteen (16 men and 2 women) right handed subjects, 18–25 years old with normal hearing participated in the study. Eleven of the subjects (all men) took part in an additional experiment comparing brain potentials to acoustic beats that differed in phase by a quarter of the beat period. 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, beginning and ending at baseline, with an intensity of 70 dBnHL 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. Tone bursts had a base frequency of 250 or 1000 Hz and beats occurred at frequencies of 3 or 6 Hz. In all there were 8 stimulus conditions: 2 Base Frequencies (250 or 1000 Hz) $\times$ 2 Beat Frequencies (3 or 6 Hz) $\times$ 2 Beat Types (acoustic or binaural).

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 binaural beats and acoustic beats were matched to these parameters.

2.2.1. Acoustic beats

Acoustic beats were generated by mixing a 250 Hz sinusoid with a 253 Hz or 256 Hz sinusoid, resulting in a composite sinusoidal signal with amplitude modulations of 3 or 6 Hz, respectively. Transducing this modulated electrical signal in both earphones, resulted in acoustic beats with an abrupt-onset envelope (Fig. 1, middle) in a 250 Hz tone burst. Similarly, 1000 Hz sinusoids were mixed with 1003 or 1006 Hz sinusoids, resulting in acoustic beats of 3 or 6 Hz, respectively. In all, there were 4 acoustic beats: 250 and 1000 Hz base frequencies with 3 and 6 Hz beats, each of which was randomly presented 400 times during the recording session with equal probability (25%).

2.2.2. Binaural beats

Binaural beats stimuli, as well as the recording and analysis procedures for the potentials evoked by them, were identical to those reported in our previous report (Pratt et al., 2009). Low frequency binaural beats were evoked by synchronous presentation of 250 Hz to one ear (left or right, randomly) and 253 or 256 Hz to the opposite ear, resulting in binaural beats of 3 or 6 Hz, respectively (Fig. 1, bottom). 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 4 binaural beats 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 fre-

Fig. 1. Time courses of the stimuli that evoked binaural beats (bottom) and acoustic beats in tones with modulations that had abrupt onset (middle) or gradual onset beginning at baseline (top). Stimuli for binaural beats consisted of tone bursts with frequencies of 250 or 1000 Hz presented to one ear, and frequencies 3 or 6 Hz higher to the other, resulting in the perception of 3 or 6 Hz modulations ('beats') of the tones. Stimuli for the acoustic beats were the same to either ear and consisted of amplitude modulated tones of 250 or 1000 Hz with modulation frequencies of 3 or 6 Hz.

quency. Each stimulus condition was randomly presented 400 times.

2.2.3. Comparing acoustic and binaural beats

The envelopes of stimuli used to evoke acoustic and binaural beats began with an abrupt onset (rectangular window), which evoked clear and synchronous onset responses, followed by beats-evoked potentials. Whereas the onset-evoked potentials to the acoustic beats stimuli and to the binaural beats stimuli had similar latencies, the respective beats-evoked oscillations appeared to be a quarter of a period apart (Fig. 2). This initial comparison might suggest that the oscillations evoked by acoustic beats were repetitive onset responses evoked by the amplitude modulation of the sound. In contrast, with binaural beats there is no acoustic modulation and therefore their mechanisms likely differ. The different phases of acoustic and binaural beats-evoked oscillations would lend further support to such a difference between beats. Therefore, in 11 of the subjects we modified the acoustic beats by uncoupling the onset response from the beat-evoked oscillations and adjusting the phase of the oscillations. In these subjects in addition to the abrupt-onset stimulus, acoustic modulation began at baseline, providing a gradual stimulus onset and beat delay by a quarter of a period compared to the abrupt-onset stimulus (Fig. 1, top compared to middle).

2.3. Procedure

Twenty-two 9 mm silver disc electrodes were placed according to the 10–20 system at: F_{p1}, F_7, F_3, F_z, F_4, F_8, F_{p2}, T_3, C_3, C_z, C_4, T_4, T_5, P_3, P_z, P_4, T_6, O_1, O_2, 1.5 cm above the left and right mastoids (M'_1 and M'_2), 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 F_z, 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. An electrode over the 7th cervical spinous process served as ground. Impedance across each electrode pair was maintained below 5 kΩ.

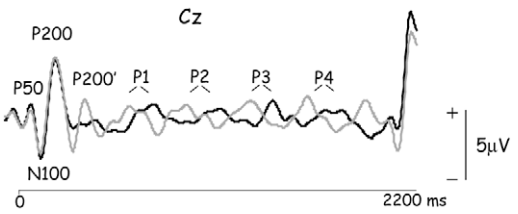

Fig. 2. ERP components evoked by the acoustic (abrupt onset) and by the binaural beat stimuli with a 250 Hz base frequency and 3 Hz beats. Following the onset response P_{50}, N_{100}, P_{200} and P'_{200}, components included periodic peaks (P_1, P_2, P_3, etc.), corresponding to the beat frequency, followed by the initial components of a tone offset response at the very end of the trace. Grand-averaged waveforms across 18 subjects.

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 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 4 subjects, the beats-evoked oscillations recorded while subjects indicated the detection of beats or their absence (attending to sound), were indistinguishable from the potentials when the same subjects were reading and not responding to the beats (actively distracted from sounds). Subjects were allowed breaks as needed and the total duration of recording was 3–4 h during which subjects remained vigilant by reading a text they were subsequently tested on.

2.4. Data acquisition

Potentials were amplified from the EEG (100,000×) and EOG (20,000×) channels, 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 and 6 Hz beats in the 250 and 1000 Hz base frequencies of each beat type (binaural and acoustic beats). Consequently, between 300 and 350 repetitions were averaged to obtain the potentials evoked by each stimulus condition. These 8 separate averages (2 Beat Frequencies × 2 Base Frequencies × 2 Beat Types) 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. This minor effect was common to all onset responses and hence did not affect the comparisons across conditions that were conducted in the waveform analyses.

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. Following the onset response P_{50}, N_{100}, P_{200} and the following positive peak between 230 and 370 ms from tone onset (P'_{200}; Fig. 2), periodic peaks corresponding

to the 3 and 6 Hz beats, were noted. The first four positive peaks of the beats-evoked oscillations were termed P_1, P_2, P_3 and P_4 and their latencies, amplitudes and source distributions were analyzed.

ERP peak amplitudes and latencies were subjected to a repeated measures analysis of variance (ANOVA) with Greenhouse–Geisser correction for violation of sphericity (when violation was indicated) and Bonferroni corrections for multiple comparisons. To assess scalp distribution effects of beat type and stimulus factors, potentials were initially analyzed for the effects of the following factors: Beat Type (2 levels: acoustic and binaural beats), Base Frequency and Beat Frequency combinations (4 levels: 250 and 1000 Hz base frequencies, each with 3 or 6 Hz beats) and 9 of the Electrodes (F_3, F_z, F_4, C_3, C_z, C_4, P_3, P_z, P_4) representing left, right and midline locations in the frontal, central, and parietal scalp areas. Subsequently, ANOVAs for the effects of Beat Type, Base Frequency and Beat Frequency, in the 9 electrodes were conducted to assess the effects of Beat Type, Base Frequency and Beat Frequency separately. In 11 of the subjects, the effects of acoustic beat phase was also evaluated by running ANOVAs for the effects of acoustic beat Onset Phase (abrupt or baseline) and Electrodes (F_3, F_z, F_4, C_3, C_z, C_4, P_3, P_z, P_4) 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 violation was indicated), were considered significant.

2.6. ERP functional imaging

An earlier study (Karino et al., 2006) on magnetic fields evoked by beats of 4.00 or 6.66 Hz in base frequencies of 240 and 480 Hz suggested multiple sources of the responses in the left and right auditory cortices and in parietal and frontal cortices. Therefore, we chose a source estimation method that makes minimal assumptions on the location of sources and no assumptions on their number.

Standardized Low Resolution Electromagnetic Tomographic Analysis (sLORETA, Pascual-Marqui et al., 1994; Pascual-Marqui, 2002) is a functional brain imaging method that estimates the distribution of current density in the brain as suggested by the minimum norm constraints. 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). sLORETA was therefore selected and applied on the 21-channel ERP records to image the estimated source current density throughout the duration of the beats-evoked oscillations, in response to the 8 stimulus conditions (2 Base Frequencies $\times$ 2 Beat Frequencies $\times$ 2 Beat Types).

In this study, differences in current density distributions between the acoustic and binaural beats-evoked oscillations, across stimulus conditions, were assessed using Statistical non-Parametric Mapping (SnPM). The SnPM method estimates the probability distribution of the data set being analyzed 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 earlier studies by comparing its results with more conventional ANOVA results (Laufer and Pratt, 2003; 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 acoustic and binaural beats-evoked components we compared them across 45 ms (11 time frames) from 5 time frames before- until 5 after the peaks of the components. The peaks used for comparison in each subject's recording were the ones with the source distribution closest to the grand-averaged source distribution of the beats-evoked oscillations. Differences were considered significant if either 11 time frames from each comparison or their average current density indicated significance.

Average current density values from the 5 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: Beat Type (binaural and acoustic), Brain Region (the surroundings of BA 11, 20, 21, 37 and 38) and Hemisphere (left and right). This analysis was conducted separately for beat frequencies of 3 and 6 Hz with a base frequency of 250 Hz. Preliminary analyses did not reveal a consistent effect of base frequency on source current density distribution. Probabilities below 0.05, after Greenhouse–Geisser corrections for violations of sphericity (when violation was indicated), were considered significant.

2.7. ERP spectral analysis

In addition to time-domain peak measures, the frequency content of the waveform following P'_{200} and preceding the offset response at the end of the waveform (Fig. 2), 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 8 stimulus conditions (3- and 6-Hz acoustic and binaural beats in 250 and 1000 Hz base frequencies). Acoustic beats were described by subjects as "somewhat more pronounced" than the respective binaural beats. All beats were localized to the center of the head. At onset, all tones evoked components P_{50}, N_{100} and P_{200}, followed by an additional positive peak labeled P'_{200}, and repetitive positive/negative oscillations at the perceived beat frequency. We identify these "beat" oscillations at their positive peaks as P_1, P_2, P_3, etc. An offset N_1–P_2 complex occurred at the end of the record (Fig. 2). The interpeak latency differences between the oscillatory peaks following the onset response corresponded 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. The oscillatory potentials evoked by the acoustic beats were a quarter of a period removed from their counterparts to binaural beats when embedded in a tone with an abrupt onset (Fig. 3). Oscillatory potentials associated with binaural and acoustic beats overlapped when the latter started from baseline at tone onset (Fig. 4).

The prominent positive component at the end of the traces is present in both types of beats, whereas in Fig. 4, in which only the binaural beats had an abrupt offset and acoustic beats have a baseline offset, this positive component is absent in the acoustic beats. Note that in both figures, this offset potential has a distinct scalp distribution being largest in midline central and frontal leads and absent in lateral and occipital leads. This would speak against a filter artifact as all leads were filtered similarly. It is also incompatible with an interaction of the filter with stimulus artifacts because stimulus artifact is largest at lateral electrodes, close to the earphones, whereas the offset potential is smallest at these sites. Interestingly, the offset N_{100} is, as expected, smaller to offset than to onset, suggesting that the large positive offset component is a composite of an offset P_{200} and some other components that need to be resolved in future studies.

The oscillations following the onset response were not the result of stimulus artifacts from the left- and right-earphones. This

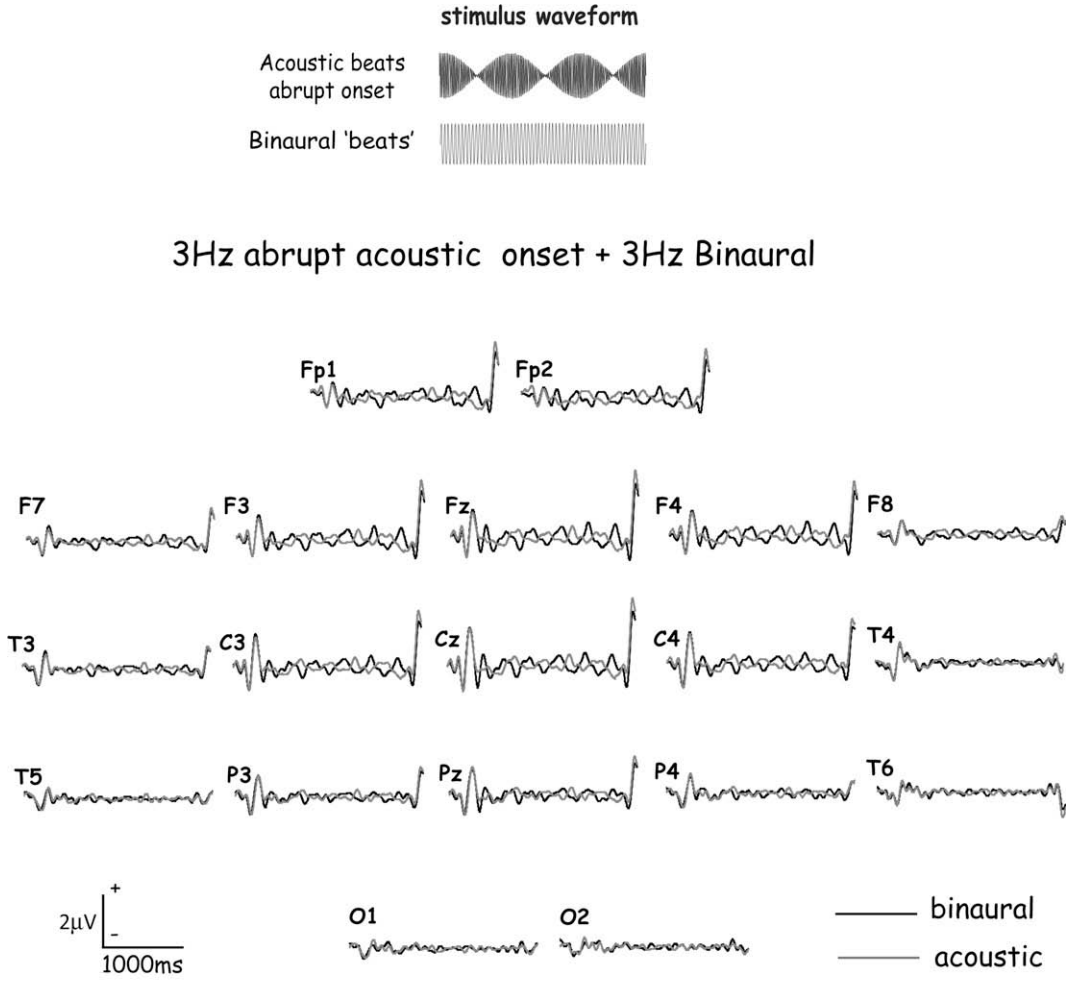

Fig. 3. Scalp distribution of the potentials to tone onsets and the 3 Hz binaural beats and acoustic beats with the abrupt onset to full amplitude beats with the 250 Hz base frequency, grand averaged across 18 subjects. The top of the figure presents the waveforms of the acoustic stimuli and the bottom – the ERPs evoked by them. The ERP plots include a baseline of 100 ms before tone onset and the initial components of the offset response. Note the synchronized onset and offset responses and the out-of-phase binaural and acoustic beats-evoked oscillations.

was determined in 5 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 electrical beats that would not have been affected by the occlusion of ears.

3.1. Comparison of binaural beats with abrupt-onset acoustic beats

When scalp distribution effects of beat type and stimulus factors were analyzed, no interactions involving the Electrodes factor were observed, in line with a similar scalp distribution across stimulus conditions. Therefore, the effects of Beat Type, Base Frequency and Beat Frequency, in the 9 electrodes were subjected to analysis of variance procedures to assess these effects on the beats-evoked oscillations.

In general, beats-evoked oscillations were larger to acoustic than to binaural beats, to 3 Hz than to 6 Hz beats, and with 250 than with 1000 Hz base frequency. These differences were more pronounced with acoustic than with binaural beats, and with the lower base and beat frequencies, as detailed below.

3.1.1. Waveform comparisons

P_1, P_2, P_3, and P_4 were the initial four positive peaks of the beats-evoked oscillations. Observing their waveform, the abrupt-onset acoustic beats-evoked oscillations and the binaural beats-evoked oscillations were different in phase relative to each other (Fig. 3). Their latencies were therefore, obviously different in their peak latencies (by approximately 85 or 40 ms with 3 or 6 Hz beats, respectively). The latencies of both types of beats-evoked oscillations were significantly affected by Beat Type, inconsistently affected by Base Frequency and, as expected, very highly and increasingly significantly affected by Beat Frequency, such that the later the peak the larger the *F* ratio (Table 1). Latencies were consistently longer to binaural than to their acoustic beat counterparts (by 150 ms on average) and with 3 than with 6 Hz beats. Interestingly, all latencies of beats-evoked oscillations were significantly affected by significant Beat Type × Beat Frequency interactions (Table 2), in all of which the effect of beat frequency was more pronounced in the binaural beats.

Amplitudes of the beats-evoked oscillations were also significantly affected by Beat Type, by Base Frequency and by Beat Frequency (Table 1), with amplitudes consistently larger to acoustic than to binaural beats (by 0.17 μV on average), larger with base frequency of 250 than with 1000 Hz and to beat frequency of 3 than to 6 Hz. The amplitudes of the third and fourth oscillation

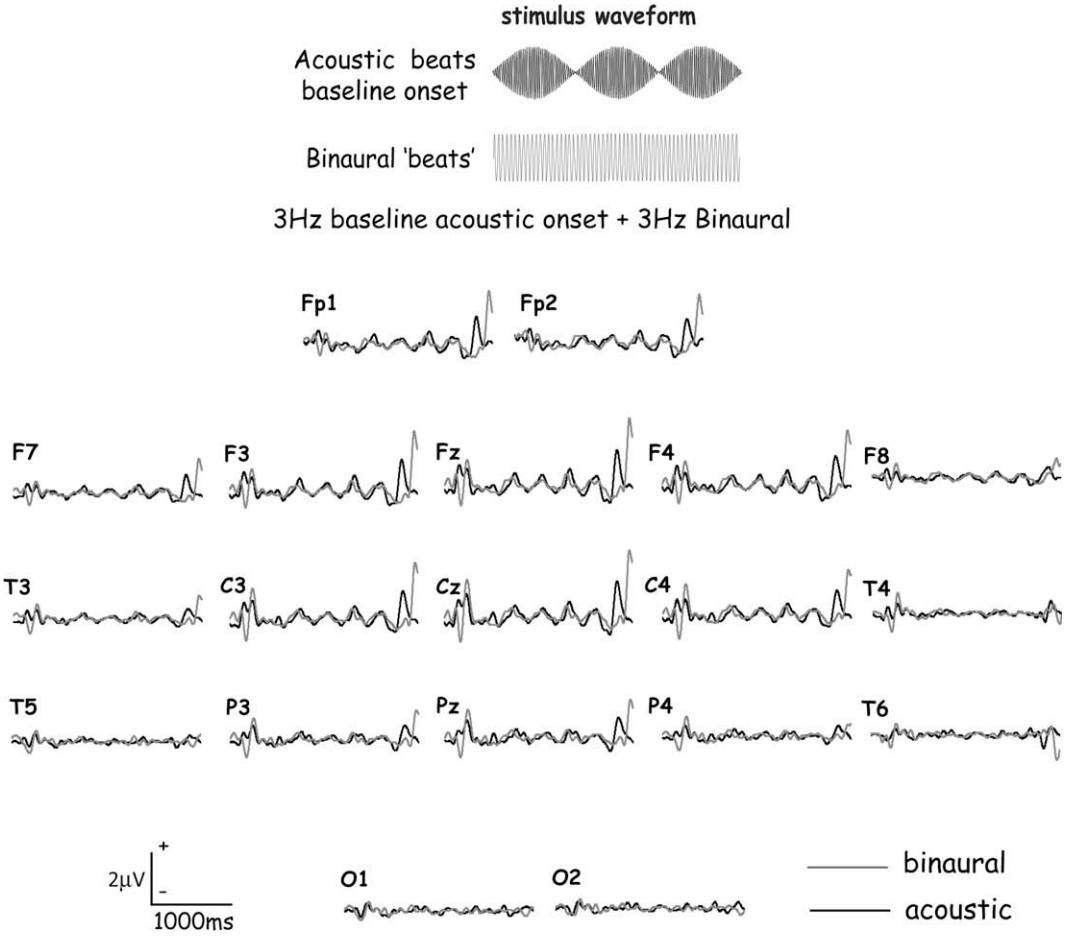

Fig. 4. Scalp distribution of the potentials to tone onsets and the 3 Hz binaural and acoustic beats with the gradual onset of modulation from baseline with the 250 Hz base frequency, grand averaged across 11 subjects. The top of the figure presents the waveforms of the acoustic stimuli and the bottom – the ERPs evoked by them, including a baseline of 100 ms before tone onset and the initial components of the offset response. Note the difference in the onset and offset responses and the almost synchronous binaural and acoustic beats-evoked oscillations.

peaks were also affected by significant interactions of Beat Type and Beat Frequency (Table 2), in which the larger acoustic beats were more evident with the beat frequency of 3 Hz. The amplitudes of the third and fourth oscillation peaks were also affected by a significant Base Frequency × Beat Frequency interaction (Table 2) in which the differences between beat frequencies were more pronounced with the base frequency of 250 Hz, and the difference between base frequencies was more pronounced with 3 Hz beats (peak P_3) or with 6 Hz beats (peak P_4).

Table 1
F-values and significance levels of the effects of Beat Type, Base Frequency and Beat Frequency on measures of the potentials evoked by abrupt-onset acoustic beats and binaural beats. N.S. denotes a non-significant effect.

Measure	Beat Type	Base Frequency	Beat Frequency
P_1 latency	$F(1, 16) = 138.60$, $p < 0.001$	N.S.	$F(1, 16) = 2634.44$, $p < 0.001$
P_2 latency	$F(1, 16) = 240.53$, $p < 0.001$	$F(1, 16) = 5.80$, $p < 0.02$	$F(1, 16) = 9697.51$, $p < 0.001$
P_3 latency	$F(1, 16) = 180.94$, $p < 0.001$	N.S.	$F(1, 16) = 21136.57$, $p < 0.001$
P_4 latency	$F(1, 16) = 127.09$, $p < 0.001$	$F(1, 16) = 4.19$, $p < 0.05$	$F(1, 16) = 37530.03$, $p < 0.001$
P_1 amplitude	$F(1, 16) = 11.14$, $p < 0.001$	$F(1, 16) = 7.09$, $p < 0.01$	$F(1, 16) = 114.54$, $p < 0.001$
P_2 amplitude	$F(1, 16) = 8.64$, $p < 0.004$	$F(1, 16) = 12.50$, $p < 0.001$	$F(1, 16) = 66.30$, $p < 0.001$
P_3 amplitude	$F(1, 16) = 53.64$, $p < 0.001$	$F(1, 16) = 114.54$, $p < 0.001$	$F(1, 16) = 141.26$, $p < 0.001$
P_4 amplitude	$F(1, 16) = 48.70$, $p < 0.001$	N.S.	$F(1, 16) = 139.39$, $p < 0.001$

3.1.2. Source estimates

Source current density distribution of the oscillatory beats-evoked potentials, to both acoustic and binaural beats (Fig. 5), 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 frontally (approximately BA 11). Notably, the left temporal lobe distribution to binaural beats was irrespective of the ear that was presented with the high-

Table 2
F-values and significance levels of the interactions between effects of Beat Type, Base Frequency and Beat Frequency on measures of the potentials evoked by abrupt-onset acoustic beats and binaural beats. N.S. denotes a non-significant interaction.

Measure	Beat Type × Beat Frequency	Base Frequency × Beat Frequency
P_1 latency	$F(1, 16) = 306.78$, $p < 0.001$	N.S.
P_2 latency	$F(1, 16) = 426.42$, $p < 0.001$	N.S.
P_3 latency	$F(1, 16) = 457.78$, $p < 0.001$	N.S.
P_4 latency	$F(1, 16) = 352.68$, $p < 0.001$	N.S.
P_1 amplitude	N.S.	N.S.
P_2 amplitude	N.S.	N.S.
P_3 amplitude	$F(1, 16) = 10.92$, $p < 0.001$	$F(1, 16) = 7.41$, $p < 0.007$
P_4 amplitude	$F(1, 16) = 57.14$, $p < 0.001$	$F(1, 16) = 6.84$, $p < 0.01$

er frequency. Source estimation of the spectral peak at the beat frequency revealed the same distributions (Fig. 5 top row compared to bottom row). Average current density values and spectral peak energy values from these 5 most active regions during the beats-evoked oscillations were subjected to repeated measures analysis of variance, for the effects of Beat Type (binaural and acoustic), Brain Region (BA 11, 20, 21, 37 and 38) and Hemisphere (left and right). This analysis was conducted separately for beat frequencies of 3 and 6 Hz. The results obtained with source current density and with spectral content were in full agreement, and in this report we detail the results of the frequency domain source analysis.

Source spectral peak energy for the 3 Hz beats was significantly affected by Beat Type [$F(1, 17) = 41.80$ $p < 0.001$], with the highest power associated with acoustic beats. Source activity was also significantly affected by the Brain Region involved [$F(4, 68) = 7.52$, $p < 0.001$] and by Brain Laterality [$F(1, 17) = 10.31$, $p < 0.002$], with the highest values in the vicinity of BA 20 and BA 21 in the left hemisphere. A significant Base Frequency $\times$ Brain Region interaction [$F(1, 16) = 5.86$, $p < 0.03$] indicated that activity in BA 21 was larger with 1000 Hz than with 250 Hz base frequency, but not so in BA 20. Similarly, a significant Beat Type $\times$ Brain Region interaction [$F(1, 16) = 7.08$, $p < 0.02$] indicated that activity in BA 21 was larger with acoustic – compared to binaural beats whereas in BA 20 activation was no different in response to acoustic and binaural beats.

When ANOVA was conducted on the 6 Hz beats, source spectral peak was also significantly affected by Beat Type [$F(1, 17) = 33.03$, $p < 0.001$], with the highest energy values associated with acoustic beats; by the Brain Region involved [$F(4, 68) = 33.03$, $p < 0.001$], with the most active regions around BA 20 and 21; and by Hemisphere [$F(1, 17) = 49.21$, $p < 0.001$], with the highest values in the left hemisphere. Significant interactions with Hemisphere indicated that Beat Type [$F(3, 51) = 3.56$, $p < 0.02$] and Brain Region [$F(4, 68) = 5.24$, $p < 0.004$] effects were more prominent on the left.

Fig. 6 shows the scalp-recorded waveform from C_z (very top trace) and the time courses of activation in key brain areas in the left and right hemispheres, estimated to be involved in the potentials to the first four 3 Hz binaural (top) and acoustic (bottom) beats, with a base frequency of 250 Hz. Note that the intracranial activity peaked approximately at the time of both scalp-recorded peaks and troughs. Also note the lateralization to the left of beats-evoked activity, particularly in lateral temporal areas.

SnPM comparison of the source distributions of the acoustic and binaural beats-evoked oscillations did not show significant differences. Similarly, when source distributions were compared between 3 and 6 Hz and between high- and low base frequency, no significant distribution differences were noted.

3.2. Comparison of abrupt-onset and baseline-onset acoustic beats

In 11 of the subjects, the potentials evoked by 3-Hz acoustic beats in a base frequency of 250 Hz with abrupt onset were compared with their counterparts whose beats onset began at baseline and was therefore more gradual and phase-shifted by a quarter of the beats period. In general, except for the expected latency differences, no significant differences were observed between beats-evoked oscillations to abrupt-onset and baseline-onset acoustic beats, except for an inconsistent finding of significantly [$F(1, 10) = 10.91$, $p < 0.002$] larger P_1 amplitude and significantly [$F(1, 10) = 7.64$, $p < 0.007$] smaller P_4 to baseline-onset than to abrupt-onset acoustic beats. These differences were very small (on average 0.1 μV), inconsistent between components, and were not found for the other beats-evoked components (P_2 and P_3) which were not different between acoustic beat types.

3.3. Comparisons of binaural- and baseline-onset acoustic beats

As detailed below, when potentials evoked by binaural beats were compared with their counterparts evoked by baseline-onset acoustic beats the results were the same as in the comparison with abrupt-onset acoustic beats detailed in Section 3.2.

3.3.1. Waveform comparisons

The beats-evoked oscillations associated with binaural beats were compared with their counterparts evoked by baseline-onset acoustic beats. P_1, P_2, P_3, and P_4 of the baseline-onset acoustic

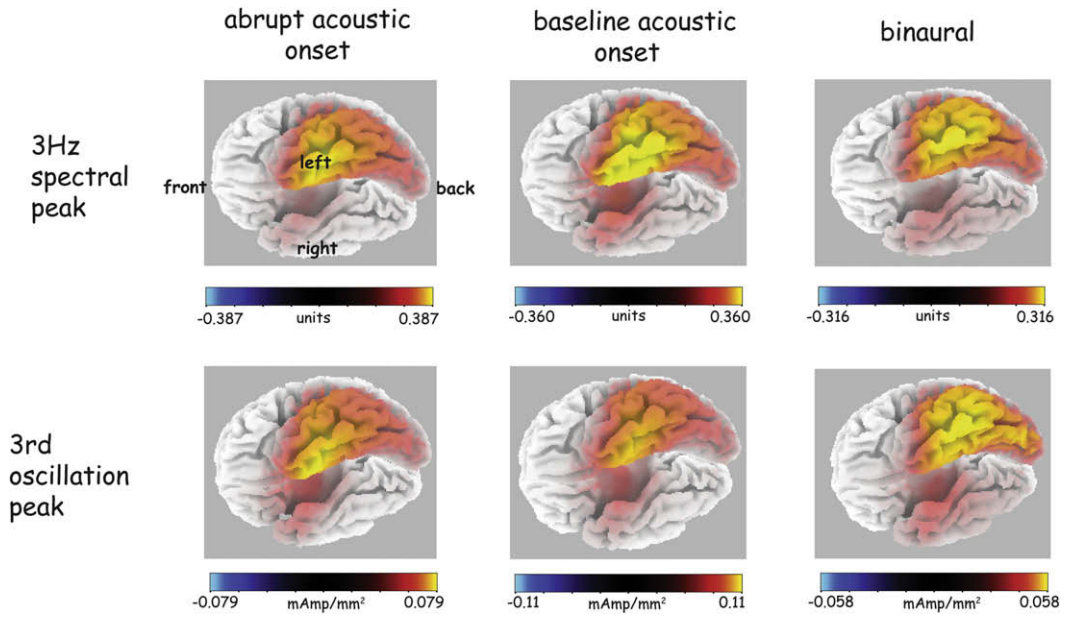

Fig. 5. Source distributions for the spectral peak (top) and for the 3rd oscillatory beats-evoked peak (bottom) across acoustic beats with abrupt beat onset (left column), acoustic beats with baseline beat onset (middle column) and binaural beats (right column). Stimuli were 3 Hz beats with a base frequency of 250 Hz. Note the tilted bottom view of the brain showing the practically identical distributions in the lateral inferior left temporal lobe across all stimulus conditions using spectral energy or source current density analyses. Only a very minor difference in distributions between acoustic and binaural beats is indicated at the posterior temporal lobe. Note the scales in current density units (bottom row) as well as in the relative arbitrary spectral units (top row) indicating somewhat lower activation by binaural beats.

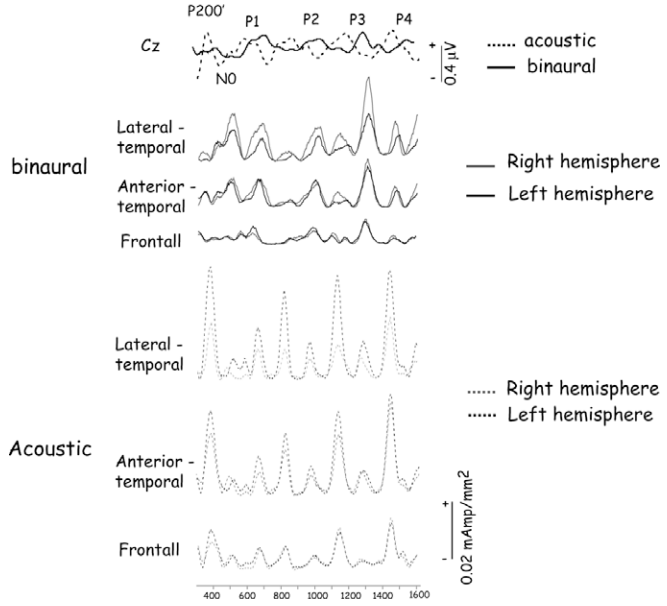

Fig. 6. Time courses of average current density values in the left and right hemisphere regions that were most involved with 3 Hz binaural beats and acoustic beats with abrupt onset from baseline: lateral temporal areas (BA 20, 21, 37) anterior-temporal lobe (BA 38) and frontal region (BA11) with a base frequency of 250 Hz. The grand-averaged waveform recorded at C_z, including final onset components P'_{200} and N_0, followed by beats-evoked oscillation peaks P_1, P_2, P_3 and P_4 are plotted at the top. Note the correspondence of current density peaks to the scalp-recorded positive as well as negative peaks. Time 0 corresponds to stimulus onset.

beats-evoked oscillations and of the binaural beats-evoked oscillations were in-phase (Fig. 4) and their peak latencies were very similar, but consistently somewhat longer (by an average of 44 ms) in response to the baseline-onset acoustic beats [$F(1, 10) = 104.08$, $p < 0.001$ for P_1; $F(1, 10) = 132.64$, $p < 0.001$ for P_2; $F(1, 10) = 35.66$, $p < 0.001$ for P_3; and $F(1, 10) = 45.98$, $p < 0.001$ for P_4].

Amplitudes of P_1, P_2, P_3, and P_4 of the baseline-onset acoustic beats-evoked oscillations were consistently larger (by 0.17 μV on average) than their binaural beats-evoked counterparts [$F(1, 10) = 32.51$, $p < 0.001$ for P_1; $F(1, 10) = 10.53$, $p < 0.002$ for P_2; $F(1, 10) = 27.53$, $p < 0.001$ for P_3; and $F(1, 10) = 18.04$, $p < 0.001$ for P_4].

3.3.2. Source current density comparisons

Source current density distribution of the three types of beats-evoked potentials were remarkably similar 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 all 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). Only a minor difference between binaural and acoustic beats may be noted in the posterior temporal lobe. Thus, source distributions of the oscillations to baseline-onset and to abrupt-onset acoustic beats were found not to be different. Therefore, analysis of variance procedures on the effects of experimental parameters on the source activity was not repeated on responses to the phase-adjusted acoustic beats.

SnPM comparison of the source current density distributions of the baseline-onset acoustic beats and of the binaural beats at 3 and 6 Hz did not reveal a significant difference. Similarly, when source distributions were compared between abrupt-onset acoustic beats and baseline-onset acoustic beats, no significant source distribution differences were noted.

3.4. Summary of results

Overall, all 12 stimulus conditions (binaural beats, abrupt-onset acoustic beats and baseline-onset acoustic beats of 3 and 6 Hz with 250 and 1000 Hz base frequencies) evoked beats-evoked oscillations, in addition to the tone onset and offset components. In general, acoustic beats-evoked oscillations were larger than binaural beats-evoked oscillations, all three beat types evoked oscillations that were higher in amplitude with the 250 Hz compared to the 1000 Hz base frequency and to 3 compared to 6 Hz beats. Sources of all the beats-evoked oscillations were located mostly in the vicinity of the left lateral and inferior temporal lobe in all stimulus conditions. Source current density analyses indicated that the differences across beat types were minor and located to the vicinity of BA 20 and 21, in the left hemisphere, with activity around BA 21 larger than around BA 20 to acoustic beats but not to binaural beats.

4. Discussion

4.1. General context

In this study brain potentials associated with amplitude modulated tones (acoustic beats) were compared to their counterparts evoked by stimulating each ear with a slightly different tone to produce a perception of amplitude modulation (binaural beats). The perception of binaural beats is not limited to the laboratory setting. Dancers report a 'beating' quality to prolonged (fermata) unchanging notes played from speakers on both sides of the stage when they move across the stage with fast head movements. The opposite Doppler effects on the inputs to the two ears, create a small frequency difference between the ears, sufficient to produce an illusion of amplitude modulation of the note.

Acoustic and binaural beats provide a unique opportunity to compare a similar perception (beats) created by either an actual acoustic event (acoustic beats) or by the effects central auditory processing (binaural beats). This comparison, and the relatively well understood effect of binaural beats provide a model to study central mechanisms of auditory perceptions such as elementary and complex auditory auras (Hogan and Kaiboriboon, 2004), hearing a favorite and familiar song (Fischer et al., 2004; Ozsarac et al., in press), the perception of "voices" in paranoid schizophrenics (Dierks et al., 1999; Shergill et al., 2000) and musical hallucinations in Alzheimer's disease (Mori et al., 2006).

The results of this study found no significant differences in the effects of stimulus manipulations or cortical distribution of the evoked potentials to acoustic and binaural beats. Such lack of differences does not prove similarity and may be related to poor statistical power. However, similar sample sizes have been sufficient to show significant differences between experimental conditions in other studies (e.g., Pratt et al., 2007; Haiman et al., 2008) using the same analysis techniques (ANOVAs of waveform measures and SnPM and sLORETA for source estimates) and number of electrodes (21 scalp electrodes). Moreover, the failure to find differences repeated across analysis methods (spectral and time domain), stimulus types (abrupt onset and baseline onset) and source estimates. Hence, the consistent lack of differences in cortical representation of acoustic and binaural beats is not spurious. Small differences that were undetected due to methodological limitations cannot be ruled out, but are most likely smaller than the effects typically seen in parametric studies using ERPs.

Although the acoustic stimuli for acoustic and binaural beats were equal in intensity, the acoustic beats were reported to be more pronounced than their binaural counterparts, and the oscillations evoked by acoustic beats were correspondingly larger than their binaural counterparts. However, both beat types were simi-

larly affected by beat frequency and base frequency and shared the same cortical sources, in line with the similar beating quality of perceptions to both. Both acoustic and binaural beats-evoked oscillations were significantly larger to 3 than to 6 Hz beats and larger with a base frequency of 250 than with 1000 Hz. Sources of both types of beats-evoked oscillations were located to the vicinity of the left lateral and inferior temporal lobe. The cortical similarities and differences of these potentials will be related to the convergence of monaural and binaural pathways on similar auditory cortical regions.

4.2. Binaural beats and acoustic beats with different onsets

The 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. In humans, the frequency following responses 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 human ascending auditory pathway, beginning at about 1 kHz. This decline parallels the decline in perception of binaural beats above 1000 Hz, suggesting that phase-locked neural activity is likely involved in binaural beats.

The rate of discharge of auditory neurons typically increases as a function of sound intensity (Goldberg et al., 1964; Kiang et al., 1973). It is thus not surprising that amplitude modulated sounds evoke correspondingly modulated post-stimulus, or cycle histograms of auditory neurons (Moller, 1973). Interestingly, the modulation of discharge frequency of the neurons as a result of intensity modulation of the sound is affected by modulation frequency, substantially decreasing at modulation frequencies above 100 Hz at the brainstem level. Human auditory steady-state potentials include modulated brainstem activity at the frequency of amplitude modulated sounds, which are optimally recorded with a modulation frequency of 40 Hz and brainstem activity which is optimally recorded with a modulation of around 80 Hz (Picton et al., 2003).

The beat frequencies recorded in this study were 3 and 6 Hz, frequencies that favor cortical activity. Neither beat frequencies nor other experimental parameters were manipulated to selectively augment brainstem contributions. Therefore, the results of this study are limited to the cortical manifestation of beats, and the precise site along the auditory pathway in which they are first generated was not determined. However, in conjunction with earlier studies, our results are compatible with subcortical activation of the temporal cortex from which the oscillations were recorded and similar cortical activity associated with both types of beats.

The results of our earlier study on cortical activity associated with binaural beats (Pratt et al., 2009) suggested that the slightly different volley frequencies from left and right ear which converge and interact in the brainstem pathways to generate beats of neural activity, which in turn, modulate activities in the left temporal lobe. Similarly, the modulated acoustic stimulus of acoustic beats generates beats of neural activity, which in turn, modulate activities in the left temporal lobe. This modulated left temporal lobe activity is associated with the sensation of beats and is recorded as voltage oscillations on the scalp. The scalp-recorded oscillations are thus phase-locked activity at the cortical level. The exact phase of the cortical beats is determined by conduction and synaptic delays from the site at which neural beats are generated (most likely at the brainstem level) to the left temporal lobe.

The smaller amplitudes of the oscillations associated with binaural- than with acoustic beats may be explained by the mechanism generating binaural beats. The sum of the electro physiological potentials to monaural left- and right ear stimulation is greater than the response to binaural stimulation, resulting in a binaural interaction component at the brainstem level, with a larger effect at cortical levels (Dobie and Norton, 1980; Wrege and Starr, 1981; Debruyne, 1984; Woods and Clayworth, 1985; McPherson and Starr, 1993; Polyakov and Pratt, 1995). Similarly, the oscillations to binaural beats, generated by convergence of left and right ear inputs on binaural brainstem neurons, would be expected to be smaller than the oscillations to the sum of monaural acoustic beats, and even more so at the cortical level.

In contrast, acoustic beats are a result of amplitude modulation of the acoustic input to each ear, and thus they do not necessarily involve convergence of activity from both ears (thus they are often called 'monaural beats'). Therefore, the afferent activity of acoustic beats to the left temporal lobe does not necessarily involve the same binaural neural substrates as binaural beats, and the latency (or phase) of acoustic and binaural beats is not necessarily the same. This assumption was tested in this study by comparing binaural beats with acoustic beats having different onset phases. Indeed, the phase of cortical acoustic beats could differ from that of binaural beats, in line with their different peripheral neural substrates.

4.3. Sources of acoustic beats and binaural beats

Sources of all the beats-evoked oscillations were located mostly to the same left temporal lobe areas across all stimulus conditions. The lateralization of all beats-evoked oscillations to the left temporal lobe are in line with behavioral, positron emission tomography evidence and clinical findings on specialization of the left-hemisphere auditory cortex 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 beats-evoked activity to the 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 sources estimated in the present study and in our previous study on binaural beats (Pratt et al., 2009) appear to be at odds with some previous reports, which, in turn, are also at odds with each other (Draganova et al., 2008; Karino et al., 2006; Schwarz and Taylor, 2005). These source differences were widely discussed in our earlier report (Pratt et al., 2009) and were reconciled by differences in methods of recording (electric or magnetic fields), beat frequency (40 Hz or a few Hz), source models (dipole, current density distribution or no model with only scalp distribution). These methodological variations resulted in differences in the relative contributions of symmetrical, high-frequency thalamocortical and lateralized, slower cortical sources to the scalp-recorded responses in addition to the relative contribution of slower cortical activity from secondary non-specific cortex and middle latency activity from specific auditory cortex (Galambos et al., 1981; Kileny and Shea, 1986; Hashimoto, 1982; Lee et al., 1984; McGee et al., 1992; Zaaroor et al., 2003). The earlier studies and this study thus complement each other, showing that beats, most probably present as early as the brainstem (human steady-state potentials and animal studies), persist through auditory processing at the thalamus and specific auditory cortex (previous beats studies) and further involve cortical non-specific areas in the left temporal lobe (this study).

Source current density analyses indicated that a minor difference in sources of binaural and acoustic beats may involve the

vicinity of BA 20 and 21, in the left hemisphere. Whereas with acoustic beats activity in the area around BA 21 was significantly larger than around BA 20, this was not the case for binaural beats. This minor difference may be evident in Fig. 5. Except for this small difference, the sources of potentials to the perception of binaural beats and to their acoustic counterparts with either onset phase were not different. This indicates that cortical processing actual amplitude modulations of sound and the illusion of such modulations involve the same temporal lobe cortical areas.

4.4. Binaural beats, acoustic beats and onset responses

The comparison of onset responses to the acoustic beats oscillations having different phases and to binaural beats can also indicate whether beats-evoked oscillations constitute repetitive onset responses to each of the sound modulation cycles. In an earlier study (Pratt et al., 2009) we compared the potentials to binaural beats with those to stimulus onset of the same stimuli. Whereas onset potentials were bilaterally located to the left and right temporal lobes, binaural beats-evoked oscillations were lateralized to the left. In the present study both types of acoustic beats (abrupt-onset and baseline-onset) as well as binaural beats-evoked activity were lateralized to the left temporal lobe whereas onset responses were bilateral. Moreover, comparing potentials to abrupt-onset and to baseline-onset acoustic beats (Figs. 3 and 4), amplitudes of the onset-evoked potentials were markedly different while the beats-evoked oscillations were of similar amplitudes (albeit a different phase). Thus, amplitudes of onset responses can be dissociated from those to acoustic beats, indicating that they are distinct.

4.5. Effects of base frequency and beat frequency on beats-evoked oscillations

All beat types evoked oscillations that were higher in amplitude with the 250 Hz compared to the 1000 Hz base frequency and to 3 – compared to 6 Hz beats. The larger amplitudes with a base frequency of 250 Hz are consistent with a previous study (Schwarz and Taylor, 2005) which showed that beats-evoked amplitudes were larger with low (400 Hz) than with the higher (3000 Hz) base frequency, to which only acoustic beats could be recorded.

The effect of base frequency on the beats-evoked oscillations may reflect the 'volley principle' of encoding frequency in the ascending auditory system. The temporal pattern of firing of auditory units 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 antero-ventral cochlear nucleus (Goldberg and Brownell, 1973) of experimental animals. Moreover, 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 beginning at about 1 kHz. The decline in the beats-evoked oscillations at 1000 Hz compared to 250 Hz probably reflects this high frequency decline in-phase locking in the human central auditory system.

The lower amplitudes of oscillations to the higher beat frequency (6 Hz) compared to the low (3 Hz) beat frequency may be related to cortical ability to follow repetitive activation (sometimes referred to as cortical 'refractoriness' or 'recovery cycle'). The auditory evoked N_1–P_2 potential decreases in amplitude at interstimulus intervals shorter than 8–10 s, is reduced to a quarter with interstimulus intervals of 1 s (Davis et al., 1966; Hari et al., 1982), and with interstimulus intervals of 200 ms its amplitude is further halved (Gilley et al., 2005; Sussman et al., 2008). Thus, although the potentials to acoustic and binaural beats are clearly distinct from onset responses, their lability to increasing stimulus rate appears to be similar.

4.6. Conclusions

The results of this study show that brain cortical activity recorded from the scalp and associated with amplitude modulation of sound (acoustic beats) and with its perceptual counterpart – binaural beats – is similarly affected by experimental manipulations and the cortical regions involved are not different. The beats of cortical activity recorded in this study in response to acoustic beats and to binaural beats originated in the temporal lobe regions and both were lateralized to the left hemisphere. These results are congruent with similar involvement, in the cortex, of 'volley principle' neural encoding of both acoustic beats and their binaural beats counterparts.

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