

# Human auditory steady state responses to binaural and monaural beats

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## Abstract

**Objective:** Binaural beat sensations depend upon a central combination of two different temporally encoded tones, separately presented to the two ears. We tested the feasibility to record an auditory steady state evoked response (ASSR) at the binaural beat frequency in order to find a measure for temporal coding of sound in the human EEG.

**Methods:** We stimulated each ear with a distinct tone, both differing in frequency by 40 Hz, to record a *binaural beat* ASSR. As control, we evoked a beat ASSR in response to both tones in the same ear. We band-pass filtered the EEG at 40 Hz, averaged with respect to stimulus onset and compared ASSR amplitudes and phases, extracted from a sinusoidal non-linear regression fit to a 40 Hz period average.

**Results:** A 40 Hz binaural beat ASSR was evoked at a low mean stimulus frequency (400 Hz) but became undetectable beyond 3 kHz. Its amplitude was smaller than that of the acoustic *beat* ASSR, which was evoked at low and high frequencies. Both ASSR types had maxima at fronto-central leads and displayed a fronto-occipital phase delay of several ms.

**Conclusions:** The dependence of the 40 Hz binaural beat ASSR on stimuli at low, temporally coded tone frequencies suggests that it may objectively assess temporal sound coding ability. The phase shift across the electrode array is evidence for more than one origin of the 40 Hz oscillations.

**Significance:** The binaural beat ASSR is an evoked response, with novel diagnostic potential, to a signal that is not present in the stimulus, but generated within the brain.

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**Keywords:** Binaural; Beat; ASSR; EEG; Auditory; Steady state response; Temporal code

## 1. Introduction

Two simultaneous pure tone stimuli of frequencies within the same critical band (Fletcher, 1940; Greenwood, 1961a,b; Moore, 2003) may be heard as one tone with a pitch at the mean frequency of both inputs, beating at a rate equal to the difference between both input frequencies (cf. Moore et al., 1998). Because both tones are summed before entering the cochlear filter, acoustic beating tones can be heard at pitches throughout the auditory frequency range. Beat stimuli are similar to amplitude-modulated (AM) tones that are commonly used in auditory steady-state response (ASSR) audiometry (Picton et al., 2003). An important difference is that, at supra-threshold intensities, the two-component beat stimulus may excite a narrower cochlear

frequency band than an AM stimulus, which consists of three spectral components, spread over twice the frequency range at an identical pitch and envelope oscillation rate. Thus, the ASSR in response to beat stimuli (*beat*-ASSR) may, in special cases, offer improved frequency resolution in objective audiometry, justifying its exploration in human subjects.

A beating tone may also be heard when each sinusoidal component is presented, in isolation, to a different ear (Tobias, 1963). This *binaural beat*, a much fainter sensation, is restricted to relatively low component frequencies of a few hundred Hz (Licklider et al., 1950) that are temporally encoded as spike firing phase-locked to the sound wave (Rose et al., 1968). In the mammalian auditory system robust phase locking, to tone frequencies up to a few kHz, has been demonstrated in cochlear nerve fibers (Palmer and Russell, 1986) and in the spherical bushy cells of the antero-ventral cochlear nucleus (Goldberg and Brownell, 1973) whose

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axons project to the medial superior olivary (MSO) nucleus (Goldberg and Brown, 1969; Smith et al., 1993). MSO neurons receive the inputs from both ears separately, each at one of their two dendritic trees (Ramon Y Cajal, 1909; Schwartz, 1977), where the sound waves are, presumably, replicated (Agmon-Snir et al., 1998). The membrane potential at the MSO neuron spike trigger zone, close to the soma, is thought to reflect a combination of the sound waves from both ears, and the output spike rate of MSO neurons is modulated at the envelope time pattern of the sum of both input waveforms (Yin and Chan, 1990). Thus, firing patterns of inferior colliculus neurons, the target of MSO axon projections, can reflect a binaural beat (Kuwada et al., 1979). In the tonotopic array of the MSO, each neuron is tuned to a narrow frequency band, similar to cochlear nerve fibers, and different tones in both ears may be combined only if they fall in the same critical frequency band. Thus, different tones in each ear can be heard as binaural beat only if they are separated in frequency by less than a critical band (like acoustic beats) and have frequencies sufficiently low to be temporally encoded (unlike acoustic beats). An EEG response to binaural beat stimuli will, therefore, functionally assess the temporal coding mechanisms in the lower auditory brainstem.

A binaural beat stimulus should, hypothetically, be able to evoke an ASSR, since it should cause firing rates in the MSO output, and thus in cortical afferents, to be modulated in a manner similar to the AM tones commonly used in ASSR audiometry. We did not doubt that we could record an *acoustic beat* ASSR from the human brain, since this response type had already been reported for non-human mammals (Dolphin et al., 1994). Our ability to detect a binaural beat ASSR was less certain, however, for several reasons. (1) The binaural beat usually is a faint sensation. Thus, a corresponding EEG signal could be small and difficult to separate from larger EEG oscillations. (2) Maximal steady state responses have been recorded at stimulus repetition- or amplitude modulation rates in the gamma range, close to 40 Hz (cf. Picton et al., 2003). The human auditory system may interpret a 40 Hz modulation rate as a distinct low pitch tone rather than a beat. (3) A human subject may have a choice to listen to two tones in different ears separately or combine them as one beating sound. Here, one perception could exclude the other, as in Edgar Rubin's famous picture showing mutually excluding contours of either two faces or a vase (cf. Kandel, 1991). During dichotic sound stimulation two mutually exclusive perceptions may also occur. For example, a dichotic presentation of the second of two noises, delayed between the ears, can cause either a noise sensation at a specific location in space or a musical pitch (Fourcin, 1970). A binaural beat ASSR could, therefore, depend upon a focus of attention on the beat sensation. Using a time domain analysis, we attempted to optimize a distinction of small steady state responses from the ongoing EEG and noise. Hink et al. (1980) reported a beating binaural frequency

following response. Here we show that two pure tone stimuli in separate ears can evoke a binaural beat ASSR at a 40 Hz difference frequency in normal hearing subjects. The binaural beat ASSR differed qualitatively from a much more robust monaural, acoustic *beat* ASSR.

## 2. Methods

### 2.1. Subjects

Eighteen healthy subjects (11 females and 7 males, ages from 16 to 47 years) without otological or neurological disease in their histories and with normal pure tone audiometric thresholds were recruited for experiments. The subjects were paid to participate in the study, informed in writing about purpose and procedures and signed a consent form, according to procedures approved by the Human Ethics Committee of the University of British Columbia, in compliance with the Helsinki Convention.

### 2.2. Stimuli

Stimuli were two simultaneous pure tones differing in frequency by 40 Hz, with frequency means of 400 or 3200 Hz. For example, a 40–400 stimulus, in our nomenclature, consisted of components at 380 and 420 Hz. Stimuli were generated with Labview-6 software on a National Instruments I/O board, in a Pentium IV-based computer, at a 200 kHz D/A update rate. The two components were either separately fed through the two channels of an Amcron stereo power amplifier into two separate channels of Etymotic ER-2 insert earphones (binaural beat stimuli), or they were digitally summed into one output channel and fed to one of the insert earphones for presentation in the right ear in 16 cases, or the left ear in two cases (acoustic beat stimuli). Attenuation between stimuli delivered by an insert earphone and the opposite ear was greater than 70 dB at all frequencies used in this study. The components were presented as tone bursts of 1.2 s duration, starting at a fixed phase of 0° and gated on and off with 5 ms cosine ramp functions. The stimulus bursts were alternated with stimulus-free intervals of 1.2 s and 180 of these 2.4 s stimulus/interval epochs were repeated per typical recording session. Individual thresholds were determined at component frequencies, and stimuli were presented at intensities of 70 dB above the thresholds in all subjects and at 30 dB in nine of these subjects.

### 2.3. Focus of attention

A few subjects with musical training attempted to assign a musical interval value to the difference between both component tones. As this effort appeared to suppress the generation of a binaural beat ASSR, we instructed subjects

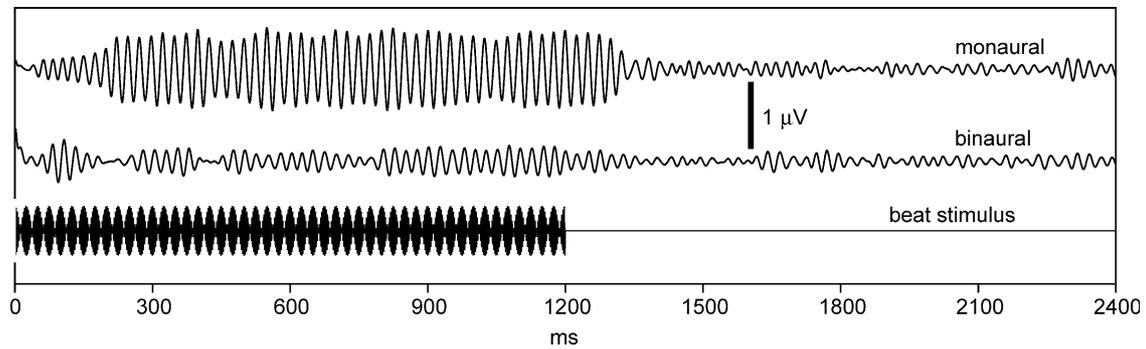


Fig. 1. Beat ASSRs. Averaged monaural *beat ASSR* (top) and *binaural beat ASSR* (middle) recorded at CZ. The beating sound wave, shown at the bottom, is the sum of two sine waves with frequencies of 380 and 420 Hz, which were presented either both in the right ear (monaural, top) or one in the right and the other in the left ear (binaural, middle). Intensities: 70 dB above component thresholds.

to attend to one warbling tone and not to try and identify separate components.

#### 2.4. Recordings and data analysis

We recorded a continuous EEG during, and, as control, before and after the repetitive stimulus presentation. Using a Neuroscan 4.3 digital system we sampled the EEG at 1 kHz per channel, initially with 27 mono-polar electrodes, referred to both earlobes and placed and labeled according to the international 10–20 system. Due to unacceptable signal/noise levels at most lateral electrodes we later restricted the recording routinely to the mid-sagittal electrode array indicated in Fig. 7. No phase shift between channels was detectable in a 60 Hz calibration sine wave introduced to all channels simultaneously. Electrode impedances were kept below 5 k $\Omega$ . We displayed the records on a computer screen, interactively removed stimulus-response epochs and control segments containing eye movement—and excessive EMG artifacts and then digitally band-pass filtered the EEG between 36 and 44 Hz with 48 dB/octave slopes (Butterworth filter). We then

averaged the remaining 80–178 stimulus epochs, with respect to stimulus onset time, to obtain records of the ASSRs during the stimuli and subsequent intervals (Fig. 1). As additional controls, we similarly averaged 1.2 s epochs from the EEG before or after the stimulus presentation. These ‘controls’ were included to test the hypothesis that stimulus-locked oscillations might persist during the intervals. Since this hypothesis was not supported by the data, the appropriate controls for stimulus epochs are, in fact, the intervals, which, in contrast to the controls, were recorded during approximately identical states of alertness. From these stimulus, interval and control epochs we subsequently computed 40 Hz period-averages over 1 s, starting 200 ms after epoch onset, in order to limit possible contaminations of the steady state responses by other potentials evoked by the sound onset or offset and to accommodate cortical integration (Roß et al., 2002). Period average means and standard errors at the 25 data points were plotted for all three conditions. Using a non-linear regression analysis with 150 iterations, sinusoidal functions were then fit to the data (e.g. Fig. 2) in an effort to compare, with *F*-tests, the amplitudes and phase angles of the fundamental frequency components

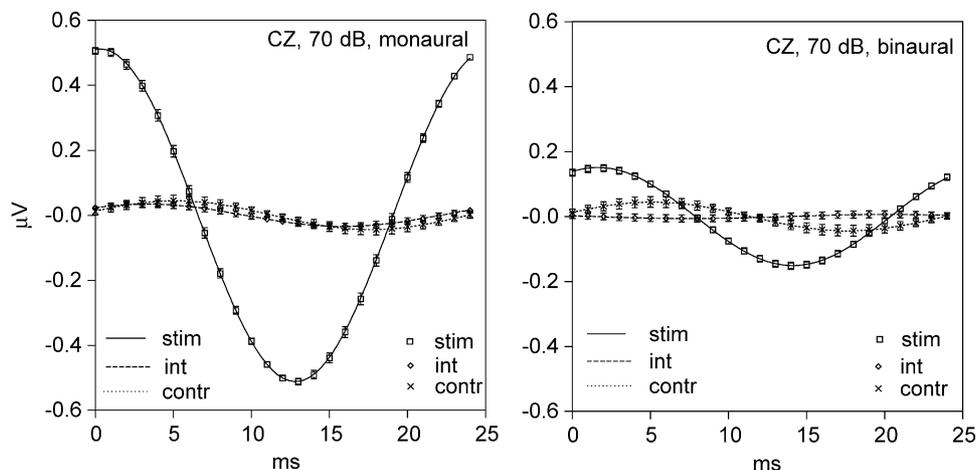


Fig. 2. Beat ASSR periods. Period averages and regression functions for stimulus (stim), interval (int) and control (contr) epochs of monaural *beat ASSR* (left) and *binaural beat ASSR* (right) recorded at CZ. Data points are amplitude means; error bars indicate standard errors. Lines are non-linear sinusoidal 40 Hz regression functions fit to the data.

( $F_0$ ) of the 40 Hz ASSR with the two control record types. Thus, amplitudes, phases and standard errors used for comparisons of records were derived from the non-linear regression analysis. If the ASSR amplitudes significantly exceeded 40 Hz oscillations during the two control epochs ( $F$ -test,  $P < 0.05$ ) we subsequently evaluated differences between amplitudes under the three conditions (stimulus, interval and control) with a one-way ANOVA, followed by the Tukey–Kramer post-test. This time domain analysis was chosen to restrict the quantification of the ASSR, which is stimulus-phase-locked by definition, to single 40 Hz sine waves at tightly defined phases. As a consequence, masking 40 Hz sinusoids that were unrelated to the ASSR, and sinusoids at spectrally adjacent frequencies, were eliminated in an effort to maximize signal/noise ratios and minimize variability.

For scalp locations yielding significantly greater amplitudes during stimulus epochs, compared with intervals and/or controls, we superimposed the regression curves to illustrate phase differences, interpreting phases to be different when their 95% confidence intervals did not overlap.

### 3. Results

#### 3.1. Beat ASSR

Stimulation of the same (right or left) ear with two tones differing in frequency by 40 Hz, with a mean frequency of 400 Hz and presented 70 dB above individual component thresholds (40–400, 70 dB stimulus), produced a strong 40 Hz oscillation in the band pass filtered averaged EEG record. This acoustic *beat ASSR*, shown for the CZ electrode in the example of Fig. 1 (monaural), gradually grew in amplitude over the first 200 ms and was sustained throughout the remaining stimulus duration. After termination of the stimulus the oscillation continued for a few cycles and waned, after ~200 ms, into the (band-pass filtered) background EEG prevailing in the inter-stimulus interval. During the sustained stimulus-response period (200–1200 ms after stimulus onset) the beat ASSR irregularly fluctuated in amplitude, as in Fig. 1, at all recording sites of all subjects, sometimes by more than 50%, in spite of its classification as ‘steady state response’.

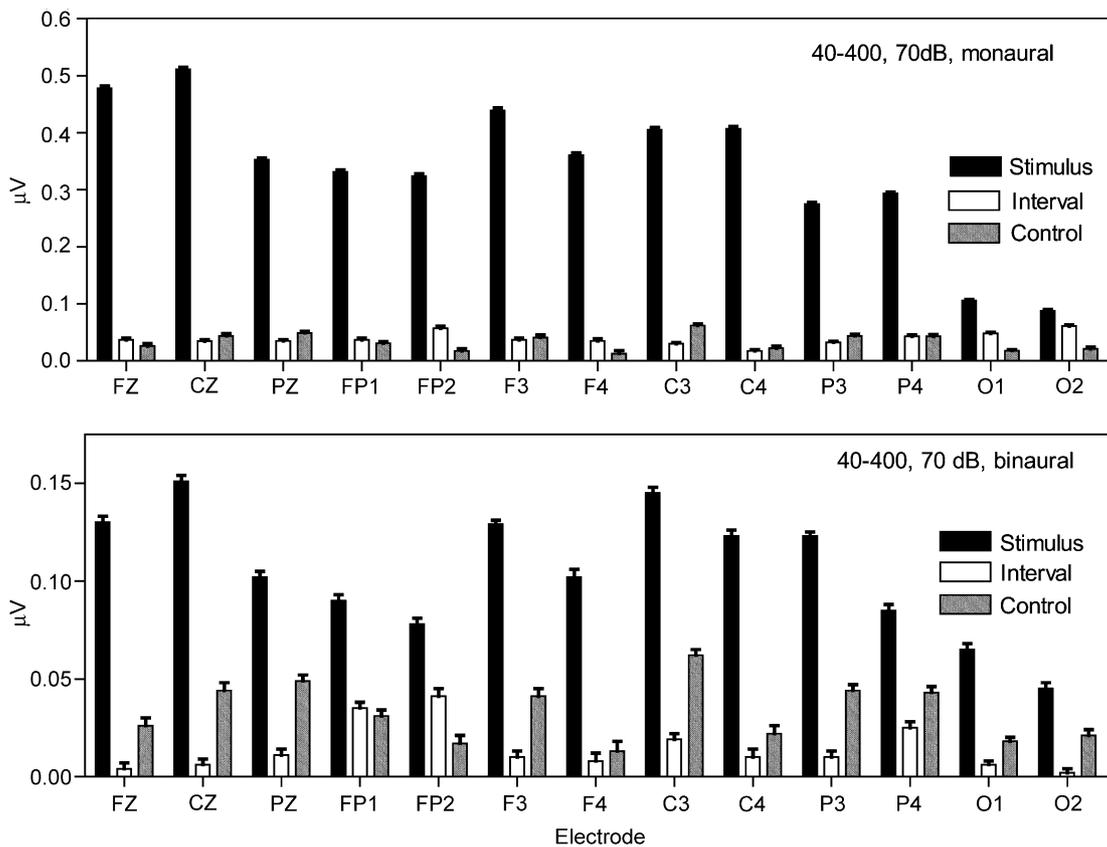


Fig. 3. Beat ASSR amplitude distribution. Amplitudes of sinusoidal regression functions computed for stimulus, interval and control epochs at a mid-sagittal electrode array in one subject. ‘Intervals’ are epochs between sequential stimuli whereas ‘control’ epochs were recorded a long time before or after stimulation, possibly during a different state of alertness. Top: *beat ASSR*, bottom: *binaural beat ASSR*. Stimulus parameters are indicated in each plot (top right). Error bars show the uncertainty in amplitude definition by the non-linear regression analysis. Amplitudes during stimuli were significantly greater than during interval- or control periods at all electrodes in this example ( $P < 0.001$ ).

### 3.2. Binaural beat ASSR

A sustained 40 Hz oscillation was also visible when the two component tones were presented separately in the different two ears, 70 dB above component thresholds in each ear. Amplitudes of this binaural beat ASSR (Fig. 1, binaural) were considerably smaller, compared with the monaural beat ASSR. A decision about its presence hinged upon a statistical comparison of 40 Hz oscillation amplitudes during stimulus and interval epochs. To this end, we computed a 40 Hz period average (25 ms) for each stimulus response record, starting 200 ms after stimulus onset and terminating with the stimulus, and for corresponding times during the stimulus intervals. In order to assess a possibility of stimulus-induced 40 Hz oscillations during intervals we also included, in our comparison, 'control' epochs of equal duration, recorded tens of minutes to hours before or after stimulation sessions. As shown in Fig. 2, the 25 averaged data points per period were well fit by 40 Hz sinusoidal regression functions for the stimulus, interval and control conditions. It is self-evident, in this example, that the  $F_0$  amplitudes of both the acoustic *beat* ASSR and the binaural beat ASSR are significantly greater than 40 Hz oscillation amplitudes in the inter-stimulus intervals and controls ( $P < 0.001$  for all four comparisons).

Changes in mental state may have occurred during the relatively long times separating stimulus and control periods. We therefore, expected variability in 40 Hz background oscillation amplitudes. Indeed, we occasionally found significant differences between 'interval' and 'control' amplitudes, as shown in the examples of Figs. 3 and 4: interval amplitudes could be either smaller or greater than control amplitudes, without significant or consistent preference of either possibility. Thus, we found no evidence for stimulus effects in the interval beyond 200 ms after stimulus termination.

### 3.3. Topographic amplitude distribution

In both the acoustic *beat* ASSR and binaural beat ASSR configurations, amplitudes of regression sinusoids during stimulus epochs significantly exceeded the amplitudes in interval and control epochs at several electrode locations. In the example of Fig. 3 this difference was highly significant ( $P < 0.001$ ) at all mid-sagittal recording sites, under both monaural and binaural beat conditions. This pattern was typical for the acoustic beat experiments (Table 1, A). However, binaural beats produced significantly greater amplitudes at fewer electrodes in many subjects, usually at central and frontal locations (Table 1, B). Maximal

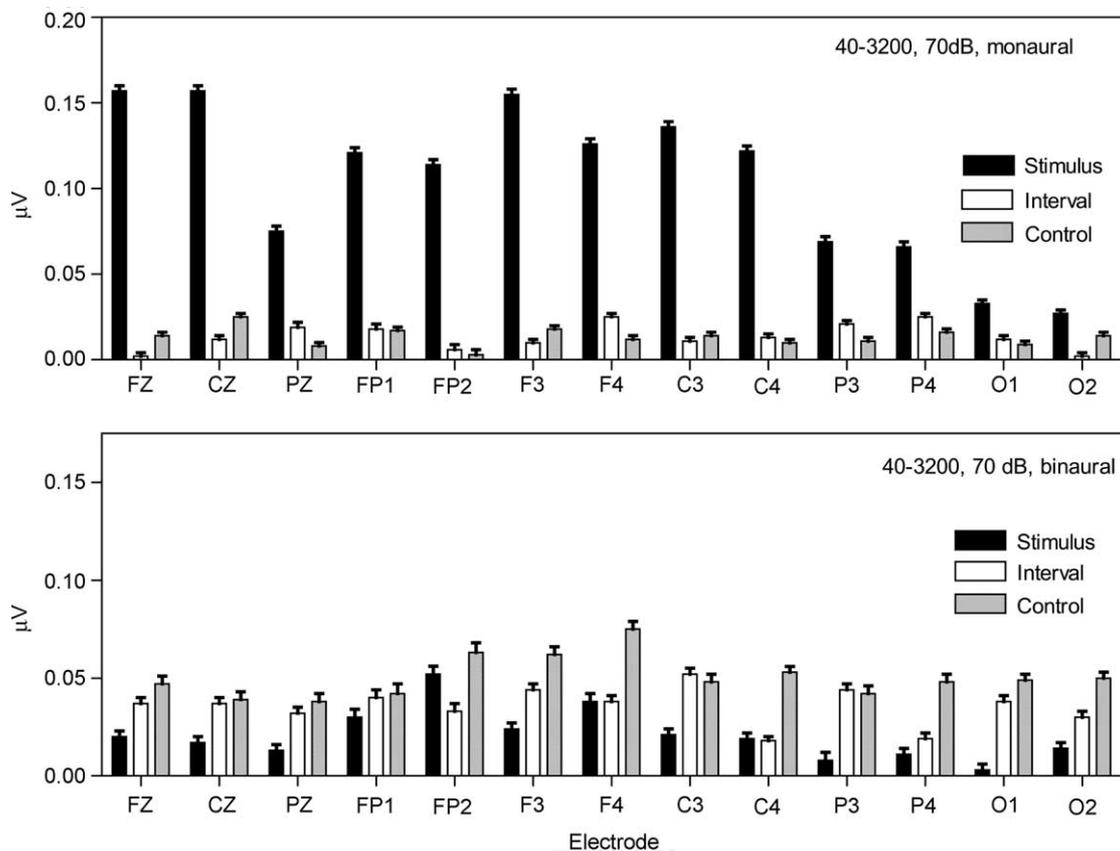


Fig. 4. High frequency beat ASSR. Monaural two-tone stimuli with a mean frequency of 3200 Hz cause a beat ASSR (top) with significantly greater stimulus-response amplitudes at all sites ( $P < 0.001$ ). No binaural beat ASSR is evoked by dichotic application of the same stimuli (bottom). Subject and error bar definition are the same as in Fig. 3.

Table 1  
Significance levels

	FZ	CZ	PZ	FP1	FP2	F3	F4	C3	C4	P3	P4	O1	O2
(A): Monaural, 40–400; 70 dB													
>0.05	1	0	0	2	3	0	1	0	1	0	0	1	2
<0.05	0	0	0	0	0	0	0	0	0	0	0	0	0
<0.01	0	1	1	0	0	0	0	1	0	0	1	0	0
<0.001	17	17	17	15	14	18	17	17	17	18	17	17	16
(B): Binaural, 40–400; 70 dB													
>0.05	6	0	4	13	12	8	8	3	4	6	6	12	11
<0.05	0	3	1	1	0	1	0	0	0	0	0	2	0
<0.01	0	1	0	1	2	0	3	1	1	2	0	2	2
<0.001	11	14	12	3	4	8	7	14	13	10	12	2	5
(C): Monaural, 40–3200; 70 dB													
>0.05	0	0	1	1	1	0	0	0	1	0	0	2	3
<0.05	0	0	0	0	0	0	0	0	0	0	0	0	0
<0.01	1	0	0	2	0	0	0	1	0	2	0	0	0
<0.001	3	4	3	1	3	4	4	3	3	2	4	2	1
(D): Binaural, 40–3200; 70 dB													
>0.05	3	4	4	4	3	3	4	4	4	4	4	4	4
<0.05	0	0	0	0	0	1	0	0	0	0	0	0	0
<0.01	1	0	0	0	1	0	0	0	0	0	0	0	0
<0.001	0	0	0	0	0	0	0	0	0	0	0	0	0
(E): Monaural, 40–400; 30 dB													
>0.05	0	0	1	2	2	0	0	0	0	2	2	2	4
<0.05	0	1	0	0	0	0	0	0	0	1	1	1	0
<0.01	0	0	1	1	0	1	0	0	1	1	0	2	0
<0.001	8	8	7	6	7	7	9	9	8	5	6	4	5
(F): Binaural, 40–400; 30 dB													
>0.05	5	5	5	4	5	4	5	2	6	5	5	7	5
<0.05	0	0	0	1	0	1	0	1	0	0	1	0	0
<0.01	0	1	1	0	1	0	0	0	0	0	0	0	0
<0.001	4	3	2	3	2	3	2	5	2	3	2	1	2

Cells show the probabilities that 40 Hz ASSR amplitudes were not different during stimulus and interval epochs for the 6 experimental conditions illustrated, as examples, in Fig. 3–5. Each cell shows how often the electrode in the column heading was characterized by the *P*-value range in the row heading. *P*-values are derived from Tukey–Kramer post tests of ANOVAs, which were computed for each condition and electrode, provided *F*-tests had indicated significant differences ( $P < 0.05$ ) between stimulus, interval and control epochs. Intervals represent the relevant controls since no systematic difference was found between interval and control epochs.

monaural-or binaural beat ASSR amplitudes were recorded consistently in the fronto-central region, usually at CZ, as in Fig. 3. Thus, steady state responses to beats produced by summation of sound waves in the external ear canal, or of temporal representations of these waves in the central nervous system, are widely distributed over the skull. However, the smaller binaural beat ASSRs are more readily masked by background EEG activity.

### 3.4. Frequency dependence of binaural beat ASSR

Our hypothesis assumed that binaural beats should not occur when the separate stimuli in each ear exceeded the upper frequency limit for temporal coding; such stimuli should not evoke a binaural beat ASSR. In contrast, the summation of two high frequency sound waves in one ear canal should still produce the beat pattern and an acoustic beat ASSR. We stimulated four subjects with two tonal stimuli of mean frequencies at 3200 Hz, leaving the difference frequencies at 40 Hz and intensities at 70 dB above component thresholds. Under these conditions

a strong *acoustic beat* ASSR was still present at all mid-sagittal electrodes (Fig. 4, monaural; Table 1, C), but there was no consistent evidence for a binaural beat ASSR (Fig. 4, binaural; Table 1, D). Since there is no reason to assume that 3200 Hz sound pressure waves are less efficiently conducted than 400 Hz tones through bone between the ears, these results provide evidence for an effective isolation of the stimuli in each ear, when presented through the insert earphones. We conclude that binaural beat ASSRs indeed depend upon tonal stimuli in the lower, temporally coded frequency range.

### 3.5. Sound intensity

Stimuli of low input frequencies (40–400 stimuli) should still evoke a binaural beat ASSR at low intensities, as long as stimulus response amplitudes exceed the background amplitudes in interval epochs. We compared, in nine subjects, monaural and binaural beat ASSRs with input tones that were 30 dB above component thresholds, in order to further exclude contaminations by inter-aurally

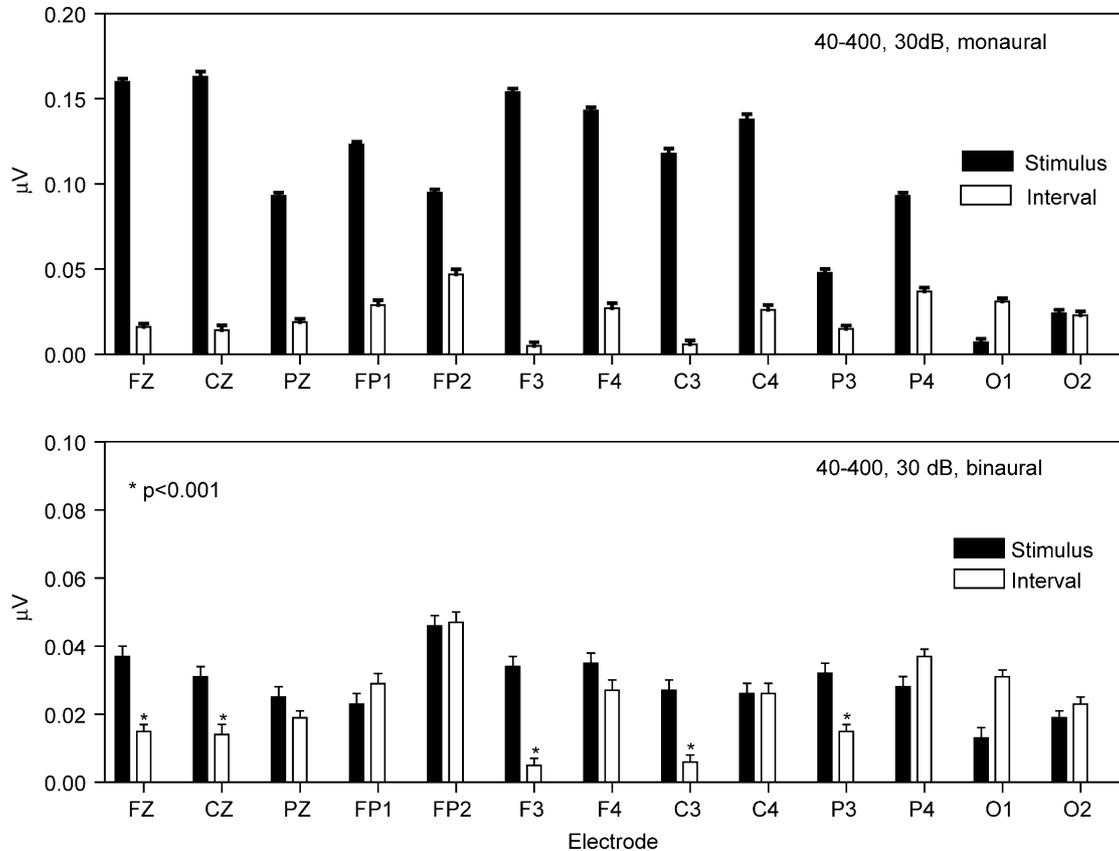


Fig. 5. Low intensity beat ASSRs. Supra-threshold component intensities of 30 dB are sufficient to evoke a *binaural beat ASSR* at a mean frequency of 400 Hz. Top: When both components are fed into the same ear (monaural) amplitudes are significantly greater at all except the occipital electrodes ( $P < 0.001$ ). Under dichotic conditions (binaural, bottom) stimulus amplitudes significantly exceed, in this example, interval amplitudes at 5 recording sites (marked by asterisks, which signify  $P < 0.001$ ). Error bars are defined as in Fig. 3.

conducted sound waves. Under these conditions acoustic *beat ASSRs* were still prominent at most electrodes. For example, amplitudes were significantly larger ( $P < 0.001$ ) for stimulus - than interval epochs in all but the occipital electrodes in Fig. 5 (monaural). However, in the binaural configuration only five electrodes yielded significantly greater stimulus amplitudes at these low intensities in this case (in Fig. 5). In all nine cases ASSR amplitudes significantly exceeded interval amplitudes at several electrodes during monaural (Table 1, E) as well as dichotic (Table 1, F) conditions. These data indicate that binaural beat ASSRs can be recorded with stimulus intensities that are too low for effective bone conduction of sound between the ears.

### 3.6. Differences between subjects

The binaural beat ASSR could not be recorded equally well at all electrodes. In Fig. 6, top, we show how often amplitudes were significantly greater during the stimulus- than the interval epochs, for all subjects, under 40–400–70 dB stimulus conditions. The only electrode providing evidence for a binaural beat ASSR in all cases was CZ. The least effective electrodes were at fronto-polar and occipital

locations, the former because of high background amplitudes, presumably originating from frontal muscles, and the latter because of small stimulus-response amplitudes. The corresponding significance levels at the different electrodes are listed in Table 1, B. In the lower panel of Fig. 6 we show global amplitude averages, of both significant *beat* - and binaural beat ASSRs, for all subjects along the mid-sagittal electrode array, again under 40–400–70 dB conditions. For acoustic *beat ASSRs* maximal averages occurred at FZ and CZ, without significant difference. Surprisingly, these electrodes did not record, on average, the largest amplitudes of binaural beat ASSRs, probably because binaural responses were more frequently masked by frontal background activity than monaural responses. The population-summary illustrated in Fig. 6 shows that it is possible to record monaural and binaural beat ASSRs from the entire mid-sagittal region of the head.

### 3.7. Phase shifts

It is reasonable to assume that a major source of the monaural-and binaural beat ASSRs is located in the auditory cortex, on Heschl's gyri within the Sylvian fissure. If there was only this one source of the oscillation we would expect

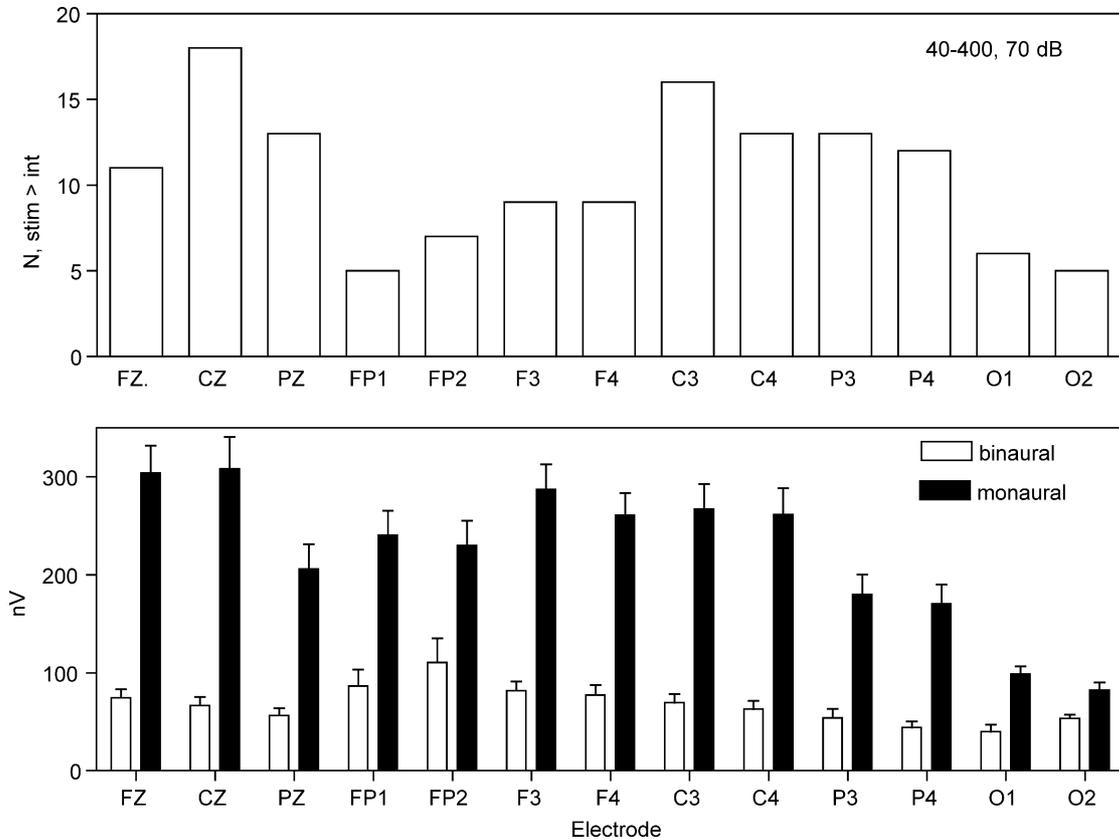


Fig. 6. Distribution of the *binaural beat* ASSR over the mid-sagittal scalp. Top: for each electrode the number of subjects is shown for which stimulus response amplitudes significantly exceeded interval amplitudes during dichotic application of the two tones (mean: 400 Hz; 70 dB). Bottom: grand averages and standard errors in the amplitude, defined by non-linear regression, of the *binaural beat* ASSR (binaural) and *acoustic beat* ASSR (monaural) at various recording sites.

the amplitudes to vary across the antero-posterior scalp dimension, as in Figs. 3–6, however, phases should remain relatively constant. We compared the phases of the 40 Hz regression sinusoids derived from records during the stimulus epochs at the various electrode locations. Several such regression curves are shown superimposed in Fig. 7, (top). A systematic fronto-occipital phase shift is evident in both the monaural (top left) and binaural beat ASSRs (top right). Frontal oscillation phases lead occipital phases by  $\sim 3$ –7 ms and regression sinusoids at intermediate locations exhibited an orderly, sequential phase progression in an anterior to posterior direction. We recorded such an orderly phase progression from locations at which stimulus response amplitudes were significantly greater than oscillation amplitudes during intervals or controls, provided the regression curve amplitudes were larger than  $\sim 50$  nV. The orderly phase progression tended to be obscured in cases of low beat ASSR signal to background noise ratios, presumably due to phase interaction of the relevant and irrelevant regression curves. In Fig. 7, (bottom) we plotted the mean phases and 95% confidence intervals at a greater number of electrode positions for one exemplary beat ASSR. The 95% confidence intervals tended to overlap for electrodes at similar levels along the anterior–posterior axis,

but not at widely different levels, illustrating significant phase differences between anterior and more posterior recording sites. The phase shifts suggest that oscillating neuron networks at different locations contribute to the acoustic and binaural beat ASSRs.

## 4. Discussion

### 4.1. Beat ASSR and beat sensation

Binaural beats originate, presumably, in the same neural circuits of the auditory brainstem that compute sound direction from an inter-aural time difference (Kuwada et al., 1979). If the tone presented to one ear differs in frequency from the tone in the other ear by less than  $\sim 2$  Hz the sound appears to move in the head from side to side. At greater frequency difference the perceived tone fluctuates in loudness instead (Moore, 2003), producing similar beating vibrato sensations during binaural and monaural presentation of the two tones. The vibrato character becomes less distinct at the relatively large frequency difference of 40 Hz used in our study, which induces a sensation of a rough or warbling tone in most listeners. Because the two

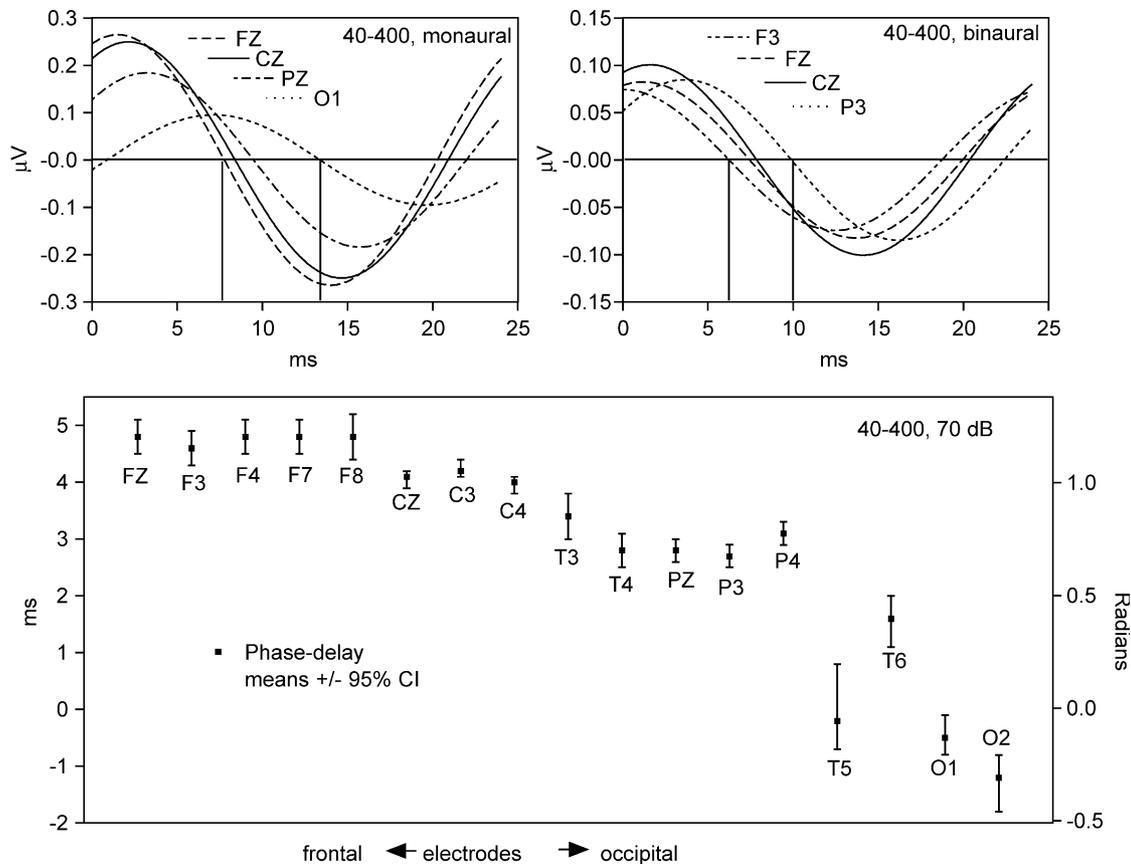


Fig. 7. Frontal-to-occipital phase shift. Top: Regression curves (cf. Fig. 2) at various recording positions are superimposed during stimulation with both tones in the right ear (left plot, monaural) and with one component in each ear (right, binaural). The lines from zero crossings to the time axis illustrate the delays between the most rostral to the most occipital of these electrodes. Different line patterns identify different electrodes in each graph. Bottom: Mean regression curve phase angles (right ordinate) and relative delays (left ordinate), with their 95% confidence intervals, are plotted for a greater number of electrodes arrayed in an approximate frontal-to-occipital sequence.

components of our standard 40–400 stimuli are separated in frequency by slightly less than one whole tone of the well-tempered musical scale, an alternate impression of two tones in disharmony may occur. A few subjects in our series, recruited from the music department of our university, initially attempted to assign a musical interval to the two tones in different ears and thus attended to the two input tones rather than one beating combination tone; while they did, we were unable to find evidence for a binaural beat ASSR. When the same subjects attended to the one warbling tone instead, binaural ASSRs were evident. Interestingly, the same subjects produced strong ASSRs under monaural, acoustic beat conditions, when a distinction of the two tonal components appeared much more difficult. Musicians may be better able to control ASSR generation in their enlarged auditory cortex (Schneider et al., 2002) than untrained people. An elucidation of the effects of attention and musical training on the magnitude of mono- and binaural beat ASSRs will require new measurements following a different experimental paradigm.

We chose the 40 Hz frequency difference as optimal stimulus for binaural beat ASSR generation, in spite of the relatively indistinct ‘beat’ sensations it evokes. Steady state

responses are best generated with oscillations around 40 Hz because at that rate middle latency evoked responses may be optimally summed (Galambos et al., 1981; Galambos and Makeig, 1988; Plourde et al., 1991; Stapells et al., 1988) and the cortex has a tendency to produce gamma oscillations, around 40 Hz, during conscious attention and perception (Basar et al., 1987; Singer and Gray, 1995). We expected to record larger monaural beat ASSRs with an average frequency of 3200 Hz where the 40 Hz difference amounts to much less than a musical half tone and the two components were not heard distinctly. However, there was a statistically insignificant trend toward larger ASSR amplitudes at average frequencies of 400 Hz in the small population of subjects tested with both stimuli.

#### 4.2. The dichotic stimuli

The *binaural beat* ASSR depends upon a combination of the two separate tonal stimuli within the central nervous system (CNS). In contrast to previously reported steady state responses, the gamma frequency (e.g. 40 Hz) signal is not present in an ear as repetition- or modulation rate of a peripheral sensory stimulus. The two different pure tone

stimuli were isolated in the two ears by insert ear-phones that attenuate the sound in a contra-lateral, un-stimulated ear by more than 70 dB, as measured with a calibrated probe microphone. Thus, at a stimulus intensity of 70 dB above component threshold an effective summation of air pressure waves, combining stimuli from both ears, did not occur. This does not rule out a summation, within the cochlea, of sound waves that are conducted from the opposite ear through bone and fluids. However, in healthy subjects with intact external and middle ears bone conduction plays a negligible role in binaural hearing (Békésy, 1932). Conduction between ears through these media is attenuated by at least 40–60 dB. An addition of a contra-lateral tone 40 dB below the ipsi-lateral tone level would not perturb neuronal phase locking sufficiently to affect directional hearing (Blauert, 2001) or binaural beat patterns. In fact, monaural, acoustic beats are inaudible when intensities between both component tones differ by 40 dB. Thus, signal summation within the CNS would appear to be essential for the generation of binaural beat ASSRs to tonal stimuli 70 dB above component thresholds, as they certainly are when components intensities are lowered to 30 dB.

The binaural beat ASSR is frequency dependent. The binaural beat sensation is distinct, for example, at 300–600 Hz but difficult to discern at frequencies beyond ~1 kHz (Licklider et al., 1950; Tobias, 1963). The reason is, that the component sound waves must be temporally encoded, by phase-locked spike firing in the MSO input, where the index (vector strength) of phase locking (Goldberg and Brown, 1969) decreases with frequency above 1 kHz toward zero at ~5 kHz in mammals. Using input frequencies with a mean of 3200 Hz we found no clear evidence of the binaural beat ASSR, at 70 dB, in the same subjects and sessions in which we recorded strong acoustic, monaural *beat ASSRs* with the same component frequencies. Since sound conduction through the skull does not dramatically improve at the lower frequencies there is no reason to assume that, during dichotic stimulation, a summation of the sound pressure waves in an ear occurs more readily at frequency means of 400 than at 3200 Hz. The fact that, in the same recording sessions, binaural beat responses were strong at 400 Hz, but not at 3200 Hz, argues in favor of a summation of most, if not all, of the sound wave signals within the CNS. At ideal signal/noise ratios the binaural beat ASSR detection limit could exceed 3200 Hz since phase locking extends to ~5 kHz, as do human listening skills that depend on it, such as spatial hearing (Blauert, 2001), vowel recognition or musical pitch perception (Pijl and Schwarz, 1995). In fact, in three isolated cases we observed small differences between stimulus and interval epochs (Table 1, D), however, only at frontal electrodes, which tend to record large myogenic signals. In a clinical standardization of the binaural beat ASSR, the optimal stimulus level may be placed somewhere between 30 and 70 dB above component thresholds: at lower intensities the response will be difficult to detect and

at higher levels an acoustic, rather than binaural beat ASSR could be evoked. The latter possibility is unlikely, however, because acoustic (monaural) beats become undetectable at large intensity differences between components, in contrast to the binaural beat, which is relatively resistant to intensity differences (Gu et al., 1995; Tobias, 1963).

#### 4.3. *Origins of beat ASSRs*

The auditory cortex in Heschl's Gyri probably contains neural sources of both the acoustic - and binaural ASSR. Models of a representation on the superior temporal plane have been able to accommodate the scalp distribution of other ASSR types (Herdman et al., 2002; Johnson, 1988). A systematic phase shift in our recordings, consistent with a fronto-occipital delay of several ms, raises the possibility of more than one dipole source along the rostro-caudal axis. A contribution of several cortical areas might be plausible because the perception of the beat requires selective attention to the vibrating sound, similar to the attention required for recognition of vowels, musical tone complexes, Fourcin's pitch (Fourcin, 1970) or Rubin's picture. Thus, the phase delay could reflect a sequential recruitment of cortical regions. A similar rostro-caudal phase shift of gamma waves has been reported (Ribary et al., 1991). Our phase data could, however, also be produced by two or more dipoles with different orientations in auditory cortex fields and in the brainstem (cf. Herdman et al., 2002).

#### 4.4. *Clinical implications*

The binaural beat ASSR assesses the ability of auditory neurons to temporally encode sound, by phase locking. This ability is of clinical interest as higher auditory functions depend on it, such as speech and musical pitch recognition, directional hearing and detecting signals in noise. Neurological diseases would be expected to perturb the temporal code if they interfere with the timing of action potentials. Examples for pathogenic factors could include demyelination, inflammation and agents or conditions affecting ion conduction, e.g. through certain Na<sup>+</sup> and K<sup>+</sup> channels. Thus, the binaural beat ASSR may enrich the diagnostic repertoire of the EEG. It reflects the generation, by neural mechanisms in the brain, of a sensory signal that is not present in the peripheral sensory organ as stimulus parameter.

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