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Comparison of binaural auditory brainstem responses and the binaural difference potential evoked by chirps and clicks

Helmut Riedel *, Birger Kollmeier

AG Medizinische Physik, Universität Oldenburg, D-26111 Oldenburg, Germany

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Abstract

Rising chirps that compensate for the dispersion of the travelling wave on the basilar membrane evoke larger monaural brainstem responses than clicks [Dau et al., J. Acoust. Soc. Am. 107 (2000) 1530–1540]. In order to test if a similar effect applies for the early processing stages of binaural information, monaurally and binaurally evoked auditory brainstem responses were recorded for clicks and chirps for levels from 10 to 60 dB nHL in steps of 10 dB. Ten thousand sweeps were collected for every stimulus condition from 10 normal hearing subjects. Wave V amplitudes are significantly larger for chirps than for clicks for all conditions. The amplitude of the binaural difference potential, DP1–DN1, is significantly larger for chirps at the levels 30 and 40 dB nHL. Both the binaurally evoked potential and the binaural difference potential exhibit steeper growth functions for chirps than for clicks for levels up to 40 dB nHL. For higher stimulation levels the chirp responses saturate approaching the click evoked amplitude. For both stimuli the latency of DP1 is shorter than the latency of the binaural wave V, which in turn is shorter than the latency of DN1. The amplitude ratio of the binaural difference potential to the binaural response is independent of stimulus level for clicks and chirps. A possible interpretation is that with click stimulation predominantly binaural interaction from high frequency regions is seen which is compatible with a processing by contralateral inhibitory and ipsilateral excitatory (IE) cells. Contributions from low frequencies are negligible since the responses from low frequencies are not synchronized for clicks. The improved synchronization at lower frequencies using chirp stimuli yields contributions from both low and high frequency neurons enlarging the amplitudes of the binaural responses as well as the binaural difference potential. Since the constant amplitude ratio of the binaural difference potential to the binaural response makes contralateral and ipsilateral excitatory interaction improbable, binaural interaction at low frequencies is presumably also of the IE type. Another conclusion of this study is that the chirp stimuli employed here are better suited for auditory brainstem responses and binaural difference potentials than click stimuli since they exhibit higher amplitudes and a better signal-to-noise ratio.

Key words: Auditory brainstem response; Binaural difference potential; Chirp; Basilar membrane dispersion; Frequency specificity

1. Introduction

The properties of the traveling wave along the basilar membrane are such that the activation maximum for higher frequencies occurs earlier than that for lower frequencies (von Békésy, 1960; Greenwood, 1990). From this dispersion it follows that an acoustic click stimulus is no longer synchronized after passing the inner ear. To compensate for the dispersion on the basilar membrane a chirp with rising instantaneous frequency was developed by Dau et al. (2000). They demonstrated that a rising chirp stimulus evokes a larger response than an equally loud click for monaural stimulation. This effect can be well understood by the enhanced neural synchronization obtained by the chirp especially for low frequencies, i.e., below 1 kHz.

Binaural interaction in auditory brainstem responses (ABR) is commonly analyzed in terms of the binaural difference potential (BD), i.e., the difference between the evoked responses to binaural and summed monaural stimulation, symbolically BD = B−(L+R) (Levine, 1981; Furst et al., 1985, 1990; Ito et al., 1988; Jones

* Corresponding author. Tel.: +49 (441) 7983528; Fax: +49 (441) 7983698.
E-mail address: hr@medi.physik.uni-oldenburg.de (H. Riedel).

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and van der Poel, 1990; Levine and Davis, 1991; Jiang, 1996; Brantberg et al., 1999a,b; Riedel and Kollmeier, 2002). The BD is thought to reflect the activity of neural units responding specifically to binaural stimulation. The amplitude of the summed monaural potential is usually found to be larger than the binaural response, i.e., the BD has an inverted polarity compared to the binaural response. At least two mechanisms can be thought to cause this reduction. (i) Contralateral inhibitory and ipsilateral excitatory (IE) cells (Goldberg and Brown, 1969) in the superior olive (SO) exhibit a reduced response to binaural stimulation. (ii) Contralateral and ipsilateral excitatory (EE) cells are driven (near) to saturation by monaural stimulation and cannot double their response for binaural stimulation. For clicks, the amplitude of the most prominent peak pair DP1–DN1 is about a fifth of the amplitude of wave V for a wide range of stimulus levels (Levine, 1981). Because the noise variance of the BD is about three times the variance of the monaural response the signal-to-noise ratio (SNR) of the BD is about an order of magnitude smaller than the SNR of the binaural response. Click evoked BD amplitudes barely exceed 0.2 μV. Therefore, it is highly desirable to provide methods which augment the SNR of the BD. An increased dynamic range of the latter could expand the possible experimental setups used to study the correlation between spatial stimulation and the corresponding BD.

In the present study we investigate whether a larger binaural potential and a larger BD (with higher SNR) can be obtained with a chirp signal in comparison to the traditionally used clicks. Furthermore, the amplitude ratio of the BD to the binaural response is analyzed as a function of stimulus level. This ratio makes it possible to draw conclusions about the cell types involved in the generation of the BD.

2. Methods

2.1. Subjects

Ten subjects, two females and eight males, aged 25–36 years participated in the experiments. They were either paid or volunteers from the staff of the University of Oldenburg. They were classified as normal hearing by routine audiometry and had no history of audiological or neurological problems. The audiometric loss was less than 10 dB for frequencies below 4 kHz and less than 15 dB for the higher frequencies.

2.2. Stimuli

Stimuli were generated digitally, downloaded to a DSP32C card in the host computer, and DA converted at a sampling rate of 50 kHz. The click was a sequence of five constant samples, and was converted to a rectangular voltage pulse of 0.1 ms duration. The spectrum of the chirp was approximated to the flat spectrum of the click by attenuating it at the lower frequencies. The flat spectrum chirp with edge frequencies of 0.1 and 10 kHz had a duration of 10.32 ms (Dau et al., 2000). Acoustic waveforms and spectra as measured with a fast Fourier transform (FFT) analyzer (Stanford Research SR780) are shown in Fig. 1. The time between two subsequent stimulus onsets was chosen to vary randomly and was equally distributed between 62 and 72 ms yielding an average stimulation rate of approximately 15 Hz.

A 700 ms segment of the stimuli comprising 11 clicks or chirps was used to determine the thresholds in quiet. They were measured three times by all subjects with a three-alternative forced-choice method in conjunction with a two-down–one-up scheme for both ears and averaged over runs, subjects and ears. The threshold level – referred to as 0 dB normal hearing level (nHL) – corresponded to 40.5 dB peak equivalent sound pressure level (peSPL) for the click and 37 dB peSPL for the chirp[1]. The standard deviation of the individual thresholds from the averaged thresholds was 2.8 dB for the clicks and 3.6 dB for the chirps.

2.3. Recordings

Ag/AgCl electrodes were used for the recordings. Three active channels were placed at the left (A1) and right (A2) mastoid as well as 1 cm below the inion (Iz). The common reference electrode was placed at the vertex (Cz), the ground electrode at the forehead (Fpz). Electrode labels are according to the 10-20 system (Jasper, 1957).

Electrode impedances were measured at a test signal frequency of 30 Hz and brought well below 5 kΩ, common values were 2–3 kΩ. Since DC recordings were performed, a second criterion for a good contact between electrodes and skin beside low impedance was the voltage drift seen in the raw EEG signal. Electrode contact was improved until any drift vanished.

During the ABR recordings, subjects lay in a sound insulated and electrically shielded room. They were instructed to relax and lie as comfortably as possible. ABRs were recorded with a DC-coupled differential amplifier (Synamps 5803). Inside the shielded room the EEG was preamplified by a factor of 150, further

[1] A sinusoid of frequency 1 kHz with the same peak-to-peak amplitude as the chirp showed 37 dB SPL in a Brüel and Kjær (B&K) amplifier type 2610. The calibration was performed using a half inch microphone (B&K 4157) with an artificial ear (1.29 cm^3) and a preamplifier (B&K 2669).

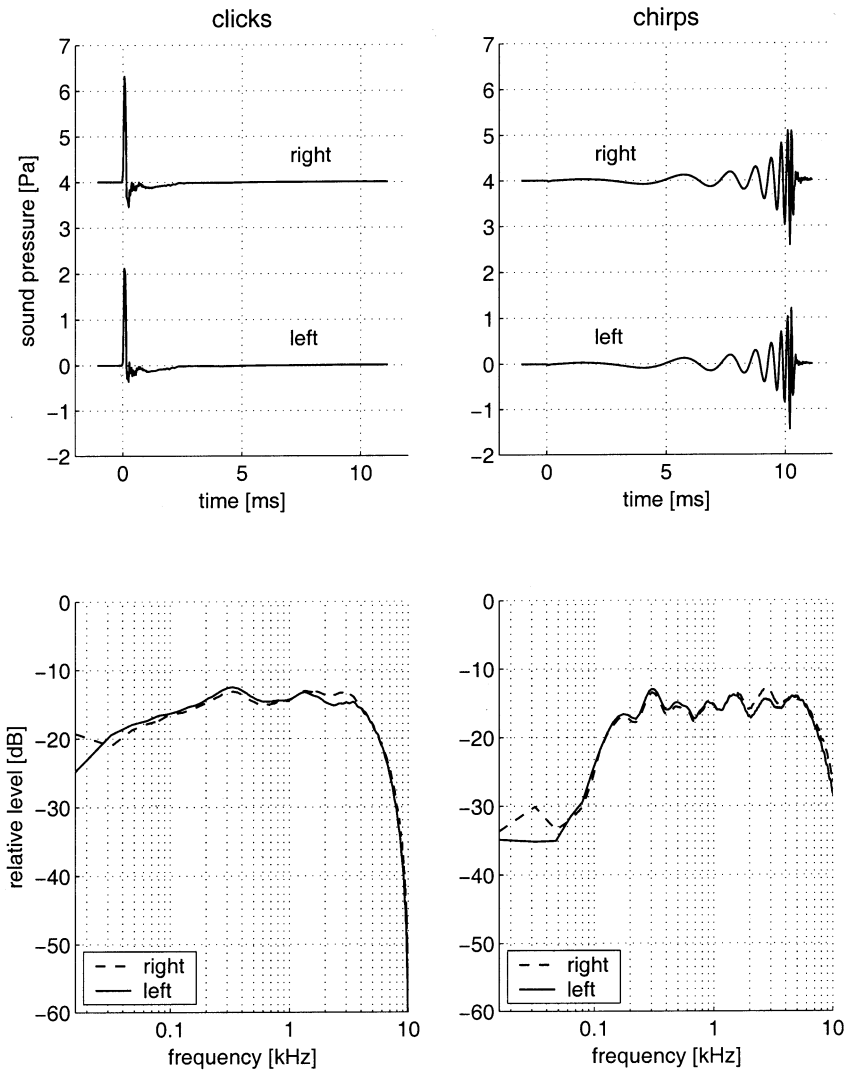

Fig. 1. (Top row) Acoustic waveforms of the click (left panel) and the chirp (right panel) measured at 60 dB nHL, corresponding to 100.5 dB peSPL for the click and 97 dB peSPL for the chirp. Right stimuli are plotted with an offset of 4 Pa. (Bottom row) Acoustic spectra of the stimuli using 625 FFT bins in steps of 80 Hz.

amplified by the main amplifier by a factor of 33 resulting in a total amplification of 74 dB. The voltage resolution was approximately 16.8 nV/bit. The sweeps were filtered by an analog anti-aliasing lowpass with an edge frequency of 2 kHz, digitized with 10 kHz sampling rate and 16 bit resolution, and stored to hard disk. The artifact level was set to ± 500 μV, since filtering, artifact analysis and averaging were done offline. The clipping level of the DA converters was ± 550 μV. The recording interval comprised 500 samples in the time interval from -15 to 35 ms relative to stimulus onset.

Left, right and binaural stimuli were presented randomly on a sweep-by-sweep basis. One run consisted of 15 000 stimuli, 5000 of each type, and lasted approximately 17 min. Two runs were performed for both stimuli (clicks and chirps) and six levels, 10–60 dB nHL in steps of 10 dB. The recordings were subdivided into four sessions with six runs each. Every session started with the highest level 60 dB nHL and successively lowered the level until 10 dB nHL was reached in the last run.

2.4. Data analysis

Before averaging the single sweeps were filtered with a linear phase FIR bandpass filter with 200 taps and edge frequencies 100 and 1500 Hz (Granzow et al., 2001). An iterated weighted average of the filtered sweeps was computed for all subjects and stimulus conditions. The residual noise of the averages was computed as the standard error of the single sweeps σ (Riedel et al., 2001).

For both stimulus types and all levels, the BD was computed channel-wise and sample by sample, symboli-

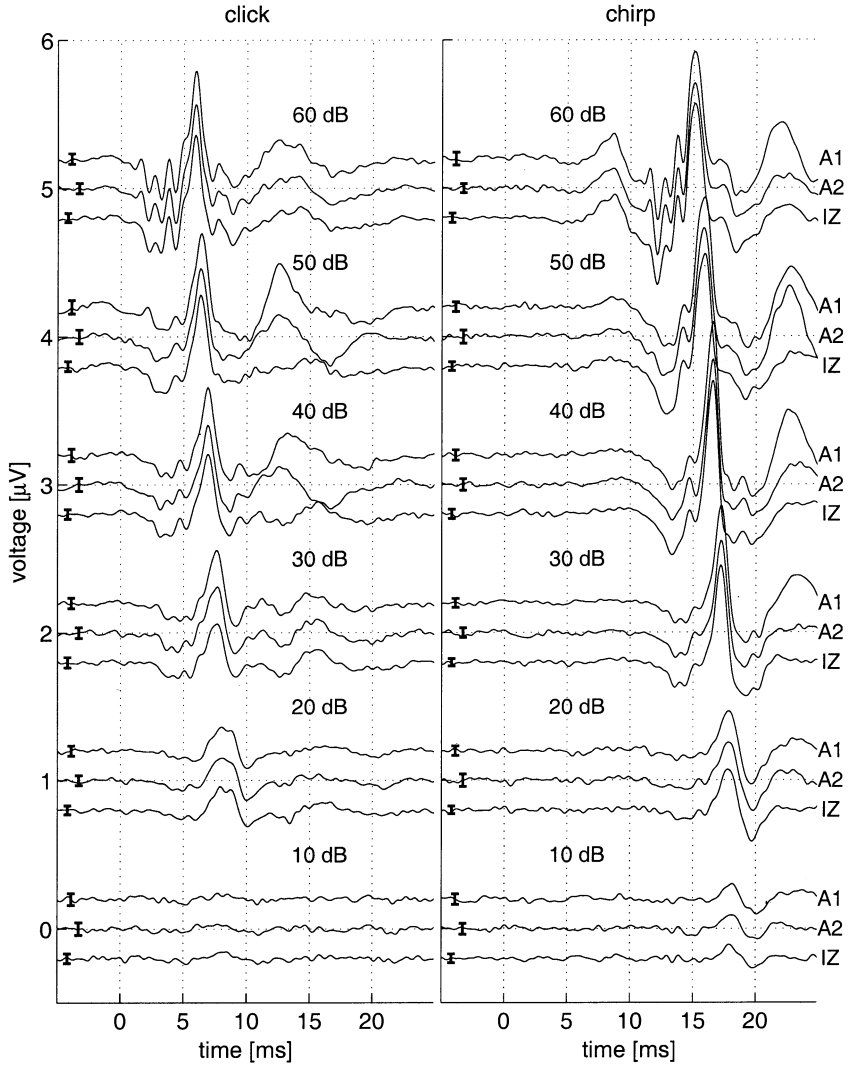

Fig. 2. Comparison of ABR evoked by diotic clicks and chirps for subject rh. Channels A1, A2 and Iz at levels from 10 to 60 dB nHL are shown. Plot offset between levels is 1 μV, between channels 0.2 μV. Error bars indicate ±3σ.

cally: BD = B−(L+R). All BDs were computed from response triplets which were recorded quasi-simultaneously. This avoids artifacts in the BD components due to long-term changes of the recording condition or subject's state. The residual noise of the BD was estimated as the square root of the summed variances of the three measurements: $\sigma_{BD} = (\sigma_B^2+\sigma_L^2+\sigma_R^2)^{1/2}$.

To increase the accuracy of amplitude and latency measurements, data were interpolated by a factor of 10, i.e., they were upsampled to convert the sampling rate from 10 to 100 kHz. This was accomplished by zero-padding in the spectral domain which in the time domain corresponds to a convolution with a sinc function. Since the original analog signal was band limited to frequencies below 2 kHz a near-perfect interpolation was possible.

Peaks in the interpolated signal were identified by a sign change in its derivative. For baseline-to-peak measurements peaks with voltages V_{bp} smaller than 3σ (99.7% confidence level for Gaussian measurement errors) were not regarded as significant and hence were discarded. For peak-to-peak measurements peaks with voltages V_{pp} greater than $\sqrt{2}\cdot 3\sigma$ were accepted. The additional factor of $\sqrt{2}$ is due to the fact that the variances of both peaks in the pair add up. Latency errors were estimated from the amplitude errors and the curvature of the peaks according to Hoth (1986).

Amplitude and latency of wave V were analyzed for each stimulus condition. The amplitude was measured baseline-to-peak because the peak-to-peak measurement from wave V to wave VI′ (the negative trough following wave VI) would yield erroneous amplitudes for three subjects exhibiting muscular artifacts in the auricular channels at the latency of wave VI′.

The first main component of the BD is the negative wave DN1 preceded by a smaller positive wave labelled

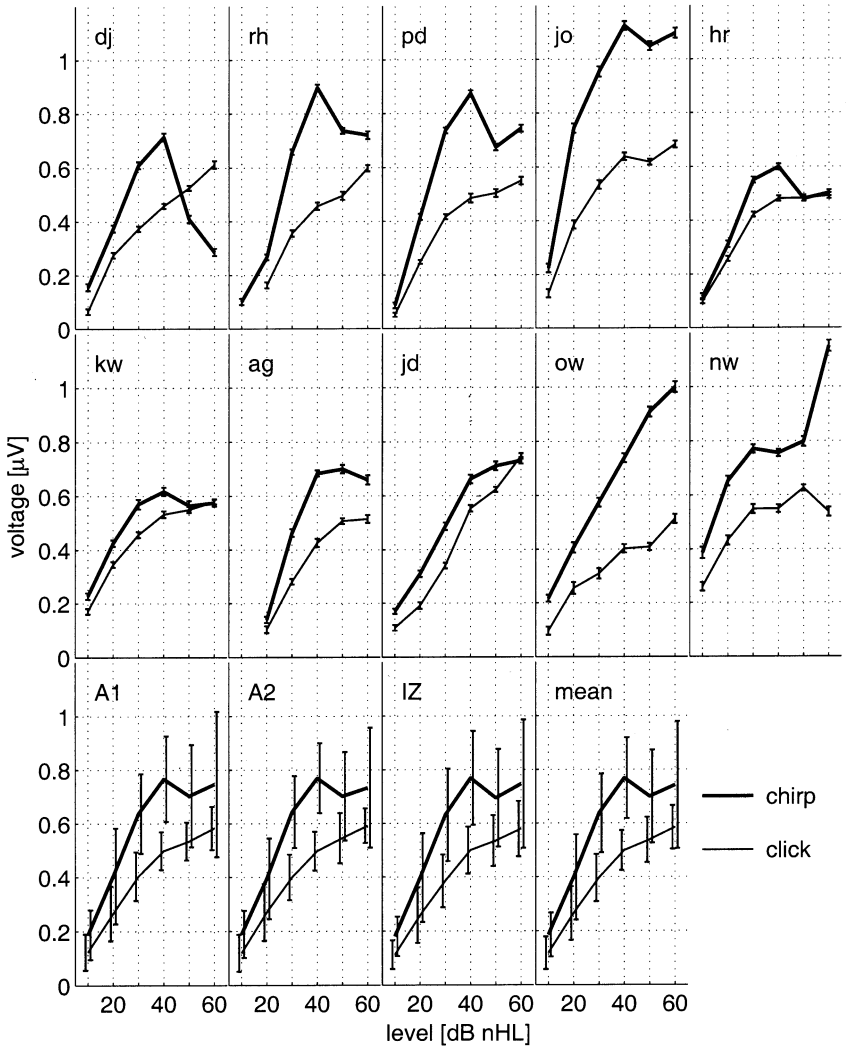

Fig. 3. Amplitudes of wave V as a function of the stimulus level, thick lines are for chirps, thin lines for clicks. (Top and middle row) Data for single subjects from channel A1 with intraindividual standard errors σ. (Bottom row) Data averaged over subjects with interindividual standard deviations, channels A1, A2, Iz and mean over channels.

DP1. The nomenclature introduced by Ito et al. (1988) is adopted here (see their fig. 1). DN1 corresponds to the β wave described by Levine (1981). BD amplitudes were measured peak-to-peak from DP1 to DN1. They are not contaminated by any muscle artifacts since the latencies of DP1 and DN1 are close to the latency of wave V. Latencies of the larger component DN1 were analyzed. Signed Wilcoxon rank tests were performed to reveal the significance of amplitude differences between clicks and chirps.

3. Results

Fig. 2 shows binaural potentials for the click (left) and the chirp (right) for levels from 10 to 60 dB nHL for one subject. For all three channels and all levels, the amplitude of wave V (A_V) is larger for the chirp than for the click. Moreover, for this subject the maximal response is found at 40 dB nHL for chirp stimulation. At 10 dB nHL there is still a visible response for the chirp, but not for the click. Relative to stimulus onset, the latency for the chirp response is about 10 ms larger compared to the click. At 60 dB nHL a positive deflection at about 8 ms latency is seen in the chirp response.

In the upper two rows of Fig. 3 the amplitude of wave V to diotic clicks and chirps is compared for channel A1 and all subjects. A_V for clicks increases with stimulation level, i.e., exhibits monotonic growth functions. In contrast, A_Vs for chirps are generally non-monotonic functions. They show a steeper increase in response than clicks up to 40 dB nHL. For higher levels there are interindividual differences: for the five subjects in the first row A_V for chirps decreases with increasing level, i.e., the maximal amplitude is reached at 40 dB nHL. For the subjects in the second row chirp ampli-

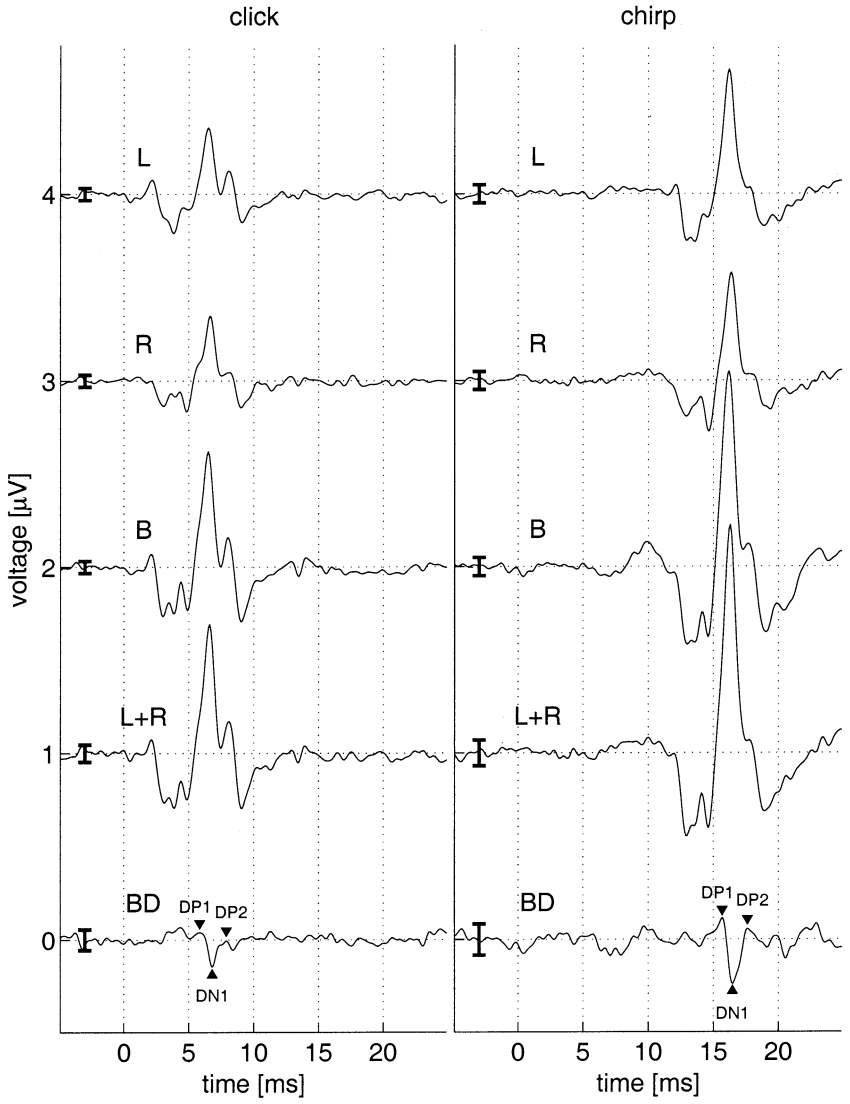

Fig. 4. Derivation of the BD. Data from subject jo for 50 dB nHL. (Top row) ABR to monaural left stimulation. (Second row) ABR to monaural right stimulation. (Third row) Binaural diotic response. (Fourth row) Sum of the monaural responses. (Bottom row) BD = B−(L+R). Error bars indicate ±3σ. Responses are plotted with an offset of 1 μV.

Table 1
Significance of the differences between amplitudes of wave V and of DP1–DN1 for clicks versus chirps as evinced by signed Wilcoxon rank tests

	Binaural wave V						BD wave DP1–DN1					
Level	10	20	30	40	50	60	10	20	30	40	50	60
A1	**	***	***	***	*	ns	ns	ns	*	*	ns	ns
A2	**	***	***	***	*	*	ns	ns	*	ns	ns	ns
Iz	**	***	***	***	*	ns	ns	ns	**	*	ns	*
Mean	**	***	***	***	*	*	ns	ns	**	**	ns	ns

Stimulus level in dB nHL. Three significance levels were tested: $\alpha < 0.05$ (*), $\alpha < 0.01$ (**) and $\alpha < 0.001$ (***); ns: not significant.

tudes level off at 40 dB nHL (kw, ag, jd) or increase even further with level (ow, nw). The small intraindividual standard errors (error bars) show that at 40 dB nHL A_V is larger for the chirp than for the click, for all subjects. In the bottom row grand average data over subjects are shown for the three channels measured as well as the mean over channels. The growth functions are very similar between channels. On the average over subjects, the chirp amplitudes reach their maximum at 40 dB nHL and level off for higher stimulation levels whereas the growth functions for clicks are monotonically increasing. To reveal the differences between amplitudes evoked by clicks and chirps signed Wilcoxon rank tests were performed for all channels and pairs of levels. The results of these tests are summarized in the left half of Table 1. For levels between 20 and 40 dB

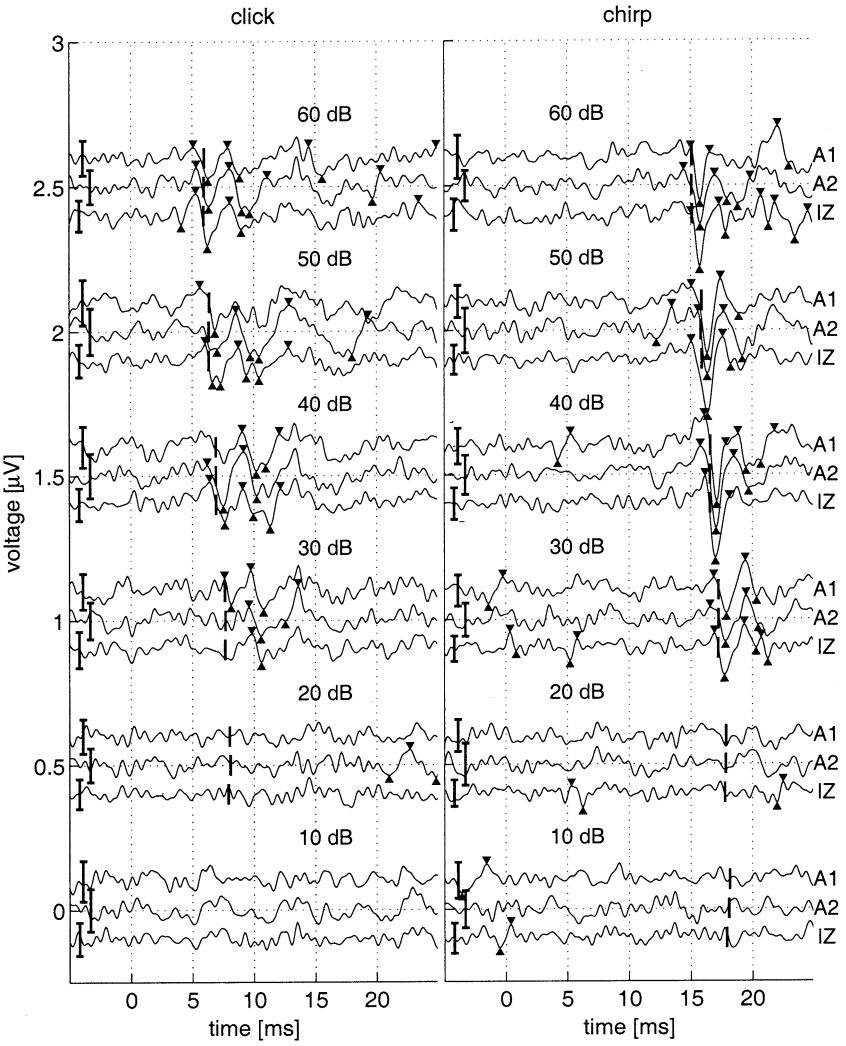

Fig. 5. Comparison of the BD evoked by diotic clicks and chirps for subject rh. Channels A1, A2 and Iz at levels from 10 to 60 dB nHL are shown. Plot offset between levels is 1 μV, between channels 0.2 μV. Error bars indicate $\pm 3\sigma$. Triangles indicate peak pairs with peak-to-peak voltages $V_{pp} \geq \sqrt{2}\cdot 3\sigma$. The vertical bars mark the latency of wave V for binaural stimulation.

nHL wave V amplitudes for chirps are larger than for clicks with high significance ($\alpha < 0.001$). For 50 dB nHL the differences are only significant at the level $\alpha < 0.05$. At 60 dB nHL only the differences for channel A2 and the mean over channels show a significant difference ($\alpha < 0.05$).

Fig. 4 shows the derivation of the BD from the binaural and monaural responses at 50 dB nHL for one subject. The error bars show $\pm 3\sigma$ corresponding to a 99.7% confidence interval for Gaussian measurement errors. The binaural responses (B) have slightly shorter latencies and smaller amplitudes than the sum of the monaural responses (L+R). This results in significant peaks in the BD, namely DP1, DN1 and DP2. DP1 and DN1 are associated with wave V of the binaural potential, DP2 with wave VI. In this example, the chirp BD has a larger amplitude than the click BD.

In Fig. 5 the dependence of the BD on stimulus type and level is depicted for one subject and all channels. Triangles are drawn for peaks whose peak-to-peak voltage V_{pp} exceeds $\sqrt{2}\cdot 3\sigma$. Only peaks satisfying this criterion are considered significant BD peaks. At the same stimulation level the chirp BDs are larger than the click BDs. Analogous to the chirp evoked binaural potential the growth function of the chirp BD is also steeper than for clicks. In this example the maximal peak-to-peak amplitude DP1–DN1 for chirps is found at 40 dB nHL.

Fig. 6 summarizes the peak-to-peak amplitude $A_{DP1-DN1}$ for all subjects and the mean over subjects as a function of stimulus type and level. Amplitudes of chirp and click BDs are marked by filled upward and open downward triangles, respectively. If for a certain stimulus and level $A_{DP1-DN1}$ failed to reach significance, no data are drawn. In analogy to Fig. 3 the first two rows show data for single subjects. There is a large variation in BD among subjects. However, similar to

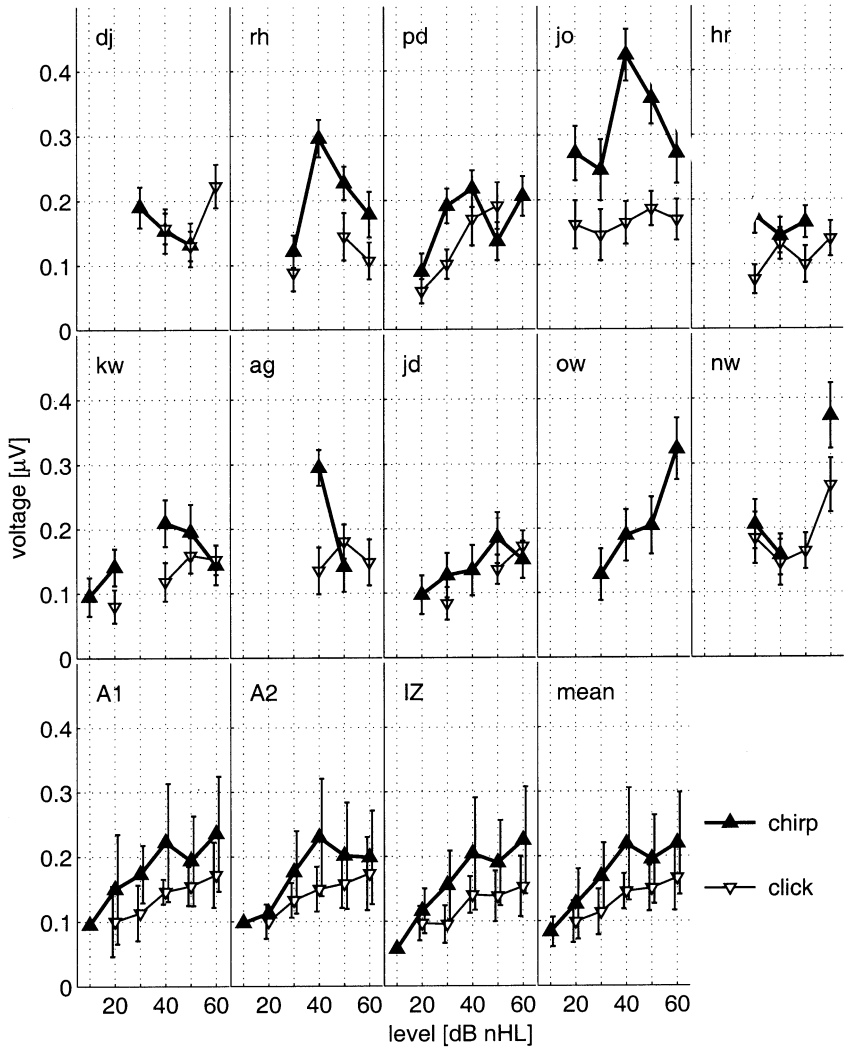

Fig. 6. BD amplitude $A_{DP1-DN1}$ as a function of the stimulus level. Thick lines with filled triangles indicate chirp amplitudes, thin lines with open triangles click amplitudes. (Top and middle rows) Data for single subjects from channel A1 with intraindividual standard errors σ. (Bottom row) Data averaged over subjects with interindividual standard deviations, channels A1, A2, Iz and mean over channels.

the binaural potentials, the chirp evoked BDs are generally larger than click evoked BDs. For one subject (ow) no BD to clicks could be detected at any stimulation level. For five subjects, the chirp BD is maximal at 40 dB nHL, for the other subjects chirp BDs level off or increase further. The grand mean data over subjects (see bottom row of Fig. 6) show that the dependence of $A_{DP1-DN1}$ on stimulus type and level is similar for all three channels measured: chirp BDs grow faster with level than click BDs and level off at 40 dB nHL.

Signed Wilcoxon rank tests were performed for all channels and pairs of levels to analyze the differences between BDs evoked by chirps and clicks. The right side of Table 1 shows that, with the exception of channel A2 at 40 dB nHL, chirp BDs are significantly larger than click BDs for 30 and 40 dB nHL. At 50 and 60 dB nHL, except for channel Iz at 60 dB nHL, no significant difference between chirp and click amplitudes is found. However, for many subjects there was no significant BD at some levels and channels either for chirps or for clicks, e.g., for subject ow there were only significant peaks in the BD for chirps. These unpaired data did not enter the above tests. If non-significant and undetectable peaks are considered to have amplitude zero, at 40 dB nHL significant differences result for all channels, and at 50 and 60 dB nHL no significant differences are found. This may be still be due to the small number of subjects ($n = 10$), but it shows that, on average, BDs to clicks grow for levels from 40 to 60 dB nHL whereas BDs to chirps saturate at 40 dB nHL. This results in a maximal difference at 40 dB nHL.

In Fig. 7 the latencies of wave V (t_V) are compared with the latencies of BD waves DP1 (t_{DP1}) and DN1 (t_{DN1}) as a function of the stimulus level. For both clicks and chirps, the latencies are ordered: $t_{DP1} < t_V < t_{DN1}$. The first two rows of Fig. 7 show single sub-

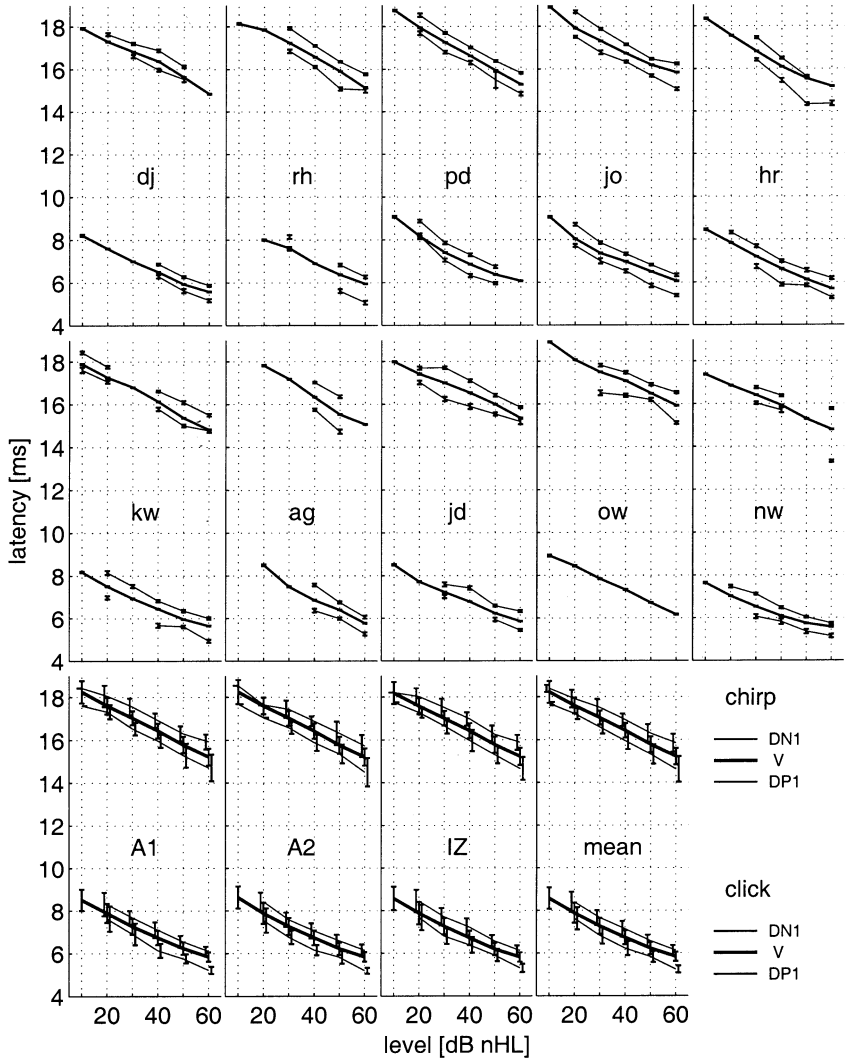

Fig. 7. Latencies of wave V (thick lines) and BD waves DP1 and DN1 (thin lines) as a function of the stimulus level. (Top and middle row) Data for single subjects from channel A1 with intraindividual standard errors. (Bottom row) Data averaged over subjects with interindividual standard deviations, channels A1, A2, Iz and mean over channels.

ject data. The error bars denote the intraindividual latency errors estimated from the intraindividual amplitude errors and the peak curvature according to Hoth (1986). In the bottom row mean latencies, averaged over subjects, are shown for the three channels as well as the average over channels. Here, error bars indicate the interindividual standard deviations of the latencies. Averaged over levels, subjects and channels, the latency difference for wave V between chirps and clicks amounts to 9.63 ± 0.14 ms. The mean time differences between DP1 and wave V amount to 0.44 ± 0.12 ms for clicks and 0.48 ± 0.08 ms for chirps. Compared to wave V, the mean delay of BD wave DN1 is 0.39 ± 0.08 ms for clicks and 0.43 ± 0.17 ms for chirps. These data indicate a strong dependence of BD latencies on wave V latencies.

In Fig. 8 the amplitude ratio of the BD and the binaural response is shown. For both stimuli and each level averages and standard deviations over 10 subjects and three channels are drawn. Except for 10 dB nHL stimulus level the amplitude ratio is nearly constant. Averaged over stimulus levels from 20 to 60 dB nHL $A_{\mathrm{DP1-DN1}}/A_{\mathrm{V}}$ is 0.28 for clicks and 0.27 for chirps. The literature value of one fifth is obtained by measuring wave V peak-to-peak to VI′, i.e., to the trough following wave VI: $A_{\mathrm{DP1-DN1}}/A_{\mathrm{V-VI'}}$ amounts to 0.20 for clicks and 0.19 for chirps. No amplitude measures were taken into account for the three subjects showing muscle artifacts in channels A1 and A2. At 10 dB nHL no amplitude ratio for clicks is given because the SNR of the BD was smaller than a triple standard error for all subjects. For chirps a higher amplitude ratio than for the other stimulus levels is found. However, the measurement of the amplitude ratio is more uncertain

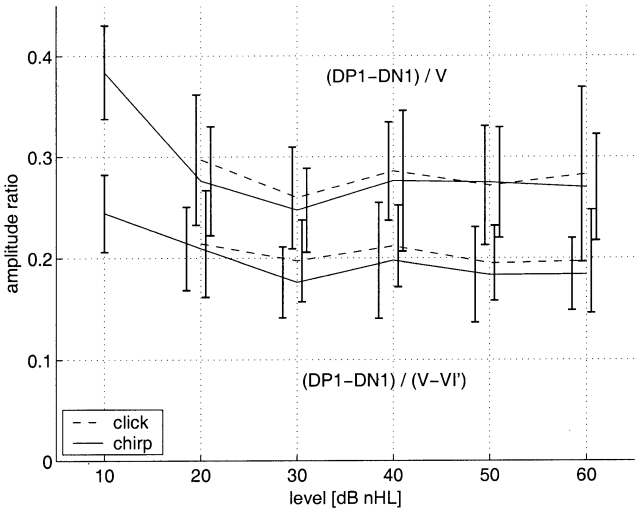

Fig. 8. Amplitude ratio of the BD to the binaural response, mean data and standard deviations over 10 subjects and three channels. Dashed lines are for clicks, solid lines for chirps. In the upper two curves wave V amplitude is measured baseline-to-peak, in the lower curves peak-to-peak from wave V to VI′ (the trough following wave VI).

at low stimulus levels due to the small amplitudes of both the binaural response and the BD.

4. Discussion

ABRs and BDs were measured using interleaved recording of left, right and binaural responses and averaging a large number of sweeps, in order to obtain a high SNR, for clicks and flat spectrum chirps. The results clearly demonstrate that chirps evoke larger binaural responses as well as larger BDs than clicks. Hence, the improved neural synchronization obtained with the monaural chirps (Dau et al., 2000) is also found for binaural chirps and propagates into an enlarged BD. This qualifies the chirp stimulus for further research work on binaural interaction with evoked potentials. However, the level range over which the chirp stimulus provides advantages over the click stimulus is limited to low and intermediate levels.

In the chirp response, a small positive deflection in the latency range from 7 to 10 ms was observed for all subjects for a stimulus level of 60 dB nHL (see, e.g., Fig. 2). This deflection is a consequence of a slight discontinuity of the chirp at time = 0 (Fig. 1, top right) caused by a shortcoming in the stimulus generation program. The magnitude of this onset transient was about −38 dB compared to the maximum amplitude of the stimulus. A control measurement using a smoothed version of the chirp was performed at levels 40 and 60 dB nHL for three subjects. No differences in the responses were detected for 40 dB nHL. However, at 60 dB nHL the early positive deflection disappeared, indicating that it was a response to the onset discontinuity. The onset transient did not mask the chirp response. Wave V amplitudes of the smoothed chirp tend to be slightly smaller compared to those obtained with the original chirp. This implies that the non-monotonicity of the chirp response could be even more pronounced, i.e., exhibiting a maximum at 40 dB nHL rather than levelling off.

The observation that the gain is largest at medium levels is consistent with the results of Dau et al. (2000), see their fig. 3. Their average monaural data showed a significantly larger wave V amplitude for the chirp than for the click at all levels. But only at medium levels (40 dB nHL) the difference was highly significant.

From animal studies it is known that the SO is the first stage at which binaural interaction occurs (e.g., Irvine, 1992). Goldberg and Brown (1969) classified the SO cells by the type of their input: IE cells predominantly found in the lateral SO receive contralateral inhibitory input via the medial nucleus of the trapezoid body and excitatory input from the ipsilateral cochlear nucleus (CN), they are believed to code the interaural level difference (ILD) at high frequencies. EE cells in the medial part of the SO obtain excitatory inputs from both CNs and are believed to code the interaural time difference (ITD) at low frequencies.

In evoked response studies the sum of the monaural responses is always found to be larger than the binaural response. According to Gaumond and Psaltikidou (1991) this can be explained by two mechanisms. (i) Contralateral inhibition in IE cells reduces the binaural response. (ii) EE cells are already saturated by monaural stimulation and can therefore not reach the summed firing rate of both monaural responses. Gaumond and Psaltikidou (1991) analyzed an EE and an IE model of binaural interaction. The constancy of the amplitude ratio $A_{\mathrm{DP1-DN1}}/A_{\mathrm{V}}$ across levels is easily explained by the IE model. Within the EE model special assumptions about the compressive non-linearity describing the saturation are needed to preserve the constant amplitude ratio. Therefore Gaumond and Psaltikidou (1991) conclude that IE cells play a larger role in BD generation.

From studies involving models of cochlear mechanics and evaluating the dispersion along the basilar membrane (e.g., de Boer, 1980; Dau et al., 2000) it is known that a click stimulus exhibits a high synchronization in hair cell deflection in the basal portion of the cochlea (corresponding to high frequencies) whereas the response to low frequencies shows a considerable temporal smearing of the deflection along different places on the basilar membrane. Hence, ABRs with clicks mainly reflect the response to high frequencies. Therefore it can be presumed that the BD measured in response to clicks represents to a larger extent the activity of the IE cells

processing ILDs than of EE cells processing ITDs. Consequently, contributions from EE cells due to saturation are not seen in the evoked response because the responses from low frequencies are not synchronized for clicks. This conclusion is in accordance with the results from Levine and Davis (1991). They measured the BD to clicks in the presence of a highpass noise masker and showed by means of the derived response technique that the BD is principally due to the high frequency components of the click.

The improved synchronization at lower frequencies using chirp stimuli yields contributions from both low and high frequency neurons augmenting the ABR and the BD. From comparing the relation of the binaural interaction and the monaural response between the click and the chirp stimulus it thus can be assessed if the lower frequencies contribute to the BD in a different way than the high frequencies. If one assumes that IE cells primarily produce the BD at high frequencies and EE cells are mainly active at low frequencies, this comparison can also be used to assess the relative contribution of both types of cell populations. Any contribution to the BD from EE cells must originate from a non-linearity after summation of the activities from the left and right side. Without such a non-linearity the contribution from EE cells would be zero. A positive contribution from EE cells to the BD, i.e., $B > L+R$, can only occur with an expansive non-linearity. However, an expansion after the summation is not very feasible physiologically. With the more plausible assumption of a compressive non-linearity describing the saturation (Gaumond and Psaltikidou, 1991) we obtain a negative contribution of the EE cells to the BD, i.e., $B < L+R$. Within this view both IE and EE cells would contribute to the BD in the same way. However, both types of non-linearity after the EE cells would alter the amplitude ratio $A_{DP1-DN1}/A_V$ as a function of the stimulus level contradicting the experimental findings. A compressive non-linearity would result in a smaller amplitude ratio at higher stimulus levels, an expansive non-linearity in a larger amplitude ratio. A possible interpretation is that binaural interaction in the ABR at low frequencies does not differ in principle from the one at high frequencies and is therefore predominantly of the IE type.

Another conclusion from the current data is that the rising frequency chirp constitutes a stimulus which makes it possible to analyze the BD with higher SNR and larger dynamic range than the conventional click stimulus. For example, the frequency specificity of the BD can be investigated by delivering chirps with different frequency contents to the two ears, e.g., a low frequency chirp to the left and a high frequency chirp to the right ear. Another useful application in future research will be the analysis of the correlation between the spatial position of a stimulus and its corresponding BD.

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