The Dependency of Chirp Evoked Binaural Difference Potentials on the ITD

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Abstract

Rising frequency chirps compensating for the dispersion of the traveling wave on the basilar membrane evoke larger monaural responses than clicks (Dau et. al. (2000) J. Acoust. Soc. Am. 107(3) 1530-1540). An analog finding holds for the binaural difference potential (BD), i.e., the difference between the evoked responses to binaural and summed monaural stimulation (Riedel and Kollmeier (2002) Hear. Res. 169 (1-2) 85-96). The BD is thought to reflect the activity of neural units in the brain stem responding specifically to binaural stimulation. In the present study the dependency of the BD on the interaural time difference (ITD) is analyzed. Chirp evoked BDs were measured for 17 ITDs in the range from 0 to 2 ms at a level of 40 dB nHL. To ensure a high signal-to-noise ratio, 10000 epochs were collected for each binaural condition, and 40000 epochs were recorded for left and right monaural conditions. In contrast to BD studies using click stimuli, considerable binaural interaction was found for ITDs larger than 1 ms. The peak-to-peak amplitude of the first components of the BD, DP1-DN1, is monotonically decreasing with ITD, again in contrast with click studies which reported a constant BD-amplitude for ITDs up to 1 ms.

Introduction

For more than half a century the prevailing paradigm to describe sound localization in the horizontal plane has been the model by Jeffress [5]. It uses a chain of coincidence detector cells which receive bilateral excitatory inputs in an ordered network of delay lines. The model provides a place code for the sound azimuth by means of the interaural time differences (ITDs) which are assumed to be represented by neural propagation delays in the auditory brain stem.
However, for cells in the medial superior olive (MSO) of the mammalian auditory system it was recently found that the best interaural phase difference (IPD) for binaurally sensitive cells is about one eighth ($\pi/4$) of the stimulus period for all stimulus frequencies, i.e., there is no distribution of IPDs (or ITDs) as required in the Jeffress model [8]. Furthermore, the best IPD occurs outside the physiological range. These data demonstrate that precise contralateral inhibition is needed and ITD coding is performed by a rate code rather than a place code [1].
Rising frequency chirps evoke a larger monaural ABR than clicks because they compensate for the dispersion along the basilar membrane [3]. While the evoked response to a click is predominantly created in basal high-frequeny regions of the basilar membrane, chirp stimulation results in synchronized activity of the entire cochlear partition. In evoked response studies, binaural interaction is commonly assessed in terms of the binaural difference potential, symbolically BD = B – (L + R) [4, 7, 2, 10, 11]. A comparative study demonstrated that larger BDs (with higher signal-to-noise ratio) can be obtained with a chirp signal in comparison to the traditionally used clicks [11], i.e., the advantage of larger chirp-evoked monaural ABRs in comparison to the click is also found for the BD.
If an ITD is applied to the stimulus, the classical Jeffress model using bilateral delay lines predicts a BD latency increase of ITD/2, a modified model using only one delay line results in a latency increase of ITD [12]. In the present study, the chirp-evoked BD is investigated for ITDs up to 2 ms. An alternative model using contralateral inhibition is proposed to explain BD amplitude and latency.

Methods

Recordings

- Stimulus: flat spectrum chirp at 40 dB nHL, see Fig. 1
- 17 ITDs: 0 to 1.5 ms in steps of 0.1 ms, and 2 ms
- 10000 sweeps were averaged for the binaural conditions, 40000 sweeps for the monaural conditions.
- Binaural difference potential BD = B – (L + R)
- 11 normal hearing subjects
- 4 electrodes (A1, A2, PO9, PO10)
- The residual noise was estimated on a single-sweep-basis as the standard error over the sweeps [9].

Model

- Adoption of the model by Ungan et al. [12] for cat
- BD generated by (multiplicative) contralateral inhibition
- χ^2-fit of 4 model parameters:
  1. difference between mean ipsilateral excitatory and contralateral inhibitory arrival time $t_e - t_i = 0.597$ ms
  2. standard deviation of the mean excitatory arrival time $\sigma_e = 0.631$ ms
  3. standard deviation of the mean inhibitory arrival time $\sigma_i = 0.629$ ms
  4. duration of the inhibition $\tau_i = 4.23$ ms

Stimuli

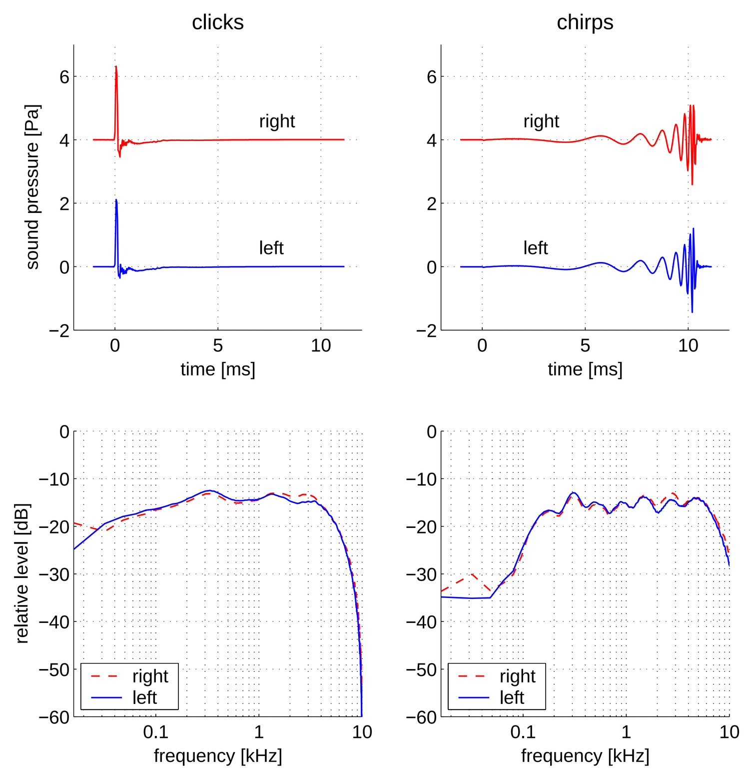

Fig. 1: Top row: Acoustic waveforms of a click with 0.1 ms duration (left panel, not used in this study and plotted only for comparison) and the chirp (right panel) measured at 60 dB nHL, corresponding to 100.5 dB peSPL for the click and 97 dB peSPL for the chirp, respectively. Right stimuli are plotted with an offset of 4 Pa. **Bottom row:** Acoustic spectra of the stimuli using 625 FFT bins in steps of 80 Hz. The envelope of the chirp was designed to result in a flat spectrum in the frequency range from 100 to 10000 Hz mimicking the spectrum of the click.

BD recording

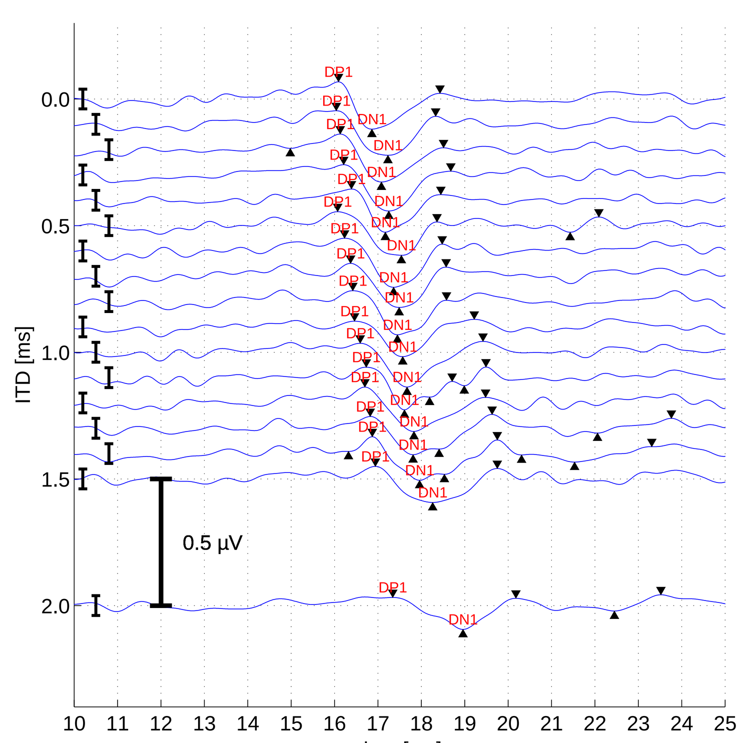

Fig. 2: BDs in dependence on the ITD: data from channel PO10, subject rb. The errorbars denote $\pm 3\sigma$ (± 3 S.E.M.). The triangles indicate peak pairs whose peak-to-peak values exceed $\sqrt{2}3\sigma$. The time axis is plotted relative to stimulus onset. Significant binaural interaction is found for all ITDs tested. The BD-peaks DP1 and DN1 occur approximately at the latency of the binaural wave V.

BD amplitude

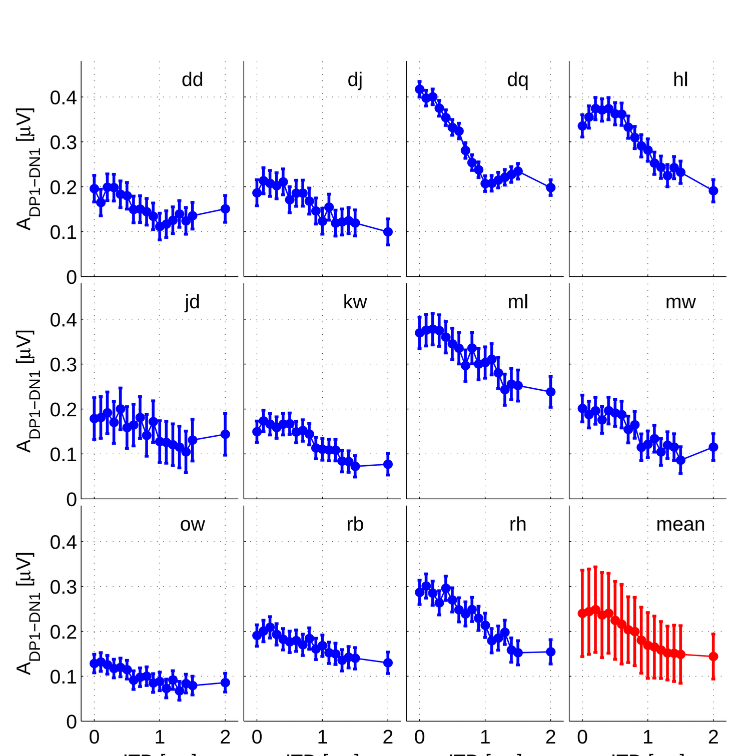

Fig. 3: Mean amplitude of BD-wave DP1-DN1 averaged over channels as function of the ITD. The first 11 subplots show single subject data, the errorbars indicate intraindividual standard errors ($\pm\sqrt{2}\cdot\sigma$). The last subplot depicts the mean over subjects, the errorbars denote ± 1 standard deviation. The BD amplitude decreases with increasing ITD. The maximum around an ITD of 0.2 ms for many subjects and the mean data is not significant. It is not due to inaccuracies in the stimulation system.

BD latency

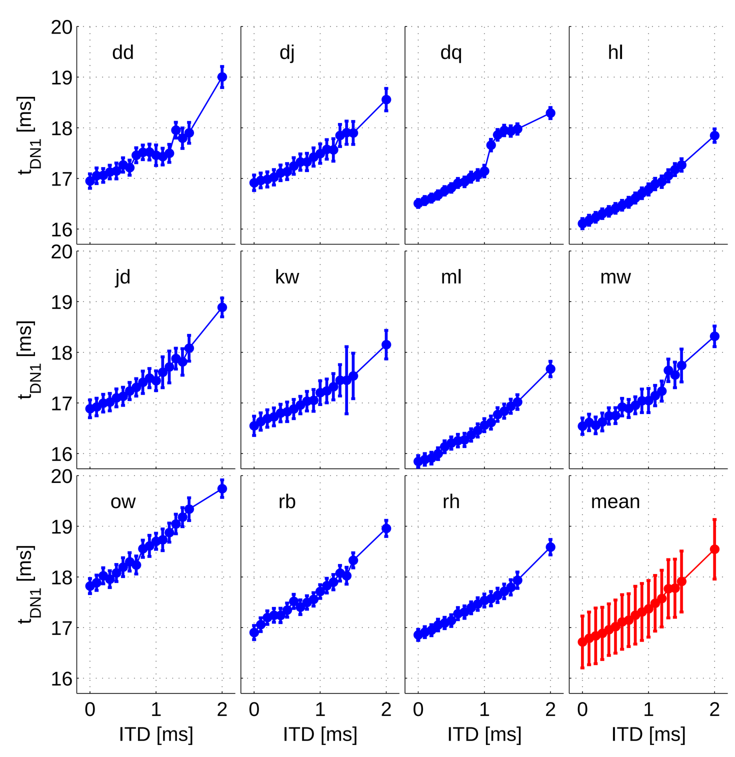

Fig. 4: Mean latency of BD-wave DN1 averaged over channels as function of the ITD. The first 11 subplots show single subject data, the errorbars indicate $\pm 3\sigma$ (± 3 S.E.M.). The last subplot depicts the mean over subjects, the errorbars denote ± 1 standard deviation. The shortest latency is always found for diotic stimulation, DN1 latency is monotonically increasing with increasing ITD. Latencies are measured from onset of the leading stimulus.

BD model

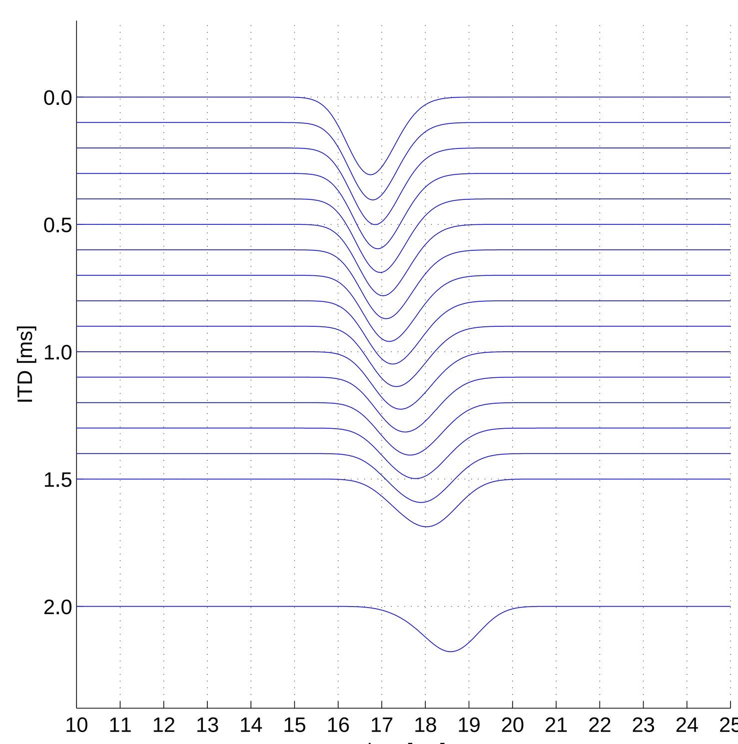

Fig. 5: Modeled BD waveforms as function of the ITD. Although the model cannot predict the small positive deflection DP1 preceding the main BD-peak DN1, measured and modeled waveforms look similar. The reduction of DN1 amplitude with increasing ITD is described correctly, see Fig. 6. The latency shift with increasing ITD is predicted properly, see Fig. 7.

Model of the BD amplitude

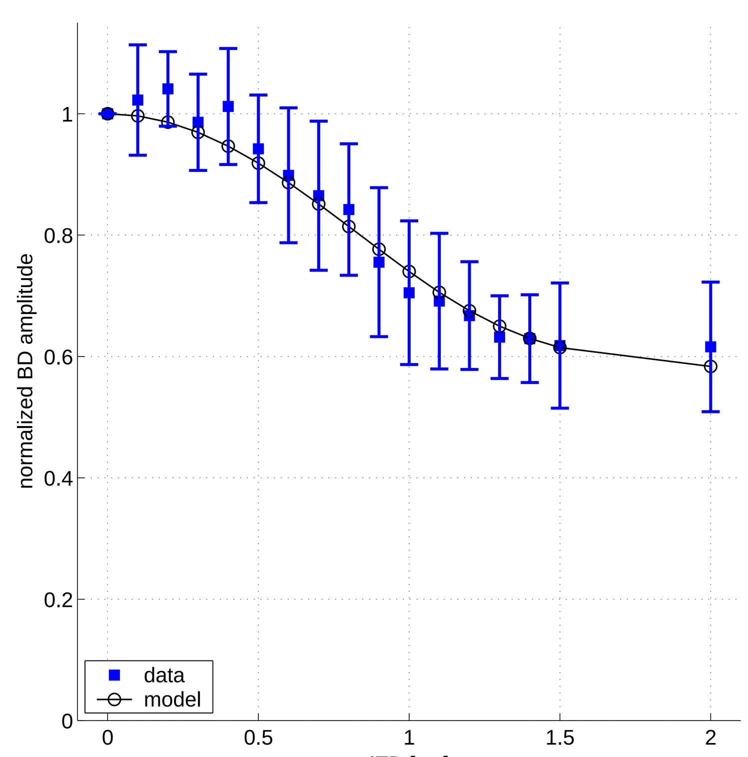

Fig. 6: Comparison of the normalized measured (blue squares with errorbars) and modeled (open black circles) BD amplitudes. The model seems to underestimate the measured amplitudes for small ITDs in the range up to 0.4 ms. In this range, the data appear to be relatively constant while the model predicts a monotonously decreasing BD amplitude. However, taking into account the standard deviation of the mean data over subjects, the model explains the data very well.

DN1 latency increase

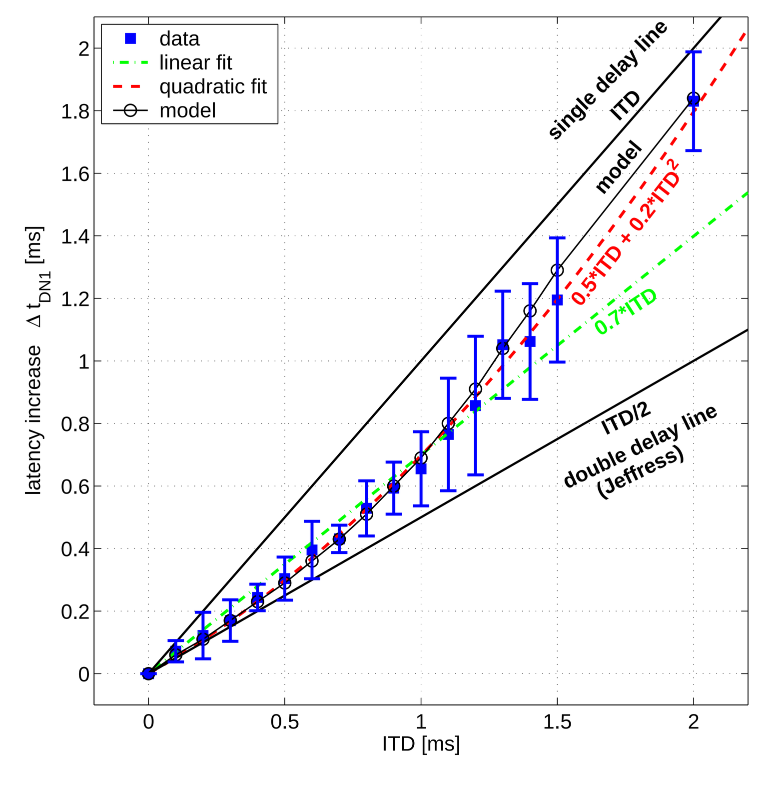

Fig. 7: Mean latency increase of BD-wave DN1 averaged over channels as function of the ITD ($\Delta t_{\mathrm{DN1}} = t_{\mathrm{DN1}} - t_{\mathrm{DN1,ITD=0}}$). Errorbars denote ± 1 standard deviation. The lower straight line indicates the latency due to the Jeffress model [6]. The upper straight line is for a modified Jeffress model using a single unilateral delay line [12]. The dash-dotted line is the outcome of a linear χ^2-fit (one parameter). The dashed line holds for a quadratic χ^2-fit (two parameters). Open circles stand for the model.

Results

- Significant BD for ITDs up to 2 ms (Fig. 2, 3)
- Decreasing BD amplitude with increasing ITD (Fig. 3)
- Increasing BD latency with increasing ITD (Fig. 4)
- BD latency increase $\Delta t_{\mathrm{DN1}} = t_{\mathrm{DN1}} - t_{\mathrm{DN1,ITD=0}}$ is between ITD/2 and ITD (Fig. 7).
- Linear χ^2-fit: $\Delta t_{\mathrm{DN1}} = 0.70$ ITD (goodness-of-fit = 0.62)
- Quadratic χ^2-fit: $\Delta t_{\mathrm{DN1}} = 0.50\ \mathrm{ITD} + 0.20\ \mathrm{ITD}^2$ (ITD in ms, goodness-of-fit = 0.999998)
- Model of A_{DN1} and Δt_{DN1}: goodness-of-fit = 1

Conclusions

- The current findings are inconsistent with the Jeffress model:
  - BDs are detectable for ITDs up to 2 ms, far outside the physiological range.
  - The latency increase in the Jeffress model is ITD/2 [6, 12], but DN1 latency grows faster than ITD/2.
- The model based on contralateral inhibition adopted from Ungan et al. [12] correctly describes the amplitude (Fig. 6) and the latency (Fig. 7) of BD wave DN1.

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