

# Modeling binaural difference potentials measured for a large range of interaural time differences

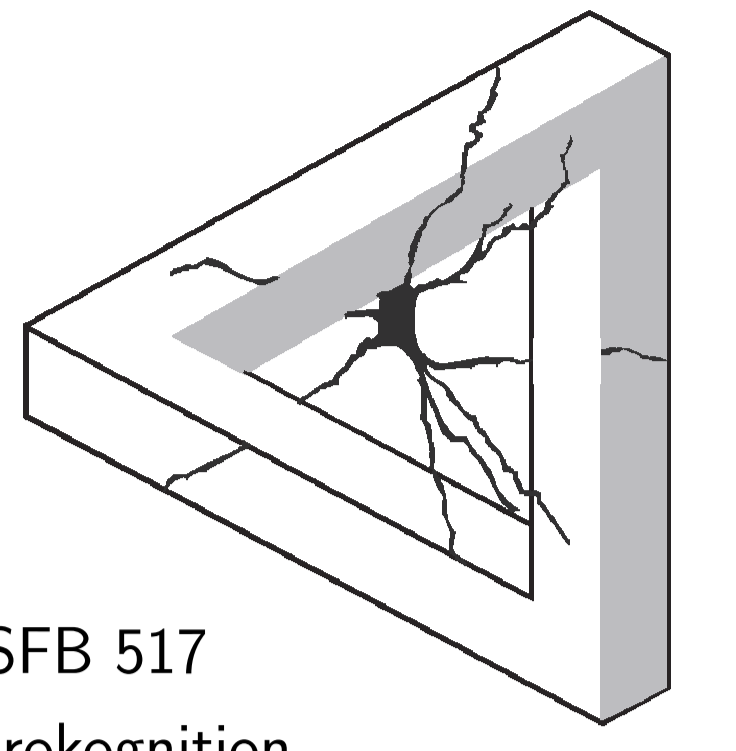
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## Abstract

**Background and problem:** Binaural difference potentials (BDs) are thought to be generated by neural units in the brain stem responding specifically to binaural stimulation. They are computed by subtracting the sum of monaural responses from the binaural response,  $BD = B - (L + R)$ . BDs in dependency on the interaural time difference (ITD) have been measured in a number of studies with conflicting results. The aim of this study is to measure, analyze and model BDs with a high signal-to-noise ratio for a large range of ITDs. **Subjects and methods:** 11 normal hearing subjects participated in this study. Chirp evoked BDs were measured for 17 ITDs in the range from 0 to 2 ms at a level of 40 dB nHL for four channels (A1, A2, PO9, PO10). For each binaural condition 10000 epochs were collected. 40000 epochs were recorded for each of the two monaural conditions. **Results:** Significant BD components are observed for ITDs up to 2 ms. The peak-to-peak amplitude of the first components of the BD, DP1-DN1, is monotonically decreasing with ITD, in contrast with click studies which reported a constant BD-amplitude for ITDs up to 1 ms. The latency of the BD-component DN1 is monotonically increasing with ITD. The classical Jeffress model assuming binaural coincidence detector cells innervated by bilateral excitatory cells via ordered delay lines predicts a latency increase of  $ITD/2$ . In the current study, DN1 latency is found to increase faster than  $ITD/2$  but slower than ITD not compatible with the Jeffress model. In order to describe the dependency of BD-latency and amplitude on the ITD, a computational model with only four parameters using ipsilateral excitatory and contralateral inhibitory inputs to the binaural cells was successfully fitted to the data. **Conclusions:** Despite its simplicity the model predicts physiologically plausible features: the inhibitory input must arrive slightly before the excitatory input and the effective duration of the inhibition must be considerably longer than the duration of the excitation.

## INTRODUCTION

In evoked response studies, binaural interaction is commonly assessed in terms of the binaural difference potential, symbolically  $BD = B - (L + R)$  [4, 7, 1, 10]. A comparative study demonstrated that larger BDs (with higher signal-to-noise ratio) can be obtained with a rising frequency chirp signal [2] 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. The first major peak in the BD, DN1, is believed to be a physiological correlate of the categorical percept of binaural fusion [4, 3]. In [4, 1] an approximately constant DN1 amplitude for ITDs up to 1 ms was found. For ITDs longer than 1.2 ms DN1 was undetectable [4]. In contrast, other studies reported a gradually decreasing DN1 amplitude with increasing ITD, the BD became undetectable for  $ITD > 1.6$  ms [6, 8]. If an ITD is applied to the stimulus, the classical Jeffress model [5] using bilateral delay lines predicts a BD latency increase of  $ITD/2$  [6, 12], 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 forgoing bilateral excitation, but using ipsilateral excitation and contralateral inhibition instead is proposed to explain BD amplitude and latency.

## METHODS

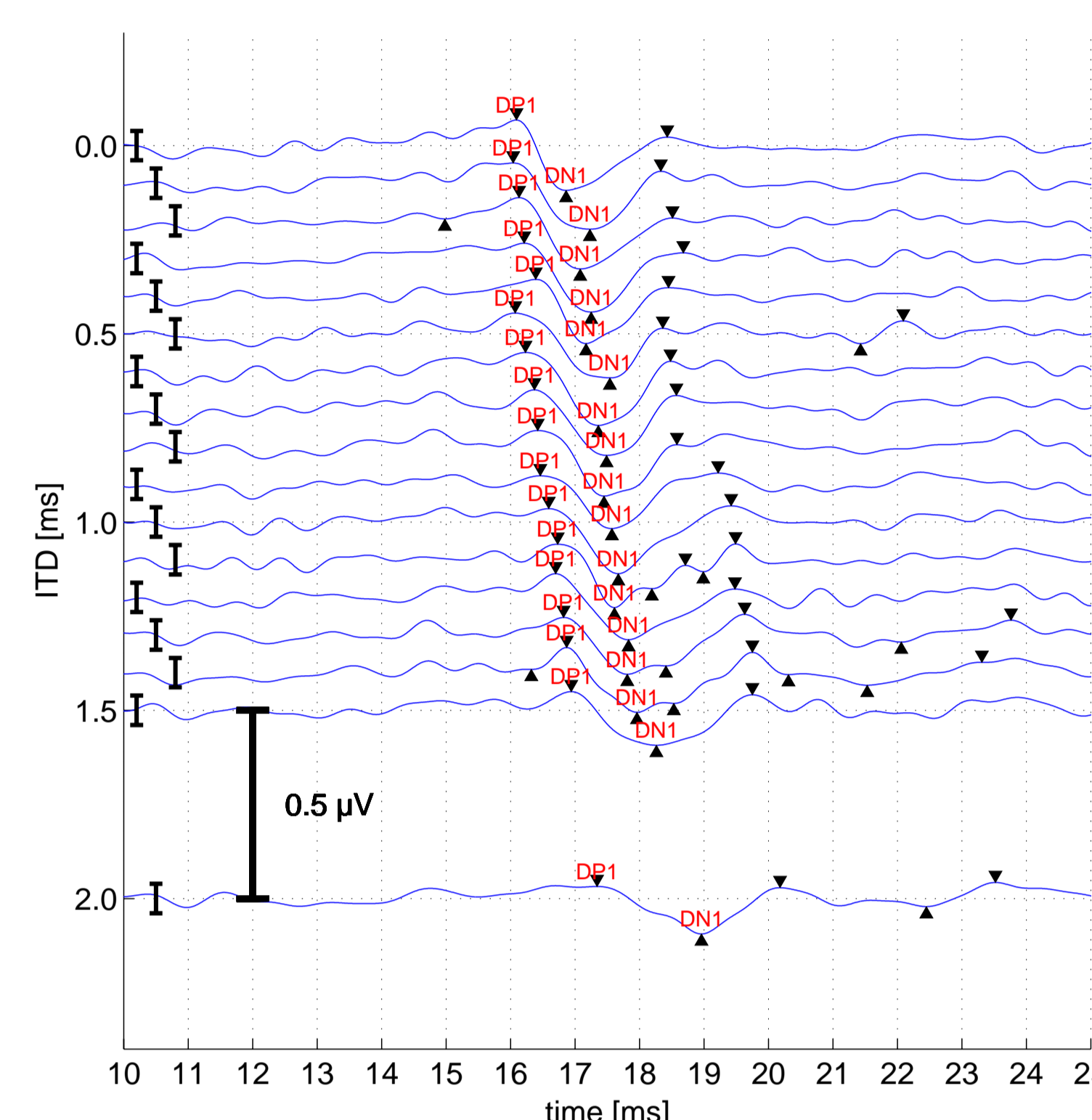
### Recordings

- Stimulus: flat spectrum chirp at 40 dB nHL ([2, 11])
- 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

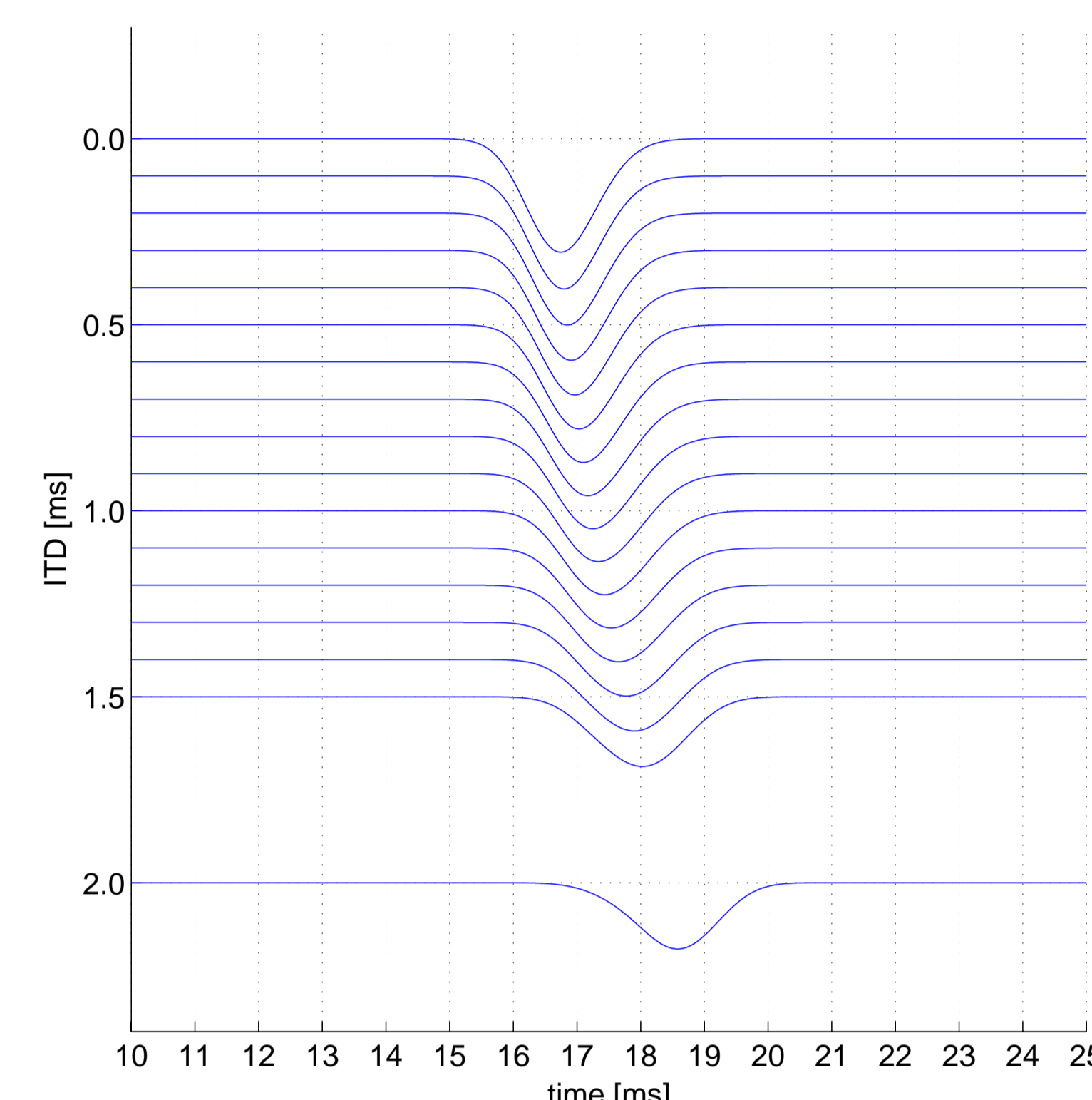
- Adoption of the model by Ugan et al. [12] for cat
- BD generated by (multiplicative) contralateral inhibition, see Fig. 7
  - $BD_L = L_{exc} * (1 - L_{inh}) - L_{exc}$
  - $BD_R = R_{exc} * (1 - R_{inh}) - R_{exc}$
  - $BD = BD_L + BD_R$
- $\chi^2$ -fit of 4 model parameters:
  1. difference between mean ipsilateral excitatory and contralateral inhibitory arrival time  $t_{e-i} = 0.597$  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

## BD recording



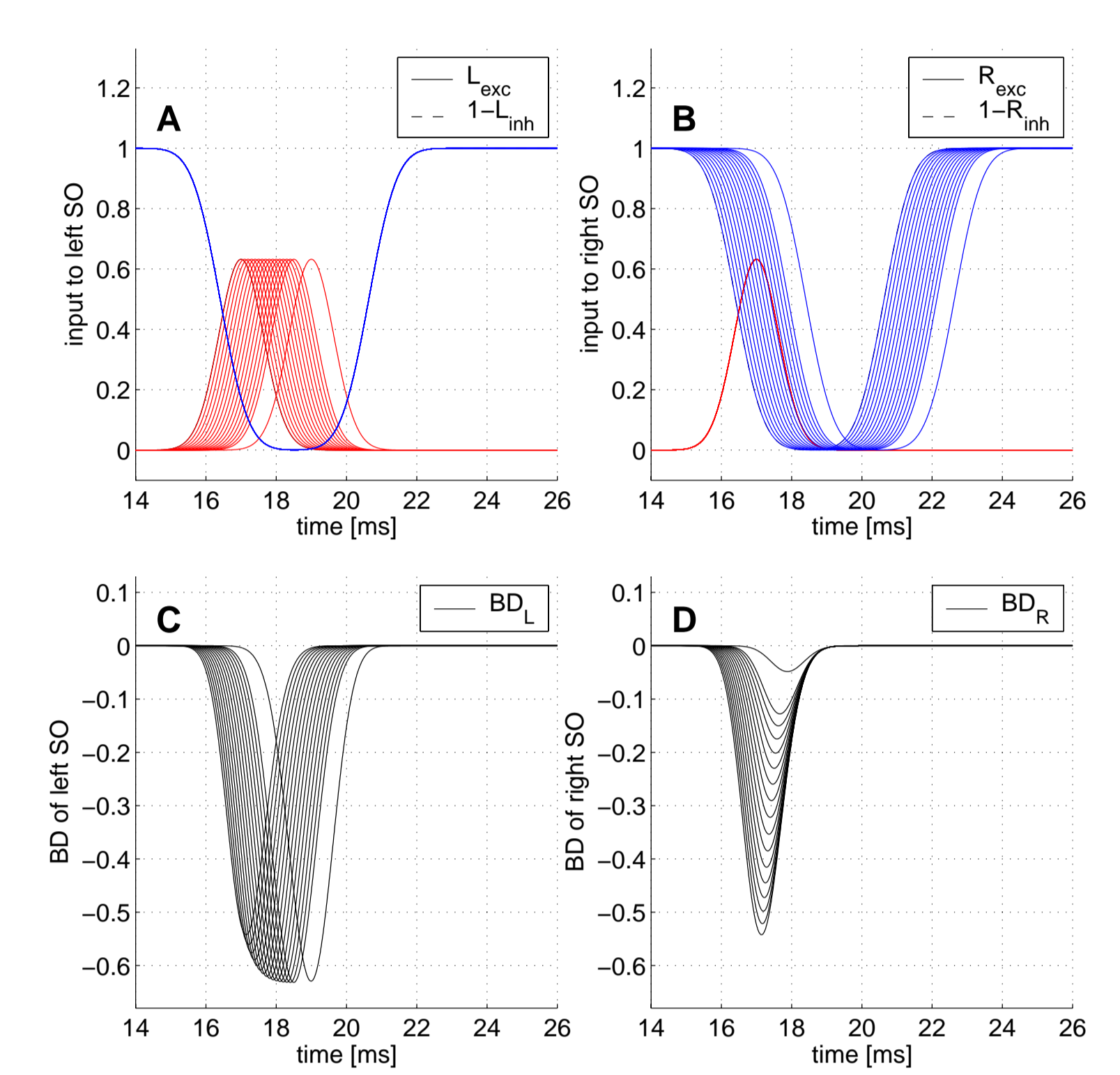
**Fig. 1:** BDs in dependence on the ITD: data from channel PO10, subject rb. The errorbars denote  $\pm 3\sigma$  ( $\pm 3$  S.E.M.). The triangles indicate peak pairs whose peak-to-peak values exceed  $\sqrt{2}3\sigma$ . The time axis is plotted relative to stimulus onset. Significant binaural interaction is found for all ITDs tested. The BD-peaks DP1 and DN1 occur approximately at the latency of the binaural wave V.

## BD model waveforms



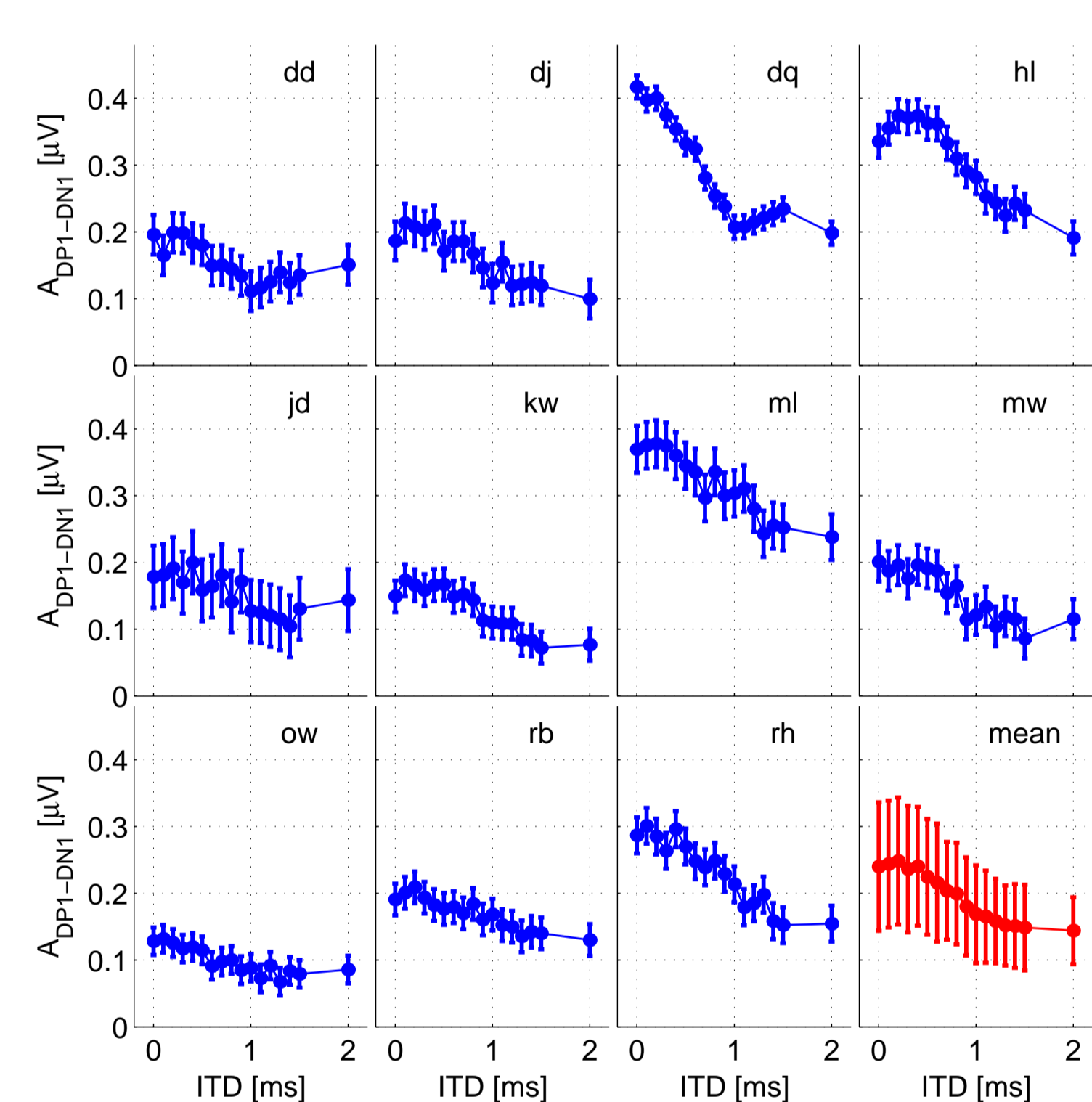
**Fig. 4:** Modeled BD waveforms as function of the ITD. Although the model cannot predict the small positive deflection DP1 preceding the main BD-peak DN1, measured and modeled waveforms look similar. The reduction of DN1 amplitude with increasing ITD is described correctly, see Fig. 5. The latency shift with increasing ITD is predicted properly, see Fig. 6.

## BD model specification



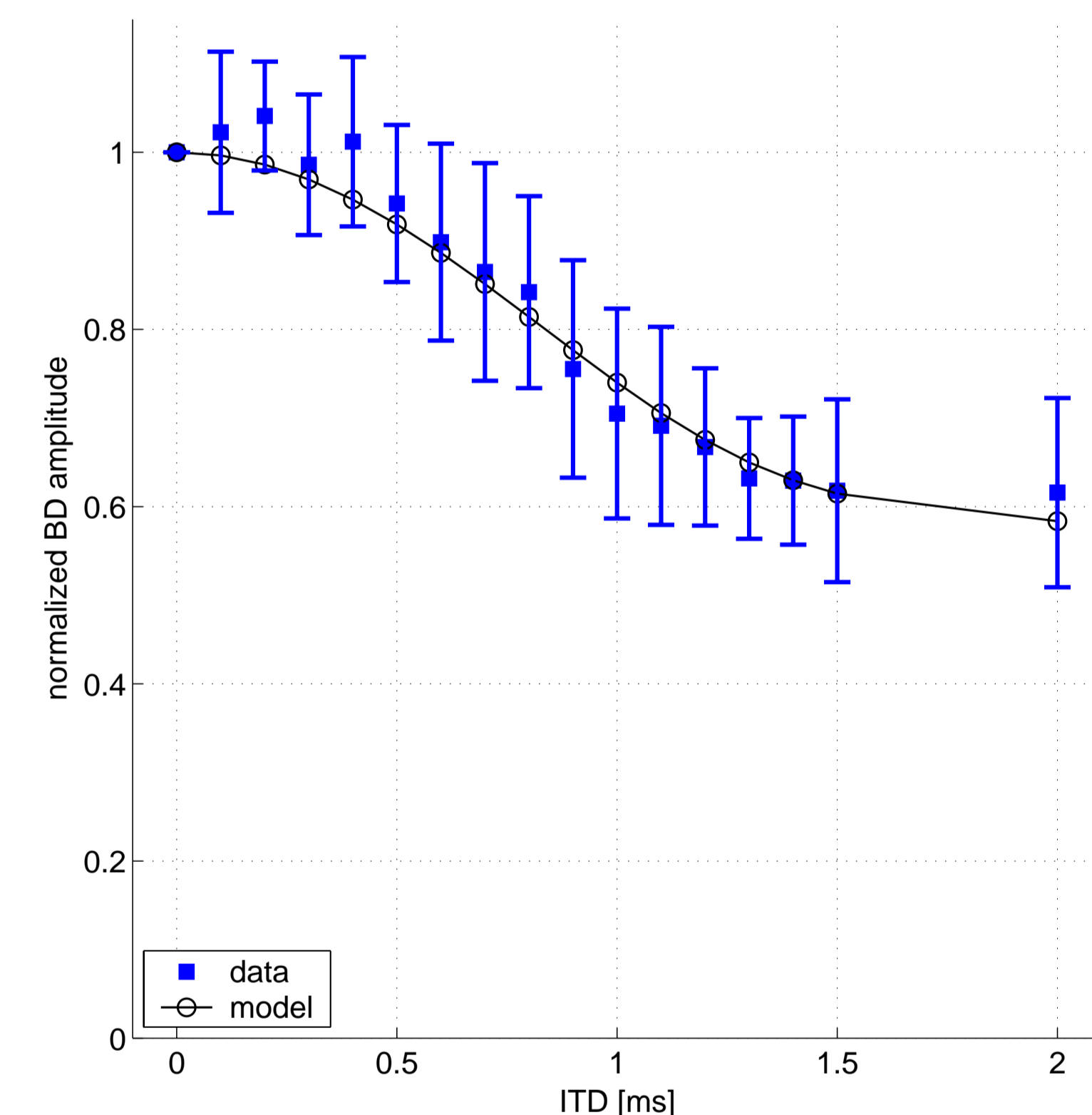
**Fig. 7:** **A:** Time courses of the inputs to the left superior olive (SO): excitation is delayed by ITD (red), contralateral inhibition (blue) is not affected by ITD. **B:** Time courses of the inputs to the right SO: excitation (red) is not affected by ITD, contralateral inhibition (blue) is delayed by ITD. **C:** BDs in the left SO as function of the ITD. **D:** BDs in the right SO as function of the ITD.

## BD amplitude



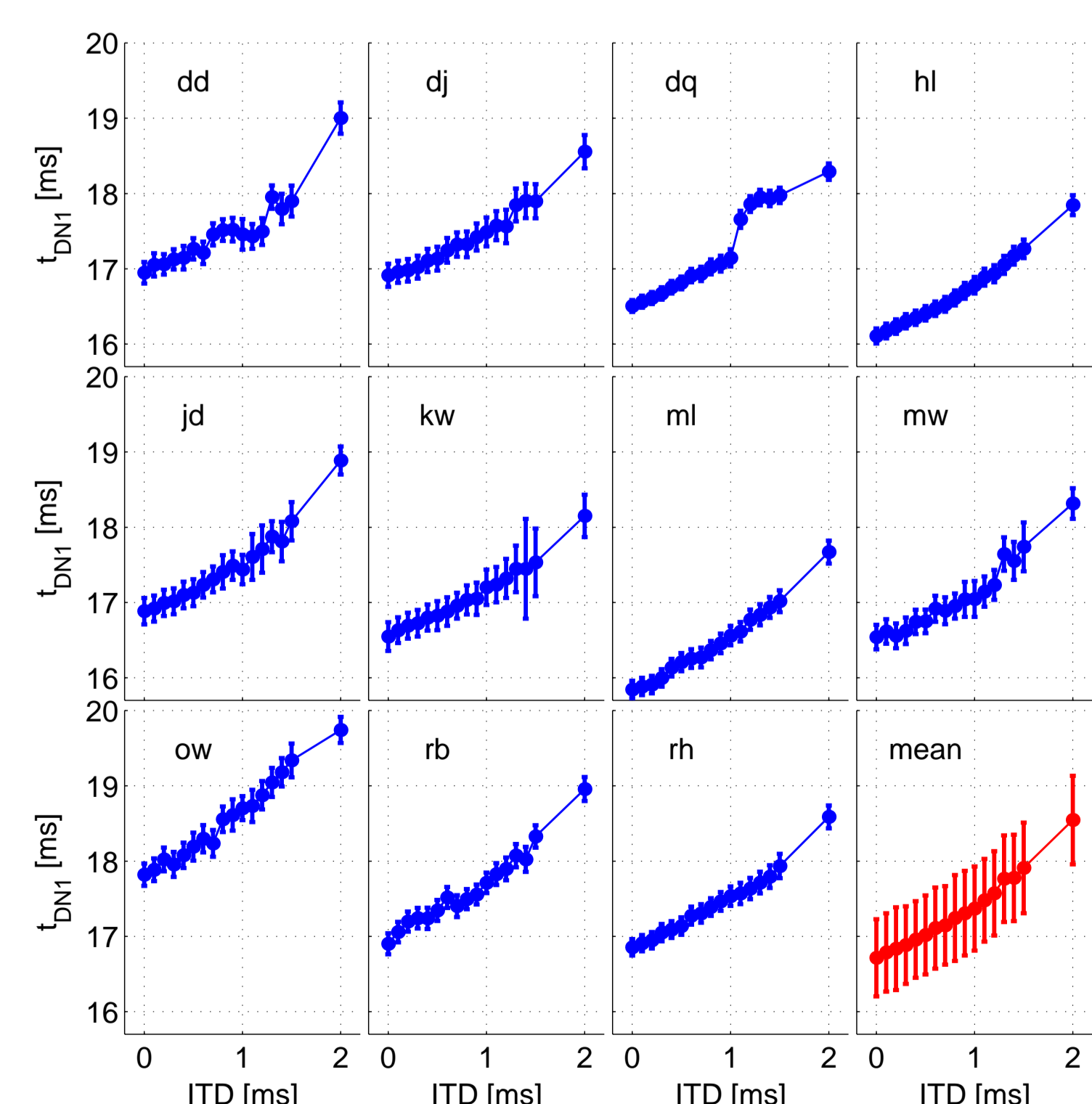
**Fig. 2:** Mean amplitude of BD-wave DP1-DN1 averaged over channels as function of the ITD. The first 11 subplots show single subject data, the errorbars indicate intraindividual standard errors ( $\pm \sqrt{2} \cdot \sigma$ ). The last subplot depicts the mean over subjects, the errorbars denote  $\pm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

## Model of the BD amplitude



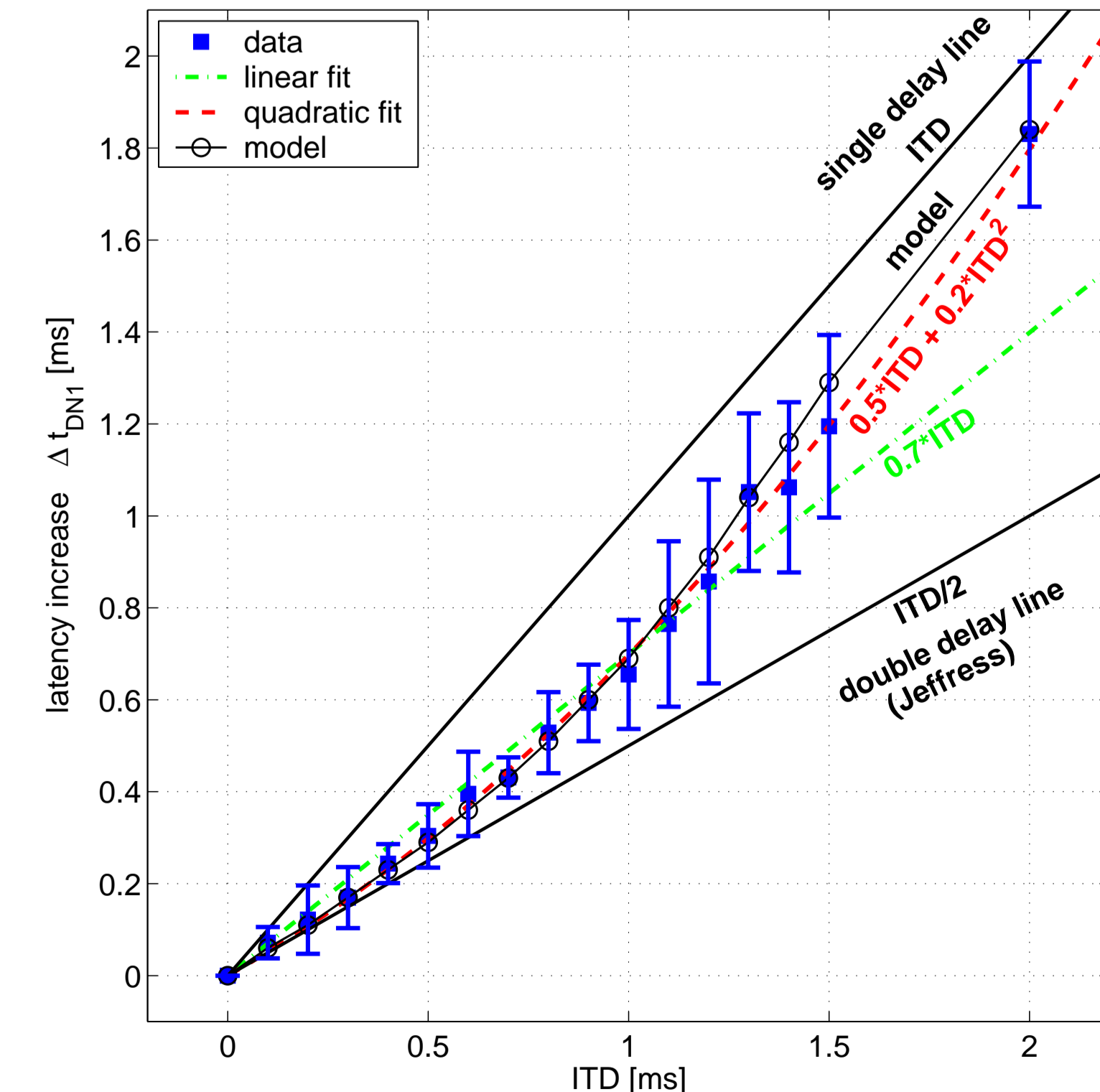
**Fig. 5:** Comparison of the normalized measured (blue squares with errorbars) and modeled (open black circles) BD amplitudes. The model seems to underestimate the measured amplitudes for small ITDs in the range up to 0.4 ms. In this range, the data appear to be relatively constant while the model predicts 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.

## BD latency



**Fig. 3:** 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  $\pm 3\sigma$  ( $\pm 3$  S.E.M.). The last subplot depicts the mean over subjects, the errorbars denote  $\pm 1$  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.

## DN1 latency increase



**Fig. 6:** Mean latency increase of BD-wave DN1 averaged over channels as function of the ITD ( $\Delta t_{DN1} = t_{DN1} - t_{DN1,ITD=0}$ ). Errorbars denote  $\pm 1$  standard deviation. The lower straight line indicates the latency due to the Jeffress model [5, 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  $\chi^2$ -fit (one parameter). The dashed line holds for a quadratic  $\chi^2$ -fit (two parameters). Open circles stand for the model.

## RESULTS

- Significant BD for ITDs up to 2 ms (Fig. 1, 2)
- Decreasing BD amplitude with increasing ITD (Fig. 2)
- Increasing BD latency with increasing ITD (Fig. 3)
- BD latency increase  $\Delta t_{DN1} = t_{DN1} - t_{DN1,ITD=0}$  is between  $ITD/2$  and  $ITD$  (Fig. 6).
- Linear  $\chi^2$ -fit:  $\Delta t_{DN1} = 0.70$  ITD (goodness-of-fit = 0.62)
- Quadratic  $\chi^2$ -fit:  $\Delta t_{DN1} = 0.50$  ITD +  $0.20$  ITD<sup>2</sup> (ITD in ms, goodness-of-fit = 0.999998)
- Model of  $A_{DN1}$  and  $\Delta t_{DN1}$ : 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 Ugan et al. [12] quantitatively describes the amplitude DP1-DN1 (Fig. 5) and the latency increase (Fig. 6) of BD wave DN1 as function of the ITD.

## References

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