Coding of speech in the intact and implanted inner ear

Werner Hemmert[1], Michele Nicoletti[1], Christian Wirtz[2], Marek Rudnicki[1]
[1] *Technische Universität München, 85748 Garching, E-Mail:Werner.Hemmert@tum.de*
[2] *MED-EL Deutschland GmbH, 82313 Starnberg, Germany*

Introduction

We develop models of neuronal sound processing of the first stages of the auditory system and evaluate neuronal spike trains with quantitative methods like automatic speech recognition or sound localization models. We have extended our modeling efforts to cover also the situation in subjects with cochlear implants (CIs), where the damaged inner ear is bypassed by direct stimulation of the auditory nerve with electric current pulses. Coding strategies used in CIs try to replicate coding of the intact inner ear, however, due to many limitations involved in electrical stimulation, the code provided by the stimulated auditory nerve fiber population differs from the coding in the intact inner ear. We have recently extend our modeling activities to the next neuronal levels and model processing in the auditory brain stem and the sound localization pathway to evaluate how precise auditory nerve spike trains are processed.

Methods

We apply two inner ear models from [1] to derive left- and right ear action potentials of the auditory nerve from stereo sound inputs. For the implanted inner ear, we have developed a framework that allows us to calculate electrically evoked auditory nerve responses from a variety of inner ear coding strategies [2]. Bushy neurons in the auditory brainstem, the first neuronal relay station, receive input from multiple auditory nerve fibers. We have implemented them as point neurons with Hodgkin-Huxley like ion channels. Model parameters for ion channels were taken from [3]. As that model did not have a realistic refractory period, we had to substitute the sodium ion channel model with the one described in [3]. We evaluated the temporal precision of the spike-trains at the level of the auditory nerve and of the bushy neurons with a correlation model developed by [5], which we modified such that it could process spike trains [2].

Results

Our evaluation of the precision of interaural time difference coding shows that the spatial position is well coded in auditory nerve responses. Analysis of the data (compare Fig. 2 and 3) suggests that globular bushy neurons significantly sharpen the spatial percept. Our assessment of fine structure coding strategies used in latest cochlear implants revealed not only that they reach a temporal precision to discriminate seven positions in space (data not shown) but also that these positions are resolved in the activity of the auditory nerve, albeit with much less salience compared to the intact inner ear.

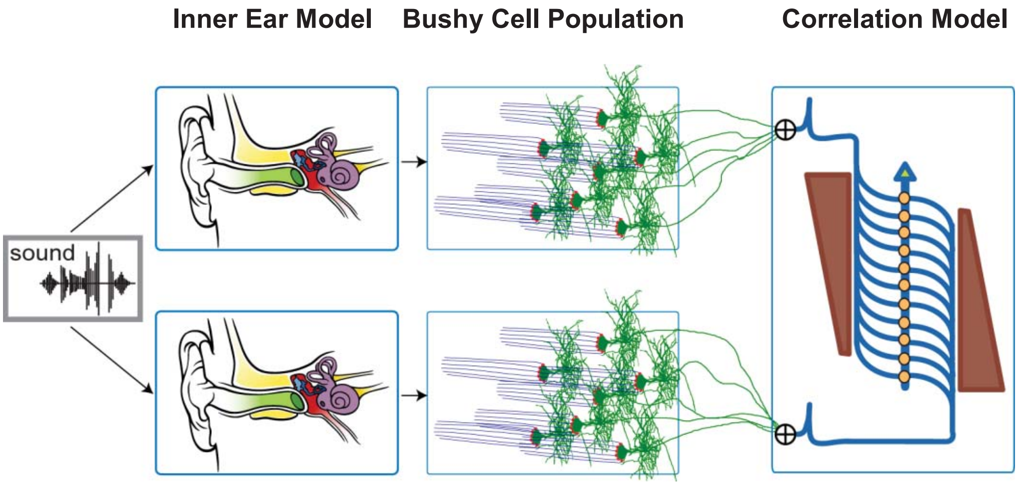

Figure 1: Model of binaural sound processing. Signals are coded in the left and right inner ear into action potentials of the auditory nerve. Bushy neurons in the auditory brainstem, the first neuronal relay station, sharpen temporal responses by coincidence detection. For evaluation of the temporal precision of the spike-trains we apply a correlation model.

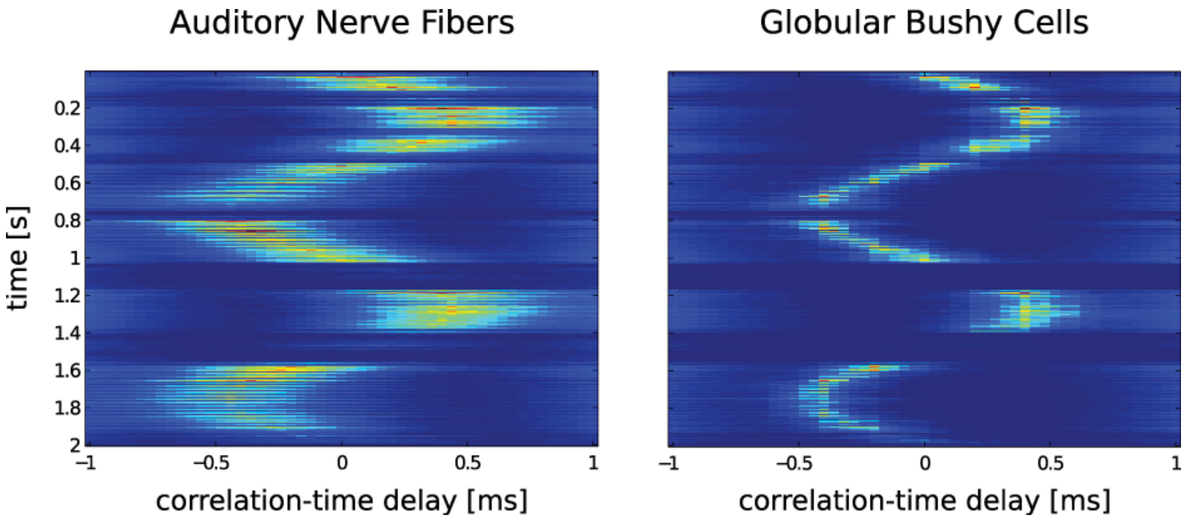

Figure 2: Evaluation of ITD coding with a correlation model with spike trains from auditory nerve fibers (left) and globular bushy neurons (right). Negative correlation time delays occur if the sound source is at the left side of the head and vice versa. Signal was a spoken sentence generated with a virtual sound source which circled around the head once per second. The source position can be tracked from both signals, but the bushy cells provide sharper localization cues.

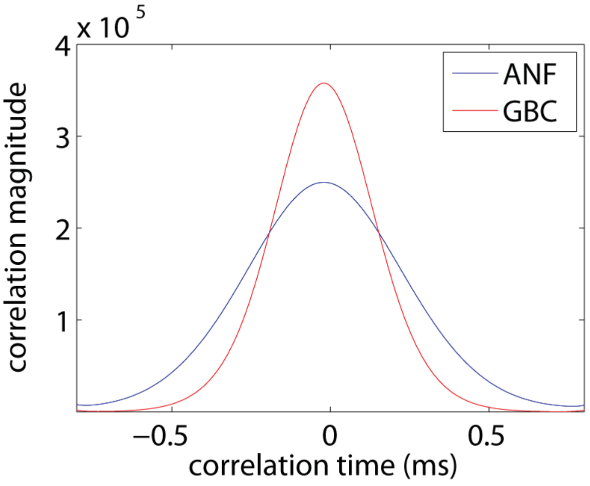

Figure 3: Comparison of correlation delay for a source position in front of the head with spike trains from auditory nerve fibers (blue line) and globular bushy neurons (red line). Responses from GBC exhibit a narrower localization distribution.

Conclusion

Modern auditory models produce very realistic spike trains of the auditory nerve, which can be used to test how sound is coded and processed – at least in the first stations – along the auditory pathway. Spiking neuron models of sound processing in the auditory pathway provide insight into details of auditory coding and processing. For example, GBC apply coincidence detection and nonlinear activation functions to sharpen sound localization cues compared to auditory nerve responses. This process does not require inhibition, which was earlier proposed e.g. by [5].

We can now use these models to evaluate how well cochlear implant coding strategies preserve sound localization cues. This model-driven approach provides large benefits, because it provides quantitative data even before a new coding strategy/implant is developed, approved, fabricated, and implanted. This procedure has therefore the potential to speed-up development cycles significantly.

Acknowledgement

This work was supported within the Munich Bernstein Center for Computational Neuroscience by the German Federal Ministry of Education and Research (reference number 01GQ1004B and 01GQ1004D), the Priority Program "Ultrafast and temporally precise information processing: normal and dysfunctional hearing" SPP 1608 (HE6713/1-1) funded by the German Research Foundation and MED-EL Innsbruck.

Literatur

[1] M. S. Zilany, I. C. Bruce, R. A. Ibrahim, and L. H. Carney: Improved parameters and expanded simulation options for a model of the auditory periphery. JASA 135:283–286, 2014.

[2] M. Nicoletti, C. Wirtz, W. Hemmert: Modelling sound localization with cochlear implants. In Jens Blauert, editor, The Technology of Binaural Listening, pp. 309–331. Springer, 2013.

[3] J. S. Rothman and P. B. Manis: The roles potassium currents play in regulating the electrical activity of ventral cochlear nucleus neurons. J Neurophysiol, vol. 89, pp. 3097–3113, June 2003.

[4] J. S. Rothman, E. D. Young, and P. B. Manis: Convergence of auditory nerve fibers onto bushy cells in the ventral cochlear nucleus: implications of a computational model. J Neurophysiol, vol. 70, pp. 2562–2583, 1993.

[5] W. Lindemann. Extension of a binaural crosscorrelation model by contralateral inhibition. I. Simulation of lateralization for stationary signals. J Acoust Soc Am, 80(6):1608–1622, 1986.