Computational model for the dynamic aspects of sound processing in the auditory midbrain

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Abstract

Recent experiments show that many of the interaural time difference (ITD) sensitive neurons in the inferior colliculus (IC) respond differently to stimuli with constant or dynamically varying ITDs. We have developed a firing rate model with an activity-dependent adaptation mechanism to study these plasticity effects. The model is highly idealized, which makes it tractable and allows clear interpretation. In our formulation, the dynamic effects originate in the IC and are not inherited from the lower level structures. The results are in a good qualitative agreement with experimental data. © 2001 Elsevier Science B.V. All rights reserved.

Keywords: Auditory processing; Firing rate model; Adaptation

1. Introduction

Mammals use interaural time difference (ITD) and interaural level difference (ILD) as principal cues for azimuthal sound localization of low and high frequency stimuli, respectively. Neurons in various structures along the auditory pathway, such as the medial and lateral superior olive (MSO and LSO), dorsal nucleus of lateral lemniscus (DNLL) and inferior colliculus (IC), are sensitive to ITD orILD. In natural environments the relative positions of a sound source and the head are free to change and, therefore, these cues are likely to be changing (dynamic) too. Recent studies show that neurons in the IC, unlike neurons in the lower level structures (such as MSO and DNLL), respond differently to binaural cues presented under static and dynamic conditions [3,5,6]. Here we develop minimal computational models to investigate
possible mechanisms of these phenomena. As a first step, we base our modeling on data recorded in the IC from ITD-sensitive or, equivalently, IPD-sensitive neurons (IPD—interaural phase disparity). Although, modulation of other stimulus properties might engage similar mechanisms, this is a subject for future investigation. Our idealized description includes a firing rate model that mimics several aspects of the experimental data. Also, the model’s simplicity facilitates mathematical and computational analyses and the results allow clear interpretations in terms of experimental findings.

2. Schematic of the experiments

We refer to the experimental work described in [4–7]. In these experiments, extracellular single unit recordings were made in the IPD-sensitive area of MSO, DNLL or IC of cats or gerbils. Presentation of a tone at different locations around the head of the animal (one at a time) generates different IPDs. By recording the responses of an IPD-sensitive neuron to these stimuli, one produces the (static) tuning curve (Fig. 2A, dashed line). It is usually a relatively smooth curve that has a clearly distinguishable peak. If now we sweep (even with relatively slow rate of modulation) through the same range of IPDs (corresponds to a sound source moving around the head of the animal), responses of cells in the medial superior olive (MSO) and DNLL follow their static tuning curves, whereas cells in IC exhibit a new profile with a different peak IPD. Similarly, if we sweep through a partial (less than a period) range of IPDs, the response may differ considerably from the equivalent portion of the static tuning curve (Fig. 2A, solid lines).

The conceptual model of the principal components of the circuit, that we use in our mathematical formulation, is shown in Fig. 1. MSO, in the brainstem, is the first auditory station where binaural inputs converge. IC, in the auditory midbrain, receives input from multiple ascending pathways. Principal source of the IPD

Fig. 1. Schematic of the circuit.
tuned input is the ipsilateral MSO (mainly excitatory) and contralateral MSO (inhibitory, through contralateral DNLL). DNLL serves as a relay and a sign inverter.

3. Model

Because little is known about the connections and biophysics of the cells in this circuit, we have chosen a less biophysically detailed model for study. Also, because the discharges of IC neurons phase-lock only to very low carrier frequencies, we use a model in which individual spikes are not represented. Our primary dynamical variable is an instantaneous firing rate, relative to the maximum possible rate, or firing probability, \( r(t) \). Our minimal model is based on the following assumptions: (i) the balance of IPD-sensitive excitatory and inhibitory inputs shapes IPD-sensitivity of the IC cells; (ii) the balance is altered in IC by local adaptation (cellular and/or synaptic mechanisms) during dynamic stimulation; (iii) a firing rate (not spike-based) description is sufficient.

In order to account for plasticity effects, we introduce an activity-dependent adaptation variable, \( a(t) \), which effectively increases the firing threshold of a unit if it has been active lately. Our two-variable model takes the form: \( \tau_r \dot{r} = -r + r_s(I_{E} - I_{I}; a) \), \( \tau_a \dot{a} = -a + a_s(r) \). Firing rate, \( r(t) \), responds fast, with a time constant related to the membrane time constant and the fast synaptic time scales, while \( a(t) \) is slow with a time constant that could be hundreds of ms or more. The cellular input–output function is sigmoidal: \( r_s(I; a) = 1/(1 + \exp(-2\beta_r(I-a))) \). The steady-state rate-adaptation relation is also sigmoidal: \( a_s(r) = 1/(1 + \exp(-2\beta_a(r-r_\theta))) \). Therefore, adaptation slowly translates \( r_s \) rightward for activity above some spontaneous rate, providing negative feedback. Typical values of parameters: \( \tau_r = 30, \tau_a = 100, \beta_r = 1.83, \beta_a = 2, r_\theta = 0.5 \). Each of the inputs \( I_{E}, I_{I} \) has the form \( I = w(0.55 + 0.45\cos(IPD - P_{MSO})) \), where \( w \) is the corresponding strength of connection and \( P_{MSO} \) is the preferred IPD of the corresponding set of MSO neurons (\( w_E = 1.8, w_I = 0.5, P_{MSO,E} = 0, P_{MSO,I} = 100 \)).

4. Results

We have explored the model’s behavior with a variety of stimuli as used in the experimental work. First of all, the static IPD tuning curve of a model IC neuron has a clearly defined peak (Fig. 2C, dashed line). Response at the ’worst’ phase (minimum of the tuning curve) can be zero or a positive firing rate. Similar variety is seen in experimental recordings. The static tuning curve’s shape can be modified in the simulations by changing the mutual strength and preferred IPDs of incoming inputs. In this spirit, Fig. 2A and C are not chosen to show qualitative agreement, but to illustrate variety.

Several other experimental observations are reproduced by the model. As shown in [5], binaural beats and full range dynamic modulation (360°) produce similar results.
Under dynamic stimulation the tuning curve becomes sharper and has a higher peak. Also, the tuning curve peak is systematically shifted with rate and direction of the modulation. For periodic, partial range, triangular modulation, up-sweeps toward the best (static) phase end in increased responses, while those away from the best phase end in decreased responses. This effect is reduced near the peak of the static tuning curve (Fig. 2A, experimental). Fig. 2B shows an example of the steady periodic response (after transient) of the model neuron to a triangular partial range modulation stimulus. Responses to the up-sweep portion of the stimulus cycle (marked with arrows in the upper panel) are plotted again in Fig. 2C as a function of IPD (star). Other curves in Fig. 2C show responses to up-sweeps of periodic stimulations, centered at different values of IPD. Dashed line is the static tuning curve. Protocol of the simulation is analogous to the experimental one.

One of the important experimentally observed phenomena in the IPD-sensitive IC cells is that the response during the down-sweep of the periodic modulation can be substantially different from that during the up-sweep (hysteresis). Our model neuron also has this property. We study the strength of the hysteresis as a function of the time-scale of adaptation. If the adaptation is fast, compared to the rate of modulation, then the system is always adapted to the instantaneous value of the firing rate and the response follows the static tuning curve. By requiring that the dynamic response deviate from the static one, we obtain a lower bound on the time-constant of adaptation, $\tau_a$. We also find, that if adaptation is too slow, then the responses to the sweeps in two directions exchange roles and do not agree anymore with experimental data (see example in Fig. 3). This sets an upper bound on $\tau_a$. Therefore, the model limits suitable values of $\tau_a$ to within approximately 100–400 ms range. This prediction can narrow a class of possible mechanisms of adaptation.

Previous modeling studies of some of these IC properties used more detailed spiking neuron models (of Hodgkin–Huxley type) with several stages of neural processing [1,2]. They also give results close to the above. Advantages of our approach, using a highly idealized model, are: (i) The model is based on minimal assumptions that closely relate to experimental evidence. For example, our formulation makes it very clear that all plasticity/dynamic effects are only in the IC because the afferents are prescribed to carry only instantaneous IPD dependence. This formulation is consistent with the evidence that there is little, if any, IPD-modulation sensitivity in the ascending pathway to the IC [7]. (ii) The mathematical and computational studies of the model are tractable and the results allow clear interpretations in terms of experimental findings. For example, requiring that our model accounts for the hysteresis effect makes clear the restrictions on the time-scale of adaptation and narrows the class of possible mechanisms.
One deficiency of the present model is that it fails to account for the strong response to a sweep over a range of IPDs, where static presentation elicits no response (‘rise from nowhere’: Fig. 2A, rightmost sweep). To overcome this problem, we have re-formulated the model, using voltage, averaged over time (to eliminate spikes), as the main dynamic variable. Our results confirm the hypothesis that the ‘rise from nowhere’ can be explained by post inhibitory rebound (PIR). These findings will be the subject of future publications.
5. Conclusion

We have presented an idealized firing rate model with a slow negative feedback, in the form of spike frequency adaptation, that can account for a number of experimentally observed properties of dynamic sound processing in the auditory midbrain. We conclude that some issues can be addressed without consideration of spike timing. Also, due to the idealized treatment of the problem, we had to make only minimal assumptions about biophysical mechanisms (yet to be determined for the IC neurons). Our results show that observed non-linearities might be a general property, dependent on the structure of connections and a presence of adaptation, not on the specifics of the IC cells. Our model also places bounds on the time-scale of the slow adaptation process. This finding narrows a range of possible adaptation mechanisms. There is also experimental evidence [3,4] of dynamic processes that take place on even slower time scales (several seconds). Neither our current model nor earlier models [1,2] account for this set of observations. This is an important direction in which we are extending our model. We also plan to incorporate slice data, as they become available, especially with regard to a spike frequency adaptation mechanism and PIR.

References


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