A cellular neural network model of the time-coding pathway of sound localization - hyperacuity in time

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ABSTRACT

This paper discusses a new cellular neural network model of the time-coding pathway of sound localization. The key feature of the model is lateral inhibition which is supposed to play crucial role in sound localization. The possible role of this inhibition is examined on the basis of our model and several conlusions are drawn concerning the expected nature of inhibition. It is also shown that by use of inhibition a group of neurons may be much more sensitive to interaural time difference than one individual neuron. Hence, our model for the first stage of the sound localization system solves a "hyperacuity in time" problem.

1 Introduction

The barn owl can catch its prey in total darkness relying on acoustic signals only. It can localize sound within 1-2° in azimuth and elevation and can detect interaural time differences as short as some tens of microseconds. The neural mechanism underlying this fascinating ability has been investigated for decades and now we know a lot about how the brain of birds localizes sounds using differences in the arrival of time and intensity [9-12,18]. However, several open questions are still waiting for answers. Two unsolved problems in sound localization we consider here are as follows:

- How can an owl detect interaural time differences as short as some tens of microseconds while a single action potential persists considerably longer, on the order of 1000 microseconds at the very least?
- How can an owl resolve phase ambiguity which results from the fact that time is measured by the phase of the input signals?

The first question concerns the operation of the sound localization system of the owl and involves a type of hyperacuity : the sound localization system can mark shorter delays of time arrivals of sound than the duration of an impulse which indicates the time arrival. With other words, the selectivity of a group of neurons is greater than that of one individual neuron. Hence, this is a kind of effect of *hyperacuity in time.*

We tried to develop a model of the time coding pathway of sound localization using *cellular neural networks* (CNN, see [3,17]). Cellular neural networks have proved to be useful dynamic spatiotemporal models for 2 1/2 D neural structures [20] as well. The key point of our model is *inhibition.* We have investigated the possible role that inhibition plays in improving the selectivity to interaural time differences and eliminating phase ambiguity. Our model considers many facts and observations neurobiologists have collected through the many years of intensive research - so we hope it is a neuromorphic model -, but it does not aim to reproducing all results found in the literature, rather illustrating that this simplified model can explain some phenomena and that it is worth drawing some interesting conclusions relating the nature of the biological connections. In this way we have a CNN model for hyperacuity in time, which, in a way, is a complement to the CNN model for hyperacuity in space [5].

As a summary, we show that (1) lateral inhibition greatly improves the selectivity for interaural time difference (ITD), (2) excitatory convergence of the different frequency channels can decrease phase ambiguity, (3) a feedback inhibition to the space-specific neurons greatly suppresses phase ambiguity, (4) a feedforward inhibition (of the same magnitude as that of the feedback inhibition) gives worse results - in each level of the sound localization system - than a feedback inhibition. In Section 2 a schematic drawing of the overall model is given. Section 3 reviews the CNN implementations of the building blocks (neurons, synapses etc.) of the model. In Section 4 the input data of the model is given. Section 5 discusses how coincidence detection is achieved in the auditory pathway and how the strength of inhibition can improve the selectivity for interaural time differences. In

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Section 6 a model is introduced for the convergence of the different frequency channels completed by a feedback inhibition which aims at decreasing phase ambiguity. For lack of space, only the main results are introduced here, the detailed description of the model and experiments is given in [16].

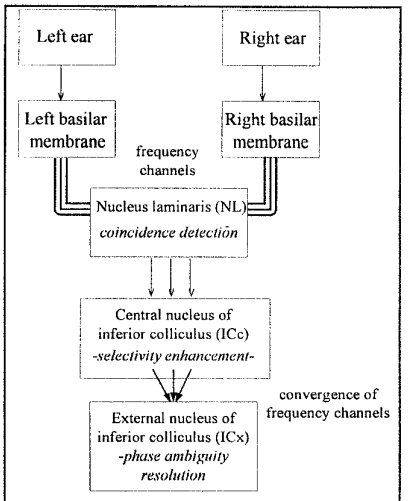

Figure 1: *Schematic drawing of the model*

2 The overall model

The schematic drawing of our model is displayed in Figure 1. Signals from the left and right ears pass through the basilar membranes and *nucleus magnocellularis* (NM) and converge in the *nucleus laminaris* (NL) where the coincidence detection of action potentials arriving from opposite directions takes place. The nucleus laminaris projects to the *central nucleus of the inferior colliculus* (ICc), where the selectivity for interaural time differences is enhanced. To this point frequency channels are strictly separated. The ICc projects to the *external nucleus of the inferior colliculus* (ICx). This is the first nucleus of the time-coding pathway where the different frequency channels converge in order to resolve the phase ambiguity problem. The description of the connections is detailed in Section 5 and 6.

3 CNN models of neurons, synapses and axons

For lack of space, the CNN models of the building blocks of our model cannot be detailed here, only a short summary is given and we refer to [6,14,15] for a detailed description.

The equivalent electrical circuit of neurons we use in our model (magnocellular neurons, NL neurons, interneurons) contains three ion channels, one is a non-gated leakage channel to maintain the resting membrane potential, the other two are voltage-gated sodium (Na) and potassium (K) ion channels to generate action potentials.

Action potentials (spikes) propagate along the magnocellular axons. Axons and signal propagation on them are modelled using a multi-compartment model well known from cable theory [6,8]. The timing of spikes is so that the time needed for a spike to travel along the axon is approximately the same as the duration of the spike. Figure 2 displays the state voltage of units along the axis *at a given moment*. Action potentials generated by a magnocellular neuron and recorded *at one point of the axon* model are displayed in Figure 3. The excitatory synaptic connections are modelled with ion channels whose reversal potential is between the reversal potentials of the sodium (Na) and potassium (K) ions, and the conductance is nonlinearly dependent on the difference of the membrane potential and the reversal potential.

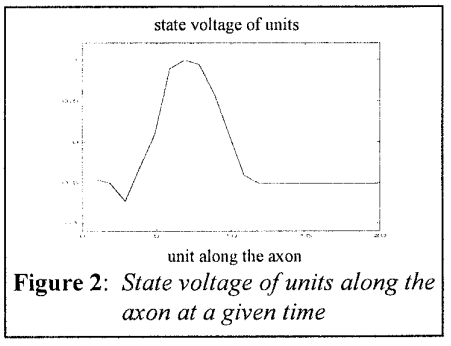

Figure 2: *State voltage of units along the axon at a given time*

4 Input data of the model

In our model we use two kinds of input data: artificial and real world data. Ideal sinusoid signals generated by a computer simulation as artificial input are used when the effect of single-frequency tones is investigated. The real world data was a stereophonic signal of a short, sharp noise captured with the SGI Indigo workstation and processed through a basilar membrane simulation. The basilar membrane accomplishes a form of bandpass filtering. The general equation for the filter shape is given by the gammatone function which was originally used by psychologists [1,7] to describe the filter responses they obtained in single unit studies with cats.

In our simulation we use 10 channels distributed logarithmically in the 300..8000 Hz frequency range. Accordingly to the Nyquist theorem we have to take the sampling rate of the sound data at least

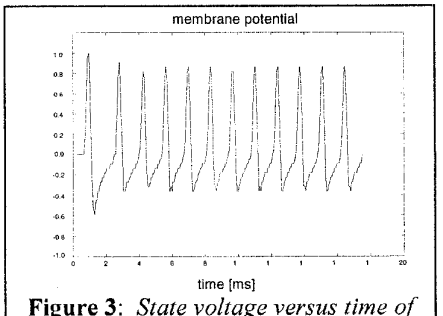

Figure 3: *State voltage versus time of one unit at a given point of the axon*

16000 Hz. We used a software implementation of the gamma tone filters.

5 Coincidence detection and selectivity for interaural time difference

As we mentioned above, there is considerable evidence that in the *nucleus laminaris* (NL) of owls interaural time differences map into a neural *place coding* via coincidence detection of signals from the left and right ear. The first model to explain the encoding of *interaural time difference* (ITD) was proposed by Jeffress in 1948 (Figure 4). Here fibers from the left and right nucleus magnocellularis (NM) converge on the nucleus laminaris (NL), and the place of the neuron responding maximally denotes the corresponding ITD. The exactness of this model was verified by the experiments and results of M. Konishi and his colleagues. Their findings indicate that the magnocellular afferents work as delay lines, and the NL neurons work as coincidence detectors [2].

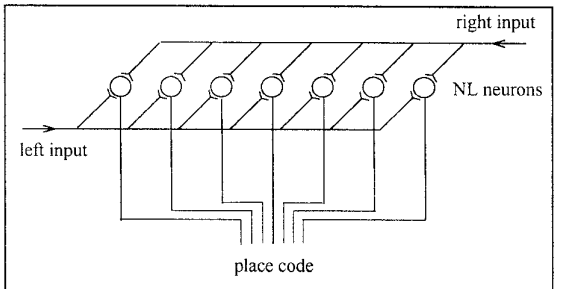

Figure 4: *The Jeffress model for encoding interaural time differences*

Action potentials pass along the magnocellular axons. It is known ([9-12]) that the velocity of spikes on the axon of a magnocellular cell is estimated to be 3-5 m/s and the NL neurons have large cell bodies (30-40 μm). If we take the velocity of spikes 4 m/s and the distance of 2 NL neurons 100 μm, then the time which is needed for a neuron to pass between two neighboring cells is 25 μm. It can be seen from Figure 3 that the duration of a spike is about 2 ms. It is also evident in Figure 2 that a spike has no distinct peak along the axon. So the place of the exact coincidence cannot be determined on the basis of this model.

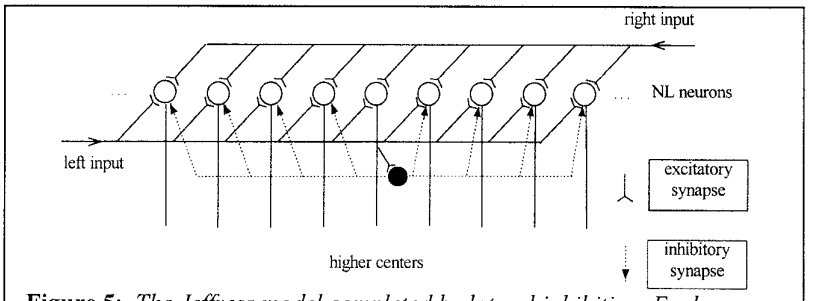

Figure 5: *The Jeffress model completed by lateral inhibition. Each neuron synapse to an inhibitory interneuron (for simplicity only one is indicated in the figure) which inhibits the neighboring cells*

We have completed the original delay line-coincidence detector model (above) with a lateral inhibition (via an interneuron) between the neighboring neurons. It is a fact that antibodies against GABAa have been discovered in nucleus laminaris which indicates many inhibitory synapses but the origin of the inhibitory axons is not yet known. It is also not clear whether inhibition occurs at all in nucleus laminaris. We assumed that there is lateral inhibition via interneurons and examined the effect of this inhibition on the neuron's ability to detect coincidence of the input signals. The new structure is displayed below in Figure 5.

In this new structure each NL neuron synapse to an inhibitory interneuron (for simplicity only one is indicated in Figure 5), which inhibits the neighboring neurons (we took the neighborhood 3). The number of NL neurons in the line is 15 in accordance with the neurophysiological data. The CNN template for the coincidence detection is given in Appendix 1 of [16]. Next we discuss the nature of the inhibitory connections.

As neurons receive several inhibitory synapses, the net synaptic current originating from the inhibitory synapses is

$$I_i = \sum_{j=-3}^{3} k_{ij}\, g(v_m - E_{REV}) \cdot (v_m - E_{REV}) \qquad (2)$$

where k_{ij} is the strength of the synapse between the ith and jth neurons, and E_{REV} is the reversal potential characterizing the inhibitory synapse.

In our experiments we used a large number of different values of k_{ij}. As a result of many experiments we have stated that the best coincidence detection was achieved when the spatial distribution of the inhibitory connection strengths was set as displayed in Figure 6. It has turned out that to obtain good results self-inhibition must be little or zero.

Next we show the results of two experiments. In the first experiment we examined how the effectiveness of coincidence detection depends on the strength of the inhibition. The effectiveness of the coincidence detection is measured by the number of spikes of NL neurons. The responses of cells 5, 7, 8, 9 and 11 were recorded at different inhibition strengths. Results are displayed in Figure 7. We used uniform inhibitory values, i.e. $k_{ij} = k$ = constant in the above equation (see Figure 7). When there was no inhibition at all, all cells responded maximally (this is not displayed in Figure 7). With increasing inhibition, coincidence detection is more and more robust. Figure 7 displays responses of cells at three different strengths of inhibition ($k = 0.1$, 0.4 and 0.5) in three columns. We can observe that in the third column (strongest inhibition) practically only Cell 8 responds. According to this simulation, it is very likely that inhibition plays a crucial role in coincidence detection.

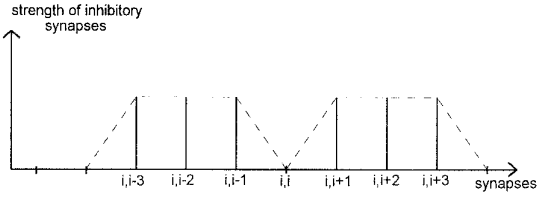

Figure 6: *Spatial distribution of inhibitory synaptic strengths*

In the second experiment we recorded the responses of 7 cells (cell 5-11) while the arrival time difference of the left and right input signals was changed. The inhibition was the same during this experiment ($k = 0.4$). Figure 8a shows the responses of the seven cells when the time difference is 0. Coincidence is detected by Cell 8 which responded most vigorously. Delaying one of the input signals by 30μs, Cell 7 detected the coincidence (Figure 8b). Further delaying the same input by the same period, Cell 6 showed the strongest response (Figure 8c) etc.

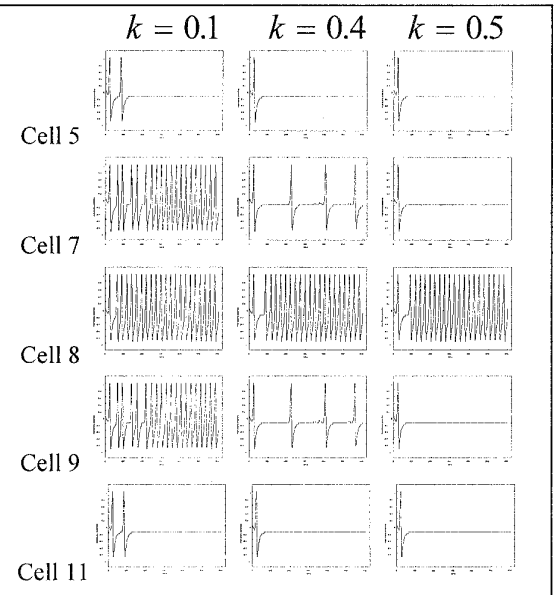

Figure 7: *Responses of cell 5, 7, 8, 9 and 11, respectively, at three different, increasing values of inhibition. A row contain the responses of the same cell at different inhibition, a column displays the responses of different neurons at the same inhibition*

The structure of Figure 5 can be considered as an aggregate model of coincidence detection. It is very likely, however, that the auditory system of owls uses several stages for this task. We developed a model which contains the next stage of the sound localizing system too (Icc). In this model, each NL neuron makes an excitatory synapse to an ICc neuron and an inhibitory interneuron (of this latter only one is indicated). The interneuron then inhibits the neighboring NL neurons (feedback inhibition) and the neighboring ICc neurons (feedforward inhibition). On the other hand, each ICc neuron again inhibits - via an inhibitory interneuron - all neighboring ICc cells (second feedback inhibition). So we model here three kinds of inhibition: two feedback and one feedforward. By applying various sets of inhibitory connections, we could observe some interesting facts:

(1) If the feedback inhibition in the NL and the feedforward inhibition is zero, the effect of the feedback inhibition in the ICc is the same as displayed in Figure 7 and 8 (the aggregate model). Therefore the assumption of inhibition in the NL is not necessary (but cannot be precluded).

(2) When only feedforward inhibition is used, with the same connection strengths as in the case of feedback inhibition, the coincidence detection is much poorer in the higher frequency band.

(3) In the lower frequency band, however, feedback inhibition is insufficient to produce the same selectivity as in the higher frequency band. In this case feedforward inhibition is indispensable.

We also concluded that by using feedback inhibition both in the NL and the ICc, the selectivity of ICc neurons to interaural time difference was better than that of NL neurons. This is in accordance with the observance that ICc neurons are more selective for time disparity than NL neurons and that application of bicuculline to ICc reduces this improved selectivity which suggests that inhibitory circuits between neurons tuned to different ITDs must be involved [4,9]. The ITD-curves measured on our model indicating the better selectivity of ICc neurons are shown in Figure 13 of [16].

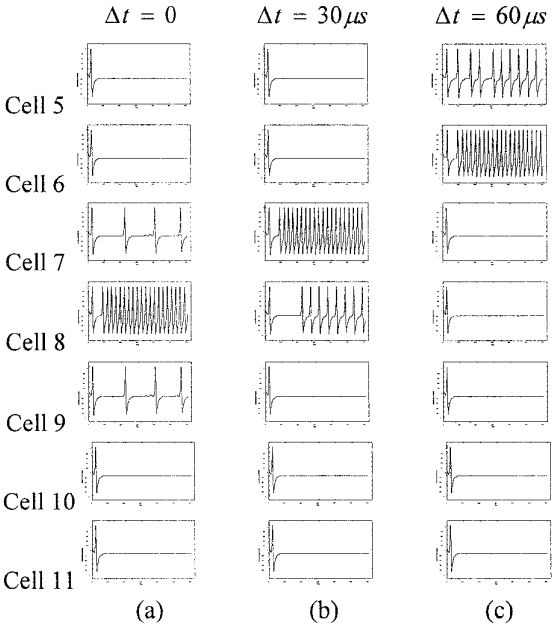

Figure 8: *Responses of cell 5, 6, 7, 8, 9, 10 and 11, respectively, at three different, increasing values of interaural time difference: (a)* $\Delta t = 0$, *(b)* $\Delta t = 30\mu s$, *(c)* $\Delta t = 60\mu s$

6 Resolving phase ambiguity

Neurons in the nucleus laminaris and central nucleus of the inferior colliculus respond not only to one ITD (τ) but also to time differences that are separated by integer multiples of the stimulus period ($\tau + nT_C$ where T_C is the period characterizing the given frequency channel). This phenomenon is called phase ambiguity which is a consequence of the fact that the NL receive phase-locked spikes [10]. We call the value τ "real ITD", and the different $\tau + nT_C$ values "virtual ITDs". Phase ambiguity can be eliminated with the convergence of multiple frequency channels, because the detection corresponding to τ takes place at the same neuron in each channel, but the values of $\tau + nT_C$ vary from channel to channel. This convergence is the key to the resolution of phase ambiguity. Experiments of M. Konishi and his co-workers show that a single space-specific neuron (in the ICx) receives inputs from a tonotopically organized array of neurons which are tuned to the same ITD and its phase equivalents [19].

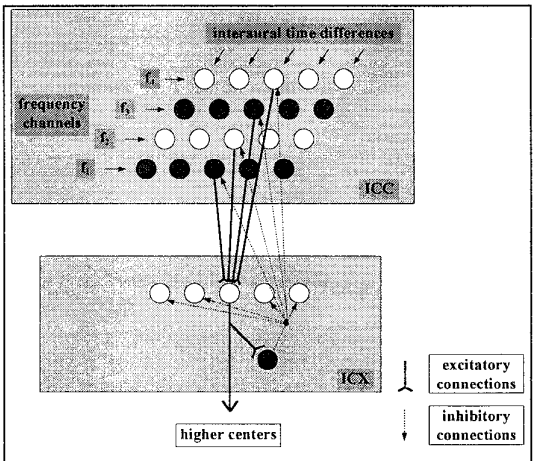

Figure 9: *Model of the circuit resolving phase ambiguity. ICc neurons coding the same ITD but tuned to different frequencies converge to an ICx neuron (space-specific neuron). This neuron excites an inhibitory interneuron which inhibits the neighboring space-specific neurons (ITD selectivity) and the neurons with different frequencies*

Here we shortly summarize our results. We have developed a model in which the different frequency channels not only converge, but there is a *feedback inhibition* in the ICx which is assumed to cancel the responses to the virtual interaural time differences. The structure of this part of the auditory pathway model is given in Figure 9 and the CNN template for the converging frequency channels with feedback inhibition is given in Appendix 2 of [16]. We used two kinds of input: first three tones of different frequencies, then a stereophonic signal of a short, sharp noise processed through a basilar

membrane simulation. In this latter experiment 10 frequency channels were used. In both cases we concluded that only by use of the convergence of the different frequency channels - without inhibition - the phase ambiguity was partly reduced. Feedback inhibition, however, further suppresses the secondary peaks corresponding to virtual ITDs (see Figure 15 in [16]). It can also be observed that the inhibition resulted in a decrease in the number of spikes which is again in agreement with physiological data (see [4]).

Conclusions

A cellular neural network of the time-coding pathway of sound localization have been presented. On basis of the model we have shown that inhibition must play a crucial role in sound localization and have drawn some interesting conclusions concerning the possible types of inhibition. In addition, we have shown that a kind of hyperacuity in time can be observed in the sound localization system.

Acknowledgements

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