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Tuning Neurons to Interaural Intensity Differences Using Spike Timing-Dependent Plasticity

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***Abstract*— Mammals are known to use Interaural Intensity Difference (IID) to determine azimuthal position of high frequency sounds. In the Lateral Superior Olive (LSO) neurons have firing behaviours which vary systematicaly with IID. Those neurons receive excitatory inputs from the ipsilateral ear and inhibitory inputs from the contralateral one. The IID sensitivity of a LSO neuron is thought to be due to delay differences between both ears, delays due to different synaptic delays and to intensity-dependent delays. In this paper we model the auditory pathway until the LSO . Inputs to LSO neurons are at first numerous and differ in their relative delays. Spike Timing-Dependent Plasticity is then used to prune those connections. We compare the pruned neuron responses with physiological data and analyse the relationship between IID's of teacher stimuli and IID sensitivities of trained LSO neurons.**

***Keywords*— Interaural Difference, Lateral Superior Olive, Spike Time-dependent Plasticity**

I. Introduction

MAMMALS use Interaural Intensity Difference (IID) to determine the azimuth of high frequency sound sources [1], [2]. Due to the shadowing of the head, the intensity perceived by the ear ipsilateral to the sound will be higher than the one on the contralateral side. This shadowing effect differs depending on the sound source position and thus the IID can code for the latter.

In one of the first brain centers to process binaural auditory information i.e., the Lateral Superior Olive (LSO), the neurons show a firing rate dependent on IID. The rate is maximum when the sound is somewhere at the ipsilateral side and decreases as the sound becomes more centered to reach zero at a certain IID value. Neurophysiological experiments [2] have shown that intensity-dependent latency of LSO inputs can explain the firing behaviour of particular neurons for changing IID. Differences between neurons i.e., the shaping of IID selectivity among LSO cells, is thought to be based on two features: the relative arrival times of the inputs from the two ears differ among cells (the latency hypothesis) and, to a lesser extent, firing thresholds between excitatory and inhibitory inputs are different. Neurophysiological experiments [5], [4] have shown that a majority of the neurons in the LSO behave conforming to the latency hypothesis whereas only a minority fit the threshold model.

LSO neurons receive excitatory inputs from the ipsilateral ear and inhibitory inputs from the contralateral one. It has been shown [9] that between birth and maturity the number of input connections, both excitatory and inhibitory, decrease with age suggesting that learning takes place by pruning connections.

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In this paper we simulate the training of LSO neurons using Spike Timing-Dependent Plasticity (STDP). The inputs from both ears differ in their delays, we therefore only characterise neurons which conform to the latency hypothesis. We compare the responses of the trained neurons to those found in neurophysiological experiments. We then investigate to what extent the IID of the teacher stimulus has an effect on the IID sensitivity of the trained neurons. Other training parameters are also varied and their variations are related to the corresponding IID sensitivity changes. This allows us to exert control over the resulting IID sensitivity curves.

II. Peripheral auditory system processing

The joint time-frequency analysis performed on the incoming signal is modeled on the transduction stage located in the inner ear (cochlea). A simple model of this analysis [1] is a filter bank consisting of parallel band-pass filters with subsequent envelope extraction (half wave rectification and low pass filter) in each channel.

The analog output of this time-frequency analysis models the operation of the Basilar Membrane (BM). Inner Hair Cells (IHC), which are situated along the BM, convert local motion of the BM into amount of neurotransmitter released. Several spiral ganglions synapse with the same IHC. The SGC fire an action potential if their membrane voltage due to the neurotransmitter released by the IHC exceeds a threshold.

We model the spike generation of one SGC as in [3] where the firing probability per unit of time conditioned on the last spike occurring at time t_{last} is

$$\rho(t \mid t_{last}) = q(I(t)).w(t - t_{last}) \quad (1)$$

$q(I(t))$ depends on the intensity of the signal and on a fixed threshold. The recovery function $w(t - t_{last})$ depends on the history of the SGC i.e., it takes into account refractory time.

III. Lateral Superior Olive

A. Physiology

The Lateral Superior Olive (LSO) is located in the brainstem in the superior olivary complex. Most of its neurons receive their excitatory inputs from the ipsilateral ear through the cochlear nucleus and inhibitory inputs from the contralateral ear through the medial nucleus of the trapezoid body (MNTB). The different connections can be seen in Fig.(1)

Neurons in the LSO have firing behaviours which depend on the IID between both ears. In [4], firing rates of neurons in the LSO of Mexican free-tailed bats were measured. Earphones were placed in the funnel of each pinna. The acoustic stimuli were 2msec duration downward frequency-modulated (FM)

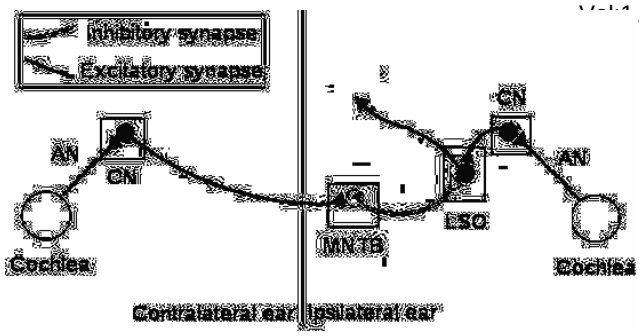

Fig. 1. Auditory pathway until the LSO. AN: Auditory Nerve, CN: Cochlear Nucleus, MNTB: Medial Nucleus of the Trapezoidal Body, LSO: Lateral Superior Olive.

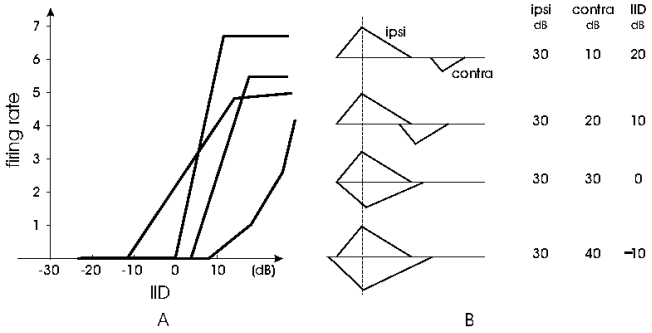

Fig. 2. A: Firing response of different LSO neurons when the IID is varied. B: Mechanisms explaining the firing rate curve of an LSO neuron with complete inhibition at 0B IID [4].

sweeps with a rise-fall time of 0.2msec. The frequency swept down from 5 kHz above to 5 kHz below the best frequency of the unit under measurement.The intensity at the ipsilateral ear was kept constant (20dB) while the contralateral intensity was varied between -10dB to 50dB in 5dB steps. Every neuron had a different response rate vs IID but all resemble the ones sketched in Fig.(2A). We adopt the convention where IID is the difference between ipsilateral intensity and contralateral intensity.

Although the responses are different they do have the same sigmoid form shifted along the abscissa. A standard response feature allowing the characterisation of its IID sensitivity is the IID of Complete Inhibition (IID_{CI}). IID_{CI} is the IID where the neuron passes from a firing to a completely silent state.

B. Model

One particular LSO neuron's response is thought to be a result of two processes [4]: the strength of the input inhibition increases with contralateral intensity and the latency of the input inhibition decreases with contralateral intensity. This is illustrated in Fig.(2B) for a cell having complete inhibition at 0 db IID. When the contralateral intensity is lower than the ipsilateral, the total post-synapse potential at the contralateral side is lower and arrive later, allowing the LSO neuron to fire. At 0 dB IID, both synapses have the same post-synaptic potential simultaneously and the neuron becomes silent.

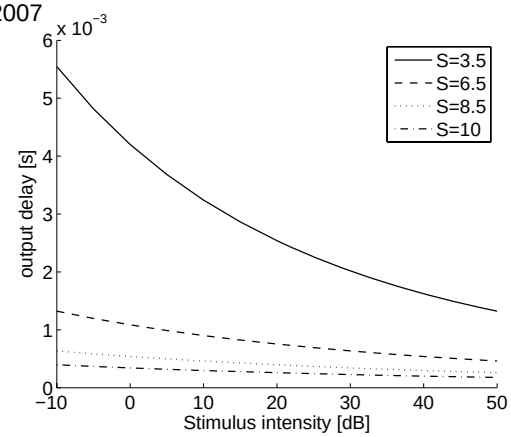

Fig. 3. Intensity-dependent output latency of 4 neurons with different transient sensitivity as a function of the stimulus intensity.

The IID curves vary greatly among cells and have complete inhibitory IID different than zero. For the majority, the shaping of IID selectivity among LSO cells relies on latencies [4]. The so-called latency hypothesis is based on two features: (1) the relative arrival times of the inputs from the two ears differ among cells and (2) changes in the relative intensity of the stimuli at the ear shift the latencies of the inputs. For example, if the contralateral input arrives after the ipsilateral, the contralateral intensity must be increased to achieve coincidence and thus the IID of complete inhibition is shifted.

Relative arrival time between excitation and inhibition is due to numerous factors like different path lengths, different axonal diameters, etc. Mechanisms for Intensity-Dependent Latency (IDL) are less well understood but it's a fact that neuron's response latency along the auditory pathway typically shorten with increasing in stimulus intensity[7]. The IDL is already present at the Auditory Nerves (AN) where the decrease in latency with increasing intensities is of the order of 25μs/dB [6]. The IDL range measured cannot be explained by the integration time of a single neuron as its membrane time constant is much too small. Hence IDL is thought to be an effect of more complex processing[6].

It has been shown [6] that IDL functions in AN fibers are invariant functions of maximum pressure acceleration of peak pressure (APP_{max}) and not of rise time or maximum amplitude. APP_{max} of a sound is the maximum of the second derivative of its envelope with respect to time. A latency-acceleration function of AN fibers has been derived from experimental data [6]:

$$L = L_{min} + 13/\left(log(APP_{max}) + S\right)^4 \qquad (2)$$

where L_{min} is the minimum which includes all the delays that are independent of the stimulus magnitude. S denotes a neuronal sensitivity to acceleration of peak pressure or transient sensitivity. Eq.(2) is plotted in Fig.(3) using the stimuli of Sec.(III-A) for different transient sensitivities S.

IV. Learning

STDP has been intensively studied in recent years [11]. Most of the focus, experimental and computational, has been

directed towards excitatory synapses. Although a large proportion of the synapses in the brain are inhibitory, not that much is known about on the learning mechanism of inhibitory synapses.

The development of the LSO of a Gerbil is largely achieved during the first 3 postnatal weeks [9]. There is a structural change in the pathway during development. The number of converging afferents decreases reaching ± 10 for the excitatory inputs and ± 8 for the inhibitory inputs [10]. Therefore MNTB afferent inhibitory synpases are also dynamic with spontaneous discharge having a substantial impact on their development. This suggests that a learning algorithm depending on relative firing times could also be used for inhibitory synapses.

The learning rule we used for the modification of the synaptic weights $w_i^{\pm}$ of an LSO neuron is

$$\frac{dw_i^{\pm}}{dt} = \eta^{\pm} \int W^{\pm}(s) S_i^{pre}(t+s) S^{post}(s) ds \qquad (3)$$

with $w_i^{\pm} \in [0, w_{max}]$. The $\pm$ sign denotes excitatory and inhibitory synapses. $\eta^{\pm}$ is the learning rate. $S_i^{pre} = \sum_{t_i^f \in F_i} \delta(t - t_i^f)$ is a presynaptic spike train. t_i^f denote the arrival times at the synapse and F_i stands for the set of all spike arrival times at synapse i. S^{post} is the output spike train. To avoid unlimited growth we impose an upper and lower bound on the weights. A synaptic change occurs if presynaptic spike arrival and postsynaptic firing time coincide within some window. The window used to train excitatory synapses (Fig.4a.) has the same formulation as in [8] i.e., for $s < s^*$,

$$W(s) = (A - B) exp\left[(s - s^*)/\tau_0\right] \qquad (4)$$

and, for $s \geq s^*$,

$$W(s) = A exp\left[-(s - s^*)/\tau_1\right] - B exp\left[-(s - s^*)/\tau_2\right] \qquad (5)$$

This window has a standard shape for STDP which is a phenomenological model based on experimental data. The window used for the inhibitory synapses (Fig.4a.) is a mirrored version (around the ordinate axis) of the one used for the excatatory synpases and has a longer time offset s^*. Therefore, inhibitory input neurons that have fired before and slighlty after an output spike will have their synaptic weights decreased. This way, we ensure that excitatory inputs which induce output firing will not be inhibited. The non-pruned inhibitory inputs will arrive later and then require a higher contralateral intensity to arrive simultaneously with excitatory inputs.

The prelearning architecture consists of an Leacky Integrate and Fire (LIF) neuron connected with 50 inputs from the excitatory ear and 50 from the inhibitory ear. All neurons have the same transient sensitivity $S = 3.5$ which gives a mean IDL of $\pm 70 \mu$s/dB. The input synapses differ in their delay which is taken linearly between 0 and 3ms. The weights are chosen randomly between 0 and w_{max} for the excitatory synapses and between 0 and $w_{max}/2$ for the inhibitory synapses to ensure initial firing which will trigger the learning process.

The stimuli parameters (signal duration and amplitude, IID range considered) are the same as in the experiment introduced in Sec.(III-A). The parameters of the network and the windows are given in [12]. First, the system is presented with 1000 stimuli corresponding to an IID of 15dB. Every time an output spike is triggered the weights are updated. After all presentations almost all weights are either zero or have reached the upper bound. The final weights of synapses of both types are shown in Fig.(4a.). The pruned network is then presented with stimuli whit IID's ranged from -30dB to 30dB and the mean firing response is computed (Fig.(4)b.). The resulting responses have the same shapes as the one measured in neurophysiological experiments.

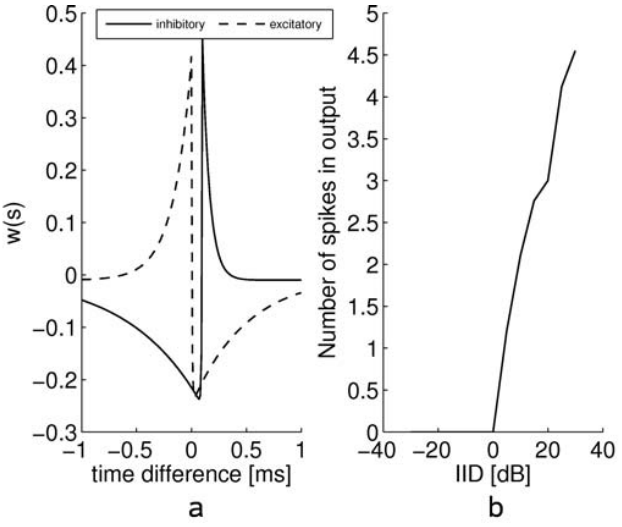

Fig. 4. a: Windows used for the STDP. b: Firing rate when neuron is presented with different IID's. Neuron taught with a stimulus of -15dB IID.

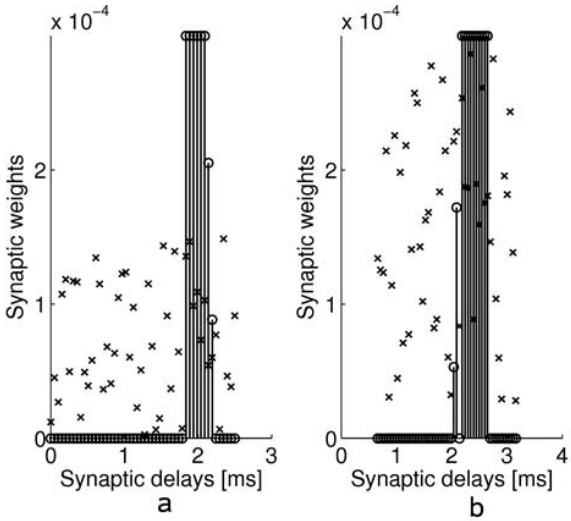

Fig. 5. Learning with a stimulus of -15dB IID. a: weights of the ipsilateral synapses (x for initial values, O for final values). b: weights of the contralateral synapses (x for initial values, O for final values).

Due to the probabilistic intialisation of the learning and the noise in the spike generation model, teaching various neurons with the same stimulus wil not yield the identical results. This can be seen in Fig.(6a.) where each curve represents the probability density function of the IID of complete inhibition (IID_{CI}) when various neurons are trained with the

same stimulus. Despite variance, the distribution has a single mode and thus can be well characterized by its mean. If we now train various neurons with different IID stimuli, we can see an almost linear relationship (Fig.(6)) between the IID of the teacher stimulus and the mean IID_{CI} of the trained neurons. Only IID values corresponding to ipsilateral positions (IID$\geq$0) have been used as teacher because neurons sensitive to ipsilateral sound are predominant in the LSO [2].

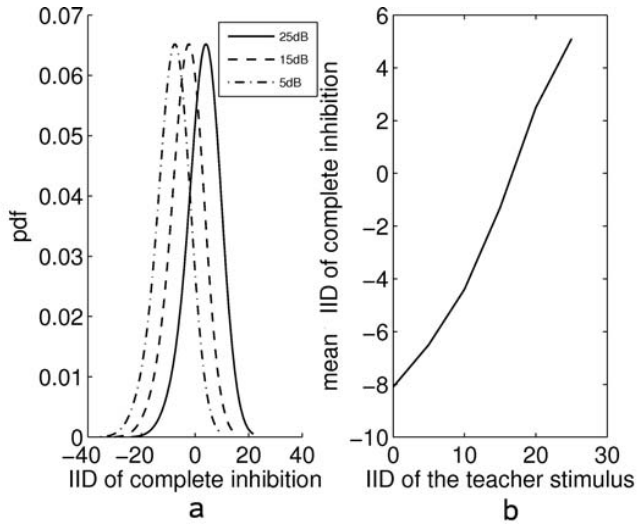

Fig. 6. a: Estimation of the probability density function of the IID_{CI} with 4 teacher IID (25dB, 15dB, 5dB). b: mean IID_{CI} as a function of the teacher IID.

Other direct relationships can be found between mean IID_{CI} and, for instance, parameters of the learning window. For example, s^* of W^-, the time offset of the inhibitory window, can be adjusted to reach a desired shift in the mean IID_{CI} for a fixed teacher IID stimulus (Fig.(7)). A longer time offset s^* of the inhibitory window keeps inhibitory synapses that arrive later (thus shifting the group of synapses in Fig.(5b.) to the right). The inhibitory inputs need a higher intensity to arrive simultaneously and thus the IID_{IC} is shifted.

V. Discussion

We have shown that it is possible to tune LIF neurons to IID using STDP. Our prelearning architecture contains input neurons with deterministicaly increasing synaptic delays and the same transient sensitivity. This allows us to relate certain parameters of the training to the shape of the resulting response (characterized by IID_{IC}). The IID of the teacher, for instance, influences the sensitivity of the trained neuron. Parameters of the learning windows can be changed as well to change the resulting sensitivity. This control over the resulting sensitivies is interesting if one wants to further process the output of those modeled LSO neurons to do azimuth estimation. Indeed, it gives one a way to be sure that the IID sensitivities of the neurons cover the whole range under consideration with a limited number of neurons to train.

Nevertheless a biological more plausible scheme would have less control (if any at all) over the learning parameters and have to rely on a larger population so that random variations would allow to cover the whole range of IID's. The resulting IID_{IC} should also have a broader distribution around a mean IID_{IC}. This can be done by changing our prelearning architecture in a more biological way. Indeed, as it is shown in [7], inputs reaching the LSO, have different transient sensitivities. We can thus use input neurons which have different transient sensitivities and random relative synaptic delays and prune those inputs. We are currently investigating to what extent this more realistic distribution of the network parameters affects the resulting IID sensitivities.

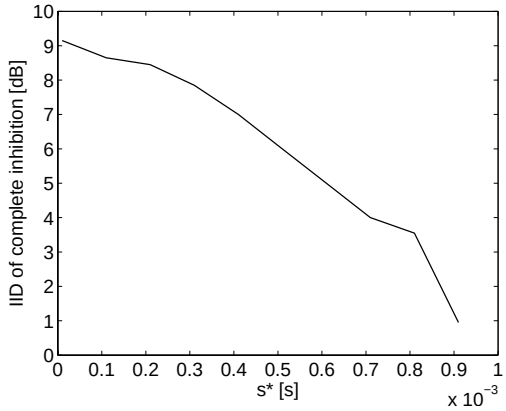

Fig. 7. Mean IID_{CI} as a function of the time offset s^* of the inhibitory window.

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[11] Gestner, W., Kistler, W.N.: Spiking neuron models. Cambridge University Press, Cambridge (2002)
[12] The networks have been simulated using the Csim Toolbox available at http://www.lsm.tugraz.at/csim The parameters of the LIF neurons are: R_m=1MΩ, τ_m=20ms, V_{thresh}=-0.045V, $V_{resting}$=-0.06V, V_{reset}=-0.06V, V_{init}=-0.06V, $T_{refract}$=1ms. The parameters of the synapses are τ_{Contra}=2ms, τ_{Ipsi}=0.5ms, W_{max}=1e-4. The parameters of the windows are A^+=2/3, B^+=0.25, η^+=50e-6, τ_0^+=1/10ms, τ_1^+=1/200ms, τ_2^+=1/2ms, s^{*+}=-1/800ms, A^-=0.7, B^-=0.25, η^-=50e-6, τ_0^-=1/5ms, τ_1^-=1/20ms, τ_2^-=1/2ms, s^{*-}=-0.1ms.