On Lateral Inhibition in the Auditory System

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Abstract. Suga (1994) has presented arguments supporting the existence of lateral inhibition in the auditory system. We developed a computational model of a lateral inhibition neural network possibly taking part in auditory stimulus processing. The behavior of the model under several hypothetical auditory stimuli matches experimental results. We have shown that lateral inhibition can be the cause of tinnitus in some conditions. 95% of subjects have phantom perceptions in the absence of auditory stimulation (acoustically shielded chambers). We suggest that the spontaneous activity in the auditory nerve (a Poisson-like neural noise), processed by lateral inhibition, is responsible for this phenomenon. Activity is generated in the output layer of the network under stimulation with a Poisson-like noise. In the presence of acoustic stimulation this neural noise is masked by the stimulus or the ambient wideband acoustic noise. The wideband noise is effectively suppressed in lateral inhibition. The shape of the probability density function of the noise determines whether it will be effectively suppressed. The edge effect has been demonstrated to be also a possible consequence of lateral inhibition.

Key words: Auditory system — Lateral inhibition — Tinnitus — Edge effect — Computer simulation — Neural networks

Introduction

Computer simulations of lateral inhibition neural networks have been used to demonstrate correlations of the model behavior with experimental data. The currently predominating opinion is that no lateral inhibition (LI) and no frequency tuning curves sharpening are present in the auditory system. It has recently been disputed by Suga (1994). He demonstrated the sharpness measure used in hearing research (Q_{10}) to be inappropriate for sharpness of tuning curves evaluation.

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We present findings supporting the existence of a LI network in higher stages of auditory processing. First we shall show the usefulness of LI in afferent signal processing and shall analyse the possible architectures of LI from that standpoint. It shall be demonstrated how the model can explain some phenomena in auditory perception. Experimental data supporting the concept of LI in the auditory system are presented.

LI is being hold responsible for a number of self-organizing processes in biological systems. It is based on the concept of suppression by a given element on its topologic neighbors. Such a very simple principle can be even responsible for e.g. the complex patterns of neurite branching (Li et al. 1992). The first description of LI dates back to the work of Hartline (1949) who studied the compound eye of the horseshoe crab. Lateral inhibition is an ubiquitous phenomenon, assumed to be operative in all sensory systems (von Bekesy 1967). It was introduced to auditory physiology due to the observed frequency tuning curves sharpening in the centripetal direction (Katsuki 1966). Even more, the traveling wave mechanism (von Bekesy 1960) seemed to be too rough to account for the high frequency discrimination of the human ear. The need for a second filter arose (Evans and Wilson 1975), which might be represented by a lateral inhibition neural network in the spiral ganglion. Computer simulations indeed confirmed such expectations. The sharpening of the input can be easily demonstrated in artificial neural networks with lateral inhibitory couplings (Majernik and Kral 1993; Rozsypal 1985).

The discrepancy in tuning between the traveling wave and the neural frequency tuning curves of auditory nerve units (and also psychophysical tuning curves) arose due to the non-physiological stimulus intensities used by von Bekesy in his experiments. He was restricted to visual microscopy. It forced him to evoke very large excursions of basilar membrane so as to be visible when illuminated by stroboscopic light. The stimulus intensities had to be high above 100 dB SPL. Rhode (1978) pointed to the non-linear behavior of the organ of Corti in respect to stimulus intensity. Similar conclusions have been drawn in psychoacoustics (Majernik and Kaluzny 1979). These were already the signs that the generalizations from von Bekesy's experiments were not justified for lower intensity stimuli. For lower intensities the tuning of the basilar membrane, as could later be demonstrated thanks to the introduction of laser velocimetry (Khanna and Leonhard 1982) and Mössbauer techniques (Sellick et al. 1982), is much sharper. In fact, it is actually the same as the tuning of auditory nerve units. Russell and Sellick (1977) managed to register the receptor potential of inner hair cells in the cochlea. The inner hair cells have essentially the same tuning as auditory nerve units. The reason for such fine frequency resolution has been found in the fast active contraction of outer hair cells (Brownell et al. 1985; Zenner at al. 1985, for review, see Keidel 1992, or Plinkert and Zenner 1992). The contractions increase the sharpness of mechanical tuning curves of the basilar membrane. These observations support the so-called negative

resistance model of the basilar membrane function developed by Neely and Kim (1983, 1986), which shows a very good match with experimental neural data. Hence we can state that the basilar membrane mechanics can explain the sharp frequency tuning of the auditory nerve units. (Even more, Braun (1994) proposed a hypothesis that the basilar membrane mechanics serves a damping function and is not concerned with frequency discrimination at all, which the author has attributed solely to hair cells.) The active processes on hair cells can be monitored by registrating of otoacoustic emissions (Kumpf and Hoke 1970; Kemp 1978). (For review on frequency tuning of the basilar membrane, see Patuzzi and Robertson 1988, for review on otoacoustic emissions see Probst 1990).

All these observations have led to the rejection of the hypothesis of the second filter in the spiral ganglion. Aitkin and Webster (1972) and Calford et al. (1983), using the Q_{10} parameter as a tuning curve sharpness measure, even contradicted Katsuki's observation about frequency tuning sharpening in the centripetal direction. The question of lateral inhibition in auditory research has been controversial since. But lateral inhibition has been demonstrated in the visual system as a mechanism responsible for contrast enhancement (Coren 1991). Similar findings supporting the existence of LI exist for the olfactory system (see e.g. Wellis and Kauer 1993), somesthesia (Kandel and Jessell 1991), hippocampus (Sloviter and Brisman 1995), neocortex and some other parts of the nervous system (see also Grossberg 1987).

The architecture of LI networks can be classified according to several criteria. Von Bekesy (1967) differentiated between 4 types of LI architectures: simple, forward, backward and central. We prefer functional criteria of classification. The function of LI can be seen in the following areas:

1. Sharpening of input excitation curves (redundancy reducing process) (Majernik and Kral 1993; Rozsypal 1985; Kandel and Jessell 1991).
2. Functional separation of distinct areas or pathways in the nervous system (e.g. in the hippocampus: Sloviter and Brisman 1995, or the cortical modules in the neocortex: Marin-Padilla 1969, 1970).
3. Maximum finders (Lippmann 1987).

A possible function of LI network in higher stages of auditory processing (central to auditory nerve) is to increase or preserve the sharpness of the frequency tuning curves despite a high convergence of afferent fibers (Rhode and Greenberg 1992). Without it the sharpness of the frequency tuning curves of neurons would deteriorate in the centripetal direction. Consequently, the frequency discrimination of hearing would be poor. LI should be related to sharpening of rate-place profiles, contrast enhancement and wideband noise suppression in the auditory system. It has also been hypothesized that a decrease of activity in only a subpopulation of neurons, possibly further enhanced by lateral inhibition, could be responsible for

tinnitus (edge-effect, Penner 1980; Salvi and Ahroon 1983). This might be of special importance for sensorineural hearing loss, a condition often accompanied by tinnitus. Such considerations are supported by the high incidence of tinnitus with sensorineural hearing loss. The patients frequently report the pitch of the tinnitus as corresponding roughly to the transition point from normal hearing to that of adjacent frequency with elevated threshold (for a detailed review on the tinnitus mechanisms hypotheses see Møller 1984; Hazell 1987; Jastreboff 1990). Such tinnitus is then further maintained by focusing attention to it, and so no habituation can take place (Hazell and Jastreboff 1990). Plastic changes of tonotopic sound representation following hearing loss, especially the reported edge frequencies representation expansion (Robertson and Irvine 1989), thus do not further influence the distressing phantom perception. Edge effect has also been demonstrated in psychoacoustics (Houtgast 1972, for review see Moore 1993), which further justifies the concept of lateral inhibition in hearing research. Houtgast's work obtained strong support by demonstrating that forward masking does not take place on the basilar membrane but is indeed the result of a central neural processes (Kemp and Chum 1980; Scherer 1984; Gobsch et al. 1992).

We suggest that the spontaneous activity of auditory nerve fibers, reshaped by a neural network with lateral inhibitory couplings, might also account for tinnitus in the absence of appropriate acoustic stimuli and ambient acoustic (white) noise. 95% of subjects (without any demonstrable hearing pathology) perceive tinnitus in the absence of external acoustic stimuli (in acoustically shielded chambers, Wegel 1931; Heller and Bergman 1953; Meyerhoff and Ridenour 1992). It might be the result of resting activity of cochlear nerve fibers processed in the afferent auditory pathway (in lateral inhibition). This spontaneous activity is a Poisson-like noise. In every-day situations it is masked by ambient acoustic noise. The ambient acoustic noise is a wideband noise, which should be effectively suppressed in LI.

Methods

The function of a natural neural network cannot be explored directly in empirical experiments yet. This restriction can be overcome by Gedankenexperiments, mainly through computer simulations (Kral 1994). This study concerns the behavior of a hypothetical LI network and the consequences it carries for the function of afferent auditory pathway. The model is based on the current knowledge of informatical aspects of LI and on physiological data. The criteria for such a model are: effective sharpening of input excitation curves and no "spurious activity" (activity not corresponding to the coding of the given input stimulus) in the network with arbitrary input (most models are designed for particular types of input). We implemented an architecture favored by Reichardt and MacGintie (1962) as it is superior in performance in the above-mentioned criteria. We do not simulate neural plasticity of

the afferent auditory pathway in the model. The plasticity is clearly documented during the development and in pathologies of the inner ear (for review see King and Moore 1991; Clarey et al. 1992; Benuskova 1988). It does not seem to be of significant importance in "normal" adult subjects. Adults have their tonotopic map fairly stable, without substantial changes in physiological conditions. The relative simplicity of the model enables the simulation of a large network and therefore a good resolution of the input and output excitation curves.

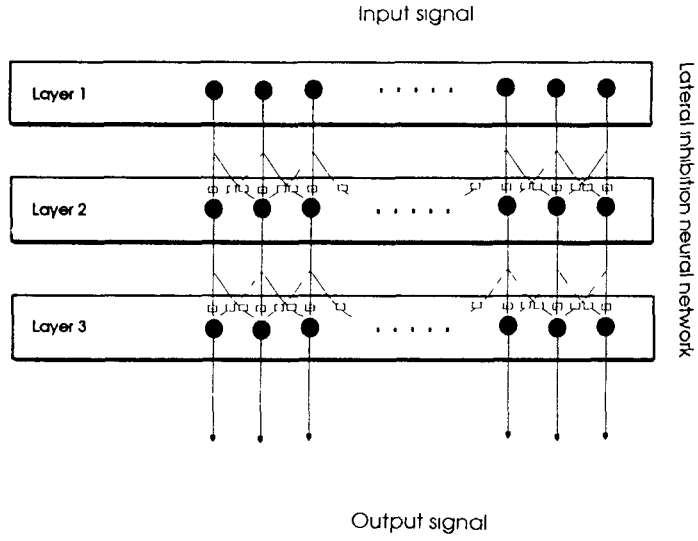

Figure 1. Illustration of the architecture of a multilayered lateral inhibition artificial neural network.

We consider artificial neural networks of L one-dimensional layers consisting of N neuron-like elements (processing elements – PEs). The processing elements of k-th layer are only connected with PEs of $(k+1)$th layer (see Fig. 1). Denoting the activity (pulsation) of x-th processing element in k-th layer as $\varphi_k[x]$ then the activity of i-th PE in $(k+1)$th layer is given by the equation

$$\varphi_{k+1}[i] = f\left(\sum_{i-M}^{i+M} w[x]\varphi_k[x] - \Theta\right) \tag{1}$$

where f is the linear threshold function shown in Fig. 2; $w[x]$ are the corresponding connection weights; Θ is the neuron threshold value. The given PE is directly connected to $2M+1$ PEs in the subsequent layer. There are different types of

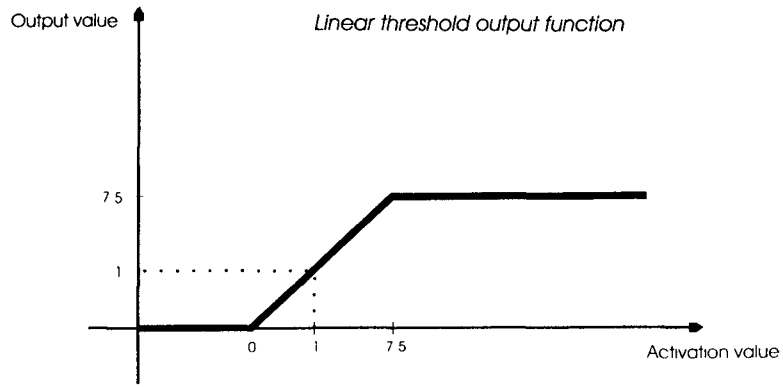

Figure 2. Input-output function of a processing element.

one-dimensional networks according to the weights taken in equation (1). If $w[i]$ is always positive (i.e. the coupling between i-th PE in k-th layer and i-th PE in $(k+1)$-th layer is an excitatory one) and $w[x]'s(x = i - M,\ i - M + 1,\ i - M + 2, \cdots\ i - 1, i + 1, \cdots\ i + M)$ (i.e. the connection between x-th PE in k-th layer and the i-th PE in $(k+1)$-th layer) is an inhibitory one, then we have a lateral inhibition network. Each one-dimensional lateral inhibition neural network is given by the following parameters:

1. the excitatory weight $w[i]$
2. the inhibitory weights $w[x]$
3. the threshold value Θ
4. the connectivity defined as $c = (2M + 1)/N$.

According to the choice of these parameters, there is a variety of possible LI neural networks. In computer modeling the real one-dimensional L-layered neural network is represented by a recursive network (i.e. the PEs' outputs are led back to the input of the network). Here the k-th iteration in the computer model represents the state of k-th neuron layer of the real network.

The model implementation consists of 1000 PE, with $c = 0.11$ $(M = 55)$, $w[i] = 1.5$, $w[x] = -0.015$, $\Theta = 0.25$. The PE thus possesses one excitatory $(w[i] = 1.5)$ and 2M inhibitory connections $(w[x] = -0.015)$ to other PEs. Such a network does match the proposed synaptic couplings architecture among cortical A cells (and possibly also among type II DCN units, Shamma and Symmes 1985). This architecture shows behavior without "wrong maxima" (a maximum on the output excitation curve which was not present in the input, Majernik and Kral 1993) or any other form of spurious activity. The excitation curves are thought to represent an analogy to rate-place profiles (firing rate versus characteristic frequency of the

neuron – Sachs and Blackburn (1991)). The neural activity in the auditory nerve was considered as input to the LI network.

Results

The sharpening of input excitation curves in the proposed network is shown in Fig. 3. The weights were chosen according to the sharpening measures suggested by Majernik and Kral (1993) (an analysis of the parameter space of the LI network can also be found in that paper). When simulating a rate-place profile with two maxima (corresponding to a spectrum of the auditory stimulus with two maxima), the network separates the evoked activity (Fig. 4). This demonstrates the contrast enhancement function of LI. Rectangular ("white") noise is suppressed in the network (Fig. 5). All these effects meet the expectation of LI behavior based on physiological observations. These are well-known areas where lateral inhibition might play an important role.

Edge effect

The edge effect responsible for tinnitus in patients with sensory-neural hearing loss can be demonstrated in a LI network (Fig. 6). The functional impairment of the inner ear results in a (more or less) step-like fall out in the rate-place profile (threshold shift). These steplike borderlines of the defect are amplified due to the contrast enhancement ability of the LI network with $c < 1.0$, provided the extent of the defect is larger than M.

Spontaneous activity of auditory nerve fibers and tinnitus

LI tends to process noise with an uneven function of probability density of interspike intervals in a nonlinear way: certain parts of the input excitation curve are enhanced whereas others are suppressed (Fig. 7), contrary to the processing of wideband (rectangular) noise. Spontaneous activity of auditory nerve fibers is a result of a Poisson-like process (Kiang et al. 1965; Eggermont 1990, but see also Teich et al. 1990), and is the generator of spontaneous activity in the auditory system (for details see, e.g. Vossieck et al. 1991). We thus have a neural noise with uneven function of probability density of interspike intervals in the afferent auditory system in the absence of acoustic stimuli, possibly being the source of tinnitus due to LI network processing. The spontaneous activity is masked by the ambient acoustic noise (a wideband noise) or other acoustic stimuli in natural conditions.

Discussion

A common question related to the LI models concerns the problem of short-range excitation in LI networks. There are several models of such an architecture (e.g.

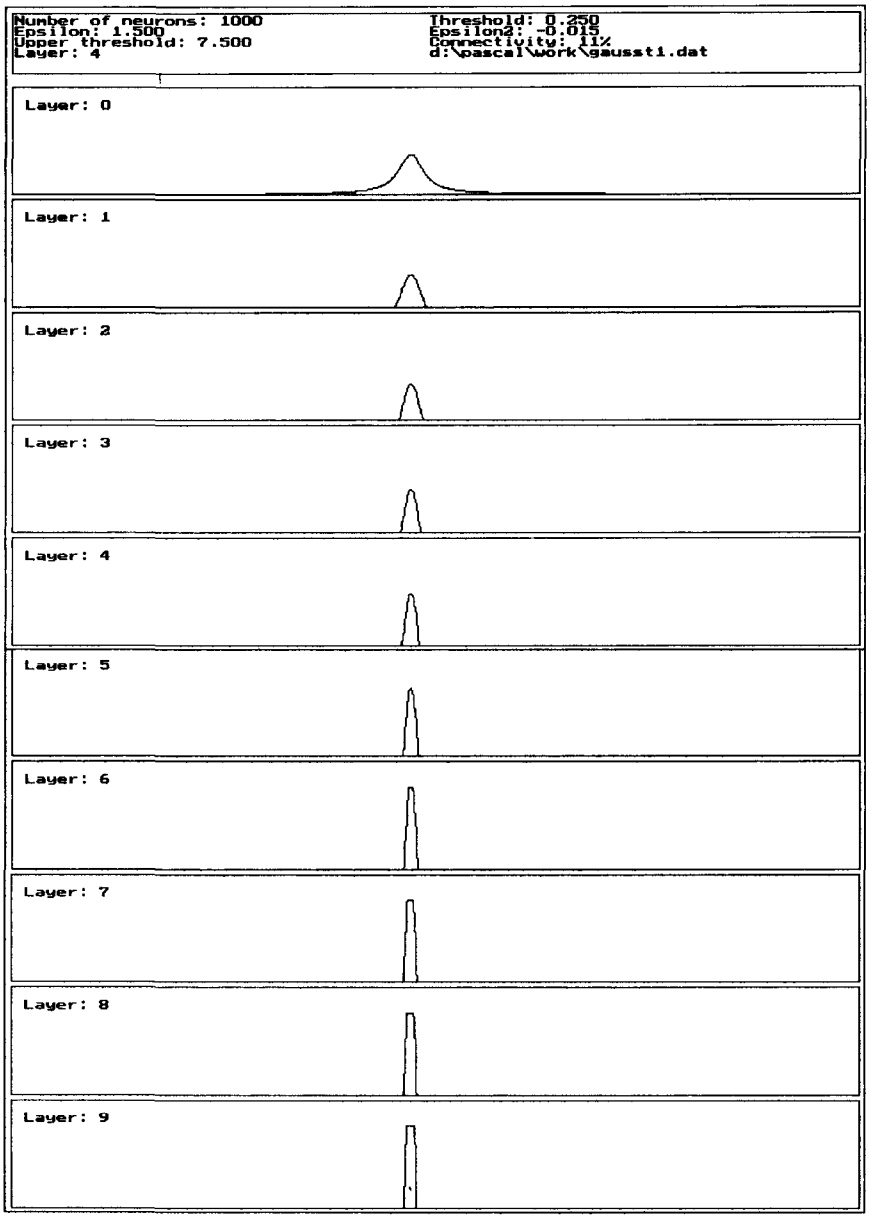

Figure 3. Sharpening of excitation curves (firing rate versus ordinal neuron number in the tonotopic array) in LI network. The network parameters are in the top window (epsilon being the value of the excitatory weight, epsilon2 being the value of inhibitory weights). Windows below depict the excitation curves (firing rate on the ordinate) in the corresponding layers of the network, whereas Layer 0 represents the input excitation curve.

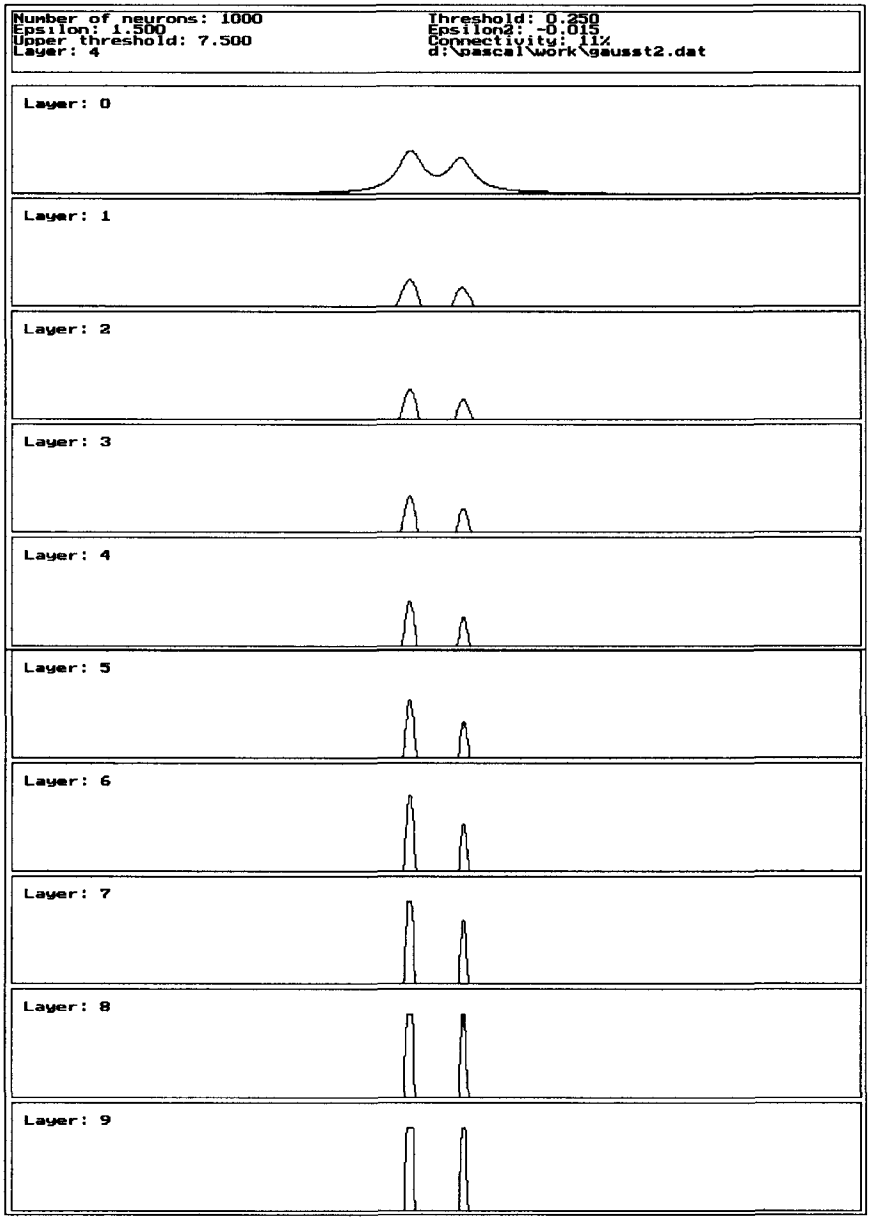

Figure 4. Contrast enhancement of the excitation curves in lateral inhibition. The merged excitation patterns in the input are separated so that the corresponding two acoustic stimuli are clearly discernible on the output layer.

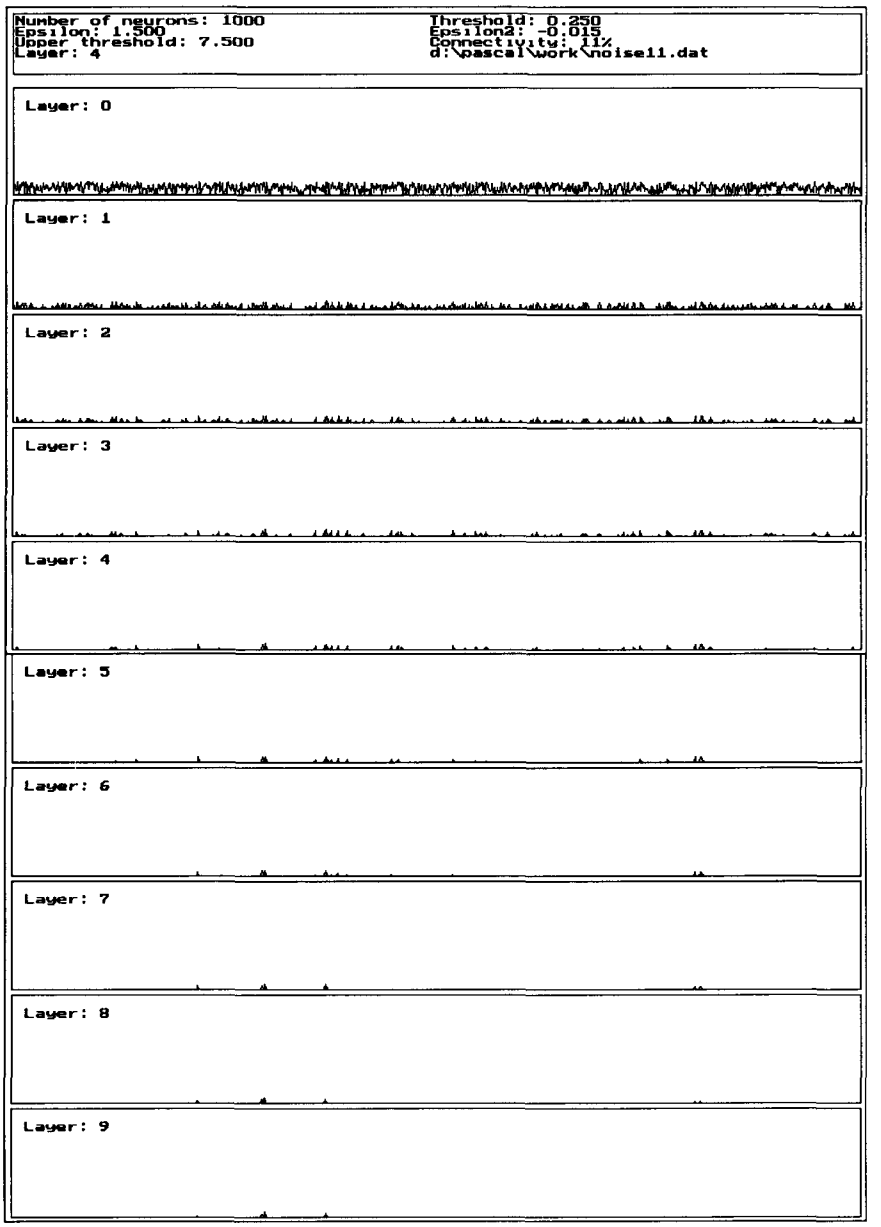

Figure 5. Wideband rectangular noise (a noise with even function of probability density of interspike intervals) is effectively suppressed in the LI network

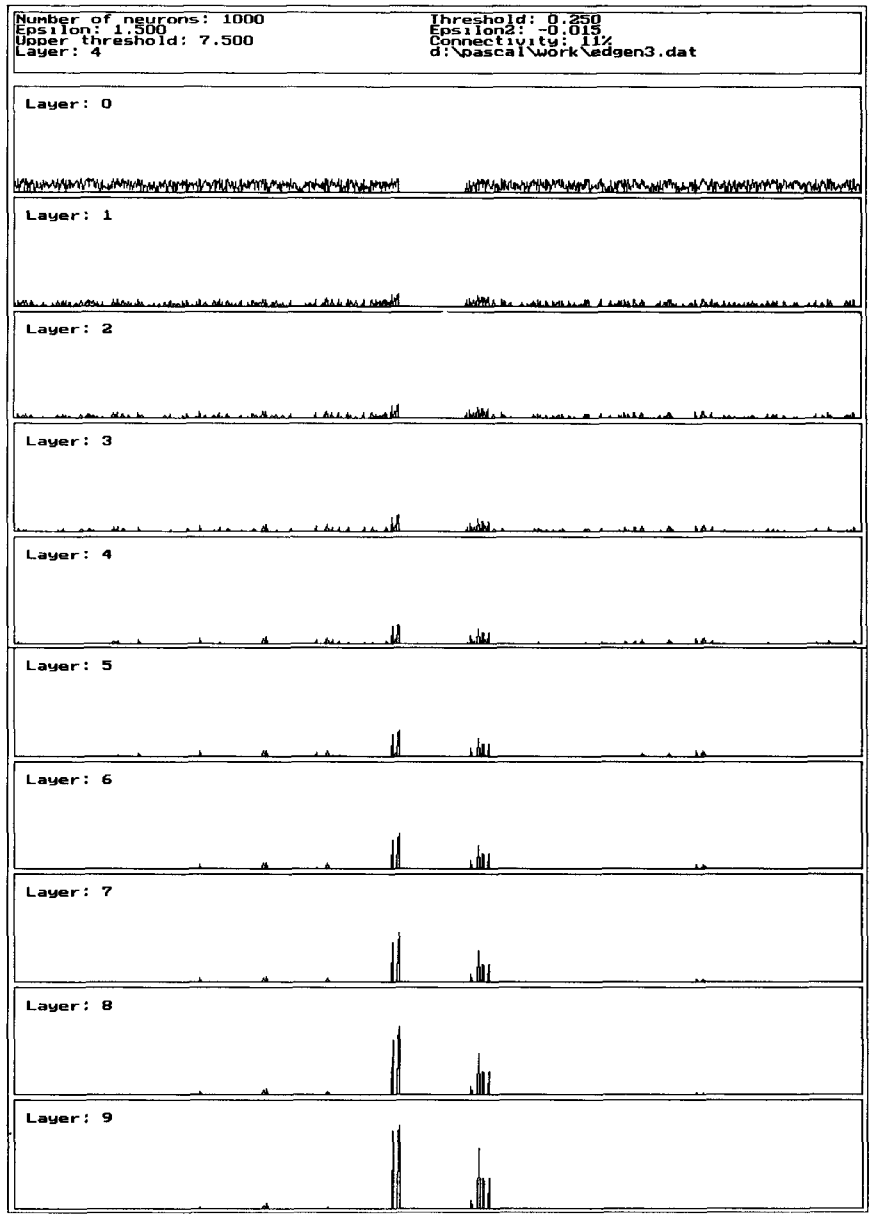

Figure 6. After disruption of a partition in the input excitation curve, the edge effect emerges in the curve processed in the LI network.

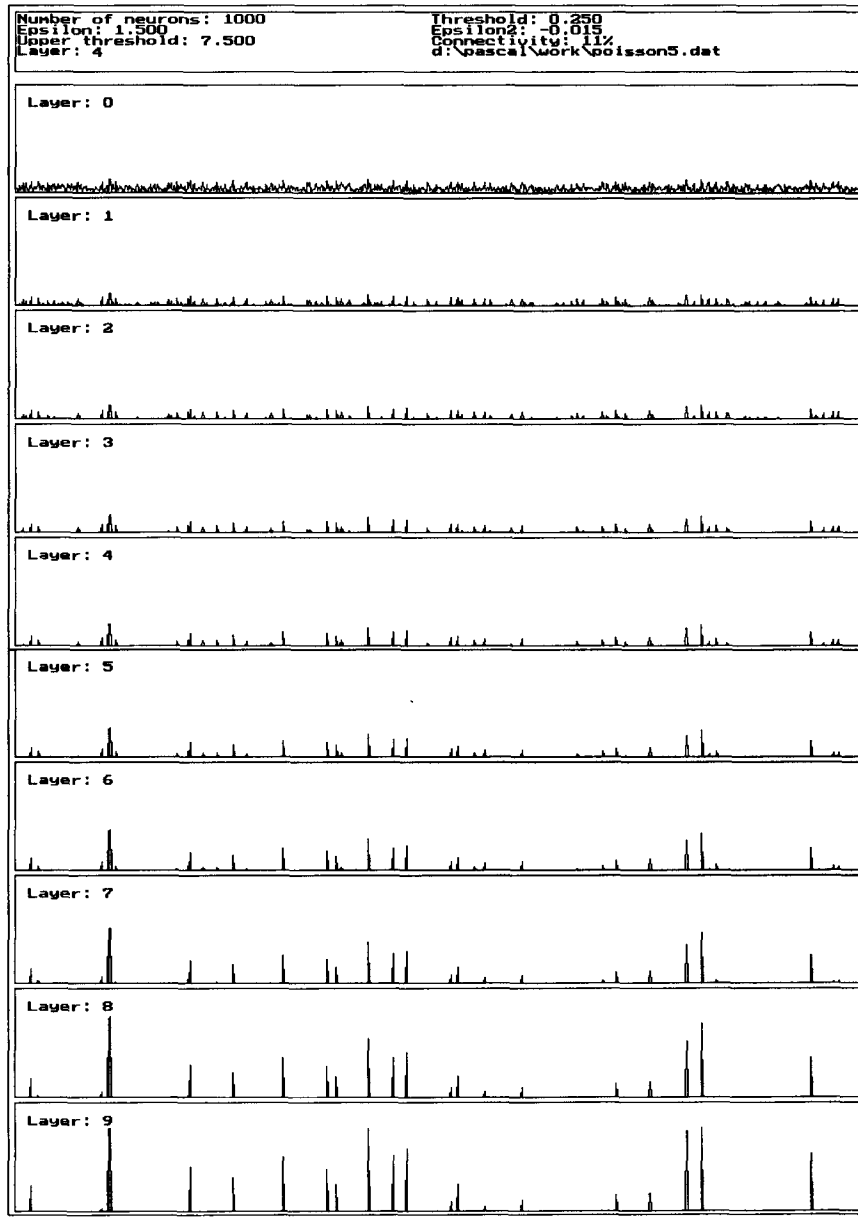

Figure 7. Processing of noise with uneven function of probability density of interspike intervals in LI network. Parts of the excitation curves are significantly amplified, in contrast to the stimulation with a rectangular noise (Fig. 5). Here a Poisson-like noise was used as input.

Kohonen 1982). According to the performance of these models we might include them in the above mentioned functional group 2 (separation of pathways). The rate-of-convergence function of such models weakens the possibility of input-excitation-curve sharpening in a significant extent (Majernik and Kral 1993). This is not surprising: the lateral excitation opposes the sharpening effect of lateral inhibitory couplings. This was the reason why we did not incorporate lateral excitation in our model. In addition, we are not aware of any physiological observations supporting such an architecture in the auditory system. One possible aim of such networks might be the functional division of the neocortex into cortical modules (for review, see Popper and Eccles 1977).

As far as maximum finders (functional group 3) are concerned (Maxnet of Hamming net, see Lippmann 1987), they basically do not differ from our network architecture. The difference is in the connectivity, which is 1.0 in these networks.

There are several observations in auditory physiology which do support the concept of lateral inhibition as outlined above:

1. The high convergence on neurons decreases the sharpness of tuning (e.g., as measured in Q_{10}) (see Rhode 1991). Despite the high convergence there are very sharply tuned units in the auditory system.
2. Several units in the afferent auditory pathway have inhibitory sidebands in their response area (see e.g., Evans and Nelson 1973a, b).
3. A possible function of inhibitory sidebands is to preserve spectral selectivity when there is a convergence of afferent fibers (Rhode and Greenberg 1992).
4. Inhibitory sidebands are not the result of the same mechanism as two-tone inhibition, which originates from the mechanics of basilar membrane motion. Inhibitory sidebands are the result of neural inhibition (for details, see Rhode et al. 1978, 1983; Hirsch and Oertel 1988). One method to selectively inactivate some of these inhibitory inputs is to present a traumatizing tone above a neuron's characteristic frequency. This treatment is thought to damage the hair cells that indirectly activate the lateral inhibitory inputs above the characteristic frequency (Salvi et al. 1990; Boetcher and Salvi 1993). The results of such studies support lateral inhibitory interactions in the auditory system (dorsal cochlear nucleus, interior colliculus).
5. Inhibitory sidebands thus incontestably show inhibitory couplings between neurons representing neighbouring stimulus frequencies, e.g. in cochlear nucleus.
6. There are neurons which are nonmonotonous, sharply tuned, with inhibitory sidebands and non (or weakly) responsive to wideband noise stimulation at several locations in the auditory system. Due to the fact that all auditory nerve fibers have V-shaped response areas, Phillips (1987) concludes that the nonmonotonous response areas in frequency-intensity plane are generated by

inhibitory processes. He further showed that the nonmonotonous response area might represent an area, which has been carved from a V-shaped one by flanking, lateral inhibitory inputs (Phillips 1993). From what has been showed so far it seems that all the above mentioned characteristics tightly correspond with a unit of an LI neural network.

7. Shamma and Symmes (1985) presented a classification of auditory cortical neurons and suggested a model of their interconnections. They included a lateral inhibitory network between type A cells in their model. Type A cells are characterized by nonmonotonous response areas, lateral inhibitory responses to two-tone stimuli, very narrow frequency selectivity (Clarey et al. 1992) and poor responsiveness to wideband noise. The architecture of their LI network matches the model presented herein. Even more, they suggested a similar pattern of interconnections for the dorsal cochlear nucleus, with type II cells corresponding to cortical type A and type IV to cortical type D. As far as DCN is concerned, it has been found that the inhibitory response areas of some (so-called output) neurons correspond well with the excitatory stimulus domains of smaller, neighboring cells. The correlation of spike discharges over time revealed that the activity in these presumed inhibitory interneurons is closely followed in time by suppression of discharges in the DCN output neuron (Voigt and Young 1980), again being in correspondence with an LI network (see also Reed and Blum 1995).

8. As far as the above mentioned sharpening of frequency tuning curves is concerned, Suga (1994) in detail explained the source of discrepancies between the work of Katsuki, who demonstrated the frequency tuning curves (FTC) sharpening in the centripetal direction (sharp, so-called pencil-like FTCs), and the subsequent contradictory results (Aitkin and Webster 1972; Calford et al. 1983). This discrepancy seems to be related to inappropriate usage of the Q_{10} parameter in auditory research. Q_{10} evaluates only the sharpness of the tip of the FTC. Due to the effect of the cochlear amplifier the mechanical FTC on the basilar membrane are very sharp in their low-threshold part. The need for sharpening (and the effect of LI) is highest on the low-steep (high-threshold) skirts of the FTC. Q_{10} can not evaluate this effect (which is the reason for the pencil-like shape of FTCs). According to Suga (1994) the lack of Q_{10}-evaluated sharpening evidence represents an artifact and the original work of Katsuki (1966) remains undisputed.

9. It has been shown that application of bicuculine (a GABA antagonist) in the inferior colliculus decreases the sharpness of the frequency tuning curves (Yang et al. 1992). The sharp shape of the frequency tuning curves is so demonstrated not to be a simple consequence of the organ of Corti mechanics. It is secured by central inhibitory influences, which corresponds to our theory.

There are other indications of LI interactions in auditory systems, e.g. from microiontophoretic studies in DCN (Evans and Zhao 1993) or from frequency resolution in the inferior colliculus (Ehret and Merzenich 1988). Blackburn and Sachs (1990) suggested that the rate-place representation observed in the response of anteroventral cochlear nucleus choppers at high SPLs may be the result of some further sharpening of the spectral representation derived from lateral inhibitory interactions. We have to mention that LI networks have been successfully included in various models of speech processing (see e.g. Kurogi 1991; Farkas 1995).

There is most probably also another mechanism participating in the generation of tinnitus in sensory-neural hearing loss. Hazell (1987) suggested that, as a consequence of the hearing loss, a negative overdampening of the basilar membrane motion by outer hair cells might appear. It is supposed to be caused by the efferent system.

There are no accepted alternative hypotheses for the tinnitus in the absence of auditory stimulation. It can not be explained by perception of the spontaneous otoacoustic emissions due to their much lower incidence (most papers claim a value around 30%, in no case exceeding 50% – see Probst et al. 1986; Probst 1990). Somatosounds can be excluded due to the character of the perception.

Conclusions

The rejection of LI architecture in the spiral ganglion does not rule out the LI concept at higher stages of auditory processing. On the contrary there are numerous papers indicating its existence. The model presented herein matches the hypothesized interconnection matrix based on experimental results. We have shown that such interconnection matrix might be responsible for the edge effect, tinnitus in hearing loss, and tinnitus in the absence of appropriate auditory stimulation (acoustically shielded chambers). There are no experimental methods at hand that could directly verify a certain neural network architecture or evaluate its weight matrix. Keeping in mind the methodological approach to computer simulations of neural networks we can state that LI is likely to significantly contribute to the information-processing function of the auditory system playing especially important role in the frequency discrimination ability and tinnitus pathophysiology.

Acknowledgements. The authors wish to thank the anonymous referees for their comments and Prof. Ivan Hulin, Head of the Institute of Pathological Physiology, Comenius University, for his support. This work was supported in part by grant No. 1/1169/95 of the Ministry of Education and Science of the Slovak Republic to Prof. Ivan Hulin.

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