

Oscillating Mobile Neurons with Entropic Assembling

Eugene Kagan^{1,2} and Shai Yona³

¹*Department of Industrial Engineering, Ariel University, Ariel, Israel*

²*LAMBDA Lab, Tel-Aviv University, Tel-Aviv, Israel*

³*Department of Industrial Engineering, Ariel University, Ariel, Israel*

Keywords: Neural Network, Oscillating Neurons, Mobile Neurons, Neurons Ensemble, Entropy.

Abstract: Functionality of neural networks is based on changing connectivity between the neurons. Usually, such changes follow certain learning procedures that define which neurons are interconnected and what is the strength of the connection. The connected neurons form the distinguished groups also known as Hebbian ensembles that can act during long time or can disintegrate into smaller groups or even into separate neurons. In the paper, we consider the mechanism of assembling / disassembling of the groups of neurons. In contrast to the traditional approaches, we set ourselves to “the neuron’s point of view” and assume that the neuron chooses the neuron to connect with following the difference between the current individual entropy and the expected entropy of the ensemble. The states of the neurons are defined by the well-known Hodgkin-Huxley model and the entropy of the neuron and the neuron’s ensemble is calculated using the Klimontovich method. The suggested model is illustrated by numerical simulations that demonstrate its close relation with the known self-organizing systems and the dynamical models of the brain activity.

1 INTRODUCTION

Artificial neural networks are mathematical models of nervous systems of living organisms. Formally, such networks are the systems of interconnected basic elements – neurons, and their functionality depends on changing connectivity between the neurons. The neurons act according to the activation function and send the output signals with respect to the sum of input signals.

In the traditional considerations of artificial neural networks (Fausett, 1995), (Russell and Norvig, 2010), connectivity between the neurons is defined by certain learning procedures that specify which neurons are interconnected and what is the strength of the connection. Then, the neurons change their states with respect to the sum of the input signals. The output signals are transmitted with respect to the states of the neurons, and after transmitting the outputs the neurons return to their initial states.

In the other approaches mainly used in synergetics (Haken, 2008), the changes of the neurons’ states are directly considered as oscillations with varying period near a neutral state (Kuzmina, Manykin and Grichuk, 2014). The variations of the period in each neuron depend on the oscillations of

the neighbouring neurons. This point of view introduces neural networks into general framework of dynamical systems (Zaslavsky, 2007) and allows their studies using the methods of non-linear dynamics and statistical physics (Klimontovich, 1991). The connectivity between the neurons in such dynamical oscillating systems is defined from “the neuron’s point of view”, where each neuron independently chooses with which neuron it prefers to connect.

Then, there arises a natural question: what factors lead the neurons to connect one with the other and to form the ensembles and what factors lead the neurons to disconnect and to disassemble the existing ensembles?

The popular Hebbian theory (Hebb, 1949) presents the reasons of such choice in the qualitative form and specifies the connection between the neurons in the terms of sparking synchronization; a brief overview of the Hebbian theory and its successors was published by Fregnac (Fregnac, 2003); one of the attempts of mathematical formalization of this theory in the terms of dynamical systems was conducted by Gerstner and Kistler (Gerstner and Kistler, 2002).

In the paper, we present the model of assembling / disassembling of the groups of neurons. Following

the dynamical systems' approach, we set ourselves to "the neuron's point of view" and assume that the neuron chooses the neuron to connect with following the difference between the current individual entropy and the expected entropy of the ensemble.

The states of the neurons are defined by the simple model of the spiking neurons developed by Izhikevich (Izhikevich, 2003, Izhikevich, 2007) on the basis of the well-known equations by Hodgkin and Huxley (see e.g. (Sterratt, Graham, Gillies and Willshaw, 2011)). The entropy of the neuron and the neuron's ensemble is calculated using the Klimontovich method (Klimontovich, 1987), (Klimontovich, 1991).

The suggested model is illustrated by direct numerical simulations that demonstrate its close relation with the Turing system (Turing, 1952), (Leppanen, 2004) and the other models of self-organizing systems.

2 GENERAL DESCRIPTION OF THE MODEL

The suggested model is based on the distributed dynamical system widely known as oscillating active media (Mikhailov, 1990). In this media, each element is considered as an oscillator (linear or non-linear) interconnected with the other elements. As a result, over the media appear the ordered wave structures created by the synchronized and desynchronized oscillations of the elements.

In the model, we consider the neurons as oscillators that can be (or can be not) connected with the other neurons. The interconnected neurons form ensembles that act as oscillating systems, and separate neurons act as point-like oscillators. The last assumption contrasts with the usual assumption applied to artificial neural networks, where each neuron spikes with respect to the input signals received from the other neurons and does not spike without such inputs. The scheme of the considered network is shown in Figure 1.

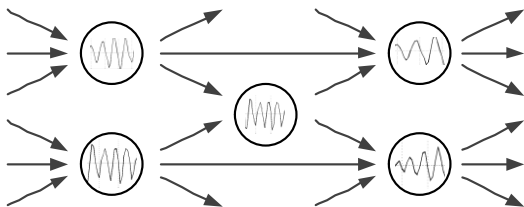


Figure 1: The scheme of the network with oscillating neurons.

Notice that similar networks already appeared in the studies of the Gorkiy physical school, especially in the works by Vedenov et al. (see e.g. Vedenov, Ejev and Levchenko, 1987)); the contemporary developments in this direction are published in the book by Kuzmina et al. (Kuzmina, Manykin and Grichuk, 2014).

The main question that arises in the studies of the networks of autonomous oscillating neurons is:

- what factors lead the neuron to connect with one neuron and to avoid connection with the other neuron?

In the other words,

- what factors lead neurons to assemble and to disassemble?

In the suggested model, we assume that the neurons' interconnections are defined by the difference between the entropies of the separate neurons and the entropies of the neurons' ensembles. The process of connecting and disconnecting is the following.

Consider two oscillating neurons i and j . The equations of their dynamics allow calculation of the entropies h_i and h_j of the neurons and the entropy $h_{ij} = h_{ji}$ of the coupled oscillator that consists of the neurons i and j . We postulate that the neurons i and j create connection between them if

$$h_i > h_{ij} \quad \text{and} \quad h_j \geq h_{ij} \quad (1a)$$

or

$$h_j > h_{ij} \quad \text{and} \quad h_i \geq h_{ij} \quad (1b)$$

Once been interconnected, the neurons act in pair that can create connections with the other neurons following the same entropic criterion. At the moment when the entropy of the pair h_{ij} reaches the value such that the inequality (1) does not hold, the neurons break their connection and continue acting separately. The same reasoning is also applied to the formation of the neuron's ensembles that include more than two neurons.

This model was inspired by the considerations of the neural networks with mobile neurons (Apolloni, Bassis, and Valerio, 2011), especially – by their application to the mobile robots control Kagan, Rybalov and Ziv, 2016). It does not require external learning processes that define the strength of the neurons' interconnections but, in contrast, forms a system that reacts to the inputs by changing internal structure.

The model requires formal equations of the neurons' activity and corresponding methods of entropy calculation. In the next section, we start with the simple model of spiking neuron.

3 THE SPIKING NEURON AND ITS CONNECTIVITY

The considered model of the network of oscillating neurons assumes that the output of each neuron in the network oscillates near some stable value. The well-known model of such oscillating neuron was suggested by Hodgkin and Huxley who considered the dynamics of its electric potential (see e.g. (Sterratt, Graham, Gillies and Willshaw, 2011)).

Starting from the Hodgkin and Huxley model, in 2003 Izhikevich suggested the simpler model that is formulated as follows (Izhikevich, 2003). Denote by u the membrane potential of the neurone and by v the membrane recovery variable that represents the negative feedback for u . Then, the spike of the neurone is defined by the system of two equations:

$$\frac{du}{dt} = 0.04u^2 + 5u + 140 - v + I, \quad (2a)$$

$$\frac{dv}{dt} = a(bu - v), \quad (2b)$$

with the reset condition

$$\text{if } u \geq 30 \text{ then } u \leftarrow c \text{ and } v \leftarrow v + d. \quad (3)$$

In the equations, I stands for the synaptic currents; parameter $a \sim 0.02$ is the time scale of the recovery; parameter $b \sim 0.2$ is the sensitivity of the recovery to the fluctuations of the membrane potential; and parameters $c \sim -65$ and $d \sim 2$ are the after-spike reset values caused by fast and slow fluctuations of the potential, respectively. In the next years, the model (2)-(3) was intensively studied and formed a basis for dynamical models of neural networks (Izhikevich, 2007).

Let us rewrite the system (2) in the form of the oscillator equation that is

$$\frac{d^2u}{dt^2} - \alpha(1 - \beta u) \frac{du}{dt} + \omega u + av = -\frac{dI}{dt}, \quad (4)$$

where following the values appeared in the system (2) $\alpha = 5$, $\beta = -0.016$ and $\omega = ab$. It is clear that equation (4) is the equation of non-linear forced oscillator with the feedback parameter av and external drive $-\frac{dI}{dt}$.

Equation (4) has the same form as the well-known van der Pol equation (see e.g. (Klimontovich, 1991)) and differs from it in the power of u in the "friction" coefficient: in the van der Pol equation it is u^2 and here it is u^1 . Nevertheless, because of its form and the bounds defined by the reset condition

(3) this equation is suitable for calculations of the entropy developed for the van der Pol equation.

Now let us consider the external drive $-\frac{dI}{dt}$. In the Izhikevich model the variable I is defined as a synaptic current, or, in the other words, as a variable that represents the flow via the neuron's inputs and outputs. Then, the value $-\frac{dI}{dt}$ in the equation (4) stands for the changes of the input/output flow that completely meets the Kawahara model of neural interactions (Kawahara, 1980); but again, notice the indicated difference between equation (4) and the van der Pol equation.

In our model, we apply the weak coupling suggested by Rand and Holmes (Rand and Holmes, 1980) and define drive $-\frac{dI_{ij}}{dt}$ between neuron i with the membrane potential u_i and neuron j with the membrane potential u_j as

$$-\frac{dI_{ij}}{dt} = \gamma'_{ij}(u_i - u_j) + \gamma''_{ij}\left(\frac{du_i}{dt} - \frac{du_j}{dt}\right). \quad (5)$$

In the considered version of the model, we assume that $\gamma_{ij} = \gamma'_{ij} = \gamma'_{ji} = \gamma''_{ij} = \gamma''_{ji}$. As a result, the connectivity between the neurons is defined by the single weight parameter γ_{ij} that also can be specified with respect to the entropies of the neurons. In the next section we define these entropies.

4 ENTROPY OF THE NEURONS AND OF THEIR ENSEMBLES

Entropy of the neurons of their ensembles is defined following the method suggested by Klimontovich; in his book (Klimontovich, 1991) this approach is considered in details. The idea of the method is as follows.

At first, consider dynamical equation of the system as the Langevin equation with certain source that describes the random walk. At second, using the Fokker-Plank equation, obtain the probability distribution of locations and velocities of the walking particles. Finally, calculate the entropy of this distribution (relatively to the distribution of the source used in the Langevin equation) that is the entropy of the considered system, in our case – of the neuron.

In the original work, Klimontovich considered the van der Pol equation; here we apply this method directly to the system (4).

For definition of the entropy of separate neuron without interactions, we assume that $-\frac{dl}{dt} = 0$. Then, the Langevin equation for the equation (4) has the following form:

$$\frac{du}{dt} = \xi, \quad (6a)$$

$$\frac{d\xi}{dt} = \alpha(1 - \beta u)\xi + \omega u + av - \sqrt{\sigma}G(t), \quad (6b)$$

where $G(t)$ is the stochastic source that is the Gaussian noise and σ is the intensity of the source. The term $\alpha(1 - \beta u)$ is a friction coefficient that defines dissipation forces and the term $\omega u + av$ represents potential field that depends on the state of the system.

For this equation, the Fokker-Plan equation of the dynamics of distribution $f(u, \xi, t)$ in the phase space (u, ξ) is

$$\begin{aligned} \frac{\partial f}{\partial t} + \xi \frac{\partial f}{\partial u} - (\omega u + av) \frac{\partial f}{\partial \xi} = \\ = \frac{\partial}{\partial \xi} [\alpha(1 - \beta u)\xi f] - \frac{\partial}{\partial \xi} [\sqrt{\sigma}G(t)f]. \end{aligned} \quad (7)$$

Because of the reset condition (3) it is rather problematic to find analytic solution of this equation. Nevertheless, it can be shown that it has the Gauss-like form:

$$f(\xi) = \frac{1}{S(u, v, \xi)} \exp(-R(u, v, \xi)), \quad (8)$$

where strictly positive function S depends also on the parameter σ and positive function R depends on the parameters α , β and ω (see equation (4)). In addition, it is assumed that $\int f(\xi) d\xi = 1$.

Finally, the entropy $h(f)$ of the neuron with the steady state distribution $f(\xi)$ is

$$h(f) = -k \int f(\xi) \ln f(\xi) d\xi, \quad (9)$$

where $k > 0$ plays a role of the Boltzmann coefficient.

The entropy of the ensemble of the neurons is defined using the external drive $-\frac{dl}{dt}$ of the neuron by the members of its ensemble. In the case of a pair of neurons, it is defined by the equation (5). The Fokker-Plank equation for the distribution $f_i(\xi_i, \xi_j, t)$ of the neuron i acting in pair with the neuron j is

$$\begin{aligned} \frac{\partial f_i}{\partial t} + \xi_i \frac{\partial f_i}{\partial u_i} - (\omega_i u_i + a_i v_i) \frac{\partial f_i}{\partial \xi_i} - \\ - (\gamma'_{ij}(u_i - u_j) + \gamma''_{ij}(\xi_i - \xi_j)) \frac{\partial f_i}{\partial \xi_i} = \\ = \frac{\partial}{\partial \xi_i} [\alpha_i(1 - \beta_i u_i)\xi_i f_i] - \frac{\partial}{\partial \xi_i} [\sqrt{\sigma_i}G(t)f_i]. \end{aligned} \quad (10)$$

Then, the entropy $h_i(j)$ of the neuron i biased by the neuron j is

$$h_i(j) = -k \int f_i(\xi_i, \xi_j) \ln f_i(\xi_i, \xi_j) d\xi_i. \quad (11)$$

where $f_i(\xi_i, \xi_j)$ is a steady state distribution over the velocities of the neurons i and j . The entropy $h_j(i)$ of the neuron j biased by the neuron i is defined by the same manner. Another method (Klimontovich, 1991) of defining the entropies $h_i(j)$ and $h_j(i)$ is to use the Kullback-Leibler entropy.

Finally, entropy of the neurons ensemble of N neurons (Klimontovich, 1991) is obtained using the average distribution \bar{f} that represents the distribution of the average velocities $\bar{\xi}$ of randomly moving (but not necessary Brownian) particles. Such distribution is governed by the Turing system (Turing, 1952), (Lepanen, 2004) of the form

$$\frac{\partial \bar{f}}{\partial t} = D_f \frac{\partial^2 \bar{f}}{\partial \bar{\xi}^2} + \varphi(\bar{u}, \bar{\xi}, \bar{g}), \quad (12a)$$

$$\frac{\partial \bar{g}}{\partial t} = D_g \frac{\partial^2 \bar{g}}{\partial \bar{\xi}^2} + \psi(\bar{u}, \bar{\xi}, \bar{f}), \quad (12b)$$

where function \bar{f} stands for the activator function and auxiliary function \bar{g} stands for the inhibitor function, and the functions φ and ψ specify the positive and negative feedback, respectively.

Then the entropy of the ensemble is

$$h(\bar{f}) = -k \int \bar{f}(\bar{\xi}) \ln \bar{f}(\bar{\xi}) d\bar{\xi}. \quad (13)$$

These formulas are widely used in statistical physics for description of the behaviour of elementary particles, but for the description of the activity of neural network the idea to use the average coordinates and velocities seems to be not the best one. More realistic method can be based on the distribution of active and non-active neurons as it is defined by the methods of population dynamics see e.g. (Kagan, Ben-Gal, 2015).

5 NUMERICAL SIMULATIONS

Numerical simulations illustrate activity of the neurons. For the simulations, we directly apply the simple numerical schemes to the equations presented in the previous section.

In all simulations, parameters have the values indicated in the previous section that are: $a = 0.02$, $b = 0.2$, $c = -65$ and $d = 2$. These values appear in the original paper by Izhikevich (Izhikevich, 2003). In the other equations we used the parameters $\alpha = 5$, $\beta = -0.016$, $s = 0.01$, $\gamma'_{ij} = 1$, $\gamma''_{ij} = 1$ and $k = 1$. In order to obtain clear illustration of the neuron activity, the value of the frequency $\omega = 1$ was chosen with respect to the time interval $t = 0, 1, 2, \dots, 50$.

Let us consider the activity of the single neuron. Figure 2 shows the graph of the velocity $\xi(t)$ and the phase portrait of the neuron defined by the Langevin equation (6).

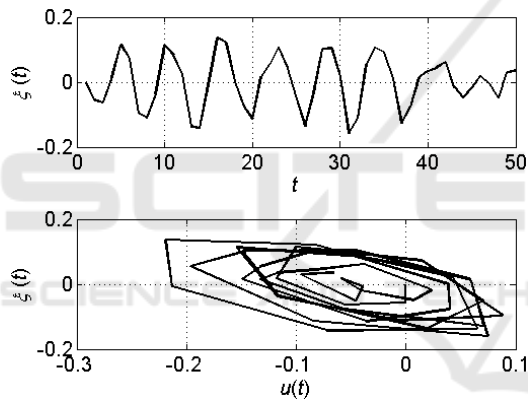


Figure 2: The graph of the velocity and the phase portrait of a single neuron described by the Langevin equation (6).

It is seen that the neuron demonstrates the oscillating behaviour with certain randomness.

The next Figure 3 shows evolution of the distribution $f(u, \xi, t)$ in time starting from the uniform distribution.

Evolution of the entropy $h(f)$ starting from the last stages of its decreasing is shown in Figure 4. As it was expected, the entropy starts with the maximal value that corresponds to the uniform distribution and exponentially decreases to some small value and then oscillates near this value.

Now, let us consider activity of the pair of neurons described by the Fokker-Plank equation (10). The first neuron in the pair is the neuron considered in the previous simulations and the second neuron is defined by the same equations with the same values of the parameters. The difference

between the neurons caused by randomness of the values generated by the stochastic sources $G(t)$.

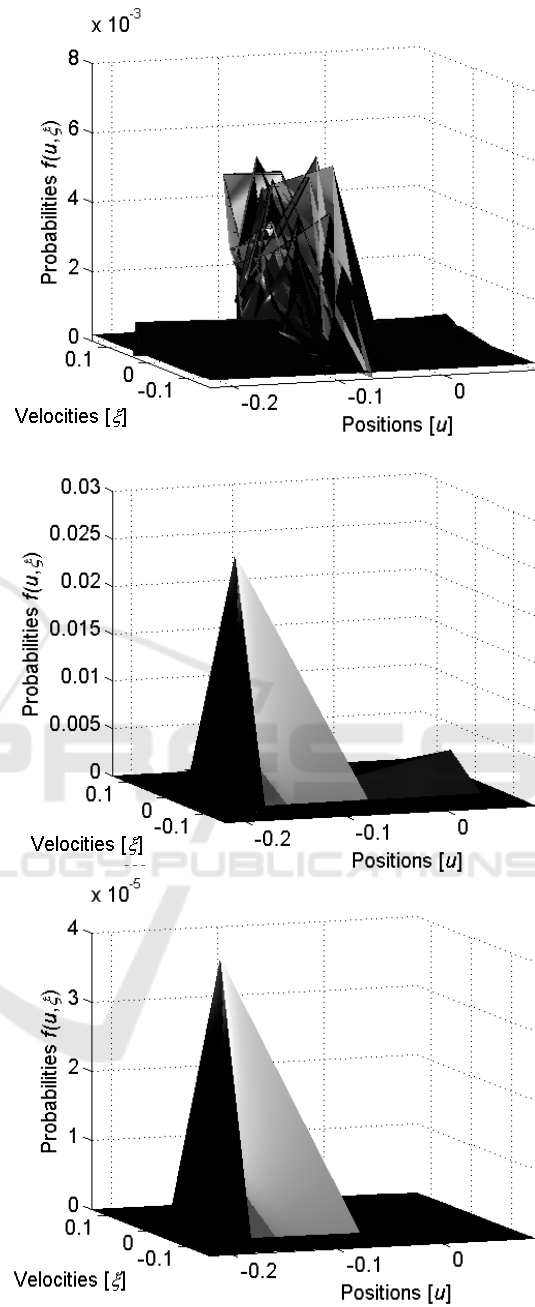


Figure 3: Evolution of the distribution $f(u, \xi, t)$ for a single neuron as it is defined by the Fokker-Plank equation (7) from the initial uniform distribution. The first graph shows the distribution $f(u, \xi, t)$ at the starting stages, $t = 3$; the second – at the middle, $t = 25$, and the last – at the end, $t = 50$, of the trial.

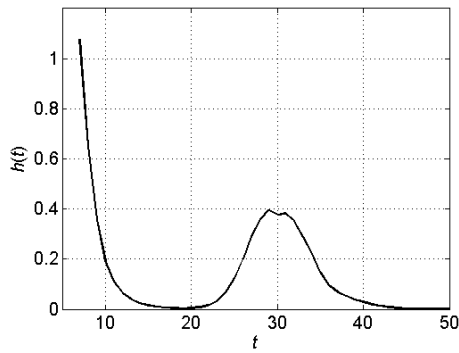


Figure 4: Evolution of the entropy $h(f)$ of a single neuron.

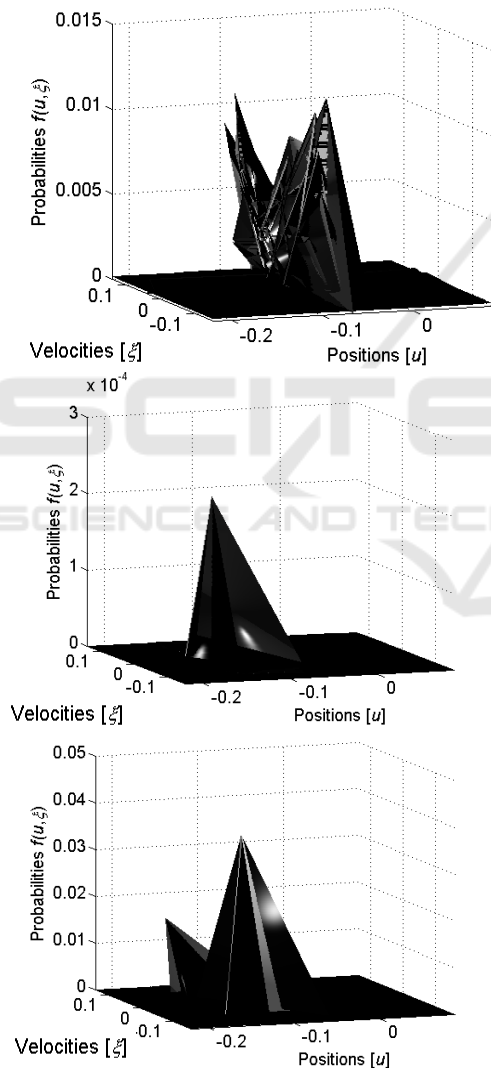


Figure 5: Evolution of the distribution $f_1(\xi_1, \xi_2, t)$ of neuron 1 acting in pair with neuron 2 as it is defined by the Fokker-Plank equation (10). The first graph shows the distribution $f_1(\xi_1, \xi_2, t)$ at the starting stages, $t = 3$; the second – at the middle, $t = 25$, and the last – at the end, $t = 50$.

Evolution of the distribution $f_1(\xi_1, \xi_2, t)$ of neuron 1 acting in pair with neuron 2 is shown in Figure 5. It is seen that this evolution is similar to the evolution of the distribution $f(u, \xi, t)$ of single neuron.

The entropies $h_1(f)$ and $h_2(f)$ of the neurons 1 and 2 acting separately exponentially decrease and oscillate near some small value, and the same holds with the entropy $h_1(2)$ of the neuron 1 biased by the neuron 2. However, the velocity of decreasing and the frequency of oscillations are different.

Figure 6 shows the last stages of the decreasing of these three entropies and their oscillations.

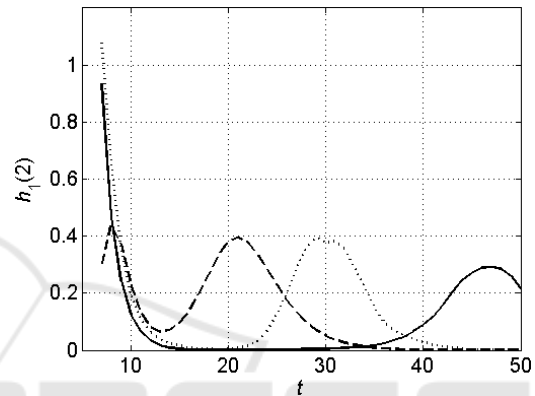


Figure 6: Last stages of the decreasing of the entropies $h_1(f)$ (dotted curve) and $h_2(f)$ (dashed curve) of the neurons 1 and 2, respectively, and of the entropy $h_1(2)$ (solid curve) of the neuron 1 biased by the neuron 2.

It is seen that starting from the time $t \approx 8$, while $h_1(2) \approx 0.4$, both $h_1(f) > h_1(2)$ and $h_2(f) > h_1(2)$. Hence, following the suggested model of creating connections between the neuron's (see Section 2, especially – inequalities (1)), the neurons 1 and 2 will interconnect and start acting in pair.

However, at the time $t \approx 38$ the entropy $h_1(2)$ of the pair of neurons becomes greater than the entropies $h_1(f)$ and $h_2(f)$ of each of the neurons acting separately. Then, the neurons disconnect and begin to act separately.

As indicated above, the neurons ensembles that include more than two agents act in the same manner, but the entropy of the ensemble should be calculated by the other methods, for example, using the models population dynamics see e.g. (Kagan, Ben-Gal, 2015).

6 CONCLUSIONS

The considered neural network consists of the oscillating mobile neurons that connect and disconnect with respect to their entropies and the entropy of the ensemble. The states of the neurons are defined on the basis of the well-known Hodgkin-Huxley model that defines the oscillations of the neurons' activity.

Such definition allows calculation of the entropy of the neuron and the neuron's ensemble using the Klimontovich method that is widely used in statistical physics.

The suggested approach contrasts with the traditional methods, where the connections between the neurons are governed by the external learning procedures, and specifies the neurons' connections on the basis of the neurons' internal properties.

Numerical simulations confirm feasibility of the suggested model and demonstrate the required properties of the entropy of separate neurons and of the neurons' ensembles. In particular, it was shown that the entropy of the single neuron periodically obtains the values greater than the values of the entropy of this neuron acting in pair with the other neuron. Following the suggested model, connection and disconnection of the neurons is governed by this inequality.

The suggested mechanism of assembling / disassembling is equal to motion of the neurons toward the other neurons or away from them, respectively, and the information about the neurons' entropies is transmitted via the glia.

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