Mathematical Model of the Impulses Transformation Processes in Natural Neurons for Biologically Inspired Control Systems Development

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Abstract. One of the trends in the development of control systems for autonomous mobile robots is the approach of using neural networks with biologically plausible architecture. Formal neurons do not take into account some important properties of a biological neuron, which are necessary for this task. Namely - a consideration of the dynamics of data changing in neural networks; difficulties in describing the structure of the network, which cannot be reduced to the known regular architectures; as well as difficulties in the implementation of biologically plausible learning algorithms for such networks. Existing neurophysiological models of neurons describe chemical processes occurring in a cell, which is too low level of abstraction.
The paper proposes a neuron's model, which is devoid of disadvantages described above. The feature of this model is description cell possibility with tree-structured architecture dendrites. All functional changes are formed by modifying structural organization of membrane and synapses instead of parametric tuning. The paper also contains some examples of neural structures for motion control based on this model of a neuron and similar to biological structures of the peripheral nervous system.

Keywords: neural network, natural neuron model, control system, biologically inspired neural network, motion control

1 Introduction

Nowadays, a lot of attention is paid to the study of the nervous system's functioning principles in the problems of motion control and data processing and the creation of biologically inspired technical analogues for robotics [1,2,3].

At the same time borrowing just part of the data processing cycle inherent to natural neural structures, seems to be ineffective. In this case, we can't avoid the step of converting the "inner world's picture" of our model, expressed in the structure and set of the neural network's parameters, set up in the narrow context in the terms of current problem. Such conversion can nullify the effectiveness of the approach. It is necessary to start with a construction of simple self-contained systems that function in an

environment model, and then gradually complicate them. For example, it is possible to synthesize the control system functionally similar to the reflex arc of human nervous system (Fig. 1).

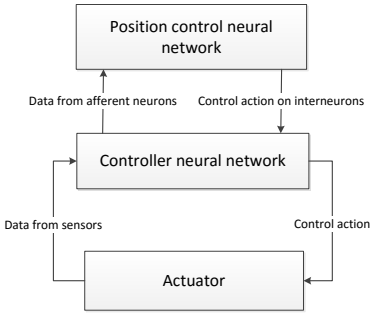

Fig. 1. Control system similar to reflex arc of human nervous system

In this case, position control neural network has input and output layers of neurons, as well as several hidden layers. Input and output layers have connections with neurons of other neural networks, while neurons of the hidden layers are connected only to the neurons of current neural network [4].

However, the most promising is the development of full-scale systems that implement all phases of the data transformation from sensors to effectors inherent to natural prototypes.

There are many models of neuronal and neural networks. These models may be quite clearly divided into two groups: for applied engineering problems (derived from the formal neuron model) [5], and models, designed for the most complete quantitative description of the processes occurring in biological neurons and neural networks [6,7] .

Considering modeling of natural neuron, we investigate the transition from formal neuron models to more complex models of neurons as a dynamic system for data transformation [8] suitable for control tasks (Fig. 2).

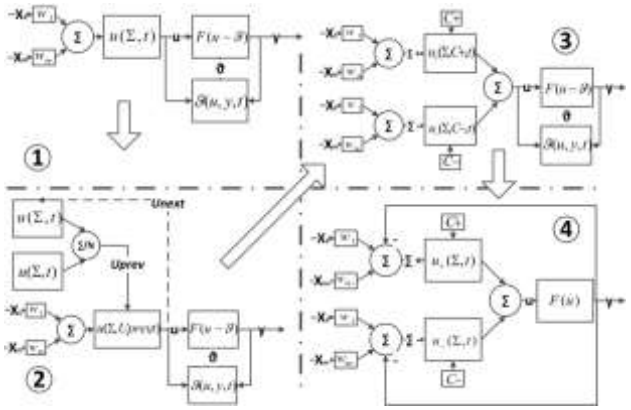

Fig. 2. Evolution of neuron model

Where $x_1 - x_m$ - neuron input signals;

$w_1 - w_m$ - weights;

y - neuron output signal;

u - membrane potential value;

ϑ - threshold function;

F - activation function;

N - number of membrane segments at the dendrite branching node;

$C+, C-$ - constants for expected level of membrane potential contribution;

$u+, u-$ - contributions to the membrane potential from depolarizing and hyperpolarizing ionic mechanisms;

Figure 2-1 represents a universal model of the formal neuron in general. Classic formal neurons can be derived from this model, if we abandon the temporal summation of signals to establish a fixed threshold and choose, for example, a sigmoid activation function.

Further development of this model may be adding a description of the structural organization of the neuron membrane (Fig. 2-2), with a separate calculation of the contribution to the total potential (Fig. 2-3) to provide at each site the ability to integrate information about the processes occurring with different speeds, as well as rejection of the an explicit threshold setting and move to the representation of the signal in the neural network as a stream of pulses (Fig. 2-4). As a result, the potential value of the neuron membrane segment is derived not only from the values of the inputs and the weights of synapses, but also from the average value of the membrane potential of other connected membrane segments. This will simulate the structure of the dendritic and synaptic apparatus of neurons and carry out more complex calculations of the spatial and temporal summation of signals on the membrane of the neuron. Thus, membrane segment should be considered as the minimal functional element of the neural network.

Given the existence of temporal summation of signals, the structural organization allows to implement separate processing of signals with different functionality on a

single neuron. To do this, may be selected a single dendrite, which will provide, for example, only the summation of signals on the current position of the control object formed by afferent neurons, as well as to the signal of corrections to position, that formed by the highest level of control. The individual dendrite will implement similar behavior, for example, the speed of the object and the body of the neuron will provide the integral combination of these control loops, which otherwise would require adding an additional neuron.

2 Neuron model

It is assumed that the inputs of the model get pulsed streams, which are converted by synapses into the analog values that describe the processes of releasing and metabolizing of the neurotransmitter in the synaptic cleft. The model assumes that the input and output signals of the neuron is zero for the absence of a pulse, and constant for the duration of the pulse. The pulse duration is determined by the time parameters of the neuron's membrane. Membrane of soma and dendrites is represented by a set of pairs of ionic mechanisms' models that describe the function of depolarization and hyperpolarization mechanisms, respectively. The outputs of the ionic mechanisms' models represent the total contribution to the intracellular potential of depolarization and hyperpolarization processes occurring in the cell. The signals from the synapses modifies the ionic mechanisms' activity in the direction of weakening their functions, which simulates the change in the concentration of ions inside the cell under the influence of external influences. It is proposed to distinguish the type of ionic mechanism in the sign of the output signal. A positive value of the output characterizes depolarizing influence, while negative characterizes hyperpolarization. Thus, the total value of the output values will characterize the magnitude of the membrane segment contribution to the total intracellular neuron potential [9].

The role of synaptic apparatus in the model is the primary processing of the input signals. It should be noted that the pattern of excitatory and inhibitory synapses are also identical to each other, and the difference in their effects on cell's membranes is determined by which of the ionic mechanisms each particular synapse is connected to. Each synapse in this model describes a group of natural neuron synapses.

More detailed model of the membrane is shown in Fig. 3.

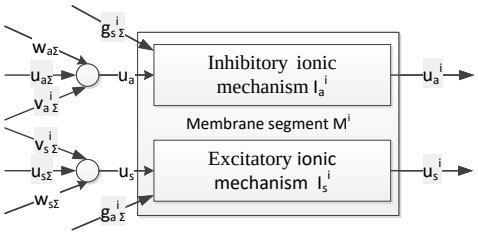

Fig. 3. Functional diagram of the *i*-th membrane segment model *Mi*

Each membrane segment $M^i, i = \overline{1, L}$ consists of a pair of mechanisms - hyperpolarization mechanism ($I_a{}^i$), and depolarization mechanism ($I_s{}^i$). Output of the membrane's segment is a pair of the contribution values of hyperpolarization (u_a) and depolarization (u_s), which determines the contribution to the total intracellular potential.

Each membrane's segment M^i can be connected to previous membrane's segment M^j taking its values $\{u_a{}^j, u_s{}^j\}$ as inputs. When specified membrane's segment is the last in the chain (the end of the dendrite or the segment of soma), as signals $\{u_a{}^j, u_s{}^j\}$ stands pair of fixed values {*-Em, Em*} simulating some of the normal concentration of ions in the cell in a fully unexcited state.

Excitatory $\{x_{sk}^i\}, k = \overline{1, M_i}$ and inhibitory $\{x_{ak}^i\}, k = \overline{1, N_i}$ neuron's inputs are inputs of many models of excitatory $\{S_{sk}^i\}, k = \overline{1, M_i}$ and inhibitory $\{S_{ak}^i\}, k = \overline{1, N_i}$ synapses, for each of the membrane's segments M^i.

The resulting values of the effective influence on the mechanisms of synaptic hyperpolarization ($g_s{}^{i}{}_{\Sigma}$) and depolarization ($g_a{}^{i}{}_{\Sigma}$) are obtained by summation:

$$g_s{}^{i}{}_{\Sigma} = \sum_{k=1}^{M_i} g_{sk}{}^{i}, g_a{}^{i}{}_{\Sigma} = \sum_{k=1}^{N_i} g_{ak}{}^{i}. \quad (1)$$

Outputs of all membrane segment models are summed by following formula:

$$u_{\Sigma} = \frac{1}{L} \sum_{i=1}^{L} u^i$$

The resulting signal is assumed as total intracellular potential of the neuron. Each pair (depolarization and hyperpolarization mechanisms), depending on their internal properties, can be regarded as model of dendrite segment or soma segment. Increasing the number of pairs of such mechanisms automatically increases the size of the neuron, and allows simulating a neuron with a complex organization of synaptic and dendritic apparatus.

Similarly, the summation of signals at branching nodes of dendrites - the total contribution of the hyperpolarization and depolarization mechanisms $\{u_a{}^j, u_s{}^j\}$ are divided by their number.

Fig. 4 contains a general view of the neuron's membrane structure [10].

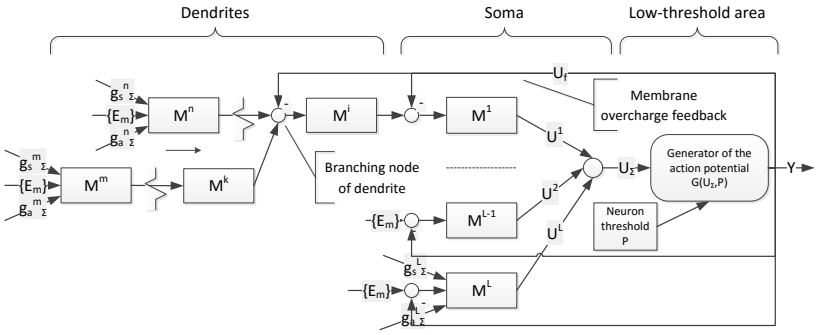

Fig. 4. Structural diagram of the neuron membrane

The body of the neuron (soma), we assume those parts of the membrane that are covered by feedback from the generator of the action potential. It should also be noted that the closer a membrane's segment located to the generator, the more effective its contribution to the overall picture of synapses in neuronal excitation.

Thus, in terms of the model:

1. carried out on the dendrites spatial and temporal summation of signals over long periods of time (a small contribution to the excitation of the neuron from each synapse), and accumulation of potential does not depend on the neuron discharges;
2. in the soma of a neuron produced summation of signals at short intervals of time (a big contribution to the excitation of the neuron from each synapse) and accumulated potential is lost when the neuron discharges;
3. in low-threshold area is carried impulse formation on reaching the threshold of generation and signal of membrane recharge.

The following discloses the mathematical description of the neuron model elements.

Synapse model. It is known that the processes of releasing and metabolizing of the neurotransmitter are exponential, and besides the process of releasing neurotransmitter, usually is much faster than the metabolizing process.

Another important factor is the effect presynaptic inhibition consists in that, when the concentration of the neurotransmitter exceeds certain limit values, synaptic influence on ion channel starts to decrease rapidly - despite the fact that the ion channel is fully open. Reaching the limit concentration is possible when synapse is stimulated by the pulsed streams with high pulse frequency.

Model that implements all three main features of the synapse's functioning can be described by the following equations:

$$\left.\begin{aligned} T_S \cdot \frac{d\rho}{dt} + \rho(t) = E_y^{-1} \cdot x, \\ T_S = \begin{cases} \tau_s, \text{ при } x > 0, \\ \tau_d, \text{ при } x \le 0. \end{cases} \end{aligned}\right| \left.\begin{aligned} g_* = 4 \cdot \xi \cdot (\rho - \xi \cdot \rho^2) \\ g = \begin{cases} R_s^{-1} \cdot g_*, \text{ при } g_* > 0, \\ 0, \text{ при } g_* \le 0. \end{cases} \end{aligned}\right\} \quad (2)$$

Where τ_s - time constant of releasing neurotransmitter,

τ_d - time constant of metabolizing neurotransmitter,

$\xi \in [0.5, \infty)$ - limit value of neurotransmitter's concentration needed to presynaptic inhibition effect,

$R_S > 0$ - synapse's resistance ("weight"), that characterizes the efficiency of synapse's influence on the ionic mechanism,

E_y - the amplitude of the input signal.

Initial conditions: $\rho(0) = 0$.

Model's input is a discrete signal *x(t)*, which is a sequence of pulses with a duration of 1 ms and an amplitude *E*. The releasing and metabolizing processes of the neurotransmitter are proposed to simulate the first order inertial element with logic control by time constant. Variable ρ characterizes the concentration of neurotransmitter released in response to a pulse. Usage of variable g_* allows us to simulate presynaptic inhibition effect.

Model's output *g(t)* is an efficiency of influence on ionic mechanism and it is proportional to the synapse's conduction. Thus, in the absence of input actions synapse conductance tends to zero, which corresponds to the open switch in the equivalent circuit of the membrane.

Model of membrane's ionic mechanism. It is known that the ion channel can be represented by an equivalent electrical circuit [11], which has three major characteristics - the resistance R_m, capacitance C_m and ion concentration $E_m = v$ maintained within the cell membrane pump function. Product $T_m = R_m C_m$ characterizes inertia of the channel that defines the rate of recovery of the normal concentration of ions E_m in the cell. Synapse's influence on the ionic mechanism consists in the loss of efficiency of the channel's pumping function and reducing the ions' concentration in the cell, with the time constant of the process:

$$T = R^I C_m . \quad (3)$$

Resistance R^I is determined from the relation:

$$\frac{1}{R^I} = g_1 + g_2 + ... + g_n + \frac{1}{R_m} = g_\Sigma + \frac{1}{R_m} . \quad (4)$$

Where $g_1, g_2, ..., g_n$ - conductions of active synapses' models that have an influence on the current ionic channel. Reduction in ions' concentration at the same time is proportional to the product $g_\Sigma \cdot R_m$ and the less, the lower the ions' concentration in the cell is.

Fig. 5a shows the dependence of the synapse's contribution in changing the membrane potential on the ratio of the synapse's channel and postsynaptic membrane's resistance. It can be seen that the effective control range of the synapse's resistance is in the range [0.1: 10] of membrane's resistance. Fig. 5b shows the change in the potential contribution to the number of active synapses in the ratio Rs/Rm = 10 (dashed line) and 1 (solid line).

The ordinate axis in both graphs - normalized postsynaptic membrane potential change in proportion to its nominal value. Fig. 5a: the dependence of the efficiency on the ratio of the synapse's channel and the membrane's resistance. Fig. 5b: the dependence of the efficiency on the number of synapses.

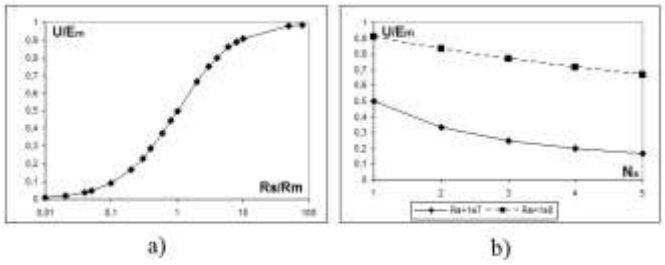

Fig. 5. Current efficiency of the synapse's model

Inertial properties of the ionic mechanism's model are proposed to describe as an aperiodic element with logic control by time constant. For the ionic mechanism of depolarization equations have the following form:

$$\begin{cases} \left.\begin{aligned} &T_I \cdot \frac{du}{dt} + (1 + g_{s\Sigma} \cdot R_m) \cdot u = v \\ &T_I = \frac{C_m}{g_{s\Sigma} + R_m^{-1}} \end{aligned}\right\}, g_{a\Sigma} = 0 \\ u = E_m^-, g_{a\Sigma} \neq 0 \end{cases} \quad (5)$$

Where $g_{a\Sigma}$ - the total efficiency of synapses influence on the hyperpolarization mechanism,

$g_{s\Sigma}$ - the total efficiency of synapses influence on the depolarization mechanism,

$R_m > 0$ - membrane's resistance,

$C_m > 0$ - membrane's capacitance,

v - the expected contribution of the model in the value of the intracellular potential in the absence of external excitation. This value is determined by the activity of neighboring membrane segments,

u - a real model's contribution to the value of the intracellular potential.

Initial conditions: *u(0)=0.*

For ionic mechanism of hyperpolarization equations are analogous up to relocation of the effects of excitatory and inhibitory synapses and Em^- on Em^+.

Action's potential generator's model. Generator's model performs the formation of rectangular pulses of given amplitude E_y as a result of exceeding fixed threshold P by the potential u_Σ. The model can be described by the following equations:

$$\left.\begin{aligned} T_G \cdot \frac{du_*}{dt} + u_* = u_\Sigma, \\ y = F_G(u_*). \end{aligned}\right\} \quad (6)$$

Where $P > 0$ – neuron's threshold,

T_G - time constant, which determines the duration of the feedback overcharging membrane and characterizing pulse durations,

$F_G(u_*)$ - Function describing the hysteresis. The output of the function is E_y, if $u_* \geq P$ and zero if $u_* \leq 0$.

Initial conditions: $u_*(0) = 0$.

Output signal *y(t)* goes to overcharge feedbacks of cell's soma.

3 Research

Setting the model's parameters was based on experimental data on the time parameters of the processes occurring in the natural neuron [10].

Fig. 6 shows a typical response of a neuron model to the exciting pulse. In the graph of intracellular potential (2) can be seen a typical region of the neuron membrane depolarization is preceded by the formation of an action potential, the zone of hyperpolarization after pulse generation and residual membrane depolarization at the end of the generation's pattern.

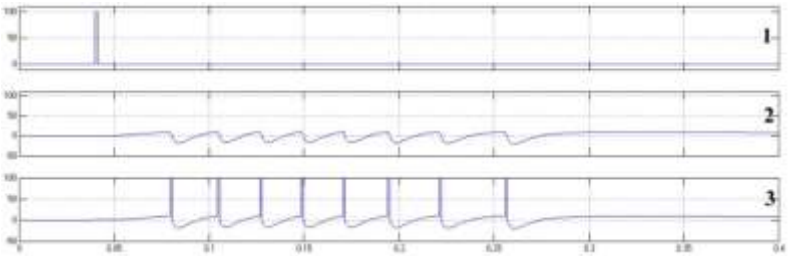

Fig. 6. Neuron with synapse on its dendrite (1 - stimulating effect 2 - intracellular membrane potential on the generator of the action potential, 3 - neuron responses combined with the graph of the intracellular potential)

One of the main characteristics of the natural neuron qualitatively affects the transformation of the pulsed streams is the size of the membrane. Unlike small neuron large neuron is less sensitive to the effects of input and generates a pulse sequence typically in a lower frequency range and generally corresponds to input effects with single pulses.

The developed model allows to build neurons with different membrane structure and location of synapses on it. Changing the number of the membrane segments neurons of different sizes can be modeled, without changing the values of the parameters.

With the increasing size of the soma at the same stimulation of the neuron number of pulses in the pattern of neuron response decreases and the interval between them increases. Fig. 7a demonstrates dependence of the response's average frequency from the number of pulses *Np* in it. Fig. 7b demonstrates dependence of response's average frequency from the number of neuron's soma segments *L*.

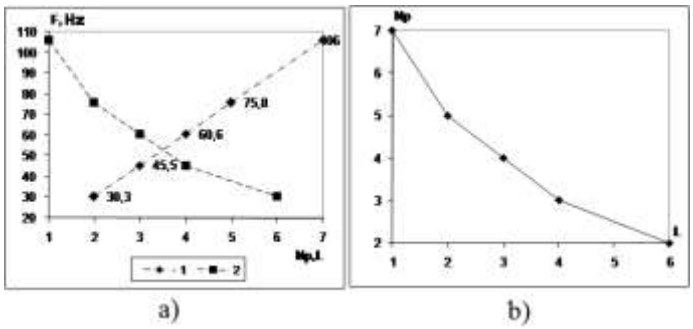

Fig. 7. Discharge frequency, depending on the neuron's soma size

As a simple neural structures with feedback considered element, which is a widely held in the nervous system connection excitatory inhibitory neurons, first studied in neurophysiological experiments, the interaction of motoneuron and Renshaw's cells (Fig. 8).

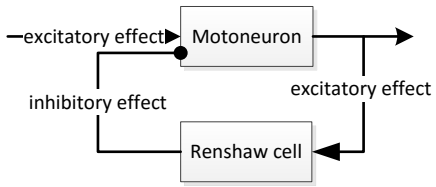

Fig. 8. The scheme of recurrent inhibition by the example of the regulation of motoneuron discharges

There are two mechanisms for increasing the strength of muscle contraction. The first is to increase the pulse repetition frequency at the output of motoneuron. Second - increasing the number of active motoneurons, the axons of which are connected to the muscle fibers of the muscle. Specialized inhibition neuron in the chain of recurrent inhibition - Renshaw cell - limits and stabilizes the frequency of motoneuron discharges. Example of such a structure shows an analog model (Fig. 9), the behavior of which corresponds to neurophysiological data [11].

Fig. 9. Recording pulsed streams in studying the interaction of motoneuron and Renshaw cells motoneuron at the excitation frequency of 20Hz (a) and 50 Hz (b): 1 - excitatory motoneuron input; 2 - Renshaw cell's discharges; 3 - motoneuron output pulses. Above - the time stamp 10 ms

The graphs show that the frequency of motoneuron stimulation enhances the inhibitory effect on Renshaw cells with motoneuron, causing, in turn, decrease the frequency of motoneuron discharges. Thus, when the frequency of motoneuron stimulation increases, the frequency of the pulses at the output of the first moments increases and then stabilizes at a low level with a duration of interpulse intervals determined by the duration of the Renshaw cell's discharge. It is essential that this limit is dependent on whether the motoneuron by recurrent inhibition "own" Renshaw cells or not. Computer simulation has allowed a more detailed study of the interaction of neurons.

The results of the experiment are shown in Fig. 10, where the top-down plotted input pulsed stream at the input of motoneurons and pulsed streams of motoneuron Renshaw cell with recurrent inhibition and, accordingly, these neurons without feedback when motoneuron excites Renshaw cell, but it does not slow motoneuron.

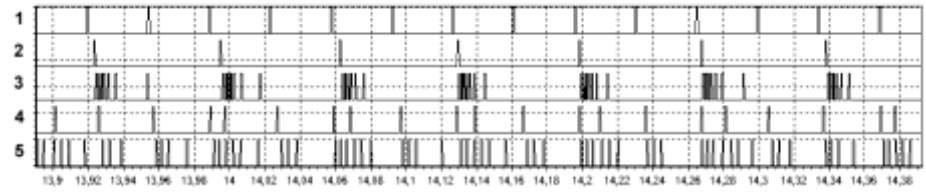

Fig. 10. Reactions of structure "motoneuron-Renshaw cell" upon excitation of motoneurons pulsed stream at 50 Hz: 1 - input pulsed stream; 2 – motoneuron's reaction with enabled FB; 3 - Renshaw cell responses with enabled FB; 4 – motoneuron's reaction without FB; 5 - Renshaw cell responses without FB

4 Conclusion

The paper presents a model of a neuron, which can serve as the basis for constructing models of neural networks of living organisms and study their applicability in solving the problems of motion control of robotic systems. The model allows to describe the structure of the neuron's membrane (dendritic and synaptic apparatus).

Plasticity model is also based primarily on changes in the structure of the membrane, rather than adjusting the parameters of the model (synapse weights, neuron's threshold, etc.), which simplifies the construction of models of specific known biological neural structures.

5 Sources

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