UDC 621.386:616-073.7(045)

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COMPUTER SIMULATION OF BIOLOGICAL NANO–GENERATOR FUNCTIONS

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Abstract*—The results of our program modeling of biological "molecular current generator" are described. Simulation was done on the base of our experimental studying of electrical currents through molecular channel-receptor complex in membrane of natural brain neurons.*

Index Terms—Program model; brain; neuron; molecular current generator; nanostructure.

I. INTRODUCTION

Ionic currents generation is spread in nature for the realization of such important phenomena like the propagation of action potentials, excitation in complexes of excitable cells in brain or muscles, etc. Such phenomena require the movement of significant numbers of ions across the membrane in a relatively short period of time. The rapid rate of ionic flow occurring during the generation of an action potential is far too high to be achieved via an active transport mechanism. Rather it results from the opening of ion channels as elements of channel-receptor complexes (CRC). Although the existence of ionic channels in the membrane has been postulated for decades, their properties and structure have only recently become known in detail. The development by Erwin Neher and Bert Sakmann of the patch-clamp technique, in which a small patch of membrane containing a single or small number of ionic channels is drawn up into a blunt microelectrode, allowed for the minuscule (10^{-9} Amps or picoamps) current flowing through single channels to be recorded in intact biological membranes for the first time [1], [2], [4], [5]. In addition, the rapid advances made in molecular biology regarding the isolation, cloning, and sequencing of the proteins making up ionic channels (and channel-receptor complexes in whole) have revealed much about their primary structure. Especially exciting is the recent crystallization of K^+ and Cl^- channels in some of their different conformations, allowing their tertiary structure to be detailed with *X*-ray diffraction. The powerful combination of electrophysiological and molecular techniques has yielded valuable insights into the structure-function relationships of ionic channels. Program modeling makes deeper our understanding of the realization of some natural phenomena [3], [6], [7], and we used this method for further studying of some CRC from the brain membrane. Activity of such molecular CRC demonstrates the similarity with current generator; so, such CRC are called sometimes "biological current nano-generator".

II. TASK STATEMENT

Basing on the results of electrical properties of natural brain neurons and their molecular channels studying to develop program models for some membrane molecular structures.

III. TRANSMEMBRANE IONIC CURRENTS IN BRAIN CELLS AS PHENOMENA

Voltage-sensitive ionic channels appear to have several shared features. First, they are large proteins that span the 6–8 nm of the plasma membrane and are typically made up of subunits. Second, through protein folding, they form a cylinder surrounding a central water-filled pore that permits the passage of only certain classes of ions between the inside and outside of the cell. The selection of which ions are allowed to pass through each different type of ionic channel is based upon the size, charge, and degree of hydration of the different ions involved. Finally, voltage-gated ion channels possess one or more gates, or voltage-sensing regions, within the ionic pore, and the flow of ions through the channels is regulated by these gates.

Neurons in the nervous system can generate occasionally action potentials that serve for information transmission. Rather, neuronal membranes are in a constant state of flux due to the presence of remarkable variety of different ionic currents. These currents are distinguished not only by the ions that they conduct (e.g., K^+, Na^+, Ca^{2+}, Cl) but also by their time course, sensitivity to membrane potential, and sensitivity to neuro-transmitters and other chemical agents [1], [2], [4], [5]. As the various ionic currents were discovered, they were divided into two general categories: those that are sensitive to changes in membrane potential

and those that are altered by neurotransmitters and internal messengers. Most currents that are sensitive to membrane potential are turned on (activated) by depolarization. The rate at which they activate and the membrane potential at which they start to become active (threshold) are important characteristics. Many voltage-dependent currents do not remain on once they are activated, even during a constant shift in membrane potential. The process by which they turn off despite a stable level of membrane potential in their activation range is known as inactivation. Inactivation is a state of the current and ionic channels that is distinct from simple channel closure. Once a current becomes inactive, this inactivation must be removed before it can again be activated. Removal of inactivation is generally achieved by repolarization of the membrane potential. Like the process of activation, inactivation and removal of inactivation are time and membrane potential dependent. Together, all of these characteristics define the temporal and voltage domain over which the current influences the electrical activity of the neuron.

IV. MATHEMATIC MODELING OF CRC – "MOLECULAR GENERATOR" FUNCTIONING IN HOMOGENEOUS NERVE FIBER

Propagation neural excitation by non-mielinized homogeneous fiber [1], [2], [8] may be described by the equation

$$\frac{\partial\varphi(x,t)}{\partial t}=\frac{1}{RC}\frac{\partial^2\varphi(x,t)}{\partial x^2}-\frac{i}{C}, \qquad (1)$$

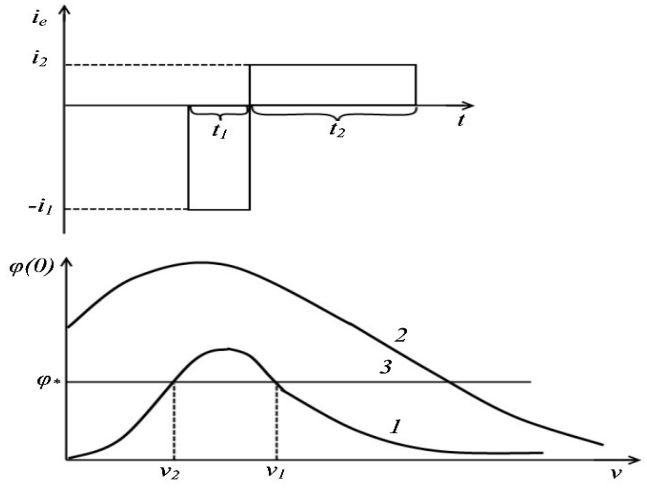
where φ is membrane potential, measured from the resting potential (this value depends on the longitudinal coordinate x and time t; R is sum of external and internal resistance per unit length of the fiber; C is capacitance of the membrane per unit length of the fiber; i is ionic current passing through the membrane per unit length of fiber, is considered a positive direction outwardly of axoplasm to the extracellular space.

From physical considerations follows that the ion current flowing through the membrane during the passage of the nerve impulse must be alternating. In other words, if the threshold is reached as soon as the current flows inside the fiber, then after some time it changes direction and flows outward. This supports the fact that the membrane potential after the pulse returns to the original value.

We approximate the ionic current that flows across the membrane by passing a pulse with two "columns" shown in Fig. 1. Suppose that at some point, corresponding to the start of excitation, the current is directed into the fiber modulus is equal to i_1. After a time t_1 current is reversed and equal i_1. This phase continues for a time t_2. Analytically, this is expressed as follows [8]:

$$i=i_1+g\varphi,\qquad i_l=\begin{cases}-i_1, 0<t<t_1;\\ -i_2, t_2<t<t_1+t_2,\end{cases}\qquad i_l>0,\ i_2>0, \qquad (2)$$

where g is leakage conductivity. Analysis begin in case of $g = 0$.



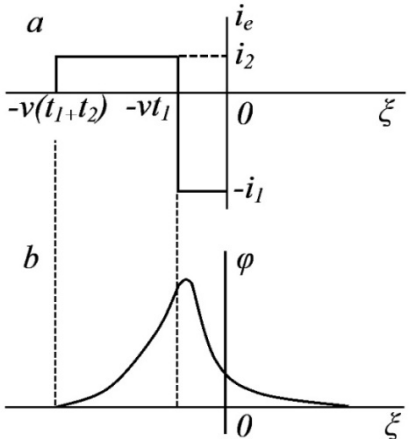


Fig. 1. Model of membrane current generator

Ionic current with respect to time is approximated by two "columns", in case of potential exceeds the threshold value. We will search for only auto-simulated (=auto-modeled) solutions of equation (1). Let's introduce a new variable $\xi = x - vt$, where v is velocity of the pulse.

Equation (1) may be transformed to the next form:

$$\frac{d^2\varphi(\xi)}{d\xi^2}+vRC\frac{d\varphi(\xi)}{d\xi}-Ri(\xi)=0. \qquad (3)$$

We choose the coordinates origin at the point where begins the excitement of membrane.

According to the definition of φ one of the boundary conditions is φ(∞) = 0. At the opposite end of the axis when $\xi \to -\infty$ imposed the requirement of limited function φ(ξ). Solution of (3) has following forms in different domains.

In domain $\xi \geq 0$.

$$\varphi(\xi) = \frac{e^{-vRC\xi}}{v^2RC^2}\left[i_1 + i_2 e^{-v^2RC(t_1+t_2)} - (i_1+i_2)e^{-v^2RCt_1}\right]. \quad (4)$$

In domain $-vt_1 \leq \xi \leq 0$.

$$\varphi(\xi) = \frac{e^{-vRC\xi}}{v^2RC^2}\left[i_2 e^{-v^2RC(t_1+t_2)} - (i_1+i_2)e^{-v^2RCt_1}\right] - \frac{i_1\xi}{vC} + \frac{i_1}{v^2RC^2}. \quad (5)$$

In domain $-v(t_1+t_2) \leq \xi \leq -vt_1$.

$$\varphi(\xi) = \frac{i_2 e^{-vRC\xi}}{v^2RC^2} + \frac{i_2}{vC}\xi + \frac{t_1(i_1+i_2)}{C} - \frac{i_2}{v^2RC^2}. \quad (6)$$

In domain $\xi \leq -v(t_1+t_2)$.

$$\varphi(\xi) = \frac{i_1t_1 + i_2t_2}{C}. \quad (7)$$

V. SOME PROPERTIES OF CALCIUM CHANNELS IN BRAIN MEMBRANE AND ELECTRICAL CURRENTS THROUGH THEM

Let's review some publications with information of calcium channels functioning. There is enough information about such type of channels of Ca-currents because of their important role for living organisms [1], [2]. Such channels may be seen as prototypes for our program model and that is why we have to examine their properties deeply. The structure of this channel is so, that Ca-channel selectivity for divalent cations are mainly determined by the intrachannel selectivity filter which, probably, contains a single carboxylic group. Therefore transition metal cations which form strong complexes with the filter are effective blockers of calcium conductance, whereas alkaline earth metal cations do penetrate. Studies revealed also the existence of anomalous mole fraction behaviour for calcium conductance. According to the one-ion model, the amplitude of mixed Ca2+ -Ba2+ current must increase monotonously from one limiting value to another when the concentration of one ion changes while keeping the total concentration constant. However, the concentration dependence observed had a minimum which could be explained by the two-ions model.

Despite its attractiveness, the two-ions model contradicts a number of experimental findings. In particular, the postulated effects of the Coulomb repulsion between cations in the calcium channel were not observed for other Ca-binding proteins with high-affinity sites. Although these proteins usually contain several Ca-binding sites not distant from each other, differences between sequential pK values corresponding to their filling by Ca ions do not exceed ± 0.5 units (Levine and Williams 1982). This difference is considerably smaller than predicted by the two-particle model (Δ pK = 3–4). Generally, the observed small increases in or decreases of sequential pK's are being attributed to some conformational transitions of Ca-binding proteins which play an important role in their function and can be revealed by different spectroscopy methods. The extent of these transitions depends on the physical and chemical properties of a given ion (Fig. 2).

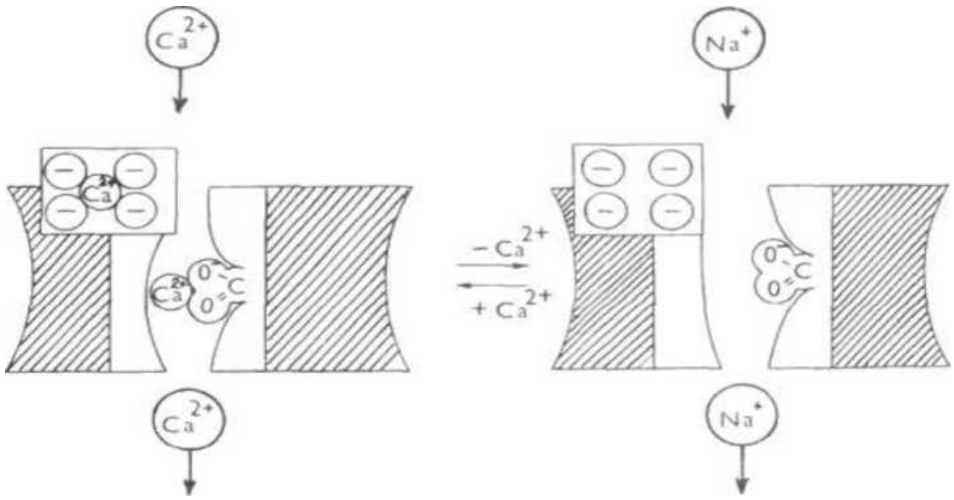


Fig. 2. Suggested Ca-binding sites in the calcium channel and their participation in its functioning (also see text) [1]

Moreover, the experimental values of the exit rate constant for the high affinity Ca binding site in proteins does not exceed $k_{\text{off}} \le 10^3 s^{-1}$ which is by 2–3 orders of magnitude smaller than the observed ion flow in the calcium channel [1]. This was used [1] to argue against the possible location of the high affinity Ca binding site on the ion pathway in the Ca-channel.

Hess and Tsien (1984) overcame this discrepancy by lowering the heights of the terminal potential barriers [1]. However, their low values ($E_{\text{barrier}} = 4RT$) lead to a high entrance rate for Caions $k_{\text{on}} \cong 10^{11} \text{mol}^{-1} \big| s^{-1}$ which is considerably lower than the corresponding diffusion limit $k_{\text{on}} \cong 10^9 \text{mol}^{-1} \big| s^{-1}$.

According to the two-ions model, the ability of Ca ions to block the monovalent ion current through the calcium channel must be approximately equal independent on the side of their application (intra - or extracellular). However, in our studies of snail neurones and model calcium channels induced by lathrotoxin in planar bilayer membranes the effect of Ca ions was strictly asymmetrical [1].

A general model of a channel with two conformational states was considered by Lauger, Stephan and Frehland in 1980 [1]. However, this model is quite complicated. Therefore, we propose here a simpler model with a kinetic scheme equivalent to the mechanism of functioning of the calcium channel shown on Fig. 3 [1], [8].

$$S'_{\text{Na}} \underset{Y_{\text{Na}}}{\overset{X_{\text{Na}}}{\rightleftarrows}} \qquad S_0 \underset{a}{\overset{b}{\rightleftarrows}} \qquad S_{\text{Ca}} \underset{X_{\text{Ca}}}{\overset{Y_{\text{Ca}}}{\rightleftarrows}} S'_{\text{Ca}}$$

Fig. 3. Model with a kinetic scheme equivalent to the mechanism of functioning of the calcium channel

Where S_0 and S_{Ca} are two different conformational states of the empty calcium channel, differing in the occupation of the high affinity Ca channel binding site, a and b being the rate constants of transitions between them. and Si > are sodium and calcium conducting states, respectively, $x_i = k^i_1 + k^i_{-2}$, $y_i = k^i_{-1} + k^i_2$ (according to Laugerer for each conformational state we considered, for simplicity, the one-site one-ion model, where k^i_j and k^i_{-j} stand for rate constants of jumps of it's ion across the jth potential barrer in the forward and backward directions). $S_0 \rightleftarrows S_{\text{Na}}$ and $S_{\text{Ca}} \rightleftarrows$ participate in Na and Ca transport through the channel. Solving the system of linear equations for steady state occupation numbers S_n we readily obtained the explicit expression for the total current

$$i_{\text{Ca+Na}} = -e_0 \frac{2\left(k_1^{\text{Ca}} - k_{-1}^{\text{Ca}} X_{\text{Na}} / Y_{\text{Ca}}\right)[\text{Ca}] / K_{\text{Ca}} + \left(k_1^{\text{Na}} - k_{-1}^{\text{Na}} X_{\text{Na}} / Y_{\text{Na}}\right)}{1 + [\text{Ca}] / K_{\text{Ca}} \left(1 + X_{\text{Ca}} / Y_{\text{Ca}}\right) + X_{\text{Ca}} / Y_{\text{Na}}}, \tag{8}$$

where $K_{\text{Ca}} = b/a$ is the dissociation constant of Ca ions with a high affinity regulatory site in the calcium channel.

The corresponding current voltage curves are shown on Fig. 4. When both Na and Ca ions are present in extracellular solution only, the theoretical curves reproduce well the effect of the depression of the sodium current through the calcium channel in the micromolar range of external Ca concentrations and the appearance of the calcium inward current at millimolar Ca concentrations.

The behavior of I–V curves with Na ions present in the intracellular solution only was somewhat unexpected. According to [1] upon lowering the extracellular Ca concentration the inward calcium current decreases whereas the outward sodium current increases. Superposition of these currents gives I-V curves with a clear reversal potential. The same effect was observed for calcium channels in chromaffine cells (Fenwick et al. 1982), isolated cardiomyocytes (Lee and Tsien 1984) and myeloma cells (Fukushima and Hagiwara 1985) when the calcium conductance could be effectively isolated from other ionic currents. Unfortunately, we could not observe this effect in neuronal membranes because their potassium conductance is difficult to eliminate using known pharmacological agents.

For these calculations we used the experimental dissociation constants and a minimum number of free parameters, assuming that the channel energy profile in each conformational state could be represented by a simple one-site model with equal and symmetrically shaped barriers [1], [8]. Our experience indicates that a quantitative description of I–V curves measured in the above works, can be obtained, e.g. by making the Na energy profile slightly asymmetrical.

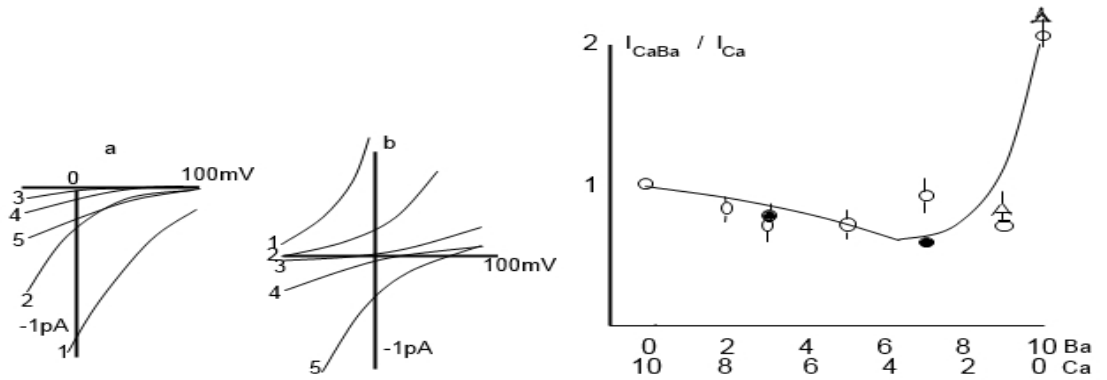


Fig. 4. I–V curves for the calcium channel with different conformational states calculated according to Eq. (1) at various extracellular Ca concentrations. The concentration of monovalent (Na) ions in extracellular (a) or intracellular (b) solutions was 30 mmol/l. The concern ration of Ca ions was 2 μmol/l (I). 20 μmol/l (2), 0.2 (3), 2 (4) 20 (5) mmol/l

At Fig.4 the height of the potential barriers for Ca and Na ions was 10 RT. the depths of the potential wells were – 5 and 0 RT, corresponding to dissociation constants K_{Ca} = 5 mmol/l and K_{Na} = 1 mol/I, respectively. The dissociation constant of Ca ions with the high affinity Ca binding site of the channel was 0.2 μmol/l.

For two penetrating alkaline earth cations (e.g. Ca and Ba), the kinetic scheme of the calcium channel with different conformational states is

$$S'_{Ba} \underset{Y_{Ba}}{\overset{X_{Ba}}{\longleftrightarrow}} S_{Ba} \underset{b_{Ba}}{\overset{a_{Ba}}{\longleftrightarrow}} S_0 \underset{b_{Ca}}{\overset{a_{Ca}}{\longleftrightarrow}} S_{Ca} \underset{Y_{Ca}}{\overset{X_{Ca}}{\longleftrightarrow}} S'_{Ca},$$

where S_{Ba} and S'_{Ba} are the empty and Ba-conducting states of the channel.

The expression of the total current is

$$i_{Ca+Ba} = -2e_0 \frac{2\left(k_1^{Ca} - k_{-1}^{Ca} X_{Ca} / Y_{Ca}\right)[Ca] / K_{Ca} + \left(k_1^{Ba} - k_{-1}^{Ba} X_{Ba} / Y_{Ba}\right)[Ba] / K_{Ba}}{1 + \left(1 + X_{Ca} / Y_{Ca}\right)[Ca] / K_{Ca} + \left(1 + X_{Ba} / Y_{Ba}\right)[Ba] / K_{Ba}}.$$

Thus, we have shown that various anomalous mole fraction effects observed recently in Na/Ca and Ca/Ba mixtures can be successfully described on postulating different conformational states of the channel. The binding of Ca^{3+}, Ba^{2+} (and possibly Na^{+}) to high-affinity) sites may switch the transition of the channel to different conducting states. These conformational transitions are an intrinsic property of Ca-binding proteins. As to the calcium channel, they may participate in its gating mechanisms. In this respect, studies of possible relationships between the gating and the ion-transporting machinery of the calcium channel should be mentioned. Recently, it was shown for calcium channels fromrat brain and Aplysia neurones (Chesnoy-Marchais 1985) that the channel conductance and its mean open time arc inversely related. That is the stronger a given ion binds to the channel the lower is its conductance and the larger is its open time. Obviously, even this relationship should be expected from the model of a channel with different conformational states.

V. MEMBRANE MOLECULAR STRUCTURES WITH FUNCTIONS OF "CURRENT NANO-GENERATOR OF CH-CRC TYPE"

Membrane in brain cells is dotted by approximately 18- to 40 thousands of channel-receptor complexes (CRCs), inquired into the membrane. Principles of their performance are in following.

1. While the CRC maintained in state of rest it doesn't let ions come across through the ionic channel. In order to make the ionic channel let the flow of ions come across it the agonist is needed to be attached to the CRC`s receptor.

2. The generation of ionic currents is useful for the propagation of action potentials, and requires the movement of significant numbers of ions across the membrane in a relatively short period of time. The rapid rate of ionic flow occurring during the generation of an action potential is far too high to be achieved via an active transport mechanism. Rather it results from the opening of ion channels.

3. An agonist is a chemical that binds to a receptor of a cell and triggers a response by that cell.

4. Antagonist is a type of receptor drug that does not provoke a biological response itself upon binding to a receptor, but blocks or dampens agonist-mediated responses. Type of CRC we picked up for modeling we call "Ch-CRC type" that means "chemosensitive CRC". For the activation of such Ch-CRC its interaction with agonist is necessary that

is followed by electric current appearance. The steady state of such Ch-CRC is demonstrated using our model on Fig. 5. Actually, at this figure the initial state of biological nano-generator is represented.

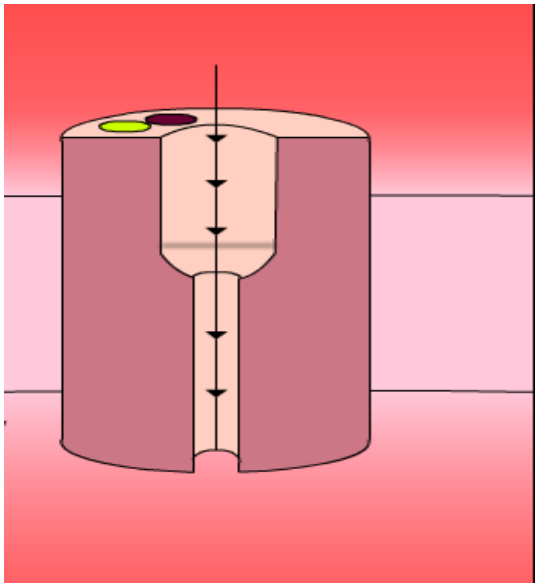


Fig. 5. A sectional view of a single Ch-CRC: yellow spot is the locus of the agonist attachment; dark red spot is the locus for the antagonist attachment

VI. DISCUSSION

On Fig. 6 the realization of functions of Ch-CRC (nano-generator) is shown in dynamics. Let's make some explanations to this program demonstration. On the base of this model information from other publications presented above is put as well as results of original experiments. Curves with results of original experiments one can see at the centre of Fig. 6. There are demonstrated the results of Ch-CRC activation by agonist kainate (KK) that caused the influx of non-inactivated inward current (on example of receptor of glutamate). At the second stage KK-activated current is blocked by specific toxin JSTX-3 – maximal amplitude of these electric current decreases to minimal values (but not to zero). At the third stage JSTX-3 is removed from Ch-CRC and KK-activated electric current amplitude increased (but not to the primary values!). Main stages of these processes one can see as 4 pictures around recorded experimental results. Indeed, expressions of these functions are similar to ones of "generator" – but biological "nano-generator". Performances of these processes permit us to see in dynamics all stages of Ch-CRC; block of KK-activated currents permit us to control of "nano-generator" at molecular level.

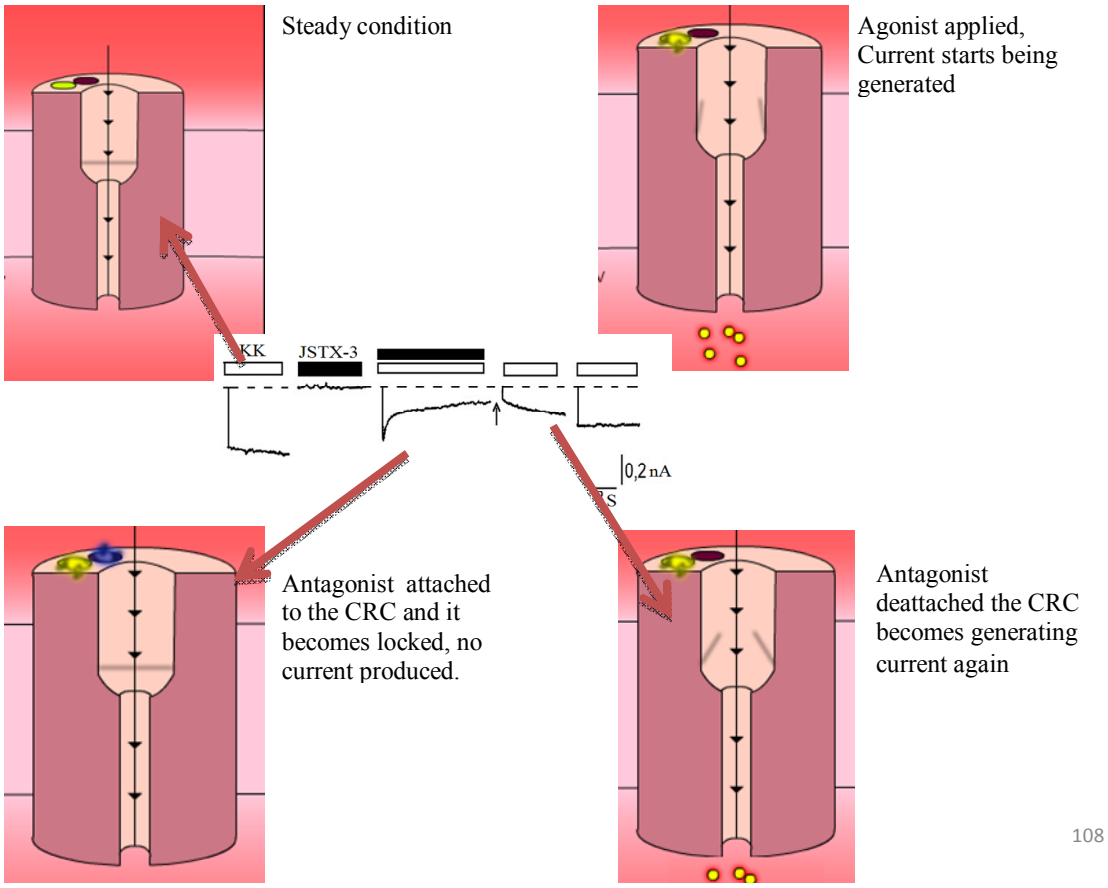


Fig. 6. Program model of Ch-CRC (nano-generator) performance in dynamics. Four phases of its action are shown (explanation see in text)

VII. CONCLUSIONS

In present article the results of following works are represented.

1. Were observed some scientific publications where the nature of electric currents through the brain membrane has been explained.

2. Were observed also some publications where were shown the functions of molecular structures responsible for electric currents generation in brain membranes, their similarity to biological "nano-generator" was revealed.

3. Mathematic model of electrical current generation by molecular structures in brain cell membranes is represented.

4. Original program model of molecular nano-generator functioning for electro-activated channel-receptor molecular complex (Ch-CRC) is described. Performances of its functioning permit us to see all stages of Ch-CRC in dynamics. Block of KK-activated currents by toxin JSTX-3 permit us to control the functioning of this "nano-generator" at molecular level.

5. Program model permits us to see functioning of Ch-CRC (nano-generator) in dynamics. Main four stages of these processes one can see as four pictures around recorded experimental results on Fig. 6.

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Received 18 May 2014

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О. М. Ключко, Д. Ю. Шеремет. Програмне моделювання функцій біологічного нано-генератора
Наведено результати виконаного програмного моделювання «молекулярного генератора струму», яке було розроблено на основі експериментальних результатів з дослідження електричних струмів через канало-рецепторні комплекси у природних нейронах мозку.
Ключові слова: програмна модель; мозок; нейрон; молекулярний генератор струму; наноструктура.

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Е. М. Ключко, Д. Ю. Шеремет. Программное моделирование функций биологического нано-генератора

Приведены результаты выполненного программного моделирования «молекулярного генератора тока», которое разработано на основе экспериментальных результатов по исследованию электрических токов через канало-рецепторные комплексы в природных нейронах мозга.

Ключевые слова: программная модель; мозг; нейрон; молекулярный генератор тока; наноструктура**.**

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