Modeling the effects of extracortical sources on the EEG-signal

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Abstract— **Extracortical activity play important role in generation of EEG-signal. Neuronal system in the brain seems to be specially sensitive to sources belonging to the class of functions: [Acos(αt)+Bsin(αt), exp(±αt), Bessel functions]. This representation could be reflect influence of external sources, for example electromagnetic fields, on the brain. In my previous papers I demonstrated the effect of electromagnetic field on the EEG-signal using nonlinear dynamics method - Higuchi's fractal dimension.**

The extracortical input from inhibitory neurons destroys the oscillatory structure of signal generated by extracortical input from excitatory neurons but do not suppresses it completely. The additional component is needed. The long-distance input from excitatory neurons take this role.

The most important parameter in the model is postsynaptic neurotransmitter rate constants. It shows a biphasic effect changes for inhibitory postsynaptic potentials (IPSP) in the EEG-signals during anesthesia. The similar behavior is visible for postsynaptic rate constants but in case of excitatory postsynaptic potential (EPSP) in epileptic EEG-signals. There are two opposed processes occur.

The average excitatory soma potential (h_e) depend on total number of excitatory and inhibitory intracortical connections. The increase of maximum value of the h_e occurs for bigger number of excitatory connections. The shape of the curve depend on number of inhibitory connections.

Keywords— **electrocortical model, EEG, extracortical activity**

I. Methods.

In this work I present some results obtained using electrocortical model of Liley [3,4]. The main computational observable in Liley's model is the average excitatory soma membrane potential, he. It is expected to be linearly related to EEG voltage. The model assumes existence of two distinct cortical subpopulations of neurons (macrocolumns): excitatory and inhibitory. Three sources give input to excitatory and inhibitory synapses: locally in the same macrocolumn of cortex, long-range but within the cortex and extracortical input, for example from the thalamus. The local interactions within the macrocolumns are proportional to the number of their connections (Nlkβ) and their mean firing rates (Sk), k,l=e (excitatory), i (inhibitory). They are estimated using a sigmoidal dependence on the local mean membrane potential. The long-range connections to other macrocolumns are formed only by the excitatory subpopulations. The model is formulated as a set of eight nonlinear ordinary differential equations (ODE): two first order ODE and six second order ODE. Each of second order ODE can be rewrite as a system of two first order ODE. Thus, the whole model is composed by 14 first order equations with 29 physiological and anatomical parameters. The set is solved numerically using MATLAB procedures (ode45) in the time range from 0 to 400 [ms]. The initial values of variables are zero. In the base model I assumed the standard set of parameters values proposed by Steyn-Ross et all. [1] and I tested the average excitatory soma potential change on different parameters within known physiological bounds [3,4].

II. Results.

A. Dependence of the average excitatory soma potential on parameters values.

The most spectacular changes are observed for postsynaptic rate constants for inhibitory postsynaptic potentials (IPSP). It shows a biphasic effect for the parameter value changes value from 0.1 to 0.2 (Fig.1b). The biphasic behavior is not observed for amplitude of inhibitory postsynaptic potential (Fig.1a).

Steyn-Ross et all. [1] suggest that this effect could be explained as a phase transition in the cortex between the activated and quiescent states at a critical value of anesthetic.

The average excitatory soma potential for different excitatory postsynaptic neurotransmitter rate constants (EPSP) demonstrate the similar behavior for epileptic signals. Wendling et all. [2] show that the model switches from spiking activity to sinusoidal alpha-like activity for specific parameter value. In my previous works I analyzed EEG-signals during sleep, anesthesia and epileptic seizure using fractal dimension method. The nonlinear character of signals is clearly visible in these cases.

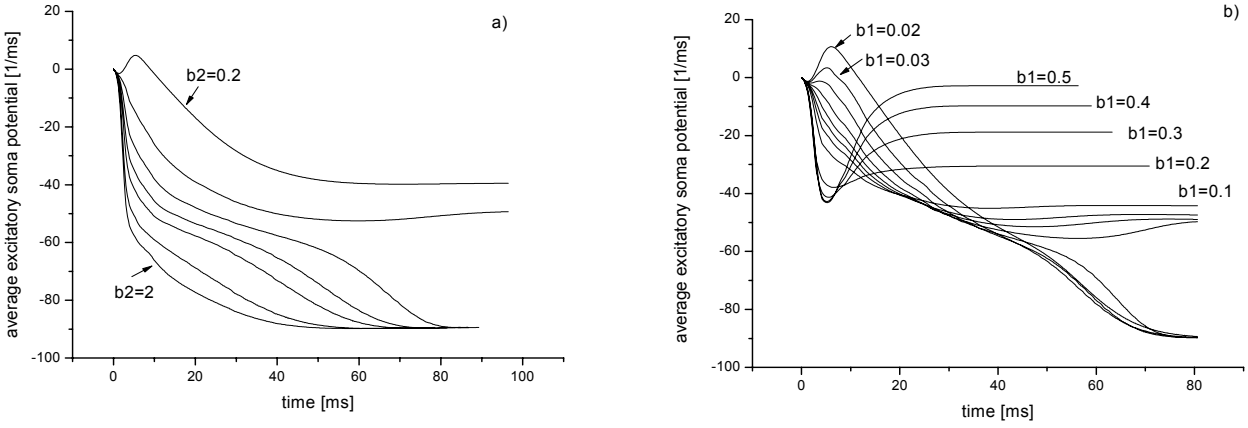

Fig.1 The average excitatory soma potential: a) for different amplitude of inhibitory postsynaptic potentials (IPSP) in the range: [0.2, 0.4, 0.6, 0.8, 1, 1.5, 2]; b) for different postsynaptic neurotransmitter rate constants IPSP in the range: [0.02, 0.03, 0.04, 0.05, 0.06, 0.07, 0.08, 0.1, 0.2, 0.3, 0.4, 0.5].

The sharp decrease of fractal dimension can be noticed at the beginning of anesthesia [10,12-13]. The change of complexity of system could be explain by the biphasic behavior caused by the phase transition in cortex between two brain states (activated and quiescent) in moment of anesthetic induction. Such behavior is not visible in whole-night sleep EEG-signal where fractal dimension decreases slowly from waking state to the deep sleep passing smoothly through consecutive sleep stages [8-10].

The phase transition between two types of EEG activity during epileptic seizure (spiking activity and sinusoidal alpha-like activity) is reflected also in fractal dimension of such signals. The fractal dimension decrease sharply when the seizure occur [10,11].

The average excitatory soma potential, h_e depend on total number of excitatory and inhibitory intracortical connections. The increase of maximum value of the h_e occurs for bigger number of excitatory connections (Fig. 2a). The shape of the curve depend on number of inhibitory connections (Fig. 2b).

B. Influence of extracortical input to excitory/inhibitory synapses and influence of long-distance effects.

One of three inputs to synapses is the extracortical input, for example from the thalamus. The primary role of thalamus is to relay sensory information from other parts of the brain to the cerebral cortex filtering out important messages from the mass of signals entering the brain. It plays role in arousal, attention, memory, pain, alertness, consciousness and expression of emotion.

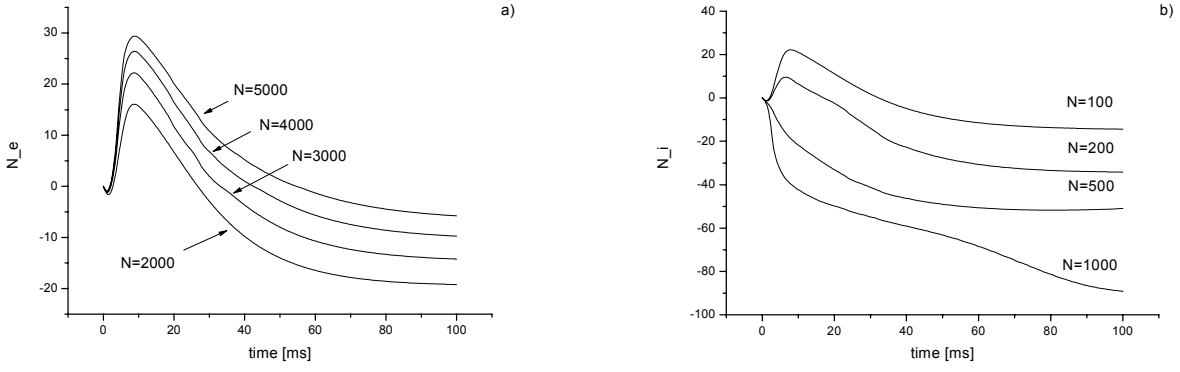

Fig.2 The average excitatory soma potential for different total number of intracortical connections. a) for excitatory (ee,ei) connections in the range: [2000, 3000, 4000, 5000]; b) for inhibitory (ie,ii) connections in the range: [100, 200, 500, 1000].

In the Liley's model extracortical activity is represented by physiologically shaped brown noise [4]. In my system this activity change in time as a sinusoid or exponentially suppresses sinusoid. This representation could be reflect influence of external sources, for example electromagnetic fields, on the brain. In the previous papers the effect of electromagnetic field on the EEG-signal using Higuchi's fractal dimension method was demonstrated [5-7] .

The average excitatory soma potential in function of extracortical input p_{ee} rate for three cases was analyzed: p_{ee} = 0; p_{ee} = 10000 * sin(t); p_{ee} = exp(-t/100) * 10000 * sin(t). I assumed p_{ei} = p_{ie} = p_{ii} =0 and absence of long-distance cortico-cortical effects (Φ=0). The structure of extracortical input function is well reflected in observed EEG-signal.

In addition to sigmoid-modulated synaptic input from the sources placed locally in the same macrocolumn of cortex, there could be present also long-range cortico-cortical contributions from distant excitatory assemblies. The effect of h_e oscillations suppression is observed in this case (Fig.3).

The long-distance input is described in the model by two additional second order ODE depending on time and spatial variables. These equations are kind of telegraph equations:

$$\frac{\partial^2}{\partial \square^2} u + \square \cdot \frac{\partial}{\partial \square} u = \square^2 \cdot \frac{\partial^2}{\partial x^2} u + b \cdot u$$

for k>0, b<0.

Substitution:

$$u(x,t) = \exp(-\frac{1}{2} kt) \cdot w(x,t)$$

give the Klein-Gordon Equation, encountered in quantum field theory:

$$\frac{\partial^2}{\partial \square^2} w = \square^2 \cdot \frac{\partial^2}{\partial x^2} w + (b + \frac{1}{4} \cdot k^2) \cdot w$$

Some of particular solutions of the Klein-Gordon equation are:

1. u(x,t)=sin(βx) [Acos(αt)+Bsin(αt)];
2. u(x,t)=exp(±βt) [Acos(αx)+Bsin(αx)];
3. u(x,t)= $AJ_0(\xi)+BK_0(\xi)$, where J_0, K_0 – the modified Bessel functions.

When complete spatial homogeneity over the region sampled by the EEG electrode is assumed, the one-dimensional Laplasian $\partial^2/\partial x^2$ is eliminated, and only time is variable in the set of ODE. All partial derivatives with time become total derivatives with time.

In this case the long-distance input belong to the class of functions: [Acos(αt)+Bsin(αt), exp(±αt), Bessel functions looking roughly like oscillating sine or cosine functions that decay proportionally to $1/\sqrt{x}$]. The same class of functions was used to describe the extracortical input p_{ee}. Neuronal system in the brain seems to be especially sensitive to sources described by such functions.

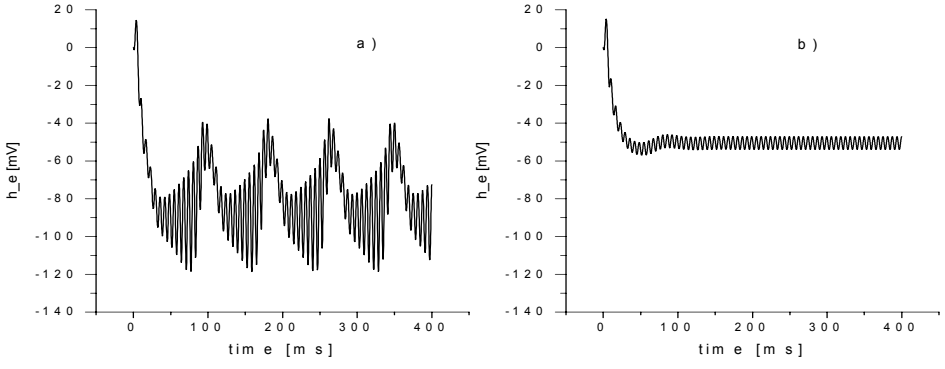

Fig.3 The average excitatory soma potential in function of extracortical input p_{ee} = 10000 * sin(t) without and in presence of long-distance input.

C. Influence of p_{ee}/p_{ie} ratio.

The role of extracortical input from excitatory (p_{ee}) and inhibitory (p_{ie}) neurons to excitatory synapses was studied. The signal is described by function Asin(t).

For the extracortical input from excitatory neurons value of amplitude is A=10000 and from inhibitory neurons amplitude change from 2000 to 10000.

The play between three sources (extracortical input from excitatory neurons, extracortical input from inhibitory neurons and input from distant excitatory neurons) carry on. The input from inhibitory neurons destroys the oscillatory structure of signal but do not suppresses it completely. The long-distance input from excitatory neurons take this role.

III. Conclusions.

Modeling of processes occurred in the brain at the cellular level permits to explain phenomena observed at the macroscopic level in EEG-signal. My previous studies of EEG-signal using nonlinear dynamics method like Higuchi's fractal dimension revealed existence of interesting behaviour of neuronal system during sleep, anesthesia and epileptic seizure. The effect of electromagnetic field on the EEG-signal was demonstrated also. The sharp decrease of fractal dimension can be noticed at the beginning of anesthesia. The change of complexity of system could be explain by the biphasic behavior caused by the phase transition in cortex between two brain states (activated and quiescent) in moment of anesthetic induction. Such behavior is not visible in whole-night sleep EEG-signal where fractal dimension decreases slowly from waking state to the deep sleep passing smoothly through consecutive sleep stages. The phase transition between two types of EEG activity during epileptic seizure (spiking activity and sinusoidal alpha-like activity) is reflected also in fractal dimension of such signals. The fractal dimension decrease sharply when the seizure occur. One of parameters in the model, postsynaptic neurotransmitter rate constants, shows a biphasic effect changes for inhibitory postsynaptic potentials (IPSP) in the EEG-signals during anesthesia. The similar behavior is visible for postsynaptic rate constants but in case of excitatory postsynaptic potential (EPSP) in epileptic EEG-signals. There are two opposed processes occur. The model shows an interesting play between three sources: extracortical input from excitatory neurons, extracortical input from inhibitory neurons and input from distant excitatory neurons. Extracortical activity play important role in generation of EEG-signal. Neuronal system in the brain seems to be specially sensitive to sources with characteristics belonging to the class of functions: [Acos(αt)+Bsin(αt), exp(±αt), Bessel functions]. This representation could be reflect influence of external sources, for example electromagnetic fields, on the brain. The extracortical input from inhibitory neurons destroys the oscillatory structure of signal generated by extracortical input from excitatory neurons but do not suppresses it completely. The additional component is needed. The long-distance input from excitatory neurons take this role. Thalamus is a very important part of the brain. It is not only filtering out useful information from other parts of the brain to the cerebral cortex, but it protects the cerebral cortex from dangerous signals. It seems to have a role of specific "firewall".

Acknowledgements

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