## STUDY OF PROPAGATION OF ELECTRIC IMPULSES IN NERVE FIBERS OF LIVING ORGANISMS

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Abstract. The propagation of solitary electric impulses in nerve fiber of living organisms is considered on the basis of the model of the generalized KdVB equation. It is shown that the nonlinear dependence of the membrane permeability on pulse amplitude leads to increasing of a steepness of the pulse front, the diffusion processes, which level the concentration on both sides of the membrane, make the front flatter, and dispersion causes the pulse to blur due to the difference in the propagation velocities of the harmonics composing the pulse. It is found that if these competing processes balance each other, then the pulse propagates through the fiber with the constant velocity without changing its shape looking like a soliton. The case when a single electric impulse can transform to a sequence of solitary pulses is noted, and that can be perceived by a living organism as another signal different from that at the input of system.

By the middle of the XX century, it was found that the propagation velocity of the electric (nerve) impulse along the nerve fiber  $v \sim \sqrt[4]{d}$  where *d* is the thickness of the central part of the fiber, i.e. it is very small, for example, for the transmission of a danger signal along the nerve fiber (in mammals  $d \leq 20$  microns, in squids and cuttlefish  $d \sim 1$  mm).

Evolution, however, "invented" another, more perfect, than the increase in the nerve fiber thickness, method of increasing the velocity of the nerve impulse: in higher animals and in humans, many nerve fibers are enclosed in the insulating shell, which gave a significant increase of propagation velocity v.

So, in the thick nerve fiber of cuttlefish v= 25 m/c, and in the mammals' fibers, which are 50 times thinner,  $v \sim 100$  m/s. In 1952, the British physiologists A. Hodgkin and A. Huxley built a theory [1] (Nobel prize 1963), which was confirmed by experiments and is based on the idea that nerve impulses are sequences of the same form of solitary electrical impulses (u, V), the form and v of which do not depend on the magnitude of irritation. Irritation is "quantized", i.e. the reaction

to it, depending on its intensity, is a series of exactly the same impulses. Moreover, if the magnitude of the irritation is below a certain threshold, the pulse is not formed. These facts are related to the Hodgkin-Huxley theory with soliton theory, in which there is a phenomenon of decay of perturbation of large amplitude on a sequence of nonlinear solitary waves – solitons [2].

In this paper, a model of the nerve fiber, including its structure and processes of salt metabolism, is constructed similarly to [3], and numerical simulation of the process of nerve impulse propagation is carried out on its basis. The model used in our numerical simulation looks as shown in Fig. 1.



Fig. 1. The model of nerve impulse propagation in the nerve fiber:(a) model of fiber, (b) form of the nerve impulse.

The fiber consists of a core enclosed in a shell (membrane) and immersed in an external plasma (Fig. 1a). The inner and outer plasmas are very different in their composition: on the outside it contains an excess of Na+ and Cl<sup>-</sup> ions formed during dissociation of NaCl; inside there are more K<sup>+</sup> ions and negatively charged ions of organic molecules. The membrane is permeable for the Na<sup>+</sup>, Cl<sup>-</sup> and K<sup>+</sup> ions, but it does not pass large organic molecules. In a quiet state all the transition processes are balanced so that the inner part of the fiber contains an excess of negative ions, and between the inner and outer plasmas voltage  $u \approx 50$  mV. When the nerve is irritated by a

sufficiently large external pulse, the membrane begins to pass Na+ ions inside, and in the site of irritation, the voltage quickly changes to the opposite. Neighboring membrane regions are involved in the process, and the voltage pulse *u* begins to propagate through the fiber (Fig. 1b). An impulse can be formed and propagated only because there is a nonlinear element in the model that suppresses small deviations from the normal state and, conversely, amplifies large ones. In the absence of nonlinear effects, the leading front of the pulse would begin to blur as a result of dispersion processes and diffusion of ions across the membrane.

The proposed model takes into account all three processes and can be described by the wellknown generalized Korteweg-de Vries-Burgers (GKdVB) equation [4]

$$\partial_t u + \alpha u \partial_x u + \beta \partial_x^3 u + \gamma \partial_x^5 u = \sigma \partial_x^2 u \tag{1}$$

corresponding to the dispersion law  $\omega = c_0 k [1 - (i\nu k + \beta k^2 - \gamma k^4)/c_0]$  [4] where the coefficient  $\alpha$  is defined by the intensity of external irritation, the coefficients  $\beta$ ,  $\gamma$  and  $\sigma$ , describing the processes of dispersion (including high-order dispersive effects) and diffusion, by the processes of salt metabolism.

Simulation of the propagation of a nerve impulse of type of

$$u(x,0) = u_0 \left[ \operatorname{sch}^2(x) - 1/2 \right]$$
(2)

shown in Fig. 1b and used as initial condition of the initial value problem (1), (2), using the implicit difference scheme with approximation  $O(\tau^2, h^4)$  [4], allowed us to obtain the following results which characterize the features of the process (see Fig. 2).

The nonlinear dependence of the membrane permeability on its amplitude leads to increasing of a steepness of the pulse front, the diffusion processes, which level the concentration on both sides of the membrane, make the front flatter, dispersion causes the pulse to blur due to the difference in the propagation velocities of the harmonics composing the pulse. At this, as one can see, the high-order dispersion correction extracts the high-frequency harmonics and the waving tails appear in the pulse structure. If these competing processes balance each other, then the pulse propagates through the fiber with v = const, without changing its shape, i.e. it is a solitary wave or soliton. In a case when the value of coefficient  $\gamma$  is rather big, the tail oscillations can have rather big amplitudes and a single electric impulse can transform to a sequence of solitary pulses. This can be perceived by a living organism as another signal different from that at the input of system.



Fig. 2. Evolution of pulse (2) with  $\alpha = 1$ ,  $\beta = 2.3 \cdot 10^{-3}$ ,  $\nu = 0.01$  at its propagation in a fiber: (a)  $\gamma=0$ , (b)  $\gamma = 1 \cdot 10^{-4}$ .

## References

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