Electromagnetic Emission Sources in the Active Nerve

Dear Sir:

In a communication published in the *Biophysical Journal*, A. Fraser and A. Frey (1) reported that the active nerves of the blue crab have an electromagnetic emission in spectral bands corresponding to a range of wavelength between 2 and 20 μ . This emission, with an intensity of 6 μ w/cm² for 20 pulses/sec, cannot be assigned either to a black body with the same temperature as the nerve or to the heating artifact from stimulation. The source of this electromagnetic emission is located at the surface of the nerve. Taking into account the major importance of this discovery, we shall discuss here the possible sources of this emission.

From electrodynamics it is well known that only an electric charge in accelerated movement can produce an electromagnetic emission. The electric charges which, during the action potential, could be in an accelerated movement at the axon surface are: (a) Na⁺, K⁺, and Cl^- ions which cross the membrane during the action potential, (b) the electric dipoles which turn during the excitation (2), (c) the negative surface charges of the axon (3, 4) which could turn.

(a) In the resting membrane of the axons there is an electric field: $E \approx 10^7$ v/m. This field accelerates the ions which cross the membrane during the excitation:

$$a = eE/m = 3.2 \times 10^{13} \text{ m/sec}^2$$
. (1)

Here $e = 1.6 \times 10^{-19}$, coulombs is the elementary charge, and m is the mass of the ion. We have taken m = 30 amu because the differences between the atomic mass of Na, K, and Cl are insignificant for these calculations.

The whole intensity of the emission of a linear accelerated charge is (5):

$$I = 2e^2a^2/3c^3, (2)$$

where $c = 3 \times 10^8$ m/sec.

With the above data we obtain $I=0.6\times10^{-26}\,\mathrm{W}$. From the Fraser and Frey measurements the energy released per impulse is $0.3\times10^{-6}\,\mathrm{W/cm^2}$. For such a value it is necessary that at least 10^{-4} moles of monovalent ions cross 1 cm² of the axon membrane during the excitation, while the experimental measurements (6) show a much smaller value: $10^{-11}\,\mathrm{moles/cm^2}$. Accordingly, it is clear that the electromagnetic emission cannot be assigned to the accelerated linear movement of the ions through the membrane.

(b) and (c) In both these cases the theoretical problem of the electromagnetic emission is almost the same. The electric dipole theory of nervous excitation (2) supposes that, during the action potential, the dipoles of the membrane surface turn. The maximum energy of the quanta, \mathcal{E} , emitted by the rotation of a dipole is: $\mathcal{E}=2dE$, where d is the dipole moment. For the turning of an electric charge, $\mathcal{E}=erE$, where r is a distance ~ 10 A. If we consider $d=1.6\times 10^{-28}$ C·m and $E=10^7$ v/m, d and E having the same direction, then, $\mathcal{E}=3.2\times 10^{-21}$ J. This corresponds to a wavelength of about $60~\mu$, which is far greater than the observed values. The same results for the electric charge rotation. The conclusion is that the electromagnetic emission of the active nerves cannot be explained by the previously discussed mechanisms, based on the classical electrodynamics. In addition, the external membrane dipoles must absorb and not release energy.

The quantum theory of the free rotation shows that the frequences of the emitted quanta are (7):

$$\nu = \frac{(j+1)\hbar}{4\pi i} \,, \tag{3}$$

where $j=0,1,2,\cdots,\dot{I}$ is the moment of inertia of the microscopic body in rotation, and $\hbar=1.054\times 10^{-21}\,\mathrm{erg}$ sec. Since, for the protein units, \dot{I} is relatively large, ν corresponds to a wavelength far greater than 100 μ . That is why we don't try to study the rotation in an electric field of the charged or dipolar protein units even if this is very intensive.

Quanta with wavelengths between 2 and 20 μ can appear only by vibrorotational transitions. The elementary negative charges belong to atomic groups. We shall consider only the vibration of these charged groups, and not for other atoms since (a) taking for the energy of an emitted quantum 2×10^{-20} J (8), and for the axonal surface charge density 4.4×10^{13} elementary charges per cm² (4), we obtain for the released energy per impulse 8.8×10^{-7} J/cm², which is of the same order of magnitude as the experimental findings. This supports the conclusion that the charged groups are responsible for the emission. (b) The emission appears only during the excitation and the most probable action of the stimulus is on charged groups.

As a first approximation, a charged atomic group may be considered to be an isotropic three-dimensional oscillator whose nonelectrostatic potential energy is $k/2(x^2 + y^2 + z^2)$, the origin of the coordinate system being the minimum of the potential well. There is also the action of the external electric field, E, in the Ox direction: $E \cdot e \cdot x$. In this case the energy operator is:

$$-\hbar^{2}/2m(\partial^{2}/\partial x^{2} + \partial^{2}/\partial y^{2} + \partial^{2}/\partial z^{2}) + k/2(x^{2} + y^{2} + z^{2}) + Eex,$$
 (4)

where m is the mass of the charged atomic group. The eigenvalues of the energy are (7):

$$\mathcal{E}_n = (n + 3/2)\hbar\omega_c - E^2 e^2 / 2k, \tag{5}$$

where $n = 0, 1, 2, \cdots, \omega_c = (k/m)^{1/2}$.

The oscillators have stable states. In the resting state of the nerve, by metabolic activity the membrane macromolecules are arranged so that the charged groups are in upper vibrational levels. The stimulus produces some local transformations of the macromolecules, the oscillators considered here passing to lower energy levels. A quantum is emitted:

$$\hbar\omega = \Delta \mathcal{E}_n = \Delta n \cdot \hbar \omega_c. \tag{6}$$

It is clear that Δn hasn't large values. For the active nerves ω emitted lies between 10^{14} and 10^{15} cycle/sec. It follows that ω_c lies between 10^{13} and 10^{14} cycle/sec, which agrees very well with the known data (9).

Our purpose here is to analyse the possible mechanisms of the electromagnetic emission of the active nerves. The conclusion is that this emission can be explained on the basis of the physicochemical theory of excitation (10, 11) which assumes that, during the excitation, a transformation of the membrane macromolecules takes place which means vibrorotational transitions.

Received for publication 16 December 1969.

REFERENCES

- 1. Fraser, A., and A. H. Frey. 1968. Biophys. J. 8:731.
- 2. WEI, L. Y. 1969. Bull. Math. Biophys. 31:39.
- 3. SEGAL, J. R. 1968. Biophys. J. 8:470.
- 4. GILBERT, D., and G. EHRENSTEIN. 1969. Biophys. J. 9:447.

Letters to the Editor 483

- 5. Jackson, J. 1962. Classical Electrodynamics. John Wiley and Sons, Inc., New York. Chap. 11.
- 6. PASINSKII, A. 1968. Biofizitcheskaia Himiia. Vyschaia Shkola, Moskva. 214.
- 7. MOELWYM-HUGHES, E. A. 1961. Physical Chemistry. Pergamon Press, New York. Chap. 4.
- 8. MÄRGINEANU, D., and D. MOISESCU. 1970. Bull. Math. Biophys. 32. (In press).
- WILLIAMS, D. H., and I. FLEMING. 1966. Spectroscopic Methods in Organic Chemistry. Mc-Graw-Hill Book Company, New York. Chap. 3.
- 10. TASAKI, I., and I. SINGER. 1966. Ann. N.Y. Acad. Sci. 137:792.
- SINGER, I., and I. TASAKI. 1968. In Biological Membranes. D. Chapman, editor. Academic Press, Inc., New York. 347.
 - D. Moisescu
 - D. MĂRGINEANU

Department of Physics, University of Bucharest Department of Biophysics, Faculty of Medicine Bucharest, Romania