

## LETTER TO THE EDITOR

### *Sodium Current Flow in Excitable Membranes*

Dear Sir:

In the November (1970) issue of the *Biophysical Journal*, Hamel and Zimmerman stated in their paper, "... we demonstrate that while changes in the voltage across the polar portions of the molecule are responsible for the negative steady-state resistance they cannot be responsible for changes of sodium flux by a factor of 250. Wei (1966) ignores the main energy barrier to  $\text{Na}^+$  permeation: the partition energy necessary to go from water to lipid. Changes in polar voltage can at most account for changes of flux by factors of only 3-5." I wish to comment that the second half of the above statement may have been deduced from a misunderstanding. In my paper published in 1966, I had suggested two methods for the calculation of ion currents in nerve membrane: the first is to employ an equivalent circuit (not of the Hodgkin-Huxley type but of a transistor type) and to take the Laplace transform; the second is to solve the two basic equations—the equation of continuity and the Nernst-Planck equation. The partition energy from water to lipid is then implied in the diffusion constant to be used in the Nernst-Planck equation. Thus my theory in essence has not ignored the role of partition energy for the ion current flow across a nerve membrane.

Although the diffusion current is not explicitly related to the height of the dipole barrier at the interface, a slight change in that barrier can result in a great change of the "total" current observed. The paradox stems from the fact that in the resting state, the drift current ( $I_f$ ) is opposite and equal (or nearly equal) to the diffusion current ( $I_d$ ) at the interface and hence the resting current

$$I_R = I_d - I_f \simeq 0.$$

Now if under stimulation the dipole barrier change reduces the drift current to one-third of its resting value while the diffusion current remains unchanged, then the total current observed will be

$$I_S = I_d - \frac{1}{3} I_f = \frac{2}{3} I_d,$$

and therefore

$$I_S/I_R = \frac{2}{3} I_d/I_R = \text{a very large number.}$$

Here we see the sort of "transistor action". The dipole barrier at the outer interface (or the "emitter" junction) not only functions like a switch but also brings about amplification following my theory (1966). A solution, however sophisticated, of the Nernst-Planck equation *alone* cannot and will not show clearly this kind of physical action. One needs to solve the continuity equation

$$\frac{\partial n}{\partial t} = G - R - \nabla \cdot J,$$

( $G$  and  $R$  are the generation and recombination rates) together with the Nernst-Planck equation in order to get a more complete physical picture. At present, little work has been done in this direction. In my view, this is a theoretical "gap" in nerve studies.

The dipole theory which I developed subsequent to 1966 is capable of dealing with a wide range of nerve phenomena, not merely electrical (Wei, 1968, 1969 *a*, 1969 *b*). Furthermore, the fundamental properties of nerve impulse can be shown in exact mathematics to derive from the quantum transitions (stimulated and spontaneous) of the electric dipoles at the membrane interface (Wei, 1971). Detailed calculations of dipole reorientations under stimulation also show unequivocally the correlation between the action potential and the birefringence change in nerve axon and thus give a deeper insight into the mechanism of action potential (Wei, unpublished data). The latent potential of the dipole theory is perhaps much greater than what its simple name might have indicated and hence deserves further and thorough investigation.

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### REFERENCES

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