Ion Adsorption and Excitation

III.

It has been demonstrated that the ions involved in the fluxes characteristic of the excitation of squid giant axons form a monolayer at the functional nerve surface and that they are not hydrated[1]. As a consequence it was postulated that the resting potential of these nerves can be regarded as the algebraic sum of the potentials as calculated separately for each of the major monovalent ions represented at the surface monolayer, the assumption proving consistent with the data found in the literature[2].

The resting potential appears thus to express solely the ionic asymmetry obtaining between the nerve surface monolayer and the outer solution. The use of the gross internal ionic concentration in the calculation of the resting potential appears to be justified because the monovalent ion composition of the surface monolayer is identical with that of the nerve interior. This identity arises from the definition of the volume Ar which at the functional nerve surface represents one half of the volume of a monolayer of a given ionic species[1].

The rapidity with which the nerve regains its ability to conduct a second impulse is apparently contradicted by the demonstration that recovery is a prolonged process, the thermal events following a single impulse lasting some 300 ms[3]. Since the nerve surface monolayer acquires the ionic composition characteristic of the outer solution as a consequence of the passage of an impulse[1], the paradox can be resolved if it is assumed that the return of the potential to its resting value is due to the establishment of a new interface some 17 Å beneath the former one. The value of 17 Å is taken since it represents the average distance between the K ions in the axoplasm. This new interface ought to appear concomitantly with the cessation of the ionic equilibrations accompanying the previous impulse, the velocity of its appearance being a function of the length of the diffusion path and velocity of the fluxing ions. A rapid train of impulses would thus cause the nerve to enter into a sort of 'debt' because of the creation of a peripheral plasma layer in which the monovalent ion atmosphere is identical with that of the outer solution in regard to composition.

Assuming the diffusion coefficient of ions in the outer neuroplasm to be identical with that in a solution of a comparable molality, the thickness of the peripheral layer of normal resting axon can be estimated from the diffusion formula[4]

$$C = C_0\left(1 - \frac{2}{\pi}\int_0^y e^{-y^2}\,dy\right) \text{ where } y = \frac{x}{2\sqrt{Dt}}$$

where x = thickness of the layer in cm

$D = 1{\cdot}534 \times 10^{-5}$ cm²/s

$t = 2{\cdot}45 \times 10^{-3}$ s (duration of the impulse as measured on an oscilloscope trace[5]

$C_0 = K_i = 345\ mM$/kg

$C = K_0 = 22\ mM$/kg

According to this calculation, the functional surface of a normal nerve, i. e. the interface across which the resting potential is manifested is situated 5 μ below the morphological surface.

The views expressed here are in a sense shared by SHANES[6] who considers such a peripheral layer to be 16 μ thick, this value resulting from tracer diffusion kinetics, and by SHAW[7] according to whom about 1/3 of the muscle fiber volume, in contrast to the rest of the cell, is regarded as being 'in physicochemical equilibrium' with the outer solution. HARRIS[8] likewise postulated the presence in frog muscle of a Na^+ rich annular layer about 3 μ thick. In red cells this peripheral layer is also believed capable of expansion under the influence of strophantin[9].

The presence of such a peripheral plasma layer in nerves would account not only for such physical effects as the 'fast' component of tracer exchange, but would also serve to interpret the 'tiring' of nerve preparations and the attendant lengthening of the spikes in terms of longer diffusion paths.

The deeply staining peripheral layer of neurons long known to appear as a consequence of stimulation[10] is assumed to be identical with the plasma layer postulated here. As a further likelihood, the molecular modifications occurring in stimulated nerves as revealed by various protein denaturation changes[11–13] may be considered as localized at the functional surface of the nerve. In fact, these structural rearrangements may well prove to be the primary cause of changes in both the ionic composition and stainability of the nerve periphery.

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Résumé

Les données expérimentales suggèrent que la surface fonctionnelle du nerf (c'est-à-dire la couche à travers laquelle le potentiel de repos se manifeste) est située à une certaine distance de la surface morphologique de l'axone et sa localisation à chaque moment dépend de l'état fonctionnel de la cellule. D'après un calcul fondé sur la durée du «spike» de l'axone du Calmar, la surface fonctionnelle de la cellule serait à 5 μ de la surface morphologique.

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