Editorial

Cardiac "Membrane Potential" as the Spatial Summation of Membrane Activities of Multiple Cells*

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While, in the heart, the individual fibers are branching and connecting with each other to complicated networks, they can not be regarded to form a simple syncytium, for the fibers which are apparently connected are separated with the intercalated disc at their mutual junction. Yet the impulse can be transmitted from one fiber to another with a high degree of safety. In this respect the cardiac muscle resembles more the smooth muscle than the skeletal muscle.

Since the cardiac muscle has such a complex fiber architecture, and an isolated single cardiac fiber is not available for experiment, it should always be borne in mind that a wave form of the "membrane action potential" recorded from one cell by means of an intracellular electrode, though having most of its origin in the excitation of the cell, can not be determined entirely by that, but is more or less influenced by the activities of adjacent cells which might not be uniform according to circumstances. Therefore, it is not a surprise to find that the configuration of the membrane action potential of the same cell recorded consecutively does not remain the same according to the way in which the excitation is induced.

Hayashi, in this laboratory, demonstrated in the canine heart that a slight but definite change in the wave form of conducted action potential in the same cell could be produced by modifying the route of entry of impulse or by making two propagating impulses collide within the cell. He considered that this effect was attributable to the lack of uniformity or symmetry in property and structure of the fibers in the adjacent area. Although these findings were observed in the normal heart, it is well conceivable that they may be exaggerated in cases when

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the excitability of the muscle is depressed. In fact such duality of the action potential pattern was observed more distinctly when the heart muscle was exposed to anoxia or changes in chemical and physical condition of the medium (e.g., low temperature, altered ion concentration or osmotic pressure) or when mechanical injury was induced. Even in the normal heart the variability of membrane potential becomes significant in the areas having lowered safety factor for impulse conduction such as in the A-V node and its vicinity, or in the phase of relative refractoriness of the muscle cell in which the safety factor for conduction is also subnormal.

In this respect, the membrane potential of the transitional fibers from the Purkinje fiber to the ventricular muscle is of interest. The features of membrane potential of the subendocardial cells in the canine ventricle are different from both those of the false tendon and of the proper ventricular muscle, but are intermediate of these two. The author has concluded, based on this fact as well as on the histological findings, that it is the action potential of the terminal Purkinje fiber (TPF). It has also been demonstrated that, in the case of conducted action potential, an abrupt and discrete increase in latency of membrane depolarization is observed in the ventricular muscle compared with TPF. A functional discontinuity seems to be present between TPF and the ventricular muscle, a situation resembling that in the junction of the atrium with the A-V-node.

The author and his collaborators have reported that the spike and plateau of the action potential of TPF appeared separated under the influence of hypothermia, anesthetics (procaine, urethane etc.), some ionic actions (Mg, low external Na) etc. Those are ascribable to a partial failure in impulse conduction. Hoshi, in this laboratory, observed an interesting fact that the plateau thus separated from the spike waned and waxed concomitantly with the action potential and contraction of the underlying ventricular muscles. In this case, at least, the genesis of plateau of TPF seemed to be dependent on the action potential of the ventricular muscles functionally connected to it. Moreover, the author experienced, not infrequently, that the transmembrane potential of a fiber which had been made unresponsive by a local injury, was so much affected by the activities of its adjoining active fibers that the inexcitable fiber appeared to be activated.

Such a possibility should be taken into consideration not only in analyzing the wave form of membrane potential, but also in discussing the cardiac excitability. The significance of “Length or area initially put into active state” which was emphasized by Prof. Yamagiwa as one of the essential attributes of stimulation is, perhaps, more important in the case of the cardiac muscle than in the case of single nerve or muscle fiber. For example, a problem whether a part of the plateau can be
abolished or not by the “ingong” current, on which there has been discrepancy in experimental results and in opinion, can not be solved without considering the effect of the adjacent fibers beside the behavior of the fiber directly polarized by anodic current.

So-called cardiac “membrane potential” picked up by an intracellular electrode does not indicate exclusively the activity of the single cardiac cell, but is also a reflection, more or less, of the temporal and spatial summation of the electrical activities of multiple cells which are connected to it and can be non-uniform in property.

This point is believed to be no less important than the elucidation of the cardiac membrane property per se or its theoretical solution, and one of the urgent problems in the electrophysiology to be investigated in the future.