7974 P

Shock Durations and Measurements of Cardiac Excitability.

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A turtle heart under vagus inhibition shows a complex of functional changes. Three of these changes will be referred to here. Consider the right vagus to be stimulated repeatedly so as to produce a constant condition of inhibition in the heart. Suppose that during this period of inhibition there is a slowing of heart rate, an increase of the time required for propagation of the cardiac impulse from sinus to atrium, and a decrease in the strength of the atrial beat. Suppose that now the sinus be stimulated by shocks such as to drive the heart at a rate faster than the above, slowed, spontaneous rate but slower than the normal rate. Unless partial or complete S-A block occurs, there will be a further increase in the S-A interval and the recorded beat of the atrium will be still further depressed. In other words, the slowed, vagus-inhibited heart appears as though the time required for recovery from a given contraction has been increased at least as much as has the interval intervening between beats. Nevertheless, by the use of induction shocks of from 2 to 5 times threshold strength, "the refractory period" of the dog's auricle is reported to be shortened markedly.[1, 2]

We have studied the threshold to electrical shocks of the turtle atrium in an attempt to discover if there exist measurable changes in electrical excitability during the later part of the recovery process. Induction shocks from a Porter coil and short condenser discharges were used. A shortened absolutely refractory period was always demonstrable when the tissue was under the effect of vagus inhibition. We then chose a fixed time interval (S-I) measured between the beginning of a normal systole (S) and the time of application of a shock to elicit an interpolated systole (I). S-I was made slightly less than the normal intersystolic interval (S-S). A shock was found having a strength just sufficient to elicit the interpolated response. Vagus stimulation was then used to produce a marked inotropic depression of the atrium, sometimes with and sometimes without concomitant slowing of the heart rate. Keeping the same S-I time as in the control determinations, no significant or con-

[1] Lewis, Th., Drury, A. N., and Bulger, H. A., *Heart*, 1921, **8**, 83.

[2] Andrus, E. C., and Carter, E. P., *J. Exp. Med.*, 1930, **51**, 357.

sistent change in threshold to induction shocks or short condenser charges could be found.

Shocks were then produced by means of a double condenser stimulator[3] having a k value of 2 and a time to maximum voltage of 0.018 seconds. With such shocks, the absolutely refractory period was shortened during periods of vagus inhibition but for a given S-I time as above, the voltage required to stimulate the atrium was invariably increased. These results were to be expected in the light of published time-intensity curves plotted from results obtained with normal and vagus inhibited hearts under conditions of controlled rate.[4]

Such findings offer another example of the need for considering not only the intensity but also the time functions of electrical stimuli used in determining tissue excitabilities. Specifically, we would point out that when measurements are made by use of induction shocks only, there may be seen only an apparent quickened recovery of the vagus inhibited tissue following a previous systole. On the other hand, with the longer shocks which were selected to approach pararesonance with the tissue, there is demonstrable both the shortened absolute refractory period which is probably correlated with the shortened period of mechanical systole, and a later prolonged period of depression. The data obtained by the use of such long duration shocks seem to be in closer accord with the observed functional changes than are data obtained by the use of induction shocks alone.

7975 P

Fate of Hexosemonophosphate During Aerobic Recovery of Frog Muscle.*

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An accumulation of hexosephosphate was produced in thin frog muscles (sartorius, ileofibularis, etc.) by keeping them anaerobically

[3] Monnier, A. M., *L'excitation électrique des tissues*, Paris. 1934, 115 ff.

[4] Ashman, R., and Garrey, W. E., *Am. J. Physiol.*, 1931, **98**, 109.

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