ACETYLCHOLINE CONCENTRATION IN THE ATRIA OF TRAINED AND UNTRAINED RATS

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The acetylcholine concentration in the atrial tissues of trained rats is indistinguishable from its concentration in untrained animals.

The object of this investigation was to study the relationship between the bradycardia characteristically found in the trained individual and changes in vagal tone. The changes in tone were judged by investigating the acetylcholine (AC) concentration in the atrial tissues of the trained and untrained animals.

EXPERIMENTAL METHOD

Experiments were carried out on albino rats. Starting from the age of 3 months, 14 animals were trained to swim for 7 months. Seventeen animals of the same age and kept on the same diet were kept in the same room as the experimental animals during these periods but were not subjected to physical exertion and acted as the control. The heart rate of the rats at rest was determined from the ECG. The animals were then decapitated, the thorax opened, and the heart was removed and placed in Ringer's solution containing eserine in a concentration of $1 \cdot 10^{-5}$ g/ml. The blood was rinsed out, all remains of the vessels and fatty tissue were removed, and the atria were separated from the ventricles. AC was extracted by Rotschuh's method [8] and determined quantitatively by the biological test [3] in Khamitov's modification [1]. The morphinized and eserinized frog's lung was used as the test object. Standard solutions of AC in concentration of $2 \cdot 10^{-8}$ and $2 \cdot 10^{-7}$ g/ml and extracts obtained from two trained and two untrained rats were tested on each preparation. Next, a calibration graph was plotted for each preparation, with the AC concentration plotted along the abscissa in μg/g 0.1 μg = 1 cm) and the upward displacement of the lever in millimeters along the ordinate (scale 1 :10). Considering that the tissue was diluted 40 times during extraction, the first of the standard solutions ($2 \cdot 10^{-8}$) corresponded to an AC concentration of 0.8 μg/gtissue and the second to an AC concentration of 8.0 μg/g. The AC concentration in the test tissue was found from the upward displacement of the lever in response to injections of the extract on the graph.

EXPERIMENTAL RESULTS

The heart rate of the untrained rats averaged 444.0 ± 16.7 and in the trained rats 338.0 ± 10.4 beats/min ($P < 0.001$). However, no significant change took place in the AC concentration in the atrial tissues under these conditions: in the untrained rats its mean value was 5.13 ± 0.63 μg/g, and in the trained rats 4.25 ± 0.77 μg/g ($P > 0.5$).

To assess the functional significance of these results, the view held by most investigators was adopted, namely that under the influence of parasympathetic influences AC is liberated from nerve endings in the effector organ, including the heart, and that the quantity of AC thus liberated depends on the strength and frequency of the flow of impulses [9], i.e., it is proportional to the intensity of the parasympathetic nervous influences on the heart. It must be pointed out, admittedly, that there is considerable evidence [2, 6, 7, 10] to show that the presence of AC in the heart is connected not only with its liberation under the

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influence of nervous impulses travelling along the vagus nerves, but also to some extent to its direct synthesis in the myocardium. However, despite this fact, the AC concentration in particular structures of the heart evidently reflects the level of vagal cholinergic influences on the functions performed by these structures of the heart for the "nonnervous" AC fraction, as some investigators believe [11, 12], is a factor which is additional to the activity and effect of the AC liberated by the vagus nerve endings. In particular, the AC concentration in the atrial tissues can be used as a criterion to evaluate the level of vagal cholinergic influences on the heart rate [4, 5].

If these views are accepted, the results obtained in the investigation described above indicate that the bradycardia characteristically found in the trained organism is not due to an increase in vagal tone.

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