DELIBERATE CARDIAC ARREST AND ITS POSSIBLE MECHANISM

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Despite the firmly established involuntary character of autonomic control of the cardiac rhythm, reports continue to appear of attempts to overcome this regulatory barrier experimentally [1, 3, 4-9]. However, changes in heart rate observed under these circumstances are very limited in character and are produced as a rule by a change in external respiration and(or) skeletal muscle tone, and also by means of autogenous emotional states [2]. Yet the arsenal of usable methods may evidently be much wider, as the case described below shows.

Subject P., a man aged 33 years, a carpenter at one of the research institutes, in the course of private conversation described his ability to stop his heart from beating deliberately. When invited to the Department of Normal Physiology of Kuban Medical Institute, he actually stopped his heart beating for 7 sec, and the phenomenon was recorded on an ELKAR-4 electrocardiograph (Fig. 1). The cardiac arrest thus observed was accompanied by marked pallor of the skin surface and by the subject's considerable general weakness for the next 10-15 min. Unfortunately, P. was reluctant to explain the mechanism of the effect he had demonstrated, but claimed that ability to control his heart beat deliberately was inherited. He also declined further investigations, on the grounds that they would be hazardous to his health. For that reason the analysis was limited to the ECG and to visual observations on this subject. It was observed that cardiac arrest took place while he was sitting, after preliminary breath holding in the expiration phase. As the heart beat slowed, and cardiac arrest supervened, sudden periodic shifts of the isoelectric line were observed in one channel of the ECG (Einthoven's second lead), the only explanation of which can be only latent voluntary movements of the diaphragm, although the posture of the clothed subject remained outwardly unchanged. After cessation of cardiac arrest, for some time the heart beat more slowly than initially. These findings suggest that this was a case of a vagus arrest of the heart, due to self-stimulation of abdominal receptors, i.e., to a well-developed Goltz reflex in a hitherto unknown modification. An important role in the mechanism of the reflex is evidently played by breath holding, increasing the central tone of the vagus nerve and facilitating voluntary movements of the diaphragm (one element of yoga exercises).

To test this suggestion, two of the co-authors of this communication carried out several training sessions, each lasting half an hour, under electrocardiographic control, in an attempt to reproduce the effect described above, in the course of which they periodically

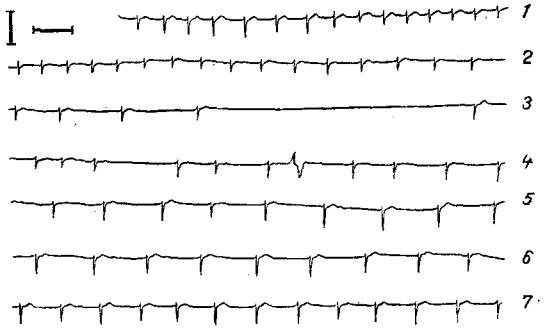

Fig. 1. ECG of subject P. in lead V_2. 1) Initial ECG; 2-5) continuous trace of slowing and arrest of the heart and restoration of the heart beat; 6, 7) ECG 1 and 3 min after cardiac arrest. Calibration: 1 mV, 1 sec.

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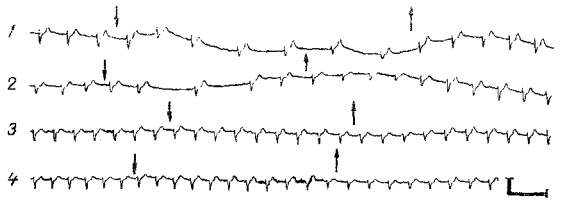

Fig. 2. Changes in cardiac rhythm as shown by ECG (lead V_2) during self-stimulation of abdominal receptors before (1) and 5 (2), 20 (3), and 40 min (4) after injection of atropine. Arrows indicate beginning and end of self-stimulation of abdominal receptors. Calibration: 1 mV, 1 sec.

compressed and distended their abdomen with the aid of the diaphragm and abdominal muscles. As a result of these exercises, after only 1 week both volunteers were able to reduce their heart rate appreciably without any particular effect (Fig. 2). Injections of atropine into the volunteers in a dose of 1 mg subcutaneously completely prevented this "voluntary" control of their heart beat. The facts described above thus confirmed once again the validity of the classical view that purely voluntary control of the cardiac rhythm is impossible, but at the same time, they showed how it can be successfully imitated over a wide range of frequencies. This procedure can evidently be used in clinical practice to explain and correct types of cardiac arhythmia, and also to produce conditioned-reflex inhibition of cardiac activity.

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TEMPERATURE SENSITIVITY OF AFFERENT RECEPTORS OF THE ISOLATED RABBIT BRAIN

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Abundant experimental evidence has been obtained by the study of physiological properties of cardiac receptors connected by afferent fibers with the CNS. Most of these receptors are atrial and ventricular mechanoreceptors, but pericardial and epicardial receptors also have been found. The adequate stimulation for activation of the afferent receptors of the heart is stretching of its chambers. Epicardial receptors also are stimulated by some chemicals (acetylcholine, veratrine, etc.) and they can respond to metabolic changes in the body [6, 8]. Previously, in experiments on cats in situ, the authors found that afferent receptor zones reponding to changes in temperature of the inflowing blood can exist in principle in the heart [1, 2]. It was shown that besides the well-known myogenic responses, a rise (or

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