PREVENTION OF STRESS-INDUCED LOWERING OF THE FIBRILLATION THRESHOLD OF THE HEART BY TRANSAURICULAR ELECTROACUPUNCTURE

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During adaptation of animals to repeated short-term stress metabolites of stress-limiting systems, namely opioid peptides [9], serotonin, dopamine [1], and GABA [8], regularly accumulate in brain structures. Meanwhile this adaptation limits the stress reaction to strong stimuli [5] and prevents lowering of the fibrillation threshold [6], distribution of the cardiac rhythm [7], and mobilization of catecholamine reserves in the myocardium [5] during severe stress and acute ischemia. Investigations in recent years have shown that a similar increase in concentration of these metabolites of the stress-limiting systems in the brain occurs with acupuncture [2, 10, 11]. We have postulated that by the use of acupuncture it would be possible to prevent the lowering of the fibrillation threshold of the heart and of the catecholamine reserves, and also, perhaps, the disturbance of the cardiac rhythm during exposure to severe stress without inducing a stress reaction.

The aim of this investigation was to assess the effect of a preliminary course of acupuncture, in the form of transauricular electrical stimulation, on the lowering of the electrical fibrillation threshold of the heart and of the catecholamine reserves of the myocardium, usually observed in immobilization stress.

EXPERIMENTAL METHOD

Male Wister rats weighing 200-250 g were used. There were four series of experiments: I) control; II) animals receiving a course of transauricular electrical stimulation (TAS); III) animals subjected to immobilization stress; IV) animals subjected to stress after a preliminary course of acupuncture. Immobilization stress was induced by fixing the animals, in recumbency in the supine position, by the limbs for 12 h. For the course of transauricular acupuncture needles were inserted into both conchae auriculae of the rat close to the external auditory meatus, after which electrical stimulation was carried out by means of a "Lasper CS-504" electrostimulator, generating pointed pulses of between 0.2 and 0.8 mA, 1.5 sec in duration, with a frequency of 3 Hz, for 15 min. These sessions were given daily for 2.5 weeks. This "dose" of TAS was close to that used for acupuncture therapy in man [4]. To determine the electrical fibrillation threshold of the heart (EFTH), thoracotomy was performed under pentobarbital anesthesia and, by means of a SEN-3201 stimulator ("Nihon Kohden," Japan), triggered by the R wave of the ECG, the heart was stimulated by premature single square pulses, 10 msec in duration, through a coaxial electrode inserted into the myocardium at the apex of the left ventricle. Scanning the S-T interval by pulses of three times the threshold value, the beginning of the relative refractory period, i.e., the moment of time when a single response to stimulation appeared, was found. The time from the R wave to this point gave the effective refractory period. The ventricular fibrillation threshold was determined as the weakest current (in mA) at which fibrillation occurred. The catecholamine level in the adrenergic fibers of the right ventricular myocardium was determined by the Falck–Owman method in Krokhina's modification [3], with measurement of the intensity of luminescence of catecholamine with the FMEL-1A instrument. The result was expressed in conventional units.

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TABLE 1. Prevention of Stress-Induced Fall of Fibrillation Threshold and of Catecholamine Concentration in Myocardium by a Course of Transauricular Electroacupuncture

Series of experiments	Threshold of electrical fibrillation, mA	Intensity of luminescence of adrenergic terminals of right ventricular myocardium, conventional units
I	8,5±0,5 (10)	17,6±1,57 (3)
II	9,5±1,5 (9)	—
III	3,3±1,0 (10)	7,0±0,87 (5)
IV	6,8±0,8 (8)	13,7±1,0 (5)

Legend. Number of animals shown in parentheses. $p_{I-III} < 0.1$, $p_{III-IV} < 0.01$.

EXPERIMENTAL RESULTS

The version of transauricular electroacupuncture which we used never elicited a pain reaction in the animals. Starting from the 2nd-3rd procedure, by the 5th-7th minute of stimulation the animals gradually fell into a semidrowsy state of immobility, in which they remained for a few minutes after removal of the needles. During the sessions the animals could be held quietly in the hand or placed on their side or back, i.e., they exhibited neither an avoidance reaction or any form of aggressiveness. Data on the effect of a course of these procedures show (Table 1) that the electroacupuncture course itself caused not only a tendency for the fibrillation threshold to rise, but at the same time it significantly prevented the fall of EFTH observed during stress. As a result the threshold in animals exposed to stress after a course of acupuncture was twice as high as in the animals exposed to stress without preliminary protection. Evaluation of the state of the adrenergic innervation of the myocardium showed that immobilization stress led to a fall in the intensity of luminescence of the adrenergic terminals by 2.5 times. In animals subjected to immobilization stress after a course of acupuncture this parameter was lower than in the control rats but twice as high as in animals subjected to stress without preliminary protection. A preliminary course of acupuncture thus largely prevents phenomena typical of stress, such as lowering of EFTH and of the noradrenalin concentration in the adrenergic terminals of the myocardium.

In principle, acupuncture can increase the concentration of GABA [2], opioid peptides [11], and serotonin [10, 11] in brain structures. Consequently, there is every reason to suppose that a course of it, like adaptation to short-term stress, may lead to activation of GABA-ergic, opioidergic, serotoninergic, and other inhibitory, stress-limiting systems of the brain. In turn, these systems limit excitation of adrenergic centers during exposure to stress, thereby reducing the stress reaction and the disturbances of electrical stability of the heart typical of it. Meanwhile, a course of acupuncture can give rise to antistressor and antiarrhythmic effects without reproducing the stress reaction itself again, evidently on account of primary reflex activation of the inhibitory structures of the brain. This hypothesis requires further testing.

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