FUNCTIONAL HETEROGENEITY OF THE ATRIAL MYOCARDIUM OF PATIENTS WITH CONGENITAL AND ACQUIRED HEART DEFECTS

S. A. Afanas'ev, B. I. Laptev, V. V. Pekarskii, V. I. Varnavskikh, and E. V. Varnavskikh

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Heart defects distort the physiological conditions under which the myocardium works. This is due to an increase in resistance to cardiac ejection or to the return of blood to the heart. The increase in load as the defect develops leads to hypertrophy of the heart, and this is reflected in the process of electromechanical coupling of the heart muscle [2].

The aim of this investigation was to study heterogeneity of the contractile activity of biopsy material obtained from the atrial myocardium of patients with heart defects in response to a change in voltage of electrical stimulating pulses.

EXPERIMENTAL METHOD

The investigation was conducted on trabeculae of the auricle of the atrium from patients undergoing surgical treatment for a congenital defect. The muscles contracted under isometric conditions at a temperature of 30°C, in response to stimulation with a frequency of 0.5 Hz. The preparation was perfused with Krebs–Henseleit solution. After adaptation of the muscles, brought about by stimulation at a voltage 20% above the threshold value, the voltage of the stimulating pulse was steadily increased. Values of the stimulating pulse (SP) and the force of contraction and its first derivative were recorded. The maximal developed tension and the maximal rate of rise and fall of tension were calculated. Changes in the parameters are given as a percentage of their values corresponding to the threshold level of SP.

EXPERIMENTAL RESULTS

An increase in the voltage of SP above the threshold value (Fig. 1) led to a change in shape of the contraction curve. When the voltage of SP was significantly higher than the threshold level, contraction was reflected by a monophasic curve with an amplitude higher than that measured initially. This relationship was observed in all the defects studied.

However, sensitivity to a change in voltage of SP and the response of parameters of contractility of the myocardial preparations from patients with congenital defects differed from those associated with acquired defects (Fig. 2). The range of changes in SP in a case of congenital defects, leading to additional activation of the contractile function of the heart muscle, was significantly ($p < 0.05$) wider than in the case of acquired defects. Meanwhile, the response of the parameters of contractility to these changes in SP were stronger in the case of acquired defects

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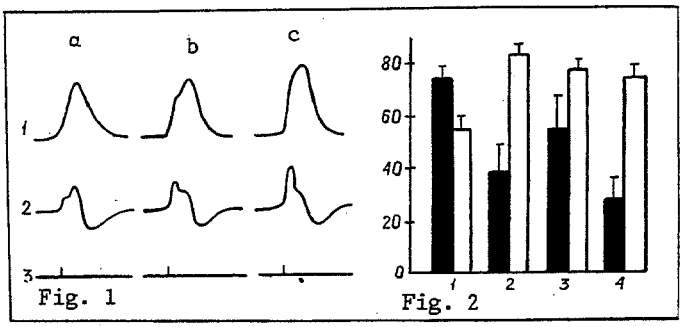

Fig. 1 Fig. 2

Fig. 1. Change in shape of contraction curve (1) and its first derivative (2) in response to increase in voltage of stimulating pulse (SP) (3). a) Threshold value of SP, b and c) SP 50% or more above threshold values.

Fig. 2. Range of changes in voltage of SP (1) and corresponding increase in maximal developed tension (2) and its maximal rate of rise (3) and fall (4). Ordinate, changes in parameters in percent of their threshold value; black columns indicate congenital, unshaded columns – acquired defects.

($p < 0.05$). In that case, changes in the maximal developed tension and the maximal rate of rise and fall of tension were virtually equal. In congenital defects, there was a significantly ($p < 0.05$) greater increase in the rate of rise of tension.

The manifestations of heterogeneity in contractile activity of myocardial biopsy specimens from the atria of patients undergoing surgical treatment for a congenital or acquired heart defect thus differ considerably. Functional heterogeneity of the myocardium may be based both on intercellular uncoupling of the cardiomyocytes [1] and intracellular changes. The latter may be associated with biochemical changes [4] or with a disturbance of calcium ionic homeostasis [3, 5-7]. Predominance of either of these changes may evidently give rise to the differences we discovered. Explanation of the nature of the functional heterogeneity of the human myocardium would enable the contractility of the pathologically changed heart muscle to be increased. This is particularly important when regression of disturbances of contractility does not begin after surgical correction of the defect.

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