# SYMPATHETIC CONTROL OF SPONTANEOUS DEFIBRILLATION OF THE HEART

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The results of research into the role of the sympathetic nervous system in ventricular fibrillation indicate that enhancement of sympathetic action on the heart provokes fibrillation [2, 5]. However, there is information in the literature that the sympathetic nervous system has a marked protective action, and that enhancement of sympathetic action on the heart may be one way of preventing cardiac arrhythmias [3].

The aim of this investigation was to assess the role of the sympathetic nervous system in the ability of the heart to undergo spontaneous defibrillation.

## EXPERIMENTAL METHOD

Altogether 28 experiments were carried out on mature guinea pigs weighing 300-350 g and 26 experiments on mature rats weighing 250-280 g, under hexobarbital anesthesia (100 mg/kg body weight) under open chest conditions. The AID-2 apparatus was used for artificial respiration. Electrical activity of cardiomyocytes was recorded on the epicardial surface of the left ventricle by means of "floating microelectrodes" [1], and synchronized with the electrocardiogram in standard lead II, on an MFJ-1 motion picture camera with MG-23 screen (Hungary). Fibrillation was induced in guinea pigs by electrical stimulation of the heart (2 sec, 50 Hz, 5 V) through a type TE1/1 coaxial electrode, by means of an ST-21 stimulator, and in rats by intraperitoneal injection of an aqueous solution of aconitine in a dose of 150 $\mu$g/kg body weight. To weaken sympathetic control of the heart, every 18-19 h for 4 days the guinea pigs received an intraperitoneal injection of Rausedil (reserpine) in a dose of 4 mg/kg body weight, and to strengthen sympathetic control a mixture of adrenalin and noradrenalin, 0.15 ml of a 0.1% solution of each, was injected intraperitoneally. In some experiments atropine also was injected in a dose of 1 ml of 0.1% solution per animal. In the experiments on rats the ventromedial (VMN) and lateral nucleus (LN) of the hypothalamus were stimulated by bipolar electrodes in glass insulation and 0.1 mm in diameter with bursts of square pulses (100-500 msec, 40 Hz, pulse duration 2 msec, duration of stimulation 2 h, stimulating current 80 $\mu$A). The ÉSL-2 stimulator with a special switching system at the output was used. The position of the electrode tips was verified morphologically. The results were subjected to statistical analysis by "Statgraphics" package on an IBM PC/XT computer.

## EXPERIMENTAL RESULTS

In the control experiments the resting potential (RP) of cells of the contractile ventricular myocardium was $-91 \pm 2.1$ mV, the action potential (AP) $102.8 \pm 1.2$ mV, and the duration of AP at the 80% repolarization level $145 \pm 15$ msec, at a heart rate of $253 \pm 12$ beats/min. Injection of Rausedil led to a decrease in RP to $-81.3 \pm 3.5$ mV ($p > 0.05$) and AP to $95.1 \pm 1.9$ mV ($p < 0.05$) and to an increase in the duration of AP to $195 \pm 12$ msec ($p < 0.05$). Reducing the parameters of the transmembrane potentials of the cardiomyocytes in response to a fall in the catecholamine levels due to adrenalectomy also was demonstrated previously [4].

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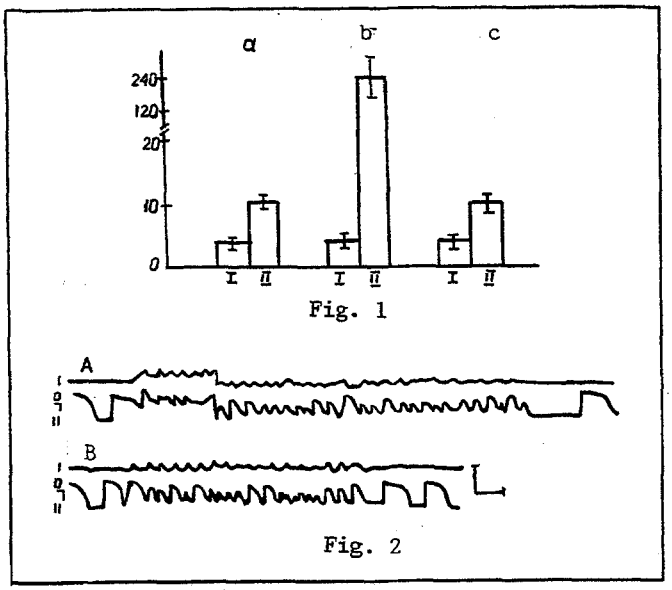


Fig. 1




Fig. 2

Fig. 1. Duration of period of SRVF in response to electrical stimulation of guinea pig heart: a) control, b) after injection of Rausedil, c) after injection of catecholamines, I, II) Nos. of stimulation.

Fig. 2. Transmembrane potentials of cells of contractile left ventricular myocardium of a guinea pig during defibrillation: A) control, B) after injection of Rausedil (3rd stimulation). Calibration 100 mV, 200 msec. I) ECG, II) AP.

After injection of catecholamines no significant changes could be detected in the parameters of the transmembrane potentials, except a decrease in the duration of AP as a result of an increase in heart rate.

After electrical stimulation of the guinea pig heart, spontaneous reversible ventricular fibrillation (SRVF) appeared in all cases without exception, and varied in duration (Fig. 1a). The first paroxysm of SRVF rarely exceeded 4-12 sec. Electrical stimulation of the heart of animals receiving Rausedil led to a sharp increase in duration of the first paroxysm of SRVF to 240-300 sec, and during repeated electrical stimulation the duration of fibrillation was a few seconds (Fig. 1B). Electrical activity of the cardiomyocytes during SRVF under these circumstances was the same as in the control experiments (Fig. 2a, b). Electrical stimulation of the heart of animals receiving catecholamines led to the development of paroxysms of SRVF of about the same duration as in the control experiments (Fig. 1c). Injection of atropine into animals of both groups shortened the duration of SRVF to a few seconds. Thus the reduced ability of the heart to undergo spontaneous defibrillation during weakening of sympathetic control is a secondary effect, due to a relative increase in the level of parasympathetic control of cardiac activity.

Injection of aconitine into rats led to phase disturbances of the cardiac rhythm: bigeminy, tachysystole, and spontaneous irreversible fibrillation of the heart. Activation of the sympathicoadrenal system by stimulation of the hypothalamic nuclei significantly delayed the development of the early stages of arrhythmia ($p < 0.05$). During stimulation of LN the latent period of onset of spontaneous irreversible ventricular fibrillation was increased, whereas during stimulation of VMN paroxysms of SRVF appeared instead of spontaneously irreversible ventricular fibrillation. The results suggest activation of the sympathetic system prevents the creation of spontaneously irreversible mode of ventricular fibrillation.

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