EFFECT OF ELECTRICAL STIMULATION OF THE DORSOLATERAL FUNICULUS ON CARDIAC RHYTHM CHANGES IN ACUTE MYOCARDIAL ISCHEMIA

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Previous investigations have shown that one of the concomitant pathogenetic mechanisms of development of cardiac arrhythmia in acute myocardial ischemia is hyperactivation of sympathetic preganglionic neurons (SPN) in the lateral horn of the superior thoracic segments [2], with the formation of a generator of pathologically enhanced excitation (GPEE) in them. Activity of SPN is known to depend on the functional state of suprasegmental structures which exercise sympathoinhibitory control over their work [3, 8, 9].

In this connection it is interesting to study changes taking place in the cardiac rhythm as a result of electrical stimulation of the descending inhibitory pathways which run in the composition of the dorsolateral funiculi (DLF) [6-9], and the investigation described below as undertaken for this purpose.

EXPERIMENTAL METHOD

Experiments were carried out on noninbred male albino rats weighing 180-200 g (21 animals). In the experiments of series I (control) a high ligation of the left coronary artery was performed and the ECG recorded for 1 h. In the experiments of series II electrical stimulation of DLF was carried out at the level of T_2-T_4 for 10 min immediately after ligation of the coronary artery. In series III electrical stimulation of DLF was carried out after a disturbance of the rhythm of the ischemic heart had already developed. For the experiments the animals were spinalized, immobilized with succinylcholine (0.2 mg/kg), and artificially ventilated. The spinal cord was divided 24 h before the experiment at level C_7. Preparations for the experiment (tracheotomy, exposure and division of the spinal cord) were made under ether anesthesia. For electrical stimuation of DLF an ESL-2 electronic stimulator and bipolar glass electrodes filled with 4M NaCl solution were used: The parameters of the stimulating current in the experiments of series II were: 200 pulses/sec, 0.6 msec, 300 μA, and in series III: 200 pulses/sec, 0.6 msec, 3 mA.

EXPERIMENTAL RESULTS

In the control, high ligation of the left coronary artery led to the development of cardiac arrhythmias, which differed in severity and the character of their course, with a latent period of onset of 37.1 ± 10.0 sec after ligation of the vessel. The duration of the arrhythmia was 5.59 ± 1.10 min (Table 1). Types of arrythmia such as ventricular paroxysmal tachycardia (135.7 ± 3.9 sec), polytopic single and grouped extrasystoles (37.7 ± 13.0 sec), nodal rhythm (31.2 ± 14.0) sec), ventricular fibrillation and flutter, etc.

Electrical stimulation of DLF for 10 min after ligation of the coronary artery sharply increased the latent period of appearance of arrhythmia to 11.0 ± 2.5 min and shortened the time of the recorded arrhythmias to 50.0 ± 3.5 sec compared with the control (Table 1). No arrhythmias such as grouped extrasystoles, nodal rhythm, or ventricular flutter and fibrillation were observed, and the duration of the single extrasystoles was reduced to 7.2 ± 1.7 sec and of paroxysmal ventricular tachycardia to 6.8 ± 3.0 sec.

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TABLE 1. Character of Cardiac Arrhythmias Developing in Spinal Animals after High Ligation of Left Coronary Artery and Electrical Stimulation of DLF for 10 min, Starting Immediately after Ligation of Artery (M ± m)

Experimental conditions	Parameter studied	Type of arrhythmia							LP	T
		SVES	GVES	NR	ALL	PVT	VFL	VFI		
High ligation of left coronary artery (control)	Number of cases of disturbances	37,7±13,4	34,5±11,1	1,1±0,4	0,7±0,4	6,4±1,5	0,5±0,2	0,5—0,2	37,1±10,7sec	5,59±1,10 min
	Time during which disturbances were observed, sec	—	15,5±9,0	31,2±14,6	13,1±8,9	135,7±33,9	11,0±5,9	6,8±2,9		
Electrical stimulation (10 min) of DLF immediately after ligation of artery	Number of cases of disturbances	7,2±1,7*	—	—	1,3±0,7	1,4±0,6*	—	—	11,0±2,5 min*	50,0±3,5sec*
	Time during which disturbances were observed, sec	—	—	—	8,2±5,8	6,8±3,0*	—	—		

Legend. SVES) Single ventricular extrasystoles, GVES) groups ventricular extrasystoles, NR) nodal rhythm, ALL) allorhythmia, PVT) paroxysmal ventricular flutter, VFI) ventricular fibrillation, LP) latent period of onset of arrhythmias from time of ligation of coronary artery, T) time during which arrhythmias were observed. *$P < 0.02$ compared with control.

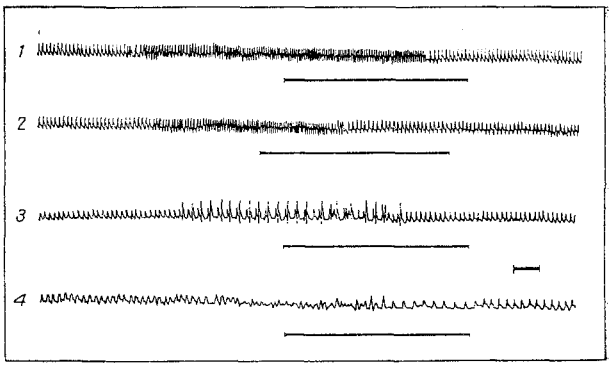

Fig. 1. Effect of electrical stimulation of DLF on cardiac arrhythmias accompanying acute myocardial ischemia. 1) Electrical stimulation of DLF during established arrhythmia; 2) electrical stimulation of DLF in same experiment after recurrence of arrhythmia; 3) electrical stimulation of DLF during development of allorhythmia; 4) electrical stimulation of DLF in ventricular fibrillation. Time of electrical stimulation indicated by horizontal line. Calibration 1 sec.

Electrical stimulation of DLF after arrhythmias had already developed led to normalization of the rhythm in all experiments 3-4 sec after it began (Fig. 1, 1). Repeated electrical stimulation under these conditions shortened the period of termination of the attack of arrhythmia by 1-2 sec (Fig. 1, 2). The same effect was observed when electrical stimulation of DLF was applied after the development of allorhythmia and ventricular fibrillation (Fig. 1, 3, 4).

Strengthening of descending sympathoinhibitory influences thus prevented the development of severe forms of cardiac arrhythmias when electrical stimulation of DLF was applied immediately after ligation of the coronary artery. In the case of established arrhythmias, transient electrical stimulation of DLF led to rapid normalization of the cardiac rhythm.

The results confirm the previous hypothesis that the development of a cardiac arrhythmia is the result of formation of pathological systems induced by the formation of hyperactive determinant structure in various parts of the central nervous system [2, 4, 5], whose activity can be suppressed by strengthening inhibitory control of their activity through activation of functionally antagonistic systems [1].

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