ROLE OF MYELINATED AND NONMYELINATED FIBERS OF THE VAGUS NERVES IN THE DEVELOPMENT OF ISCHEMIC VENTRICULAR FIBRILLATION

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The role of the vagus nerves in the development of ischemic cardiac arrhythmias still remains unexplained. A decrease in the frequency of development of ischemic cardiac arrhythmias following both stimulation of the vagus nerves and vagotomy has been reported [7, 9, 10]. Other workers have noted the opposite effect of vagotomy [2, 15]. The flow of afferent information traveling along fibers of the vagus nerves into the CNS is known to change during the development of an ischemic process in the myocardium [1, 12]. This effect is one of the leading factors in the restructuring of neuronal activity of the bulbar cardiovascular center in myocardial ischemia, complicated or uncomplicated by fibrillation of the heart [4].

The aim of this investigation was to study the frequency of development of ischemic myocardial fibrillation during step by step blocking of the conduction of excitation along different types of vagus nerve fibers.

EXPERIMENTAL METHOD

Experiments were carried out on 46 male and female cats weighing 3-4 kg under pentobarbital anesthesia (40 mg/kg body weight, intraperitoneally). Myocardial ischemia was induced in naturally breathing animals by occluding the circumflex branch of the left coronary artery close to its origin from the main trunk for up to 15 min. For this purpose thoracotomy was performed, the pericardium was opened, and after a loop had been passed beneath the coronary vessel, airtight closure of the chest was achieved by suturing the pericardium to the thoracic muscles. The character of development of disturbances of the cardiac rhythm was analyzed throughout the period of myocardial ischemia and during 15 min of subsequent reperfusion. Analysis of the developing arrhythmias considered the following idioventricular arrhythmias: single and grouped ventricular extrasystoles, allorhythmias, ventricular tachycardia, and ventricular fibrillation. The blood pressure (BP) was recorded in the femoral artery by means of an EMT-35 manometric transducer. The cervical portions of the vagus nerves below the ganglia nodosa were placed on thermodes, with a thermistor mounted in them, and carefully insulated from surrounding tissues. To block the flow of afferent information along thick myelinated fibers, the vagus nerves were cooled to 6-7°C [8]. Conduction along nonmyelinated fibers also was blocked by cooling the nerves to 0°C. The temperature of the cold block, the pressure in the femoral artery, and the ECG in standard lead II were recorded on a "Biocomb-8" recorder (Hungary). In all experiments the statistical significance of the responses was assessed by Student's t test, the chi-square test, and the signs test.

EXPERIMENTAL RESULTS

Within the scope of the present investigation the development of ischemic cardiac arrhythmias was first analyzed in experiments (17) of the control (I) series. In animals of this group, occlusion of the coronary vessel led to the following disturbances of the cardiac rhythm: single extrasystoles were observed in 82.3% of cases, grouped ventricular extrasystoles in 35.3%,

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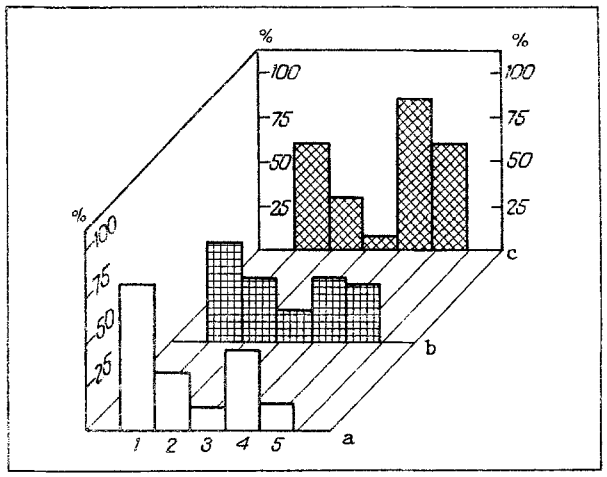

Fig. 1. Frequency of development of ischemic cardiac arrhythmias in control (a) and during blocking of myelinated (b) and nonmyelinated (c) vagus nerve fibers. Abscissa: 1) single, 2) grouped ventricular asystoles, 3) allorhythmias, 4) ventricular tachycardia, 5) fibrillation; ordinate, number of experiments in which arrhythmias were observed (in %).

allorhythmias in 11.7%, and ventricular tachycardia in 47% of cases. In 17.6% of cases myocardial ischemia was complicated by fibrillation of the heart (Fig. 1a). The development of ischemia in the myocardium led to a significant of the systolic BP toward the 30th second of occlusion ($p < 0.05$) by 15.5% of the initial level (175 ± 6.5 mm Hg). During further development of myocardial ischemia (deformation of the QRS complex of the ECG) BP was lowered by 27%.

In the experiments of series II myocardial ischemia was induced against the background of blocking of conduction in the thick myelinated fibers of the vagus nerves 5 min after their cooling to 6-7°C (16 experiments). Blocking the conduction of excitation along thick myelinated fibers of the vagus nerves led to a fall of BP ($p < 0.05$) by 5.9% of its initial level (164.3 ± 6.6 mm Hg). Other investigators also observed a similar response of BP to cooling the vagus nerves to 6°C [3, 13]. In the animals of this group no significant increase in the frequency of development of ischemic cardiac arrhythmias, including myocardial fibrillation, was observed compared with animals of the control group ($p > 0.05$). For instance, single ventricular extrasystoles were observed in 56.3% of cases, grouped extrasystoles in 37.5%, allorhythmias in 18.8%, ventricular tachycardia in 37.5%, and fibrillation of the heart in 31.3% of cases (Fig. 1b; Fig. 2). Myocardial ischemia was accompanied by lowering of BP, which was significant in the presence of changes in the QRS complex of the ECG ($p < 0.001$).

The results of this series of experiments indicate that blocking conduction in thick myelinated fibers of the vagus nerves led to elevation of BP. The development of myocardial ischemia against this background was accompanied by changes in BP similar to the experiments of the control series and by the development of cardiac arrhythmias.

In the experiments of series III the development of ischemic cardiac arrhythmias associated with the blocking of conduction of excitation along thin nonmyelinated vagus nerve fibers also was analyzed (13 experiments). BP in the animals of this group (170.7 ± 5.4 mm Hg) remained virtually unchanged during cooling of the vagus nerves to 0°C for 5 min (170.1 ± 9.1 mm Hg), in agreement with data in [10]. Occlusion of the coronary vessel was accompanied by the development of single ventricular extrasystoles in 61.5% of cases, of grouped ventricular extrasystoles in 30.8%, and of allorhythmias in 7.7% of cases. No significant differences were found in the frequency of development of these disturbances of the cardiac rhythm compared with animals of the control group. Meanwhile there was a significant increase in the frequency of development of ventricular fibrillation compared with experiments of the control series ($p < 0.05$). A tendency also was observed for the frequency of development of ventricular tachycardia to increase ($p < 0.1$, Fig. 1c, Fig. 3). Myocardial ischemia in this series of experiments did not cause any significant changes in BP until the appearance of cardiac arrhythmias.

The results of this series demonstrate that myocardial ischemia, superposed on cooling of the vagus nerves to 0°C, while not changing BP, was accompanied by an increase in the frequency of development of ventricular fibrillation.

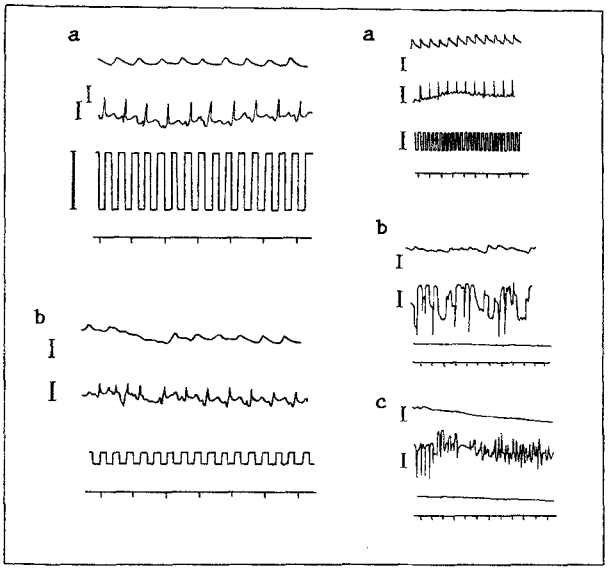

Fig. 2 Fig. 3

Fig. 2. Development of myocardial ischemia during cooling of the vagus nerves to 6°C. a) Background, b) development of ischemic arrhythmias. Legend: from top to bottom, pressure in femoral artery, ECG in standard lead II, temperature of vagus nerve, time marker (0.5 sec). Calibration: BP 50 mm Hg, ECG 1 mV, temperature 36°C.

Fig. 3. Development of myocardial ischemia during cooling of vagus nerves to 0°C. a) Background, b) development of ischemic arrhythmias, c) development of fibrillation. Legend and calibration the same as in Fig. 2.

It can thus be concluded that myocardial ischemia during blocking of thick myelinated fibers of the vagus nerves does not lead to significant changes in BP before the appearance of arrhythmias, but does cause a considerable increase in the frequency of development of severe disturbances of the cardiac rhythm, including ventricular fibrillation.

It can be tentatively suggested that thick myelinated vagus nerve fibers, carrying information mainly from the mechanoreceptors of the heart and blood vessels, have no significant influence on the development of severe cardiac arrhythmias, whereas blocking the vagus nerves and, in particular, blocking the thin nonmyelinated fibers, evidently leads to a considerable increase in the frequency of development of severe ischemic cardiac arrhythmias. This result may probably be linked both with activation of the sympathoadrenal system, which is observed in myocardial ischemia and in blocking of the vagus nerves [5, 6, 11], and also with the blocking of afferent impulsation, reaching the CNS along thin nonmyelinated fibers of the vagus nerves, of which they constitute the principal part.

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