INFLUENCE OF EXTRACARDIAC NERVES ON CARDIOVASCULAR FUNCTION IN GENERAL HYPOTHERMIA

A. I. Smirnov, E. A. Belyavskaya, and T. N. Kovaleva

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During moderate hypothermia the vagus nerve continues to exert its trophic influence on the heart.

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The influence of the vagus nerve on the heart in various functional states has been studied for many years in the authors' laboratory [2-6]. Changes in the functional state of the heart have been found to activate specific influences on the vagus nerve on activity of the heart as a heterogeneous excitable and muscular system.

As electrocardiographic data [7, 11, 12, 15-17, 20] show, changes in the cardiac function during hypothermia and an increased tendency for the ventricles to fibrillate [1, 8, 10, 14, 19] formed the basis for the study of the influence of extracardiac nerves on the heart in general hypothermia. Reports in the literature on this problem are contradictory. Some authors [8, 9] have concluded that the inhibitory influence of the vagus nerve on the heart is depressed during hypothermia, but others [13] assert that cooling increases excitability of the vagus nerve. It has also been found [18] that division of the vagus nerves in dogs under hypothermia increases the tendency for ventricular fibrillation to develop.

The object of the present investigation was to study changes in cardiovascular function under hypothermia when the influence of either the vagus or the sympathetic nerves was predominant.

EXPERIMENTAL METHOD

Acute experiments were conducted on 35 adult dogs anesthetized with morphine and urethane. General hypothermia (rectal temperature 32-24°) was induced by cooling the animal with ice. In the experiments of series I the peripheral end of one vagus nerve was subjected to prolonged stimulation, the other vagus nerve being left intact. In series II the ansa of Vieussens or the central end of the divided sciatic nerve was repeatedly stimulated. Six control experiments, without stimulation of the nerves, yielded identical results. The stimuli were square pulses (duration 0.1 msec, frequency 25 cps) from a type ÉI-1 pulse generator. The strength of the current varied from 1-5 mA. The arterial pressure and tracheal respiration were recorded on a kymograph and the ECG in standard lead 2 on a type ÉKP-S2 electrocardiograph.

EXPERIMENTAL RESULTS

Comparison of the results of the experiments of series I and II revealed certain differences in the dynamics of the changes in heart rate and mean arterial pressure developing under hypothermia (Table 1). These differences were especially marked in deep hypothermia (25-24°).

In the experiments of series I, before hypothermia and after prolonged stimulation of the peripheral end of the divided vagus nerve a slower heart rate was established than in the experiments of series II with predominance of the sympathetic influence; in the latter, under mild hypothermia (33-32°), this difference in heart rate disappeared. A further fall of temperature caused gradual slowing of the heart, and in the experiments of series I the heart rate fell more rapidly at the beginning of observation and the rhythm was more stable at a low temperature (25°), whereas in the experiments of series II the rhythm was more stable in mild hypothermia (30°) and the heart rate fell to a greater degree at 25°.

The dynamics of the changes in arterial pressure under hypothermia also differed depending on whether influences of the vagus nerve or cardiac accelerator nerve were predominant. As Table 1 shows,

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TABLE 1. Changes in Heart Rate and Mean Arterial Pressure in Dogs under General Hypothermia with Predominance of Parasympathetic or Sympathetic Influences

Series	No. of experiments	Heart rate						Mean arterial pressure (in mm)					
		before hypother.	hypothermia (in deg)					before hypother.	hypothermia (in deg)				
			32	30	28	26	25		32	30	28	26	25
I. Predominance of parasympathetic influence	15	129	135	112	102	70	64	139	123	120	112	90	68
II. Predominance of sympathetic influence	14	140	131	132	102	62	51	132	116	103	88	62	57

TABLE 2. Dynamics of Appearance of Supernumerary Waves on QRS Complex during Deepening of Hypothermia in Series I and II

Series	No. of experiments	Depth of hypothermia (in deg)										
		34	33	32	31	30	29	28	27	26	25	24
I. Predominance of parasympathetic influence	Total	12	12	12	12	12	11	11	6	5	3	3
	With supernumerary waves	0	2	4	5	5	7	7	5	4	3	3
II. Predominance of sympathetic influence	Total	11	11	10	10	10	10	10	4	4	3	—
	With supernumerary waves	5	6	7	7	7	8	9	4	4	3	—

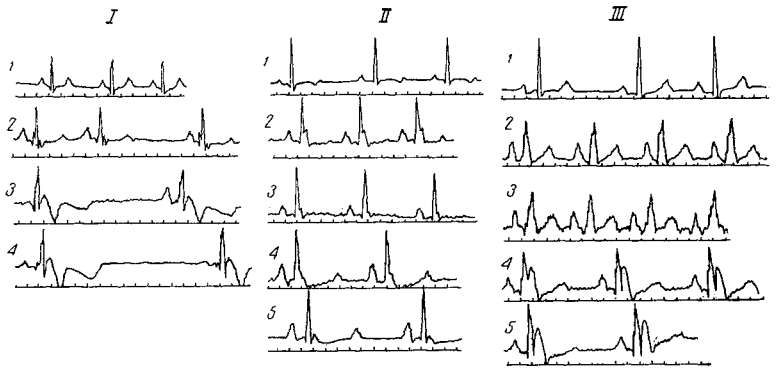

Fig. 1. Changes in ECG under hypothermia of different depths after stimulation of peripheral end of divided vagus nerve or ansa of Vieussens. I) Changes in ECG (lead 2) under different depths of hypothermia without stimulation of nerves: 1) original ECG; 2) at 30°; 3) at 28°; 4) at 26°. II) The same in experiments with stimulation of peripheral end of divided vagus nerve: 1) original ECG; 2) at 30°; 3) after stimulation of peripheral end of divided vagus nerve at 30°; 4) at 28°; 5) after stimulation of peripheral end of divided vagus nerve at 28°. III) The same after stimulation of ansa of Vieussens: 1) original ECG; 2) at 30°; 3) after stimulation of ansa of Vieussens at 30°; 4) at 28°; 5) after stimulation of ansa of Vieussens at 28°.

in series I and II the initial level of the mean arterial pressure was almost the same. As hypothermia deepened, a gradual lowering of the arterial pressure was observed in all the experiments, but the dynamics of this process varied: it was more stable in the experiments of series I than in those of series II. In individual experiments the animal was cooled to 23°; in the experiments of series I the mean arterial pressure was 64 mm, but in the experiments of series II under the same conditions the mean arterial pressure fell to 36 mm.

Analysis of the electrocardiographic data showed that as hypothermia deepened, the changes described in the literature grew in intensity: an increase of the P wave, increase in amplitude and widening of the T wave, prolongation of the P-Q, QRS, and S-T intervals, displacement of the ST segment, appearance of supernumerary waves on the ascending or descending part of the QRS complex, indicating slowing and delay of the conduction of excitation in the specific musculature of the ventricles. The first difference to appear in the dynamics of increase in severity of the ECG changes in the experiments of series I and II was the onset of supernumerary waves on the QRS complex.

It may be seen in Table 2 that in the experiments of series I supernumerary waves began to appear on the QRS complex rather later than in series II, and they were found less frequently. In experiments with repeated and prolonged stimulation of the peripheral end of the divided vagus nerve, the supernumerary waves often disappeared or became smaller. The return to a normal ECG during prolonged stimulation of the peripheral end of the divided vagus nerve also was marked by a very slight reduction in size of the P wave, correction of this wave in case of previous inversion, and narrowing of the QRS complex. However, the positive influence of vagal stimulation was manifested as a rule in moderate hypothermia; under deep hypothermia stimulation of the peripheral end of the vagus nerve had no positive effect, and in some cases it actually led to the onset of ventricular fibrillation.

Stimulation of the ansa of Vieussens or of the central end of the divided sciatic nerve in all cases caused aggravation of the functional changes in the heart, as shown by an increase in the number of supernumerary waves on the QRS complex and by an increase in their height, by enlargement of the P wave, a progressive widening of the QRS complex, and so on. The ECG changes during predominance of parasympathetic or sympathetic influences against the background of hypothermia are illustrated in Fig. 1.

Under moderate hypothermia the vagus nerve thus retains its positive trophic influence both on the functional state of the heart (helping to restore the normal ECG) and on stability of the arterial pressure. Only in deep hypothermia has stimulation of the peripheral end of the divided vagus nerve a negative influence on cardiac activity. Stimulation of the sympathetic nerves under general hypothermia intensifies functional disturbances of the cardiovascular system caused by hypothermia, as is shown by instability of the mean arterial pressure and by worsening of the ECG findings.

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