CONTROLLED SINUS ARRHYTHMIA DURING BURST STIMULATION OF THE VAGUS NERVE

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KEY WORDS: vagus nerve; burst stimulation; cardiac arrhythmia.

Neurogenic disorders of the cardiac thythm are widely known in experimental and clinical practice [1-3, 5, 6, 9, 11], but the concrete mechanisms of their development have not yet been adequately explained.

In view of the close connection between these disturbances and parasympathetic nervous influences [2, 3, 6, 11], the writers have undertaken an analysis of sinus arrhythmia observed during burst stimulation of the vagus nerve.

EXPERIMENTAL METHOD

The test objects were 30 cats and 13 rats, anesthetized by intraperitoneal injection of chloralose and pentobarbital (75 and 15 mg/kg body weight respectively) and artificially ventilated. After division of both vagus nerves the right nerve was stimulated with bursts of 3 or 4 pulses from an ÉSU-2 electrostimulator with a following frequency of 40 Hz in the burst, each stimulus being 2 msec in duration and 5-6 times the threshold strength. The ECG or electrogram of the right atrium and marker of nerve stimulation were recorded on an ÉLKAR-4 electrocardiograph.

EXPERIMENTAL RESULTS

The initial heart rate was 173 ± 6 min^{-1} (2.88 ± 0.10 Hz) in cats and 326 ± 11 min^{-1} (5.43 ± 0.18 Hz) in rats (M ± m). A characteristic feature of the chronotropic effect observed during burst stimulation of the vagus nerve was stable synchronization of cardiac contractions with the following frequency of the stimulation bursts (Fig. 1b, c), observed in cats over the range from 1.90 ± 0.05 Hz (3 pulses in the burst) and from 2.43 ± 0.15 to 1.90 ± 0.18 Hz respectively in rats (4 pulses in the burst). An increase in the number of pulses in the burst widened and shifted the range of synchronization downward, whereas a decrease in the number of pulses or in their amplitude, conversely led to narrowing and an upward shift of the range along the frequency scale. However, as soon as the frequency of stimulation went outside the upper or lower limits of this range, synchronization of the rhythms disappeared and sinus arrhythmia, which was definitely periodic in character, appeared (Figs. 1 and 2). In this case the period of repetition of the arrhythmia was inversely proportional to the difference between the following frequency of the bursts and the nearest boundary of the synchronization range. In the case of the lower boundary, if the difference in frequency was sufficiently great, the arrhythmia resembled trigeminy, or even bigeminy in type (Fig. 2).

It follows from Fig. 1 that a change in heart rate within the synchronization range, and also a change in the duration of the cardiac cycle during sinus arrhythmia were accompanied invariably by a change in the interval between the P wave of the ECG and the stimulation artefact (S). Plotting a graph of dependence of the P-P interval on the P-S or S-P interval (Fig. 3) revealed a periodic change in sensitivity of the cardiac pacemaker to the vagal stimulus, which was observed during each cardiac cycle. The picture thus revealed was found in all experiments without exception, and only the degree of the effect varied.

A similar dependence of function also was obtained during application of single stimuli to the vagus nerve in different phases of the cardiac cycle [4, 7, 10], from which it follows

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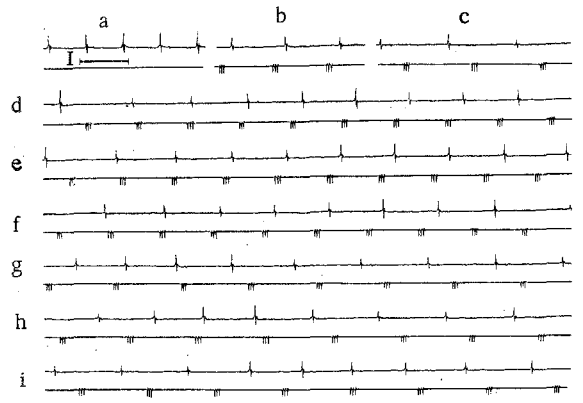

Fig. 1

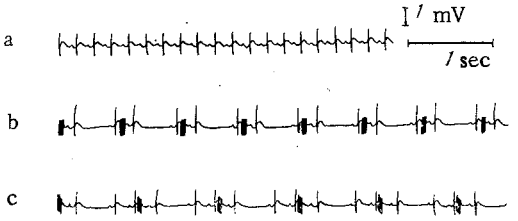

Fig. 2

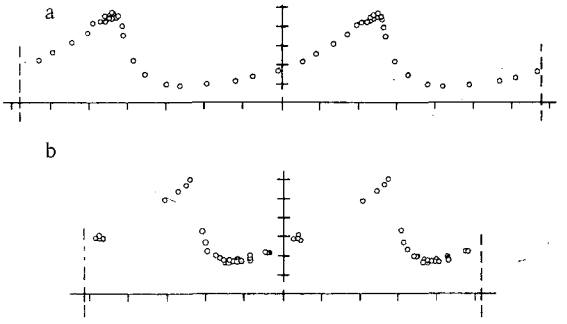

Fig. 3

Fig. 1. Sinus arrhythmia during vagus nerve stimulation in cat. Top curve of each trace is electrogram of right atrium, bottom curve marker of stimulation. a) Initial electrogram; b, c) upper and lower limits of synchronization of rhythms respectively; d, f) continuous recording of arrhythmia beyond limit of synchronization range; g-i) the same, beyond lower limit. Calibration: 5 mV.; 0.5 sec.

Fig. 2. Sinus arrhythmia during vagus nerve stimulation in rat. a) Initial ECG (lead II), b, c) different types of arrhythmias. Atypical complexes against background of ECG are artefacts of nerve stimulation.

Fig. 3. Dependence of P-P interval on atrial electrogram (in msec) on duration of P-S and S-P intervals (in msec). Vertical lines show duration of cardiac cycle bounded by lower (a) and upper (b) limits of synchronization range. a) Analysis of fragments g-i in Fig. 1; b) the same, but of fragments d-f.

that controlled reduction of the heart rate is possible only as a result of fine coordination of the power and time of arrival of the burst of pulses at the heart. Considering evidence of a cardiac rhythm in efferent impulsation in the vagus nerve [9, 12], it is logical to suppose that sinus arrhythmia of parasympathetic origin is the result of discoordination of the cardiac pacemaker and the pacemaking mechanism in the cardiovascular center. However, since a strict rhythm is not necessarily exhibited in spontaneous arrhythmia, it may be postulated that the central rhythm of excitation, unlike experimental conditions, is not strictly determined in character but is exposed to constant correction under the influence of changing efferent impulsation. In turn, the effectiveness of this correction is a guarantee of the appearance or abolition of sinus arrhythmia.

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EFFECT OF HYPOKINESIA AND MUSCLE TRAINING ON MYOCARDIAL CATECHOLAMINE LEVELS DURING POSTNATAL DEVELOPMENT IN RATS

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The study of myocardial catecholamine levels is of great importance for an understanding of the mechanisms of regulation of cardiac activity, especially under extremal conditions [3, 11-13]. An increase in the specific noradrenalin content in the myocardium, i.e., expressed as a ratio of weight of the heart, has been observed [1] during postnatal development under the influence of muscle training, whereas the adrenalin concentration remained unchanged. Data on the effect of hypokinesia on catecholamine levels in the myocardium of the developing animal could not be found in the literature.

The aim of this investigation was to determine the effect of hypokinesia and muscle training on the catecholamine concentration in the myocardium of rats during postnatal ontogeny.

EXPERIMENTAL METHOD

Experiments were carried out on noninbred albino rats. Rats aged 3 weeks were divided into three groups. The animals of group 1 were kept under conditions of restricted movement (according to the writer's own program) in individual restraining cages specially made for this purpose. Animals of group 2 were kept for the same period in ordinary cages, six to eight rats in each cage (control). Rats of group 3 were subjected to an increasing muscular load due to swimming six times a week for 50 days [1]. Some animals were decapitated 24 h after the end of the last period of training and of hypokinesis to determine catecholamines in their myocardium at rest. The remaining animals were compelled to swim carrying a load equal to 5% of body weight, until exhausted. The maximal duration of swimming was indicated by the time when the rat could no longer rise to the surface of the water to breathe. Animals which had reached this state were quickly removed from the water and killed. After decapitation, thoracotomy was performed and the beating heart removed. By immersion several times in distilled water blood was removed from the heart, which was then freed from the remains of the great vessels. Before weighing, the heart was dried with filter paper. The adrenalin and noradrenalin concentrations in the myocardium were determined by a fluorometric method and expressed in μg/g [6]. Catecholamines were oxidized with aluminum oxide by Brockman's method.

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