PATHOLOGICAL PHYSIOLOGY AND GENERAL PATHOLOGY

THE EFFECT OF PROLONGED STIMULATION OF THE PERIPHERAL PORTION OF THE VAGUS NERVE ON THE HEART IN ASPHYXIA

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

Physiology Group, USSR Academy of Medical Sciences, Moscow
(Presented by A. T. Smirnov)

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In our laboratory a positive trophic action of the vagus nerve on the intact heart of the dog has been observed as well as the disruption of this effect in the pathologically altered heart [5, 8, 9, 11, 12, 13]. It is known that asphyxia evokes a profound reorganization both in the central nervous system [1, 4, 16, etc.] and in the periphery, in particular, in the heart [1, 2, 7, 19, 21, 23].

The goal of the present investigation was to study the peripheral action of asphyxia on the functional state of the specific musculature of the heart during profound and prolonged vagus inhibition.

METHODS

Acute experiments were performed on 20 adult dogs under morphine-urethane anesthesia. Asphyxia was produced for three minutes by the method of rebreathing from a plastic balloon of 500 cc volume (until respiration was halted by asphyxia). The peripheral portion of the vagus nerve was stimulated by an electrostimulator (EI-1) by direct current impulses at a frequency of 25 gauss and duration 0.1 msec. For each experiment a current strength was chosen which produces profound inhibition of cardiac action. In experiments on 15 dogs the left vagus nerve was stimulated and in five dogs, the right (one of the vagus nerves always remained intact).

The arterial pressure and respiration were recorded on a kymograph. EKG was recorded as the Standard lead II on an EKP-S2 electrocardiograph. The percent of oxygen saturation of arterial and venous blood was determined (oximeter) in seven dogs.

For an objective assessment of the extent of the effect of the vagus nerve on the heart under conditions of asphyxia we used the following indices: duration of cardiac standstill, length of total vagal effect, cardiac rhythm, mean arterial and pulse pressures (per minute of vagal stimulation) and EKG changes.

RESULTS

Stimulation of the peripheral portion of the vagus nerve, which evokes, as a rule, a profound inhibition of cardiac activity in the dog, did not appear to have such a marked effect during asphyxia.

The duration of vagal standstill during stimulation of the peripheral portion of the vagus nerve by an optimal current, against a background of normal respiration varied in different dogs within the limits of eight to 140 sec. During asphyxia a significant shortening of vagal standstill after application of the same stimulation was noted (in 14 out of 20 experiments).

In five out of 20 experiments the duration of vagal standstill did not change and only in one case during asphyxia did vagal standstill increase from five to 15 sec; in this case almost all experiments had a very short initial duration of vagal standstill (2-5 sec).

The duration of the total vagal effect on the heart was 547.5 ± 106.6 sec before asphyxia, whereas during asphyxia it was 181.1 ± 40.3 sec, i.e. it became three times shorter ($P < 0.02$).

Asphyxia, as a rule, provokes slowing of the cardiac rhythm [2, 3, 4, 16, 19], which is observed as well in

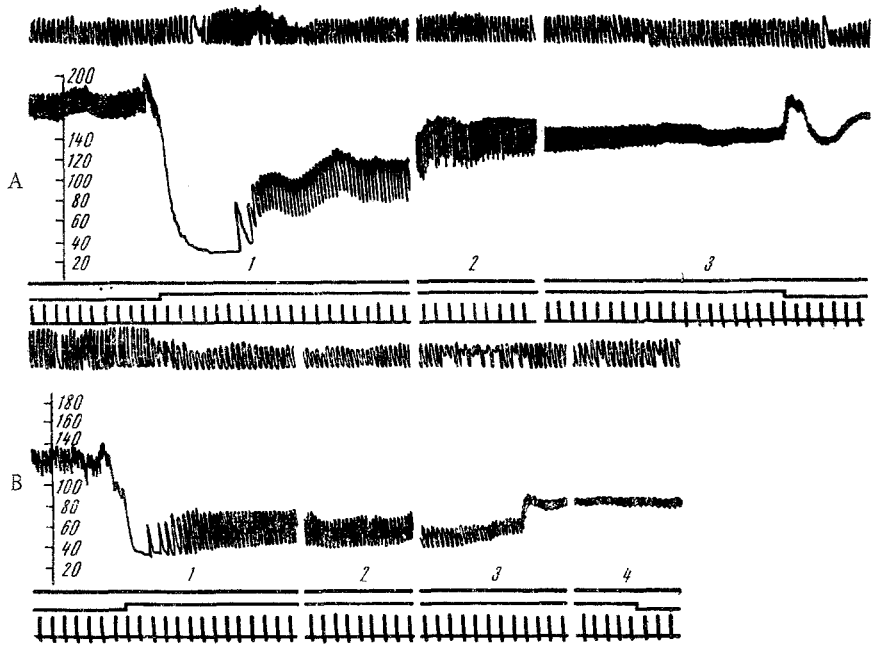

Fig. 1 Dynamics of changes in arterial pressure and respiration during stimulation of the peripheral portion of the vagus nerve in normal respiration and in asphyxia. Significance of curves (from above downwards): respiration, arterial pressure, zero line, stimulation marker, time marker (5 sec). A) Stimulation of the peripheral portion of the vagus nerve (2.8 milliamp, 25 gauss, 0.1 msec) during spontaneous respiration. 1) Start of stimulation (duration of standstill 32 sec, cardiac rate 20-40 per min); 2) seventh min of stimulation (cardiac rate 62 per min); 3) thirteenth min of stimulation (cardiac rate 70 per min), after effects (rate 120 per min). B) Stimulation of the peripheral portion of the vagus nerve (2.8 milliamp, 25 gauss, 0.1 msec) during asphyxia. 1) Start of stimulation (length of standstill 10 seconds, cardiac rate 32 per min); 2) second min of stimulation (cardiac rate 40 per min); 3) third min of stimulation (cardiac rate 40 per min, stopping of vagal inhibition—cardiac rate 128 per min); 4) end of stimulation (fourth min) and after effects (cardiac rate 128 per min).

separate experiments. During stimulation of the peripheral portion of the vagus nerve the cardiac rhythm (after vagus-escape) asphyxia is noted to slow less than in animals with normal respiration. The greatest slowing of the cardiac rhythm, noted in the first two min of vagal stimulation, was less marked during asphyxia (with normal respiration the cardiac rhythm fell by 80%, with asphyxia by 74%). Upon subsequent stimulation the difference in the change in cardiac rhythm increased (in the last min of stimulation during normal respiration the cardiac rate slowed by 53% and during asphyxia, by 38%). However, these changes did not achieve statistical significance.

Vagal stimulation in the dog produces a significant, positive inotropic effect. Therefore it is of interest to compare the pulse pressure during prolonged stimulation of the vagus nerve during asphyxia and during normal respiration. During normal respiration an increase in pulse pressure was noted: it was sharply apparent and prolonged.

During asphyxia the rise in pulse pressure when the peripheral part of the vagus nerve was prolongedly stimulated was less in height and duration.

Change in the level of arterial pressure during vagus nerve stimulation may be an indirect index of the effect of the vagus nerve on cardiac activity. In our experiments the fall in the mean arterial pressure during the first min of stimulation in asphyxia was considerably less than the fall at the same time interval during normal respiration (in asphyxia the fall in pressure was twofold, in normal respiration, more than threefold, $P < 0.02$). In later minutes no significant difference in the height of the arterial pressure was noted (Fig. 1).

These data indicate the significant decrease in inhibitory effect of the vagus nerve on the heart (with maximal stimulation) during asphyxia.

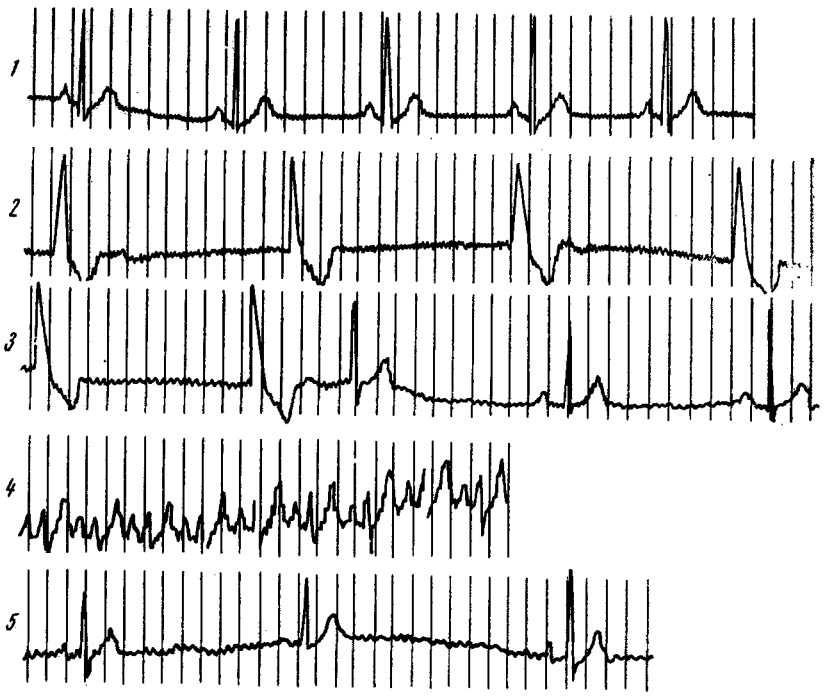

Fig. 2. EKG changes during stimulation of the peripheral portion of the vagus nerve in normal respiration and in asphyxia. 1) EKG (Lead II) before the start of vagal stimulation; 2) EKG (Lead II) at one min of stimulation; 3) EKG (Lead II) at the end of vagal stimulation (eighth min); 4) EKG during asphyxia before start of stimulation of the vagus nerve; 5) EKG (Lead II) at one min of stimulation during asphyxia.

Usually asphyxia evokes an increase, distortion, or disappearance of the P wave, lengthening of the PQ interval, and drawing together of the R and T waves (transference to a monophasic curve), increase in T wave and sometimes its distortion [4, 6, 9, 10, 15, 17, 18, 20, 22]; a number of authors note displacement and prolongation of the ST segment [6, 15, 19]. Similar EKG changes have been observed under the influence of asphyxia in our experiments.

Prolonged stimulation of the vagus nerve during normal respiration in most of our experiments (14 out of 20) provoked a depression of the sinoatrial node and appearance of automaticity of lower-lying portions of the cardiac musculature. These changes remained for $2^2/_1$ to $19^1/_2$ min of vagal stimulation. The appearance of sinus rhythm on the EKG (P waves) was always accompanied by acceleration of the cardiac rate and the increase in mean blood pressure, as has been previously reported from our laboratory [14].

Vagus nerve stimulation during asphyxia was accompanied by different changes in the bioelectrical activity of the heart: in several experiments after cardiac standstill in the first min of stimulation, normal EKG complexes appeared or atrio-ventricular dissociation, which signified a less severe inhibition of the cardiac musculature; occasionally atrio-ventricular or nodal rhythms of these forms rapidly changed to normal EKG complexes (Fig. 2).

The illustrated electrocardiographic material also characterizes the less marked inhibition of the cardiac musculature during prolonged vagal stimulation in asphyxia.

In asphyxia the oxygen saturation of the blood decreases in arterial blood from 84.2 to 64% (mean), in venous blood from 52 to 36%. These data are statistically valid ($P = 0.01$), indicating the relative depth of asphyxiation.

Thus, by studying the effect of prolonged stimulation of the peripheral portion of the vagus nerve on the heart, the functional status of which has been altered by asphyxia, we observed a decrease in the capacity of the heart to manifest profound inhibition. The results of our studies are in agreement with the data of Sloan [21]. Evidently, the profound form of depression as the most strenuous physiological process for the organism against the given pathological background is impossible for the heart as a heterogeneous stimulatory system.

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All abbreviations of periodicals in the above bibliography are letter-by-letter transliterations of the abbreviations as given in the original Russian journal. *Some or all of this periodical literature may well be available in English translation.* A complete list of the cover-to-cover English translations appears at the back of this issue.
