DYNAMICS OF CARDIAC ACTIVITY AND THE P WAVE OF THE ELECTROCARDIOGRAM WITH STIMULATION OF THE VAGUS FOLLOWING DISRUPTION OF THE INHIBITORY EFFECT

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Translated from Byulletin' Éksperimental'noi Biologii i Meditsiny, Vol. 52, No. 11, pp. 7-13, November, 1961
Original article submitted March 15, 1961

A number of works exist representing studies of the electrocardiogram dynamics during prolonged stimulation of the vagus nerve. In short term experiments on dogs involving prolonged stimulation of the vagus the investigators observed lengthening of the P–Q interval, a reduced and biphasic P wave, an increased T wave with a negative sign, and growth of the S wave [3,5,6,8]. In long term experiments on dogs, where the entire trunk of the vagus nerve was stimulated, associated with slowing of cardiac activity the investigators observed changes in the waves of the electrocardiogram: heightening of the R wave, lengthening of the P–Q interval, atrioventricular dissociation, and appearance of a negative T wave [1,2,4]. In experiments on frogs involving vagal inhibition of the heart, workers noted atrioventricular blockade, lengthening of the P–Q interval, shortening of the duration of electrical ventricular systole, changes in the form of the T wave, and heightening of the T wave [7].

Similar observations were made in clinical practice during intrathoracic operations on patients. Mechanical and electrical stimulation of the peripheral end of the vagus nerve caused lengthening of the P–Q and R–R intervals, and reduction and inversion of the P wave [10,15].

In this work we studied the dynamics of the P wave of the electrocardiogram as dependent upon the magnitude of the arterial pressure and the rhythm frequency of the cardiac action during prolonged stimulation of the peripheral segment of the vagus nerve, in short term experiments on dogs following the appearance of vagus-escape.

EXPERIMENTAL METHOD

We carried out 74 investigations on 16 adult dogs of both sexes, weighing from 6 to 25 kg, on a short term basis. The operations were performed under morphine-urethane narcosis. The morphine was injected subcutaneously, using 3–4 ml of a 1% solution. The urethane was administered intravenously, in the form of a 25% solution, employing a dosage of 1 g/kg.

Arterial pressure (mercury manometer) and tracheal respiration were recorded on a kymograph. With the aid of needle electrodes at three standard leads, we recorded the electrocardiogram (during prolonged observations we recorded the II lead) before, during, and after stimulation of the peripheral segment of the vagus nerve. The peripheral segment of the transected vagus in the neck was stimulated by means of an induction current. The intensity of the stimulating current was higher than the threshold level by 5 mm in the spacing of the induction coils.

EXPERIMENTAL RESULTS

The results of the experiments were divided into several groups, depending on the presence or absence of auricular contraction (P wave of the electrocardiogram) during stimulation of the peripheral end of the vagus nerve. They are the following:

First group. In experiments of this group with stimulation of the peripheral end of the vagus nerve following disruption of cardiac inhibition (vagus-escape) the P wave was completely absent during the continuous stimulation. In this case the mean arterial pressure was equal to 45–60% of the original level, and the rhythm of the cardiac activity was equal to an average of 30–40 contractions per minute. In the electrocardiogram we observed a nozhkovyi* rhythm, atrioventricular rhythm, and, in certain trials, the T wave enlarged and became higher than the R wave.

* Transliteration of Russian—Publisher's note. Possible Translation—"crurul."

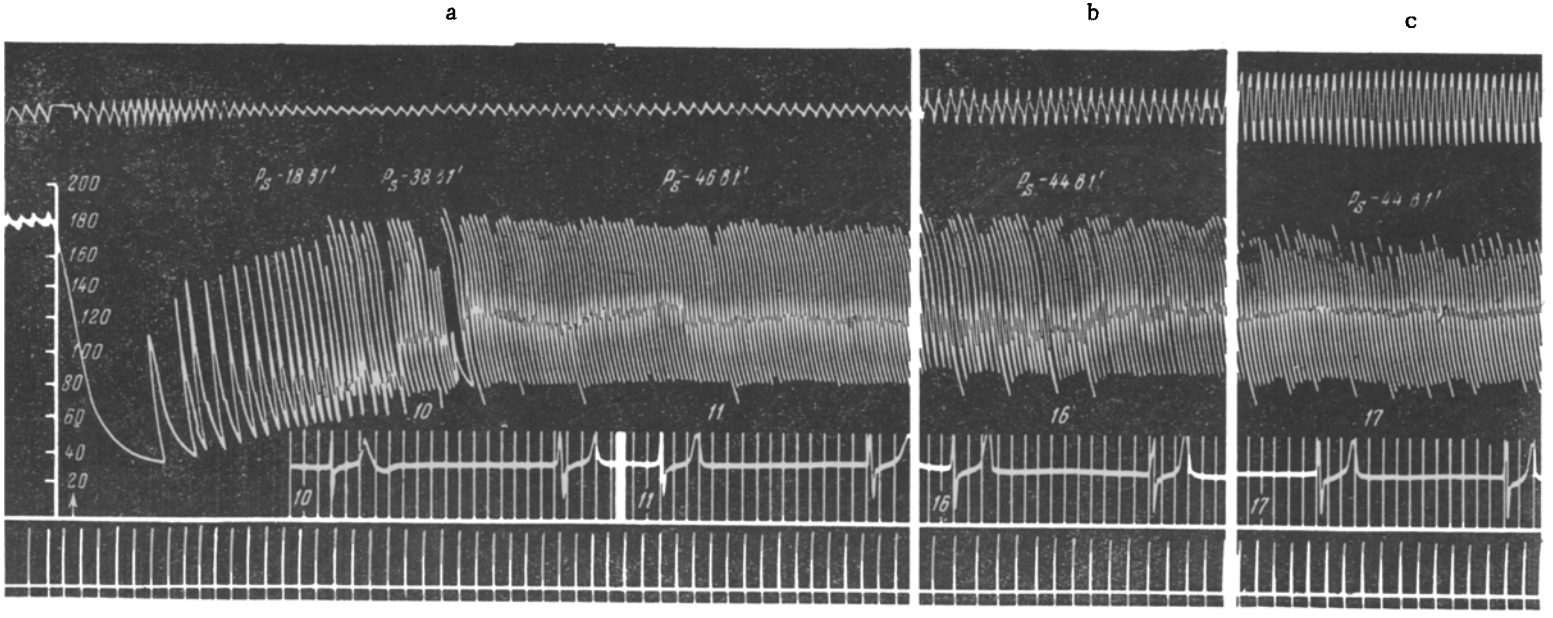

Fig. 1. Dynamics of changes in the EKG waves (lead II), respiration and the arterial pressure during prolonged stimulation of the peripheral segment of the vagus nerve. Absence of the P wave throughout the entire period of stimulation, with a slow cardiac rhythm. Meaning of the curves (from above downward): tracheal respiration; arterial pressure, recorded in the femoral artery; electrocardiograms; stimulation markings (the zero line); time markings (5 seconds). a) Initial period of stimulation of the peripheral segment of the right vagus nerve; b) after 12 minutes 55 seconds; c) after 18 minutes 15 seconds from the start of stimulation. Duration of the stimulation was 24 minutes. Time at which the electrocardiogram was recorded is noted by the numerals. The arrow indicates the beginning of stimulation of the peripheral segment of the vagus nerve.

As an illustration, we show the results of trial No. 4 from October 13, 1960 (Fig. 1). As can be seen from Fig. 1, prior to stimulation of the vagus nerve the mean arterial pressure was equal to 183 mm Hg; during stimulation the mean arterial pressure at various periods was equal to 103–135 mm Hg (56–73% of the original level) and was maintained at that level throughout the course of the entire stimulation period–124 minutes. The cardiac rhythm at the beginning of stimulation was 18 contractions per minute, and rose to 44 per minute, where it stayed throughout the entire stimulation period. In the electrocardiograms presented it is obvious that the P wave does not appear right up to the end of the stimulation. In this trial the amplitude of the T wave rose from 8 to 14 mm, which is apparently related to heightening in the intensity of the metabolic processes within the ventricular myocardium. We also observed changes in respiration: during stimulation it first increased in frequency from 18 to 22, 36 and 46 respiratory movements per minute, and then also deepened–the amplitude increased from 9 to 23 mm. We never observed similar respiratory changes in those trials where, following vagus-escape, the cardiac contractions were accompanied by a P wave. When the stimulation ceased the mean arterial pressure, the cardiac rhythm, and the electrocardiogram immediately returned to the original levels.

Second group. In this group of experiments, during stimulation of the peripheral segment of the vagus nerve, with resumption of contractions the P wave initially was absent and then appeared. With a cardiac rhythm averaging 20–40 contractions per minute the P wave was absent, despite a sufficiently high level for the mean arterial pressure (50–80% of the original level), and the P wave only appeared with acceleration of the cardiac rhythm to 50–70 contractions per minute. In this case the mean arterial pressure rose and reached 80–90% of the original level. With vagus-escape, prior to appearance of the sinus rhythm we could observe an atrioventricular rhythm in the electrocardiogram, with a negative P wave, then a nozhkovyi rhythm, then atrioventricular blockade, and, finally, the complete complex of electrocardiogram waves. As an illustration we show the data of experiments No. 2 and No. 15 (Fig. 2 and 3).

In Fig. 2 we show the kymogram and electrocardiograms of a trial in which, with stimulation of the peripheral end of the vagus nerve, the P wave was recorded in the very first contractions, but was negative in sign. The rhythm

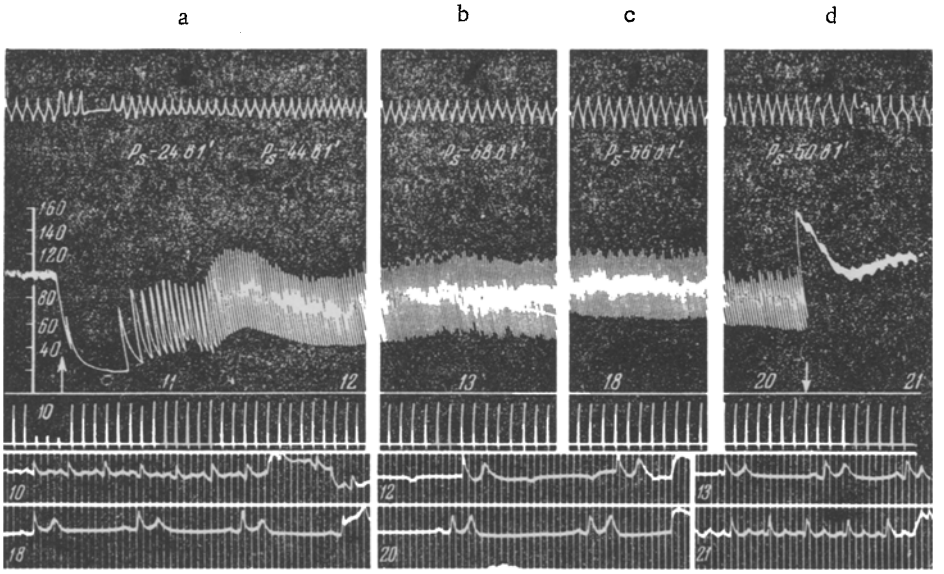

Fig. 2. Dynamics of the changes in the EKG waves (lead II), respiration, and the arterial pressure during prolonged stimulation of the peripheral segment of the vagus nerve. Appearance of the P wave with acceleration of the cardiac rhythm. Meaning of the curves (from above downward): tracheal respiration; arterial pressure, recorded in the femoral artery; stimulation marking (the zero line); time marking (5 seconds). a) Beginning of stimulation of the peripheral segment of the right vagus nerve; b) after 3 minutes 50 seconds; c) after 13 minutes 45 seconds; d) after 17 minutes 50 seconds from the beginning of stimulation. Duration of stimulation was 18 minutes 30 seconds. Arrows designate the beginning and end of stimulation.

of the cardiac contractions in this case was equal to 24–44 per minute, and the mean arterial pressure consisted of 69% of the original level. The artrioventricular rhythm was replaced by a sinus rhythm (the P wave became positive)

with a cardiac rhythm of 68 per minute and with a mean arterial pressure equal to 86% of the original level. The sinus rhythm was retained in this trial up to the end of stimulation, which lasted 18 minutes 30 seconds. The cardiac rhythm was equal to an average of 60 contractions per minute, and the mean arterial pressure consisted of 80–90% of the original. In this trial, with stimulation of the peripheral segment of the vagus nerve we observed an increase in the voltage of all the electrocardiogram waves, which is apparently related to the influence of the vagus nerve in increasing the intensity of the cardiac metabolic processes. It must be noted that in this trial the electrocardiogram was of the coronary type even before stimulation of the vagus. After cessation of the stimulation the arterial pressure, cardiac rhythm and electrocardiogram (including the voltage of the EKG waves) returned to the original values.

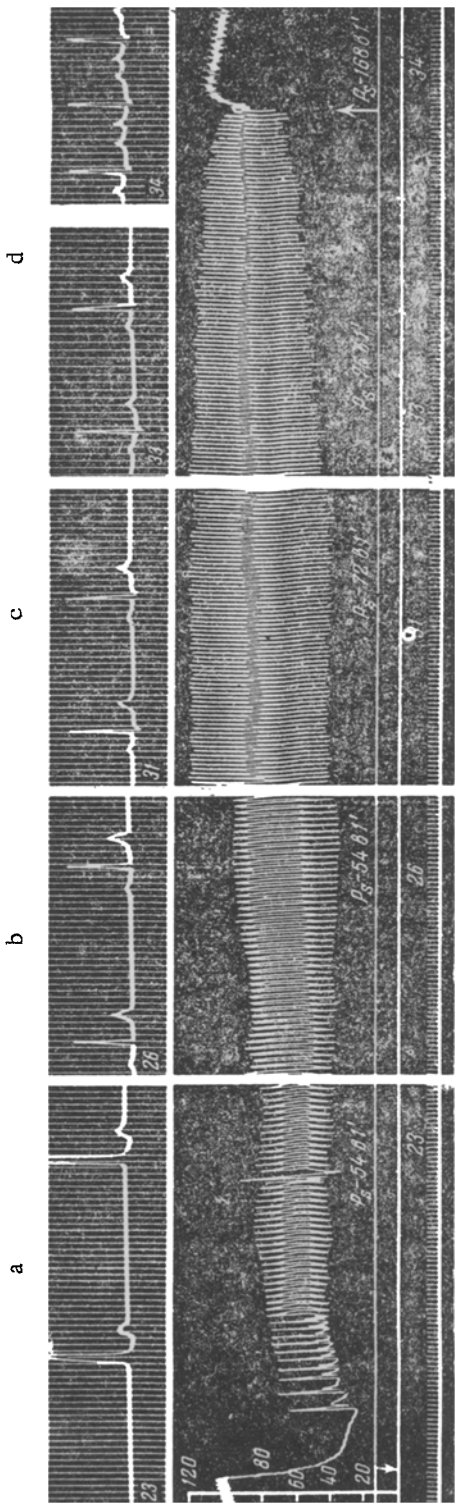

Fig. 3. The same. Appearance of the P wave with elevation of the mean arterial pressure. Meaning of the curves (from above downward): electrocardiogram; arterial pressure, recorded in the femoral artery; stimulation marking; zero line; time marking (1 second). Arrows indicate the beginning and end of stimulation. a) Initial period of stimulation of the peripheral segment of the right vagus nerve; b) after 3 minutes 50 seconds from the beginning of stimulation; c) after 7 minutes 17 seconds from the beginning of stimulation; d) after 10 minutes 44 seconds from the beginning of stimulation. Duration of the stimulation 11 minutes 24 seconds.

In Fig. 3 we show the kymogram and electrocardiograms of experiment No. 15. The first cardiac contractions following the appearance of vagus-escape, at a rhythm of 24 per minute, occur without a P wave, the ventricles alone contracting. The mean arterial pressure, in this case, is equal to 60% of the original level; after 4 minutes 25 seconds, when the mean arterial pressure reaches 66–70% of the original level and the rhythm increases to 54 contractions per minute, the P wave appears, and is retained throughout the entire period of stimulation of the vagus nerve (11 minutes 34 seconds).

Third group. In experiments of this group, during stimulation of the peripheral end of the vagus nerve, after stoppage of the heart and subsequent continued stimulation, the first cardiac contractions were accompanied by a P wave in the electrocardiogram. Included in these observations were instances where stimulation of the vagus nerve was accompanied by prolonged inhibition of the respiratory center. In this case the first contractions of the heart following vagus-escape were so intense that the mean arterial pressure exceeded the original level. It may be postulated that in these trials the action of asphyxia was represented, caused by cessation of respiration. However, this hypothesis must be rejected, since observations were made where, with prolonged inhibition of the respiratory center during stimulation of the peripheral segment of the vagus nerve, the first cardiac contractions following disruption of the inhibitory effect were not accompanied by a sinus rhythm and intensification of the ventricular contractions of the heart. In addition, there were cases where the first cardiac contractions following the appearance of vagus-escape showed P waves despite the absence of prolonged inhibition of the respiratory center.

As a result of the investigations performed it was established that with stimulation of the peripheral segment of the vagus nerve, following the appearance of vagus-escape, a sinus rhythm appears with a frequency of cardiac contractions equal to 50–60 per minute, and when the mean arterial pressure reaches 70–80% of the original level. At a lower mean arterial pressure and a slower cardiac rhythm an atrioventricular rhythm is observed. This may be seen in trials where, with stimulation of the peripheral segment of the vagus nerve following disruption of

the inhibitory effect, an atrioventricular rhythm is observed throughout the entire extent of the stimulation, in trials where, during the period of stimulation, the atrioventricular rhythm is replaced by a sinus rhythm, and in trials where the sinus rhythm appears immediately after disruption of the inhibitory effect. The stable form of the cardiac contractions, which is established with prolonged stimulation of the peripheral segment of the vagus nerve following appearance of vagus-escape (at a cardiac rhythm of 50–60 contractions per minute and a mean arterial pressure equal to 70–80% of the original level), suggests the influence on the heart of the tonus of the vagal center; this influence is characterized by slowing of the cardiac rhythm with maintenance of a large systolic blood volume, supporting the mean arterial pressure at an adequately high level. With stimulation of the peripheral segment of the vagus nerve, such cardiac contractions may be observed for up to 40 minutes or more. In this case, the heart does not fatigue, since after cessation of the stimulation the mean arterial pressure, cardiac rhythm, and electrocardiogram immediately return to the original levels.

There are a number of works, confirming the viewpoints of our laboratory [9,11,12,14, et al], in which data are presented indicating that under the influence of the vagus nerve oxygen consumption decreases and its assimilation improves, and in which the action of the vagus nerve on the heart is regarded as favorable for metabolism and as enabling a more economic blood circulation. With direct and reflex stimulation of the cardiac branches of the vagus nerve, the authors indicated above obtained a decrease in oxygen consumption independent of the negative chronotropic effect of the vagus nerve, and, in this case, the energy efficiency increased. Removal of the effect of the vagus nerve on the heart, under these same conditions, strengthened the oxidative processes in the heart [13].

The data of our experiments, involving prolonged stimulation of the vagus nerve, where the rhythm of the cardiac contractions was equal to 50–60 per minute with a sufficiently high level for the mean arterial pressure (70–80% of the original level) and maintenance of the complete electrocardiogram complex (presence of the P wave), permits postulating that such cardiac contractions represent an economic form of cardiac activity.

SUMMARY

Acute experiments were performed on dogs under morphine-urethane anesthesia. A study was made of the cardiac activity and dynamics of the ECGP wave with relation to the value of the mean arterial pressure and the rhythm of cardiac activity in prolonged stimulation of the peripheral section of divided vagus after inhibition disruption (vagus-escape). As demonstrated, in prolonged stimulation of the peripheral section of the vagus after vagus-escape the sinus rhythm appears with the frequency of cardiac contractions of 50-60 per minute and with the achievement of the mean arterial pressure of 70-80% of the initial level. The stable form of cardiac contractions thus established resembles the effect on the heart of the vaguscenter characterized by slowed down rhythm of cardiac activity with retention of large systolic blood volume, maintaining the mean arterial pressure at a sufficiently high level. Atrioventricular rhythm and artioventricular block were observed with a lower mean arterial pressure and a slower rhythm of cardiac contraction.

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