CHANGES IN THE T WAVE OF THE ECG DURING AN INCREASE

IN TONE OF THE VAGUS CENTER IN NORMAL DOGS

AND IN DOGS WITH EXPERIMENTAL MYOCARDIAL INFARCTION

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This paper describes a continuation of the study of the influence of the vagus nerves on the healthy and pathologically changed heart, at present being undertaken in our laboratory.

When the tone of the vagus center is raised, changes are known to occur in the excitation processes in cardiac muscle, and these are reflected in the dynamics of the waves and intervals of the electrocardiogram (ECG) [6, 12, 13]. During stimulation of the peripheral cut end of the vagus nerve or during an increase in the tone of the vagus center, metabolic changes take place in the myocardium [2, 7, 9, 10] and are reflected in the T wave [3]. When the tone of the vagus center is increased or the peripheral end of the divided nerve stimulated, the T wave may become negative instead of positive, and it may increase in amplitude to exceed the R wave [1, 5, 6, 8].

In the present investigation the dynamics of the T wave of the ECG was studied during an increase in the tone of the vagus center in dogs in chronic experiments in normal conditions and after the development of experimental myocardial infarction of the ventricles of the heart.

EXPERIMENTAL METHOD

The investigations were conducted on four adult dogs in chronic experimental conditions (observations on each dog continued for 3-6 months). The tone of the vagus center was increased by means of morphine, injected subcutaneously in a dose of 0.01-0.03 g. The tone of the vagus center was raised still further by intravenous injection of a 10% solution of calcium chloride in a dose of 0.05 g/kg body weight or by injection of 1.0 ml of 0.1% adrenalin solution.

The ECG was recorded by means of a type EKPS-2 electrocardiograph and a type ANEK vectorcardioscope for analysis of the electrical activity of the heart, in three standard leads (during prolonged observations lead 2 was recorded). The pneumogram was recorded and the heart rate was measured. An experimental myocardial infarct was produced in two dogs by ligation of the descending branch of the left coronary artery. Observations were maintained for 19-25 days after development of the infarct.

EXPERIMENTAL RESULTS

In experiments on intact dogs, an increase in the tone of the vagus center was accompanied by obvious changes in the T wave, whereas no changes were recorded in the other waves, or they were inconsistent. In most experiments the P-Q and S-T intervals were slightly lengthened in all the dogs, which corresponds to findings reported by many other workers [4, 6, 11].

In the initial state, the $T_{2,3}$ wave in two intact dogs was negative or biphasic, and in the other two it was positive or biphasic. After injection of morphine (when the heart rate fell from 100-120 to 50-60 beats per minute) different changes took place in the $T_{2,3}$ wave in different animals. It should be noted that in our first experiments no changes in the $T_{2,3}$ wave were observed when the tone of the vagos center was increased. In the subsequent experiments changes took place in the magnitude and direction of the $T_{2,3}$ wave during a slowing of the heart rate. In dogs with an initially negative $T_{2,3}$ wave it became biphasic or positive; if the initial $T_{2,3}$ wave was positive, when the tone of the vagus center was raised the wave remained unchanged or became biphasic.

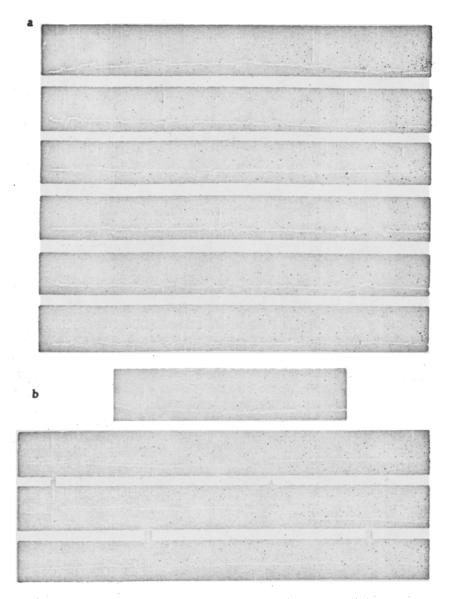


Fig. 1. Changes in the T₂ wave during an increase in the tone of the vagus center in intact dogs with an initially positive T wave. A) Experiment with the dog Laska on March 22, 1962: 1) ECG (lead 2) before injection of morphine; 2) after injection of morphine; 3,4) immediately after injection of calcium chloride; 5) 5 min after injection of calcium chloride; 6) 1 h 5 min after injection of calcium chloride. B) Experiment with the dog Reks on May 4, 1962; 1) ECG (lead 2 before injection of morphine); 2) after injection of morphine; 3) 7 min after injection of adrenalin; 4) 45 min after injection of adrenalin.

With a further increase in the tone of the vagus center after injection of calcium chloride, in the dogs with an initially negative $T_{2,3}$ wave for the first 5 min this became more strongly negative, and then changed through biphasic to positive, which it remained for 1.5-2 h. It should be noted that a strongly negative $T_{2,3}$ wave was formed immediately after injection of calcium chloride (when the heart rate had slowed to 32-44 beats per minute) irrespective of its nature during a moderate increase in the tone of the vagus center (when the heart rate was 50-60 per minute).

In one of the dogs with an initially positive $T_{2,3}$ wave, when the heart rate was slowed (after injection of calcium chloride or adrensiin) the $T_{2,3}$ wave remained positive and increased in amplitude (Fig. 1, B). In another dog with an initially positive $T_{2,3}$ wave, as in the two dogs with an initially negative $T_{2,3}$ wave, starting from the 12th experiment it became negative after injection of calcium chloride, and then changed to biphasic (Fig. 1, A).

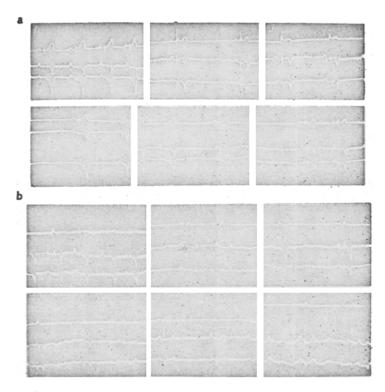


Fig. 2. Changes in the T wave during an increase in the tone of the vagus center in a dog with experimental myocardial infarction. A) Experiment with the dog Dzhek on June 7,1961 (on the 10th day after operation): 1) ECG (in three leads) before injection of morphine; 2) after injection of morphine; 3) immediately after injection of calcium chloride; 4) 11 min after injection of calcium chloride; 5) 41 min after injection of calcium chloride; 6) 1 h 10 min after injection of calcium chloride. B) Experiment with the same dog on June 26 (29 days after operation): 1) ECG (in three leads) before injection of morphine; 2) after injection of morphine; 3, 4) immediately after injection of calcium chloride; 5) 18 min after calcium chloride; 6) 56 min after injection of calcium chloride.

In two dogs an infarct of the anterior wall of the left ventricle developed after ligation of the descending branch of the left coronary artery. Significant changes were observed in the ECG, consisting of the appearance of polytopic ventricular extrasystoles and atrioventricular block. During the first 5 days after the operation the influence of the vagus nerves on the heart when the tone of their centers was raised was expressed, not by a slowing of the heart rate, but by an increase in the intensity of the extrasystoles. These observations are in agreement with our previously reported findings [5]. On the 5th day the effect of the tone of the vagus centers began to be shown by a slowing of the heart rate. One dog showed no changes in the T2,3 wave until the 16th after the operation, and another until the 14th day. With an increase in the tone of the vagus center the T2,3 wave was negative before and after injection of morphine and after injection of calcium chloride (at this period the heart rate had fallen to 44 per minute)(Fig. 2, A).

In one dog a slowing of the heart rate during an increase in the tone of the vagus center led, on the 16th day and later, to the appearance of a biphasic T_{2,3} wave (when the heart rate had fallen to 44-56 beats per minute after injection of calcium chloride) the wave became strongly negative during the first 5 min, after which it changed to biphasic or positive (Fig. 2, b).

In another dog with myocardial infarction changes in the $T_{2,3}$ wave during an increase in the tone of the vagus center became apparent on the 14th day, in the form of an increase in the negativity of the $T_{2,3}$ wave.

There results show that until the cardiac function is restored some time (14-18 days) after myocardial inforction, the vagus releves have no apparent effect on metabolism in the heart, although their inhibitory action on the heart rate is restored by the 5th-6th day.

SUMMARY

In chronic experiments on 4 dogs a study was made of the dynamics of the ECG T-wave with a rise of the vagus nerve tone; this was done for the purpose of indirect evaluation of the effect produced by the vagus on the metabolic processes in the cardiac muscle. The T-wave dynamics was studied in 2 dogs both in normal conditions and in experimental myocardial infarction. A rise of the vagus center tone was induced by subcutaneous injection of morphine, and a further rise of the tone was attained by intravenous administration of calcium chloride. With an increase of the tone of the vagus center in the intact dogs both the value and the direction of the T-wave changed. In dogs with experimental myocardial infarction T-wave changes resulted from rise of vagus center tone only on the 14th day after the ligation of the descending branch of the left coronary artery. On the basis of the data obtained it is suggested that the effect of the vagus nerves on metabolism is not shown until restoration of the functional state of the heart in myocardial infarction, although their inhibitory effect on the heart rate is already restored on the 5th-6th postoperative day.

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