THE EFFECTS OF ELECTRICALLY STIMULATING THE VENTRICLES OF THE HEART AT VARIOUS RHYTHMS

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In previous investigations [1—5], performed on normal dogs, we determined the rhythm limits within which the heart will assimilate electrical stimulation in the area of the sinoauricular node.

In this report we present the results of investigating various rhythms of electrical stimulation delivered to the ventricular myocardium.

EXPERIMENTAL METHOD

The experiments were carried out on adult dogs, weighing 10—20 kg, under morphine-urethane anesthesia. The ventricles of the heart were stimulated electrically. To do this, the animal was placed on artificial respiration, the chest cavity was opened in the fourth or fifth interspace on the left, the pericardium was incised, and the bared ends of two thin separated wires were fastened to different areas of the myocardium, serving as myocardinal electrodes. Usually, one electrode was fixed on the wall of the left ventricle, and the other, on the wall of the right. Stimulation was carried out with the aid of a GPF electronic stimulator, constructed in the All-Union Institute of Medical Instruments and Equipment, or an SIF—1 stimulator, designed by the Institute of Normal and Pathological Physiology. The impulse form was straight-angle or straight-angle with slightly rounded peak. The force of each impulse was 3—4 v, lasting 2—3 msec.

We recorded the electrocardiogram from the standard lead II. In recording an electrocardiogram during electrical stimulation of the heart it is not possible to use an electrocardiograph with two-way capacitors in the booster tract, and thus, we used an electrocardiograph with a direct current amplifier. Simultaneously with the electrocardiogram, we recorded pressure fluctuations in one of the ventricles of the heart or in the aorta, by means of an electromanometer with wire transducers having ohmic resistance. Catheterization of the left ventricle or the aorta was carried out via the left carotid artery, and of the right ventricle, via the right jugular vein. To prevent blood coagulation, heparin was injected intravenously.

EXPERIMENTAL RESULTS

The effects of the various electrical stimulation rhythms applied to the ventricles were investigated in multiple trials involving 20 dogs.

Stimulation rhythms applied to the ventricles, which were several beats per minute slower than the spontaneous rate, were sometimes assimilated by the heart. Complete assimilation of the slower rhythms did not occur as a rule. With slow stimulation rhythms, the impulses arising in response to electrical stimulation of the myocardium sometimes caused ventricular extrasystoles and a varying form of disturbance of the cardiac rhythm: ventricular bi-, tri- and quadrigemini (Fig. 1).

With a stimulation rhythm close to the spontaneous one but slower than it, one could see the phenomenon of dissociation with interference, i.e., transfer from a sinus rhythm to a ventricular one and vice versa (Fig. 2). Dissociation with interference could also be observed at a stimulation rhythm coinciding with the sinus rhythm or exceeding it, but under conditions where the stimuli were of low intensity.

An electrical stimulation rhythm (with a suitable stimulation intensity) corresponding to the spontaneous frequency of cardiac contractions or normally not exceeding 180 per minute, was immediately assimilated by the ventricles. The electrocardiographic complexes and the fluctuations in the intraventricular pressure exactly corresponded to the stimulation rhythm, and were equal in amplitude. The form of the electrocardiographic ventricular complexes, arising under the influence of the electrical stimuli, were determined by the site at which the cathode of the stimulating current was located.

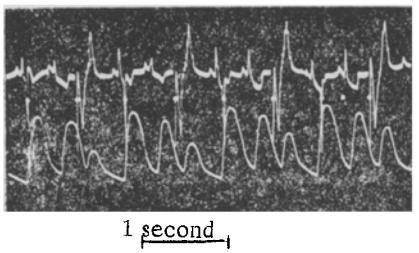

Fig. 1. Venticular trigemini at a slow stimulation rhythm of the left ventricle. Recording of the electrocardiogram and pressure in the left ventricle. Moments of stimulation are marked by dots.

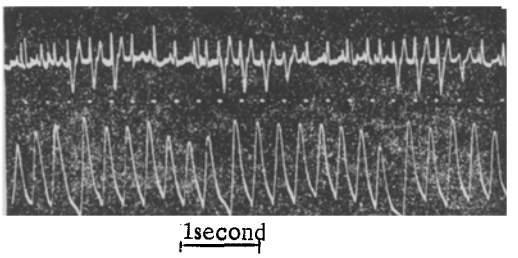

Fig. 2. Dissociation with interference associated with stimulation of the left ventricle at a slightly slower rhythm than the spontaneous one. Recording of the electrocardiogram and pressure in the aorta.

When the ventricles were stimulated with a frequency of from 150 to 200 stimuli per minute the electrocardiographic complexes and the fluctuations in the intraventricular pressure followed the stimulation rhythm. The amplitude of the pulse waves in the intraventricular pressure during this ventricular tachysystole was usually lower than the amplitude of these pulse waves during spontaneous rhythm. This is explained by the rapid rhythm of electrical stimulation delivered to the ventricles, and their diminished filling during the period of diastole.

With even more rapid electrical stimulation of the ventricles (180–300 stimuli per minute), we could observe, as a rule, transient or more prolonged signs of transformation of the contraction rhythm and electrical and hemodynamic alternations. In the beginning of the electrical stimulation, in response to every two stimuli there arose two electrocardiographic complexes, and only one intraventricular pressure wave. Transformation of the contraction rhythm was followed by alternation in the amplitude of the intraventricular pressure waves; in this case, there occured a regular alternating pattern of a stronger and a weaker contraction (Fig. 3).

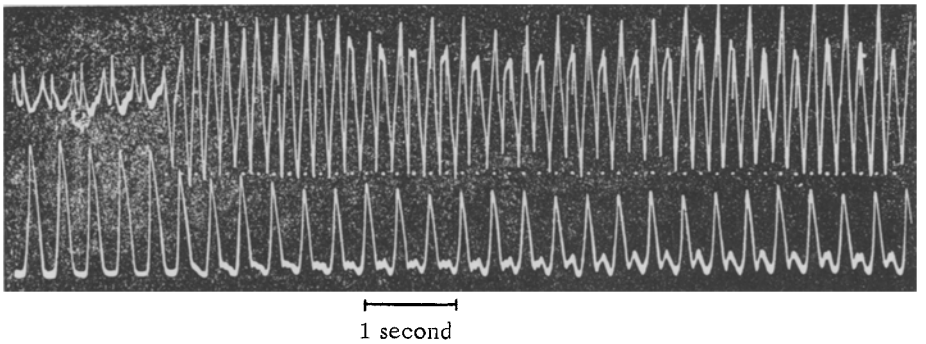

Fig. 3. Transition from transformation of the contraction rhythm to its alternation, accompanied by electrical alternation. Stimulation rhythm–340 per minute. Legend is the same as in Fig. 1.

Transformation of the contraction rhythm was sometimes completely absent, or was rather transient. The hemodynamic alternation following right behind it gradually decreased through increments in the amplitude of the weaker systole of the alternating complex. As a result, equalization of the ventricular contraction amplitudes occured, and the onset of complete assimilation of the stimulation rhythm. Transformation of the contraction rhythm or the

subsequent clearly manifested hemodynamic alternation could last for a long time. In this case, complete assimilation of the rhythm did not occur.

When the ventricular myocardium was stimulated with rapid rhythms we often observed alternation of the electrical signs manifesting excitation of the ventricles, expressed by changes in the amplitude and form of every 2nd electrocardiographic complex. Alternation of the electrocardiographic complexes was normally accompanied by transformation of the ventricular contraction rhythm or by its alternation. However, often the changes in the contracting activity of the ventricles occured in the presence of electrocardiographic complexes that were absolutely uniform in form and amplitude.

If the ventricular myocardium was stimulated at even more rapid rhythms (300–350 stimuli per minute and higher), as a rule, ventricular fibrillation ensued. In isolated cases only, with stimulation of the ventricles at a rate of 380–400 per minute, fibrillation did not occur or did not appear until sometime after initiation of the electrical stimulation. At this rapid stimulation rhythm (380–400 per minute) we sometimes observed transformation of the rhythm of the electrocardiographic complexes by a factor of two, i.e., every second stimulus was not accompanied by the generation of a ventricular complex on the electrocardiogram, and, therefore, by an intraventricular pressure wave (Fig. 4). Even in these cases, increasing the stimulation frequency invariably led to ventricular fibrillation.

There is a great deal in the literature devoted to electrical stimulation of the ventricles, but the authors essentially investigated the effects of stimulation during complete atrioventricular blockade. Only a few works involved studies of the cardiac activity during electrical stimulation of the ventricular myocardium in an intact heart [8–11].

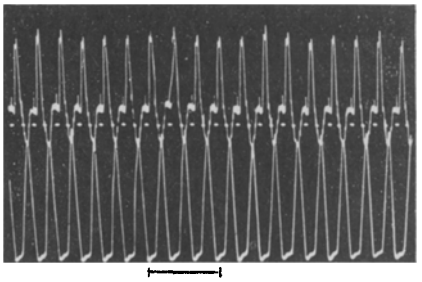

Fig. 4. Transformation of the rhythm of the electrocardiographic complexes with stimulation of the right ventricle at a rhythm of 380 per minute. Legend is the same as in Fig. 1.

The majority of investigators did not observe assimilation of the slow electrical stimulation rhythms by the heart [8,10,11]. Under these conditions we noted the possibility of a resultant dissociation with interference, as well as disturbances in the cardiac activity in the form of ventricular bi-, tri-, and quadrigemini.

With electrical stimulation of the left or right ventricle, a heterotopic focus of excitation arose at the side of the negative electrode, which functioned simultaneously with the homotopic one, i.e., the phenomenon of pararhythmia or parasystoles occured. As a result of the pararhythmia, it was possible for the dissociation with interference to occur. Impulses from the sinoauricular node interacted with impulses from the heterotopic focus of excitation. As a result of predominance of one of the two foci of excitation, the cardiac activity followed either a sinus or a ventricular rhythm.

The phenomenon of pararhythmia also explains why the electrical stimulation rhythms that were slower than the spontaneous one were not assimilated by the heart. In the presence of the pararhythmia, impulses from the heterotopic focus of excitation periodically wedge into the path of the natural impulses, causing a ventricular extrasystole. As a result of this, rhythmic disturbances occur in the form of ventricular bi-, tri-, and quadrigemini.

At a certain frequency and intensity of the electrical stimuli being delivered to the ventricular myocardium, the heterotopic focus of excitation begins to predominate over the homotopic one, and the heart transfers to a new, artificially created, contraction rhythm.

At a high frequency of ventricular stimulation (greater than 180 per minute) the stimulation rhythm is not immediately assimilated. Usually, a transformation of the cardiac rhythm occurs, then passing into an alternation of the myocardial contractions. Only later does complete assimilation of the electrical stimulation rhythm occur. Transformation of the ventricular contraction rhythm and its alternation, as well as the definite sequence relationship of these phenomena in the process of assimilation of the rapid rhythms by the heart, have already been described and explained by us in previous reports dealing with investigations of the effects of electrical stimulation delivered to the sinoauricular node. As can be seen from the data presented, these phenomena also take place with direct stimulation of the ventricular myocardium in a rapid rhythm. In addition, analysis of the changes in the mechanical and electrical manifestations of the cardiac activity, during rapid stimulation rhythms being administered to the ventricles, confirms the hypothesis advanced earlier [5], stating that the functional lability of the processes of excitation and contraction may be different. Thus, during transformation of the cardiac contraction rhythm one may observe a char-

acteristic dissociation: at the same time that the ventricular myocardium reproduces the stimulation rhythm in the form of a corresponding excitation frequency, the cardiac contractions occur at half the rate of the stimulation. In this case, alternation of the ventricular electrocardiographic complexes may be completely absent. It is also often absent in the presence of a clearly manifested hemodynamic alternation.

With direct electrical stimulation of the ventricular myocardium at a rhythm exceeding 300–350 per minute, fibrillation occurs as a rule [6,7]. In connection with this, we note that, according to our previous investigations, ventricular fibrillation does not arise with rapid stimulation rhythms delivered to the sinoauricular node (up to 1500–2000 per minute). At these rapid stimulation rhythms we observed incomplete atrioventricular blockade, protecting the ventricles from the action of the rapid impulses coming from the auricles. This justified our maintaining that atrioventricular blockade is a defense mechanism [5].

SUMMARY

Slow rhythms of electric stimulation of the ventricles are not assimilated by the heart. With this a phenomenon of dissociation with interference takes place, as well as periodic ventricular extrasystole and disturbances of cardiac rhythm in the form of ventricular bi-, tri- and quadrigemini. Frequent stimulation rhythms (up to 300-350 per minute) may be reproduced by dog heart. A change to the new rhythm of cardiac activity with high stimulation frequencies is effected through the transformation of the rhythm and alteration of cardiac stimulations. In high stimulation rhythms an alternation of the ECG ventricular complexes may take place; their rhythm may also be twice as slow as the stimulation rhythm. Fibrillation of cardiac ventricles is as a rule seen with the stimulation frequency of over 300-350 per minute.

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