

# SUPPRESSION OF THE AUTOMATIC ACTIVITY OF THE VENTRICULAR PACEMAKER IN VENTRICULAR FIBRILLATION

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Previous investigations [1,2] have shown that, during electrical stimulation of the sino-auricular node or ventricles of the heart at a fast rhythm, the automatic activity of the ventricular pacemakers is suppressed. The faster the rhythm of stimulation acting on the ventricles or arising in them, the deeper the depression of the automatic activity of the ventricular pacemakers. The depression of their automatic activity is manifested by asystole with a sudden onset of complete atrioventricular block or with a sudden cessation of the fast rhythm of stimulation against the background of a pre-existing atrioventricular block. Ventricular asystole is a preautomatic pause, at the end of which the automatic activity of the ventricular pacemakers is restored, giving rise to idioventricular contractions.

In face of these facts, it was postulated that ventricular fibrillation, characterized by a fast rhythm of excitation, must also cause suppression of the automatic activity of the pacemakers. The object of the present investigation was to verify this hypothesis.

## EXPERIMENTAL METHOD

Experiments were carried out on 25 adult dogs anesthetized with morphine and urethane. Thoractomy was performed. With a "dry heart," and with temporary interruption of the venous return, the right atrium was opened and a ligature applied to the upper part of the atrioventricular node. This produced a permanent and complete atrioventricular block, so that the ventricle contracted at its intrinsic slow rhythm. Against the background of the atrioventricular block, ventricular fibrillation was produced by an induction current. After the lapse of 10-40 sec, or in some experiments of several minutes, the ventricular fibrillation was stopped. This was done by means of the SKTB defibrillator, built by "Biofizpribor," which supplied 1-3 pulses of alternating current with a voltage of 300 V to the exposed heart; the duration of each pulse was 0.1 sec. Similar experiments with the creation of fibrillation and subsequent defibrillation were performed for control purposes on animals with an intact heart (with normal conduction of impulses from atria to ventricles).

The results obtained may accordingly be divided into two groups. One group was composed of the results of experiments with fibrillation and subsequent defibrillation conducted on animals with an intact heart (31 observations); the other of the results of similar experiments but conducted on animals with complete atrioventricular block (37 observations). In all the experiments, the electrocardiogram was recorded in one of the standard leads.

## EXPERIMENTAL RESULTS

Ventricular fibrillation in the intact heart lasting for between 10 and 120 sec did not cause depression of the automatic activity of the atrial pacemakers. Accordingly, defibrillation was not accompanied by ventricular asystole, and in all the experiments the contractions of the ventricles were soon restored by impulses arriving from the sino-auricular node. As usual in these cases, a brief tachycardia was observed immediately after defibrillation. The results of one of these experiments are shown in Fig. 1. The initial rhythm of the heart was 190 beats/min. Ventricular fibrillation lasting 15 sec was produced. After defibrillation, the heart rate began to rise quickly, and the initial rhythm was then soon restored.

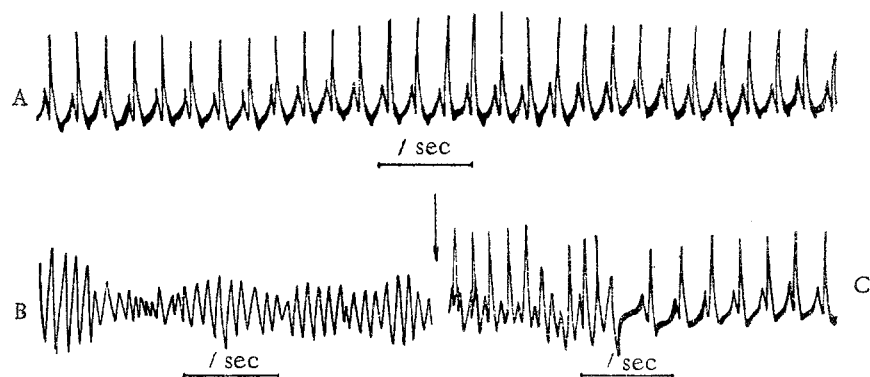


Fig. 1. Restoration of the cardiac activity by defibrillation of the ventricles of the intact heart: A) initial heart rate 190 beats/min; B) ventricular fibrillation lasting 15 sec; C) brief tachycardia and restoration of the initial rhythm after defibrillation. ECG recorded in standard lead II. The arrow denotes the time of defibrillation.

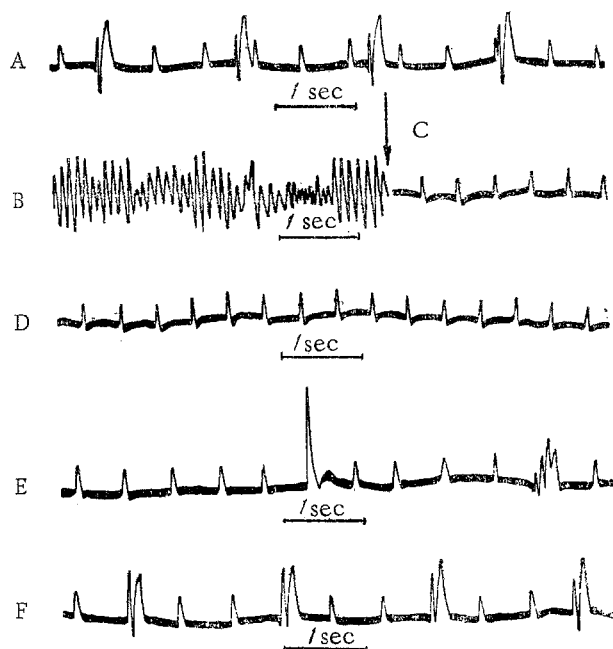


Fig. 2. Restoration of the idioventricular rhythm after defibrillation of the ventricles against the background of complete atrioventricular block: A) initial state; B) ventricular fibrillation lasting 15 sec; C,D) ventricular asystole following defibrillation and lasting 20 sec; E,F) gradual restoration of initial idioventricular rhythm. Interval between tracings D and E, 7 sec.

Different results were obtained after ventricular defibrillation against the background of complete atrioventricular block. In these cases, a temporary ventricular asystole (15-40 sec) developed after defibrillation and preceded the restoration of the idioventricular rhythm. The results obtained in the second part of the same experiment (which is illustrated in Fig. 1) are given in Fig. 2. In the second part of the experiment, complete atrioventricular block was produced before fibrillation; the duration of fibrillation was 15 sec. After it had ceased, ventricular asystole took place for 20 sec, and only then was the initial idioventricular rhythm restored.

The longer the duration of fibrillation, the longer the duration of the ventricular asystole. For example, after the cessation of fibrillation lasting 20 sec, ventricular asystole lasted 15 sec, after fibrillation for 35 sec — 22 sec, after fibrillation for 45 sec — 25 sec, and after fibrillation for 1 min 15 sec the duration of asystole was 27 sec.

In most experiments, the duration of ventricular fibrillation was not allowed to exceed 30 sec; if fibrillation continued for longer (1 min or more) severe anoxic changes could develop in the myocardium as a consequence of the disturbance of the coronary circulation and thus would complicate the interpretation of the results. For control purposes, the effects of defibrillation of the ventricles were compared after fibrillation of equal duration in the conditions of normal conduction in the heart and in the conditions of an experimentally produced complete atrioventricular block. In these circumstances, the effect of anoxia was the same but the course of restoration of the cardiac activity was different.

Comparison of all the experimental results showed that fibrillation gives rise to suppression of the automatic activity of the ventricular pacemakers just as is observed under the influence of a fast rhythm of excitation. After defibrillation of the ventricles of the intact heart, this suppression of the automatic activity is not seen for they immediately begin to contract in the rhythm of impulses arriving from the sino-auricular node, whose automatic activity is not suppressed. In the case of defibrillation against the background of complete atrioventricular block, on the other hand, a preautomatic pause is observed, and the automatic activity of the ventricular pacemakers is only gradually restored.

The results obtained explain the observations [3] that ventricular asystole arises after defibrillation in patients with complete atrioventricular block. They show that when fibrillation develops in such patients, immediately after defibrillation electrical stimulation must be applied to the heart in order to prevent ventricular asystole.

#### LITERATURE CITED

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2. E. B. Bab'skii and L. S. Ul'yaninskii, *Doklady Akad. Nauk SSSR*, 152, 5, 1263 (1963).
3. P. M. Zoll et al., *New Engl. J. Med.*, 254, 727 (1956).

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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.

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