GENESIS OF ARRHYTHMIAS AND MECHANISM OF ELECTRICAL DEFIBRILLATION OF THE HEART

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Atrial arrhythmias were induced in experiments on dogs by electrical stimulation or by local application of aconitine and methacholine to the atrium. The action of the defibrillator discharge on these arrhythmias was studied. The defibrillator discharge abolished the arrhythmias maintained by the circus movement of the excitation wave over the atria but did not abolish sinus tachycardia or ectopic aconitine tachysystoles. The threshold of the defibrillating effect depends on the existence of micro- or macro-reentries. The mechanism of defibrillation consists of excitation of the atrial myocardium with a consequent decrease in the pathway for the circulation of excitation to below the critical size for maintaining the circus movement of the excitation wave. The action of the defibrillator does not inhibit the automatism of the nomotopic and heterotopic cardiac pacemakers.

KEY WORDS: heart – arrhythmias, defibrillation.

Since there are at present two theories of the genesis of arrhythmias (the theory of circus movement of the excitation wave and the theory of heterotopic tachysystole) the existence of two opposite mechanisms of electrical defibrillation must also be postulated.

A single electric pulse can abolish fibrillation, flutter, and tachycardia as a result of synchronous excitation of the cells of the atrial or ventricular myocardium with the consequent abolition of the circulation of excitation over these parts of the heart [3, 4, 8, 16]. Abolition of arrhythmias of different origin by defibrillation can be explained by simultaneous suppression of sources of heterotopic pacemaker activity by the action of a strong current. Finally, the mechanism of defibrillation can be assumed to be the same in all cases and to be independent of the genesis of the arrhythmias.

In order to study these theoretical alternatives the effect of the defibrillator discharge was studied experimentally on atrial arrhythmias induced by the circus movement of excitation or by the presence of an ectopic focus.

EXPERIMENTAL METHOD

Experiments were carried out on 30 dogs weighing 8-15 kg anesthetized with pentobarbital (30 mg/kg, intravenously). Thoracotomy, incision of the pericardium, and exposure of the heart were carried out under mechanical ventillation. Monophasic potentials from various parts of the atria (detected by suction electrodes), the ECG in standard lead II and, in some experiments, the intracavitary electrogram (recorded by means of a bipolar catheter-electrode introduced into the chamber of the right atrium) were recorded on a polygraph. Arrhythmia was induced in five dogs by applying aconitine crystals to the right atrium or to its auricle, followed by electrical stimulation of these structures [18, 19]. Atrial stimulation in this and other methods was carried out in diastole through bipolar electrodes with square pulses with a duration of 2 msec, a frequency of 10-50 Hz, and a voltage 2-5 times above threshold.

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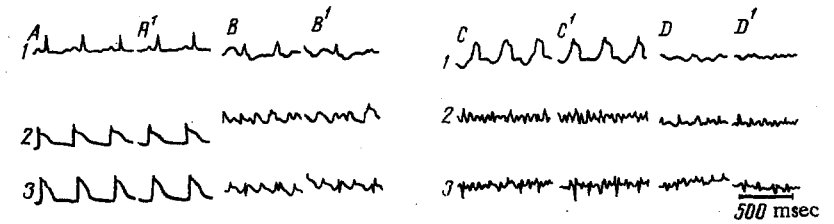

Fig. 1. Action of defibrillator discharge on cardiac electrical activity during arrhythmias induced by quickening automatism of nomotopic and heterotopic pacemakers: 1) ECG; 2) activity of right atrium; 3) of left atrium; A, B, C, D) activity before, and A^1, B^1, C^1, D^1) activity after discharge of 5 kV; A, A^1) sinus tachycardia; B, B^1) artial flutter after application of aconitine; C, C^1) ventricular tachycardia and atrial fibrillation after additional application of aconitine to ventricles; D, D^1) fibrillation of the heart as a result of repeated application of aconitine.

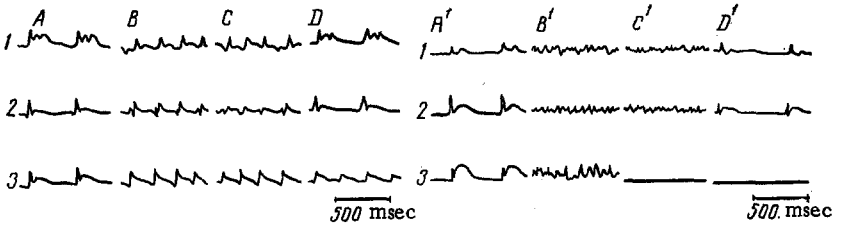

Fig. 2. Comparison of action of defibrillator discharge and local isolation of site of application of aconitine and methacholine on atrial electrical activity: 1) activity of left atrium; 2) of right atrium; 3) of right auricle (site of application of preparations). A, A^1) sinus rhythm before application of substances; B) aconitine flutter; B^1) methacholine fibrillation; C, D^1) activity after discharge of 3 kV; D, C^1) activity after application of clamp to right auricle.

Arrhythmia was produced in 12 dogs by the application of two or three drops of methacholine (solution containing 1 g/ml) to the surface of the atrium, followed by electrical or mechanical stimulation [15].

In four dogs a method of inducing double arrhythmias was used. For this purpose aconitine arrhythmia was produced, after which a clamp was applied to the atrium separating the site of aconitine application (the right auricle) from the rest of the atrial tissue, to which methacholine was applied. When conductivity was disturbed by the clamp, the two arrhythmias existed independently.

In five dogs, after bilateral vagotomy continuous electrical stimulation of the right vagus nerve was carried out with square pulses with a duration of 1 msec, a frequency of 10 Hz, and a voltage of 2 V; against this background arrhythmia was induced by electrical stimulation.

In 13 dogs atrial arrhythmia was induced by brief stimulation of the atria after mechanical destruction of the sino-atrial node [1, 2].

The threshold of atrial defibrillation was measured in the various forms of arrhythmias. The Prema defibrillator was used and its electrodes, 7 cm in diameter, were fixed under the skin at the sides of the chest wall, and for this reason the thoracotomy was carried out as sparingly as possible.

EXPERIMENTAL RESULTS AND DISCUSSION

Under the influence of thoracotomy and barbiturates the frequency of the sinus rhythm reached 100-150 beats/min. In some experiments sinus tachycardia with a frequency of more than 170 beats/min was observed. A defibrillator discharge of 1-6 kV did not abolish the sinus tachycardia but, on the contrary, increased the frequency of the nomotopic rhythm (Fig. 1).

Local application of aconitine induced tachycardia, flutter, and fibrillation of the atria. In the first

two forms of arrhythmias atrial activity with a frequency of 200 to 500 beats/min was observed and the impulse spread over the atria from the site of application of the aconitine. When a clamp was applied to the auricle (the point of application of the aconitine) the sinus rhythm was quickly restored and conducted over the atria, whereas flutter or tachycardia continued in the auricle (Fig. 2). After application of additional aconitine to the ventricles, arrhythmia of the atria and ventricles simultaneously was observed. A defibrillator discharge of 4-6 kV did not abolish the aconitine arrhythmias of either the atria or the ventricles (Figs. 1 and 2).

Against the background of the maximal effect of cholinergic factors (action of methacholine or constant stimulation of vagus nerve for 1-3 min) the automatism of the sino-atrial node was depressed and the monophasic potentials were irregularly shortened. Atrial fibrillation appeared either spontaneously or under the influence of electrical or mechanical stimulation. Application of multiple clamps to the atrium did not abolish the atrial fibrillation but merely abolished the fibrillation locally, for example, in the isolated auricle to which methacholine had previously been applied (Fig. 2). With disappearance of the methacholine effect (after its action for 3-5 min) or discontinuation of vagus nerve stimulation flutter was observed, ceasing spontaneously. After the more localized action of methacholine dissociation of the atrial rhythm was observed: in the right atrium and its auricle, into which the methacholine penetrated, fibrillation was observed, with flutter in the left atrium and auricle; ultimately flutter developed in all parts of the atria. Shortening of the duration of the action potentials (shortening of the refractory period), increased heterogeneity of the atrial tissue, and suppression of automatism under the influence of cholinergic agents are among the most important conditions for the development of micro-reentry (fibrillation) [7, 10, 14]. Abolition of fibrillation of the isolated auricle is evidence that the tissue is too small to maintain this process in accordance with the criterion of critical mass [20]. With a decrease in cholinergic influences (lengthening of action potentials) the micro-entries changed to macro-entries (flutter), one feature of which was the appearance of successive depolarization of different areas of the atria during the period of flutter for 100-200 msec [17]. If a source of pacemaker activity was present (heterotopic, or in the sino-atrial node) this sequence of events could be traced for the first 70 msec after the beginning of the impulse in these pacemakers. The defibrillator discharge abolished all forms of arrhythmias arising under cholinergic influences. The threshold of defibrillation for local and systemic fibrillation was 2-3 kV (mean 2.5 ± 0.15 kV). The threshold voltage for flutter was lower, namely 1.0-1.7 kV (mean 1.2 ± 0.17 kV).

By the use of brief high-frequency stimulation of the atria after destruction of the sino-atrial node flutter could be induced in the dogs and, in the absence of any additional measures, it lasted more than 50 min. The presence of circus movement of the macro-reentry type in this case was indicated by the sequence of depolarization of the atria during the period of flutter and a change in the polarity of the intracavitary atrial electrogram (catheter near the atrio-ventricular node) in two consecutive cycles of flutter. Repeated destruction of the tissues in the search for the sino-atrial node prevented the onset of flutter. The test was thus positive [13] and it accordingly follows that destruction of the pathway or creation of an obstacle in the pathway of macrocirculation prevents its onset and maintenance. The electrical discharge abolished the flutter in 100% of cases. When induced a second time the arrhythmia was indistinguishable in its characteristics and the threshold of defribillation was stable. It varied in different dogs from 0.8 to 1.5 kV (mean 1.1 ± 0.13 kV).

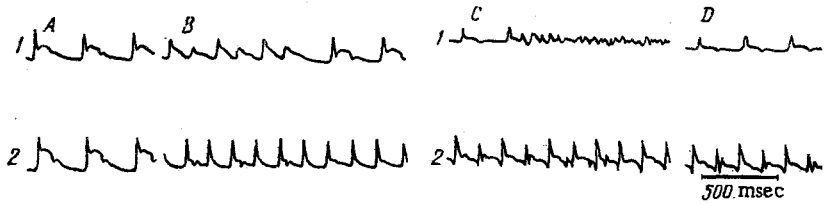

Fig. 3. Atrial electrical activity during induction of double, separate arrhythmias and action of defibrillator discharge: 1) activity of right atrium; 2) of right auricle. A) sinus rhythm; B) effect of clamping auricle, conduction block between right atrium and auricle leads to dissociation of rhythm, sinus rhythm in atrium and flutter in auricle, to which aconitine had previously been applied; C) subsequent application of methacholine to atrium leads to appearance of fibrillation in the atrium but flutter continues in the auricle, dissociation of rhythm as a result of the presence of two arrhythmias; D) action of discharge of 3 kV, defibrillation of atrium and preservation of aconitine flutter in auricle.

By producing separate double arrhythmias, in response to aconitine flutter or tachycardia was observed in the isolated auricle of the dogs, but either fibrillation or flutter in the rest of the atria depending on the time of action of the methacholine. The defibrillator discharge abolished the atrial arrhythmia but did not abolish the arrhythmia of the isolated auricle (Fig. 3). The defibrillation thresholds were similar to those in the experiments with methacholine alone. When application of the clamp did not disturb conduction between the sites of application of methacholine and aconitine, fibrillation of all parts of the atria was observed. The frequency of the fibrillations reached 2000/min. This arrhythmia was not abolished by a dishcarge of 5-6 kV, but intravenous injectionof novocainamide in a dose of 10 mg/kg did abolish the arrhythmia. Novocainamide also abolished both arrhythmias when they existed separately. This effect can be explained by the ability of novocainamide to depress aconitine automatism and also to lengthen the refractory period of the atrial tissue [11].

The results of these experiments indicate that the defibrillator discharge does not suppress the automatism of true and heterotopic pacemakers and does not abolish arrhythmias arising with an increase in the frequency of automatism of these pacemakers. With respect to the automatism of aconitine origin, Antoni [9] reached a similar conclusion in his investigations using a microelectrode technique. It was found both clinically and experimentally that ectopic ventricular tachycardias after toxic doses of digitalis and also after injection of adrenalin not only are not abolished by the discharge but are actually induced by it [5, 6, 12]. The present experiments also showed that the arrhythmia caused by interaction between the circus movement of excitation and an ectopic focus is not abolished by the discharge.

The experimental results showed that the defibrillator discharge abolishes atrial arrhythmias with features only of the circus movement of excitation over the atria and restores a nomotopic rhythm. The threshold of defibrillation depends on the presence of macro-reentry (flutter) or micro-reentry (fibrillation resulting from cholinergic influences). The mechanism of defibrillation consists of excitation of the atrial myocardium with a consequent shortening of the path of circulation of excitation to something below the critical limit for maintenance of the circus movement of the impulse. Accordingly with macro-reentry single excitations are sufficient, but with micro-reentry it is necessary to excite a large number of cells, in order to produce a sufficiently high current density throughout the myocardium of the atria. If the refractory period is short (cholinergic influences) cells must be excited at a short distance in accordance with the criterion of critical mass during fibrillation, whereas if the refractory period is long a single excitation in the path of circulation (flutter) will create a sufficient obstacle for macro-reentry.

The genesis of arrhythmia thus determines the positive or negative effect of the action of the defibrillator discharge; the conditions of existence of micro- and macro-reentry determine the strength of the defibrillator action. The mechanism of defibrillation is the same, excitation of the heart. The method of electrical defibrillation can be used to study the mechanism of maintenance of arrhythmias.

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