Cardiac Arrhythmias Following Condenser Discharges and Their Dependence Upon Strength of Current and Phase of Cardiac Cycle

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With the technical assistance of Z. Blásek, M. Rábl, and E. Sládková and the statistical evaluation of Z. Roth

The effect of electric currents upon cardiac activity has attracted increasing interest in connection with electrical defibrillation of the heart. In many cases resuscitation depends largely upon cardiac massage and ventricular defibrillation, whether the chest is open or closed.\(^1\)\(^-\)\(^12\) Although defibrillation of the exposed heart has been adequately studied theoretically as well as practically\(^13\)\(^-\)\(^19\) there is still no uniform opinion on transthoracic defibrillation.

It is generally agreed that both high voltage and high intensity of current are needed to stop ventricular fibrillation in a closed chest.\(^7\)\(^-\)\(^20\)\(^-\)\(^22\) This puts great demands upon the construction of a suitable apparatus, the output of which must range from tens to hundreds of watts. At present a condenser discharge, which enables us to provide within a short time the quantity of energy necessary to induce defibrillation, seems to be the most satisfactory solution. It also seems to be the only solution for portable defibrillators with their own power source, independent of electrical power lines, the urgent need of which is increasingly apparent in clinical practice as well as in field emergencies.

Defibrillation in a closed chest can be achieved by various types of electrical impulses. Akopyan et al.\(^23\) used a condenser discharge (16-24 \(\mu\)F) up to 6000 volts, Hosler and Wolf\(^5\) an alternating current up to 1000 volts, Kouwenhoven et al.\(^7\) 450-900 volts, Peleška\(^10\) a condenser discharge of 16 \(\mu\)F up to 5000 volts, etc. Such high voltages are capable of damaging the myocardium and the prognosis of resuscitation may thus be adversely affected.

Several authors\(^21\),\(^24\),\(^25\) have attempted to clarify the question of the effect of the high voltage itself, whether of alternating current or condenser discharges, upon the myocardium, but the problem is complicated by lack of agreement as to what strength and type of impulse is most suitable for transthoracic cardiac defibrillation. Our earlier work\(^26\) demonstrated that defibrillation impulses may, under special conditions, cause a certain degree of either morphological or functional damage. Some authors described various degrees of morphological changes ranging from insignificant damage to burns, necroses, and various degenerative changes of individual areas or fibres. Other authors categorically deny the possibility of any damage. The cardiac effects of electric currents of the intensity necessary for defibrillation seem to depend upon the voltage, the density of current per cm\(^2\), the duration of the impulse, the overall energy, the condition of the myocardium and, particularly, upon the presence or absence of repeated applications.

We began to work upon this problem several years ago. Although we were unable to check thoroughly all of the forms of defibrillation impulses employed, we believe that our work portrays the general aspects of the effects of electric currents upon the heart. At the outset we did not know whether damage
to the tissues is caused by the total quantity of electric energy, by the voltage, or by other variables. Therefore we chose a wide range of energy (from 0.06 to 1800 watts), as well as of voltage (from 500 to 6000 volts). We used condensers with capacities of 0.5 to 100 μF, which cover the range of capacities hitherto employed for defibrillation. The calculation of energy for certain values of voltage and capacities is shown in table 1. In the present study we were particularly interested in the pathophysiology of the effect of a high voltage electric current upon the myocardium at different phases of the cardiac cycle, a question which is interesting in relation to sequelae of injuries by electric current and the explanation of their mechanism.

**Methods**

To carry out this work we constructed an apparatus which made it possible to apply a condenser discharge in any phase of the cardiac cycle. Its components are shown in figure 1. The apparatus works on the following principles: The electric potentials of cardiac activity are amplified by the electrocardiograph and led to the discriminator, which passes only the highest amplitude, i.e., the R wave. This amplified R wave is used as the impulse for the trigger and delay circuit which controls the timing of the impulse.

**TABLE 1**

Values of Electric Energy (in watts) Used in Experimental Discharges.

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<thead>
<tr>
<th>kV</th>
<th>0.5 μF</th>
<th>1 μF</th>
<th>2 μF</th>
<th>4 μF</th>
<th>8 μF</th>
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<td>360</td>
<td>432</td>
<td>576</td>
<td>900</td>
<td>1800</td>
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applied to the dog by an electronically controlled switch from the condensers.

The electrocardiograph is protected from damage by a relay connected to the input lead, and is also controlled electronically from the delay circuit which at the same time synchronizes the switching off and on of the electrocardiograph. The apparatus is put into operation by the trigger circuit which, after releasing the impulse, blocks the further activity of the apparatus from accidental impulses. The application of the impulse in the pre-set phase of the cardiac cycle is checked by the electrocardioscope and recorded by the electrocardiograph during the experiment.

By means of this apparatus we studied the effect of condenser discharges upon cardiac activity in the three phases shown in figure 2, i.e., I, absolute refractory period (ARP); II, relative refractory period (RRP); III, period of excitability (EP). The designations of the phases are the same for figure 5.

We carried out the experiments on mongrel dogs (20 to 25 kg) because the cardiac physiology of these animals closely resembles that of man, according to our own experience and to that of others. The dogs were anesthetized with thiopental intravenously, repeated as required during the entire experiment. The standard electrocardiographic leads of the extremities were recorded on the Mingograph 42.

The electrodes for the application of the condenser discharge measured 15-17 cm². They were placed on opposite sides of the thorax, at sites previously shaved and treated with electrocardiographic paste. Blood pressure was recorded directly from a femoral artery. At the beginning of the experiment an electrocardiogram was made and by a test discharge into an artificial load we set the parameters of the delay circuit so that the discharge would be applied to the chosen phase of the cardiac cycle. The discharge was applied to the dog only after the delay circuit was accurately set. About 12 discharges were tested in every dog, beginning with discharges of high energy which were then gradually lowered. Thus the dog survived a greater number of experimental discharges. After every discharge there was an interval of 5-15 minutes to allow the cardiac rhythm to recover.

When changes of cardiac rhythm of a permanent character or of long duration occurred, we discontinued the experiment. We also discarded those dogs in which ventricular fibrillation occurred after the discharge. In every case of fibrillation we carried out and evaluated statistically a total of 2160 discharges in 240 dogs.

Method of Evaluation

Changes of cardiac rhythm were classified from electrocardiographic recordings made during and after the experimental discharge. The recorded arrhythmias were divided according to their severity into five groups, as follows: (1) no changes of rhythm following the discharge, with regular sinus rhythm immediately afterward (fig. 3A); (2) mild arrhythmias of brief duration, e.g., several extrasystoles, temporary tachycardia or bradycardia (fig. 3B); (3) moderate but fully reversible arrhythmias involving longer lasting changes of rhythm such as bigeminy, ventricular extrasystoles, idioventricular rhythm, ventricular tachycardia, bradycardia, atrioventricular dissociation, etc. (fig. 3C); (4) severe changes of rhythm (fig. 4D) such as marked electrocardiographic deformations and ventricular rhythm lasting up to several minutes, which usually caused a decrease of blood pressure, (5) severe arrhythmias changing into ventricular fibrillation. Figure 4E shows such an arrhythmia changing after approximately two contractions into fibrillation. In the majority of cases, however, the transition to fibrillation required several seconds up to several minutes, but in some instances (fig. 4F) ventricular fibrillation occurred immediately after application of a condenser discharge.

Results

The individual results are summarized in figure 5. For evaluation we always took five values from different experimental animals in one column with identical parameters of voltage and capacity. For each phase of the cardiac cycle we obtained and evaluated 720 experimental discharges. We are presenting this figure to draw attention to two types of
FIGURE 3
Electrocardiogram and blood pressure record before and after condenser discharge. A. No significant change, no arrhythmia. B. Several interpolated extrasystoles, example of slight arrhythmia. C. Ventricular rhythm and bigeminy evaluated as moderate arrhythmia.
FIGURE 4

Electrocardiogram and blood pressure record before and after condenser discharge.
D. Marked electrocardiographic abnormality, ventricular rhythm, alteration of blood pressure following discharge; evaluated as severe changes. E. Ventricular rhythm changing after several contractions into fibrillation. F. Ventricular fibrillation beginning immediately after discharge.
mechanism involved in fibrillation. In one type, fibrillation sets in with relatively low voltages (about 1 kv) and is of a benign character, easily restored to normal. The other type follows high voltages (over 5 kv) and is more serious because it is often associated

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CARDIAC ARRHYTHMIAS

Statistical evaluation of 720 experimental discharges and their effect upon cardiac rhythm in the ARP. I: area without arrhythmias. II: area of slight arrhythmias. III: area of moderate arrhythmias. IV: area of severe arrhythmias. V: area of fibrillation or changes of severe arrhythmia into fibrillation.

with inhibition of the electrical activity of the conduction systems and with morphologically demonstrable damage to the myocardium. These morphological changes will be described in another paper.

The results of our experiments were statistically evaluated and expressed in graphs by Z. Roth. Figure 6 shows the results of condenser discharges applied in the absolute refractory period (ARP). The vertical axis shows \( kv \) linearly, the horizontal axis \( \mu F \) in a logarithmic progression. The individual curves limit the areas of different degrees of functional damage of the myocardium. This division was obtained by statistical methods, i.e., by calculating for each capacity (Körber's method for LD-50), a voltage at which 50% of the animals had arrhythmias more severe than one point, e.g., one and two points; or one, two and three points, etc. The term "point" is explained in the following paragraph. Up to four values were thus obtained for each capacity and these, when connected by a continuous line in the figure, gave five different areas in which there is always one dominating type of arrhythmia. Area I is without arrhythmias. Area II includes slight arrhythmias and Area III moderate arrhythmias. An important part of the figure is the area of severe arrhythmias, area IV, and the smallest is area V, i.e., ventricular fibrillation. Figure 7 demonstrates a corresponding statistical evaluation of discharges applied in the relative refractory period (RRP) and figure 8 shows the results in the period of excitability (EP).

RELATION OF PHASE OF CARDIAC CYCLE TO THE EFFECTS OF STIMULUS

We first tested the hypothesis that the arrhythmia produced by a condenser discharge is unrelated to the phase at which it is applied. For this purpose a response without arrhythmia was given one point, slight arrhythmia two points, moderate arrhythmia three points, severe arrhythmia four points, and fibrillation five points. For each group a score of points was calculated. Decision depended upon the score of points that was the
Statistical evaluation of 720 experimental discharges and their effect upon cardiac rhythm in the RRP. I: area without arrhythmias. II: area of slight arrhythmias. III: area of moderate arrhythmias. IV: area of severe arrhythmias. V: area of fibrillation or changes of severe arrhythmia into fibrillation.

Statistical evaluation of 720 experimental discharges and their effect upon cardiac rhythm in the EP. I: area without arrhythmias. II: area of slight arrhythmias. III: area of moderate arrhythmias. IV: area of severe arrhythmias. V: area of fibrillation or changes of severe arrhythmia into fibrillation.
CARDIAC ARRHYTHMIAS

Statistical evaluation of 2160 experimental discharges and their effect upon cardiac rhythm regardless of the phase of the cardiac cycle. I: area without arrhythmias. II: area of slight arrhythmias. III: area of moderate arrhythmias. IV: area of severe arrhythmias. V: area of fibrillation or changes of severe arrhythmias into fibrillation. The curves in the figure intersect points of identical energies (25, 50, 100, and 150 watts) with various voltage and capacity values.

Discussion

Arrhythmias produced by the effect of electric current upon the myocardium depend upon two factors. The first is the height of absolute voltage. This dependence is much more pronounced than the second one, the total quantity of energy acting upon the heart. Both these relations are illustrated in figure 9 which is a statistical evaluation of all three phases of the cardiac cycle, obtained by the process just described.

If we trace a series of curves in this figure connecting the points of identical energies, we find that the same curve intersects almost all grades of arrhythmia, from severe to none at all. If the degree of functional damage to cardiac activity is approximately parallel to the degree of morphological damage, we can derive a clear idea of the proper parameters for the impulses of condenser defibrillators. Morphological studies made during these experiments indicate that the functional changes are indeed parallel to morphologically demonstrable changes in the myocardium. It follows that the defibrillation impulse should have parameters which will avoid all but the least functional damage to rhythm.

Our studies also cast some light on the dynamics and the sequelae associated with cardiac arrhythmias. Injuries by electric current, particularly the frequent accidents caused by undischarged condensers when repairing transmitters or other high voltage sources either in laboratories or industrial plants, have characteristic time courses. As demonstrated in figure 5, two kinds of ven-

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tricular fibrillation may be caused by condenser discharges. One of these, sudden fibrillation, occurs most frequently with lower voltages and lower energies, as shown in the upper half of figure 5. According to our experience, such fibrillations can easily be stopped by one adequate defibrillation impulse. Usually there is no subsequent recurrence of fibrillation.

The other type of fibrillation caused by the application of high voltage (5-6 kv) and high energies (300 watts) is more serious. In such cases there are marked changes of rhythm, such as bradycardia, even total inhibition of the electric activity of the heart and severe ventricular tachycardia, which often precedes fibrillation. In many of these cases we observed, immediately after a high energy discharge, a normal sinus rhythm which, after several seconds, was interspersed with supraventricular or ventricular extrasystoles. Gradually thereafter a picture of severe arrhythmia developed, ending within one or more minutes in ventricular fibrillation. We observed 28 such cases of transition of severe arrhythmias into fibrillation.

After some discharges of great intensity we could still record the electrical activity of the heart but mechanical contractions were weak or absent, and blood pressure was low or not recordable. These types of heart failure are reminiscent of some of the fatal cases after injury with electric current which were encountered by us and by others.20–34

Our findings indicate that the appearance of fibrillation following a condenser discharge is most likely when it occurs in the phase of the cardiac cycle corresponding with the T wave. In our cases we observed the development of fibrillation 56 times in the RRP, 37 times in the ARP, and only 15 times in the EP. Our finding that the relative refractory period of the cardiac cycle is most dangerous for the development of fibrillation confirms the conclusions drawn by Kouwenhoven et al.,24, 35 Milnor et al.,25 Mackay, Leeds21 and others. The period of excitability is the least favorable for fibrillation. The damage to cardiac rhythm at this time is smaller than in either of the two former phases.

We should like to add that in constructing condenser defibrillators induction coils of various types are sometimes incorporated in the discharge circuit. This inductance changes the shape and the duration of the condenser discharge and considerably lowers the voltage delivered. The electrical resistance of the coil also reduces the energy of the impulse as applied to the heart. The intensity of arrhythmia caused by a condenser discharge is different if there is an inductance connected to the circuit. These questions will be considered in more detail elsewhere.

Summary

To improve technics for electrical defibrillation of the heart, an apparatus was constructed for the application of a condenser discharge at any selected phase of the cardiac cycle. With this apparatus 2160 discharges were applied through the intact chests of 240 dogs under thiopental anesthesia. The voltage ranged from 0.5 to 6 kv, the condenser capacity from 0.5 to 100 μF, the total energy from 0.06 to 1800 watts. The associated changes in cardiac rhythm were graded from one (no abnormalities) through two (mild, brief extrasystoles), three (more protracted but fully reversible periods of bigeminy, extrasystoles, block, etc.), four (severe but reversible changes lasting several minutes, usually with fall in blood pressure), to five (severe arrhythmias changing into ventricular fibrillation).

The severity of the arrhythmias increased progressively as the voltage was increased, and to a much smaller extent with increases in the total amount of electrical energy. Arrhythmias also were significantly more severe when the shock was delivered during the refractory period (S-T interval) than during the excitable (P interval) stage of the cardiac cycle. Two types of ventricular fibrillation of very different prognosis were revealed. One came on suddenly after low voltage shocks and could easily be terminated by a single adequate defibrillation impulse. The
other came on more gradually after high volt-
ages and energies and tended to be irrever-
sible.

Observations thus far made indicate that the severity of the arrhythmia following con-
denser shocks gives an approximate indication of the severity of pathological changes in the heart. Excessive currents could damage the heart to such an extent that myocardial con-
tractions were weak or absent although elec-
trical activity persisted. Therefore the pa-
rameters of condenser discharges used for clinical defibrillation should be such that they will produce only the mildest and briefest arrhythmias.

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