RESTORATION OF REGULAR RHYTHM IN THE MAMMALIAN FIBRILLATING HEART*

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IT IS WELL KNOWN that under the influence of a strong tetanic electrical stimulus, the normal activity of the ventricles of the heart of warm-blooded animals may cease and ventricular fibrillation appear (Hoffa and Ludwig; Prevost and Battelli). The latter found that fibrillation in dogs and adult cats, brought on by a sinusoidal current, do not stop spontaneously, and that death follows arrest of the circulation. Regular rhythm may return, however, under the influence of a stronger sinusoidal current. A dog with a closed thoracic cage requires a current of 1200-4800 volts, but with direct application of one of the electrodes to the heart, 240 volts are sufficient.

At the suggestion of L. S. Stern, we studied the mechanism for restoring normal activity of the mammalian heart after fibrillation was produced by a strong sinusoidal current of low frequency. We used a condenser discharge through electrodes, placed usually upon the body of the dogs or cats with unopened thoracic cage. Repeated experiments could be conducted on the same animal for several days. The recorded blood pressure and electrocardiogram served as criteria of restitution of cardiac action. More than 40 experiments were conducted on dogs. Preliminary experiments were made on cats, which have the advantage of possible spontaneous arrest of ventricular fibrillation with restitution of regular sinus rhythm.

METHODS

Oval metallic electrodes were placed upon the closely shaved thoracic skin—the active electrode, 6 cm. square, on the apical region, the indifferent electrode, 30 cm. square, usually in the right subscapular area. These electrodes served first to transmit a sinusoidal current of low frequency (50 cycles from the city lighting current), which produced ventricular fibrillation, and secondly to transmit a condenser discharge with a potential difference of 3000-3600 volts and a capacity

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of 3 mf. The condensers were charged either from a rectifier, fed by a transformer (600 volt capacity) or from a number of anodal batteries (600 volt) connected in series. In either case a rise in potential difference of the charged condensers followed their connection in series. Our scheme permitted a quick change of the condensers from parallel linkage at the moment of charging, to connection in series at the time of discharge.

Observations in the first series of experiments consisted in recording the blood pressure (femoral artery) and in the second series (dogs) in recording lead 3 of the electrocardiogram by the Boullitte string galvanometer. The electrocardiogram of each dog was registered at the beginning of the experiment for control, after the action of the sinusoidal current to register the ensuing ventricular fibrillation, and after the condenser discharge to visualize the return of normal heart action. An automatic and bipolar switch excluded the string of the electrocardiograph at the moment of discharge of the sinusoidal current or the condenser in the circuit.

These electric stimuli produce an intense (more than 90 millivolts) and prolonged skin current, which swings the string out of the illumination field. Compensation for this changing current is extremely difficult. Hence, in the majority of experiments, we included a condenser with a large capacity in the circuit of the string (after Kremer). Although the condenser somewhat altered the form of the electrocardiographic waves, it did not interfere with our work.

EXPERIMENTAL RESULTS

1. A sinusoidal current of low frequency was passed for one-fifth second through the thoracic cage of a dog or cat. It carried 15-40 volts, depending upon the size and weight of the animal. Ventricular fibrillation resulted as registered by the blood pressure and electrocardiogram. If the duration of the current is less, this does not occur, as previous investigators, Prevost and Battelli, and Yuniev and Gerchikova, have shown. In occasional cases ventricular fibrillation arose from weaker currents. In an old dog weighing only 5 kg., a 4-volt current sufficed. If the duration of current is longer, lower voltage is needed to produce this ventricular arrhythmia. In one experiment with gradual increase to 6 volts, fibrillation began at the fifteenth second. Short duration of current for 0.5 second caused ventricular fibrillation in this animal with only 25-30 volts. For cats and young dogs up to one year of age a much higher voltage than expected for size and weight is needed to produce fibrillation.

In the electrocardiogram ventricular fibrillation is shown by a characteristic picture of continuous waves with a rate of 500-600 per second, like those recorded in similar experiments of Yuniev and Gerchikova.

2. A single discharge of a condenser (3 mf.) through the thoracic cage, with occasional exceptions, may stop the ventricular fibrillation of the unexposed hearts of dogs and cats, if the discharge occurs not later than 25-30 seconds after the onset

of the fibrillation. Occasionally discharges of the condenser are effective after 45-50 seconds and in exceptional cases even after one minute or more. The required potential differences depend upon the size and weight of the animal.

In small dogs, 5-6 kg., (2 experiments), fibrillation could be abolished at 2000 volts; for 6-12 kg. dogs, 3000-3600 volts were required. If the dogs weighed more than 12 kg. this potential difference did not stop the fibrillation. If the discharge occurs within 30 seconds of the onset of the fibrillation, a marked rise in blood pressure occurs, higher than the control level, and a temporary tachycardia results. When the condenser discharge occurred later—40-50 seconds after the onset of the fibrillation, and, hence, at the time of anoxia of the animal, and was still successful in restoring normal rhythm, the blood pressure rose more slowly, with a slow heart rate. Consequently the effectiveness of the condenser discharge depends upon (1) the potential difference and capacity of the condensers, (2) the interval between the onset of the fibrillation and the moment of condenser discharge.

When the delayed condenser discharge did not stop fibrillation, it was possible occasionally to revive the animals by cardiac massage, produced by rhythmic pressure upon the thoracic cage for five or more minutes. We could repeatedly produce fibrillation and stop it by a condenser discharge in one and the same dog. After the ligation of the femoral artery and suture of the wound such dogs showed no signs of cardiac abnormalities. The electrocardiogram of the revived heart is comparable to the control registered before onset of fibrillation.

Control experiments showed the influence of single condenser discharges upon the normal unexposed heart, not previously influenced by the sinusoidal current. It was found that a condenser discharge, even with a high potential difference of 3600 to 5000 volts, does not produce fibrillation or other abnormalities of the heart action in dogs. Only a temporary fall in blood pressure, followed by a rise, and transient tachycardia, were observed.

DISCUSSION

1. Momentary electrical stimulation of the fibrillating heart by a condenser discharge abolishes fibrillation. This may be explained by the supposition of Lewis that in the fibrillating state the myocardial excitation wave repeatedly follows a closed circus pathway. Such a circus rhythm, as is well known, was experimentally produced in the heart of the turtle by Mines, and thoroughly investigated electrocardiographically by Samoiloff. Vetokhin in 1927 demonstrated the circus rhythm in a preparation cut from the ring of the medusa. He confirmed the experiments of Meyer in 1908. The arrest of fibrillation under a single stimulus corresponds with the observation of Samoiloff who noted that circus rhythm in the turtle heart stops as a result of an induced extrasystole. The circus rhythm in the preparation from the ring of the medusa also stopped, in Vetokhin's experiments, after a single stimulus.

2. The momentary electrical excitation from a condenser discharge and the lasting but short excitation from a sinusoidal current, in certain intensities, possess a

similar physiologic effect, in the sense of restoring normal action in a fibrillating heart. The same factors act upon the heart of the frog (Yuniev, Gerchikova, and Nikolskaya, Yuniev and Mershchikov; 1937, 1938), and upon the skeletal musculature (Battelli and Stern; Stern and Battelli).

3. There are also substantial differences between the two modes of electrical excitation. In our experiments the condenser discharge did not produce ventricular fibrillation in any of the strengths up to 5000 volts. Likewise no signs of heart block were observed. On the other hand, the sinusoidal current easily produced in warm-blooded animals not only fibrillation, but not infrequently block, as previously registered electrocardiographically by Yuniev and Gerchikova. Furthermore, in the frog heart, block is very often elicited by the sinusoidal current, but almost never following a single intense stimulus (Yuniev and Mershchikov).

4. For experimental restoration of the fibrillating heart condenser excitation seems to be preferable to the use of the high intensity sinusoidal current. (a) The latter may produce paralysis of the respiratory center and general depression of the central nervous system resembling "electronarcosis" (Stern and Battelli, Tolmasskaya), and also a severe and prolonged contraction of the skeletal muscles; (b) the sinusoidal current of high intensity, in the presence of occasional technical defects, may cause ventricular fibrillation, because of the drop in potential difference between the electrodes placed upon the body.

The possibility of applying this electrical method for abolishing fibrillation caused by other conditions of clinical importance is of considerable interest. This question requires further study.

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