Susceptibility to Defibrillation of the Hypoxic Heart

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Susceptibility to defibrillation of the hypoxic dog heart was investigated. As a whole heart without influences of innervation and endocrine function and without work by contractions, Langendorff’s fibrillating dog heart preparations with cross circulation were used. Electrical fibrillation-defibrillation sequences were performed after coronary occlusion with ECG monitoring obtaining the following results.

Despite linearly progressive increase of myocardial hypoxia, susceptibility to defibrillation did not decrease proportionally but did recover to some extent as myocardial hypoxia progressed. This is analyzed that, contrary to impression, the severely hypoxic heart (C stage) was more susceptible to defibrillation than the moderately hypoxic one (B stage) in spite of progressing of suggestive myocardial damage. Of course, defibrillation could be more easily established in less hypoxic stage (A) than in moderately one (B). The anoxic heart (D stage) was refractory to any electrical stimuli including artificial pacemaker stimulation. ECG changes of defibrillation in above noticed 4 stages (A, B, C, and D) were illustrated.

SINCE capacitor discharge type defibrillator has been put into practice, the method of defibrillation has been reviewed by many investigators. Nevertheless it is still clinically noted on several occasions that the ventricular fibrillation cannot be resuscitated by either DC (direct current) or AC (alternating current) countershock in various pathological conditions. Although a lot of studies on comparison of DC countershock with AC countershock were reported, it is as yet in question which method is more effective for defibrillation except for the fact that DC countershock has less side effects. Difference of defibrillation efficacy between mono- and multipulse is also in question up to present. Therefore, it is considered that in order to obtain better success of defibrillation, fundamental study of susceptibility to defibrillation of the heart itself should be more important rather than improvement of the methods.

Although the fibrillation threshold of the hypoxic heart was reported by Wiggers and it might be considered that the threshold of fibrillation differed
from that of defibrillation, few research work on the defibrillation threshold has been performed. In this report the changes of susceptibility to defibrillation of the heart was investigated with "the percent defibrillation curve" on the canine heart. The experiments were in a series fibrillation-defibrillation sequences at several levels of myocardial hypoxia.

**MATERIALS AND METHODS**

Fifteen isolated Langendorff's preparations of the dog hearts$^{12}$ weighing 98 to 104 Gm. were used, and the coronary blood supply of the preparations was maintained by cross circulation$^{13}$ with donor dogs of 17 to 20 Kg. anesthetized with intravenous sodium pentobarbital. To maintain the preparation at normal temperature of 35° to 36°C, the thermoelectric heat exchanger$^{14}$ was applied in the circuit. Constant coronary perfusion, at about 80 ml./min. of blood flow and 70 mm.Hg of pressure, was continued (Fig. 1).

To induce artificial ventricular fibrillation, 9 volts—50 cycles alternating current generated by a blocking oscillator was applied to the myocardium of the preparations for a short period. To terminate the induced fibrillation, a capacitor discharge type defibrillator, 400 volts and 10 μF, was used. One discharge of the capacitor gave a power of 0.8 watt-sec. to the myocardium. Two metallic disc electrode paddles were directly attached to the right and left ventricular walls.

A course of each experiment with increasing hypoxic level of the myocardium consisted of the following procedures. At the beginning, constancy of the coronary inflow blood and that of the outflow was measured in order to maintain the conditions of the heart preparation as constant as possible before coronary occlusion. The preocclusion conditions were as follows: the coronary blood flow, 68 to 96 (average 79) ml./min.; the inflow blood pH, 7.356 to 7.520 (average 7.432); the
outflow blood pH, 7.330 to 7.425 (average 7.342); the inflow blood Po2, 373 to 466 (average 398) mm.Hg; the outflow blood Po2, 36.1 to 52.9 (average 44.6) mm.Hg; the inflow blood Pco2, 19.2 to 25.8 (average 23.4) mm.Hg; the outflow blood Pco2, 26.2 to 37.4 (average 30.2) mm.Hg. Then the coronary inflow was interrupted and, as soon as possible, the fibrillator was switched on to induce ventricular fibrillation. After fibrillation lasting 30 sec., the DC shock was applied to defibrillate. At this time, it was determined whether or not defibrillation was successful by ECG. When it was successful, fibrillation was again induced as soon as possible. When defibrillation was not successful, another defibrillation was tried 30 sec. later. These procedures were repeated until the myocardium became irresponsive to any stimuli. The coronary occlusion was then released. Results of defibrillations, ECG changes were recorded.

Fig. 2. ECG changes of defibrillation showing different examples in the four stages (II lead in the supine position).
RESULTS

Susceptibility to defibrillation did not decrease linearly, but did recover to some extent as the myocardial hypoxic level increased. ECG changes of defibrillation seemed to indicate 4 stages of defibrillation, which were named A, B, C and D (Fig. 2).

In stage A, the fibrillation was terminated spontaneously or by DC countershock without any remarkable change of QRS complex.

In stage B, the fibrillation remained persistently despite countershock. In stage C, the fibrillation was terminated spontaneously or by countershock, followed by asystolic stage and then the heart preparation began spontaneous contraction. Several abnormalities were observed on ECG, such as aberrant QRS complex, atrioventricular conduction block and ventricular tachycardia. At this stage, even if asystolic state continued, the myocardium was still responsive to artificial pacemaker or some other stimuli.

In stage D, a standstill state continued, or fibrillation could not be induced any more. At this stage, the myocardium did not respond to any artificial stimuli.

These A, B, C, and D stages appeared in this order with few exceptions, one after another in one course of fibrillation-defibrillation sequence. The "percent defibrillation curve" shows transition of percentage of 4 defibrillation stages (Fig. 3): the duration of coronary occlusion is along the

![Graph showing cumulative percent defibrillation curves](Fig. 3. Cumulative "percent defibrillation curves": Change of susceptibility to defibrillation in progressive myocardial hypoxia. Each line of 4 stages showing percent of each stage that appeared in duration of hypoxia.)
abscissa and the percentage of each defibrillation stage is plotted along the ordinate.

Because the A and C stage are the states in which ventricular fibrillation can be terminated the doubled line which is made by the sum of A and C represents the percent success of defibrillation. In other words, it indicates the curve of susceptibility of the heart to defibrillation.

**DISCUSSION**

The nature of fibrillation is regarded as nonuniformity of the electrophysiological properties of myocardial fibers. From this point of view, electrophysiological study on a single myocardial fiber is not adequate for the study of defibrillation and therefore, the experimental material for defibrillation must be a mass of the myocardial fibers. As a mass of the myocardial fibers electrophysiologicaly excited or not excited, a strip of myocardium may be used, but, for the purpose of clinical application, investigation on the whole heart preparation should be better. The influence of work by contraction, however, confuses the results, and therefore it has to be avoided. Fibrillating heart preparation should be adequate to study the susceptibility of the heart to DC defibrillation from electrophysiological aspect.

The isolated Lagendorff’s preparation of dog heart is convenient to eliminate innervation and endocrine influences. Cross circulation is necessary to avoid influences of accumulating metabolic substances as well as to supply blood to the coronary artery of the preparation.

Each fibrillation-defibrillation sequence of the preparation was done by electrical procedures which has less side effects on the electrophysiological property of the myocardium.

In a process of fibrillation-defibrillation sequence along the gradually increasing hypoxia of the myocardium, the 4 stages of defibrillation appeared in succession. The “percent defibrillation curve” was proposed by the author to manifest the changes of susceptibility to defibrillation. The duration of coronary occlusion represented the hypoxic levels of the myocardium on the abscissa. The percentage of one defibrillation stage at each level of myocardial hypoxia was plotted along the ordinate. It is considered that the percentage indicated a quantitative rate of each stage that appeared.

The nature of each defibrillation stage was speculated as follows:

In stage A, DC current stimulus brings all elements of the myocardium to a uniform state and normal sinus rhythm appears after the shock current ceases. If changes in refractoriness, excitability and conductivity of the heart are little, the uniform state of these properties is spontaneously established.
and termination of the fibrillation can be followed.

In stage B, changes of the electrophysiological properties become less uniform, therefore, the fibrillation remains persistently despite countershock.

In stage C, changes of the properties proceed more severely in themselves, but the relative difference in various parts of the myocardium becomes less. Therefore, defibrillation is more easily established in the C stage than in the B stage. But the decrease in conduction velocity results in the QRS complex and aberrant A-V conduction block and the reduction of excitability may result in the transient asystole.

In stage D, excitability of the myocardium is deeply decreased and it becomes irreversible to any stimuli.

In this paper, susceptibility to defibrillation is only electrophysiologically discussed, but the hemodynamic improvement after defibrillation in the C stage, for instance, still remains in question. The difference of the susceptibility to defibrillation between the heart preparation separated from normal circulation and the heart in normal circulation should be further studied.

The influences of electrolytes, acid-base balance and catechol amines in the myocardium to defibrillation remain unsolved.

CONCLUSION

Susceptibility of the dog heart to DC defibrillation was investigated electrophysiologically using the Langendorff’s fibrillating dog heart preparation with cross circulation in several levels of myocardial hypoxia.

Four stages of defibrillation during complete coronary occlusion were identified. It may be concluded that, despite linear increase of myocardial hypoxia, susceptibility to defibrillation does not decrease proportionally. The susceptibility decreases in the certain early period after coronary occlusion and then it recovers to some extent as myocardial hypoxia progresses.

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