Patterns of Ventricular Activity During Catheter Defibrillation

By Morton M. Mower, M.D., M. Mirowski, M.D., Joseph F. Spear, Ph.D., and E. Neil Moore, D.V.M., Ph.D.

SUMMARY

In order to clarify the mechanism of ventricular catheter defibrillation in which the electrode distribution and the low energy requirements make a simultaneous depolarization of the entire myocardium unlikely, the electrocardiograms recorded during 120 catheter fibrillation-defibrillation episodes in 39 dogs were analyzed. Three distinct, equally distributed defibrillation patterns were observed: 1) immediate resumption of a coordinated rhythm, thought to reflect complete depolarization of the myocardium; 2) increasing coarsening of the fibrillation waveforms interpreted as progressive reduction in the number of fibrillating fibers with reversion when a critical mass of myocardium with synchronized activity is reached, and 3) production of more coordinated “flutter-like” ventricular complexes probably representing a rhythm distinct from fibrillation and convertible to sinus rhythm by a second subthreshold shock. These observations suggest that total depolarization of the entire myocardium is not a prerequisite for ventricular defibrillation.

Additional Indexing Words:
Low-energy defibrillation
Catheter countershock
Critical mass for ventricular defibrillation
Multiple mechanisms of defibrillation

Electrical conversion of ventricular fibrillation to normal rhythm has been traditionally explained by simultaneous depolarization of all ventricular fibers, thus allowing the pacemaker with the highest degree of automaticity to assume control of the heart. \(^1\) Although experimental evidence supporting such a hypothesis is still lacking, the concept satisfactorily explains defibrillation by means of transthoracic and internal paddles. The recent finding that ventricular defibrillation can also be accomplished with low-energy catheter-delivered countershock\(^3\) suggests, however, that additional mechanisms capable of terminating this arrhythmia may exist. Indeed, the very nature of catheter defibrillation appears to preclude creation of an electrical field large and strong enough to simultaneously depolarize the entire heart. The purpose of the present study was to clarify the nature of ventricular catheter defibrillation and, by inference, that of ventricular fibrillation itself, by an analysis of the electrocardiographic waveforms observed immediately following the countershock delivery.

Material and Methods

Electrocardiographic tracings recorded during 120 catheter fibrillation-defibrillation episodes, performed in 39 closed-chest mongrel dogs, were reviewed. In addition, the characteristics of ventricular movement during catheter defibrillation were studied in three open-chest mongrel dogs by means of motion pictures.

The animals weighed between 12 and 25 kg. Pentobarbital anesthesia was induced with an initial dose of 0.5 cc per kg intravenously and maintained with additional 1–2 cc doses intraperitoneally, as required. The animals were intubated and the three open-chest dogs were also artificially ventilated.

A specially built No. 11 French bipolar catheter (fig. 1) was introduced under fluoroscopic control through a jugular vein into the heart. This catheter contained two sets of interconnected platinum rings, each set forming one defibrillating electrode. The distal defibrillating electrode was located at the tip of the catheter, while the second electrode was 12.5 cm farther proximally.

Two electrode positions were used for defibrillation (fig. 2). In the first, the distal electrode was wedged into the right ventricular apex, the proximal being located in the superior vena cava (panel A). In the other position, the distal electrode was in the pulmonary artery, the proximal one then being in the right ventricular cavity (panel B).
CATHETER DEFIBRILLATION

Ventricular fibrillation was induced with a 2–3 milliamperere alternating current passed through the defibrillating catheter. After a period of more than one minute, during which the presence of true fibrillation was verified from the electrocardiographic tracings in the closed-chest dogs and by direct visualization in the open-chest dogs, defibrillation was performed by delivering a countershock through the two intravascular electrodes. The energy source was a specially-built defibrillator, delivering accurately calibrated 5, 10, and 15 watt/second pulses.5, 11 A truncated exponential discharge,7 4 msec in duration, was used for defibrillation, with energy levels of 5 watt-seconds in most of the episodes. During the procedure, an electrocardiographic tracing (lead II) was continuously recorded at a speed of 25/mm/sec.

The open-chest animals underwent a sternal splitting procedure, the pericardium then being opened and sutured to the incision line. Sixteen millimeter motion pictures were made at a speed of 27 frames/second. Frames clearly showing sequential displacements of the ventricular apex immediately following the countershocks were selected for analysis.

Results

The review of the electrocardiographic tracings recorded during catheter defibrillation revealed three distinctive patterns of reversion to sinus rhythm. The incidence of these respective patterns was virtually equal among the material studied, and there was no correlation between body size and a particular pattern of defibrillation.

The first pattern was characterized by an immediate abolition of ventricular fibrillation following the delivery of the countershock. A sinus rhythm was promptly resumed, although the reversion was at times preceded by a brief period of asystole or a few ectopic beats. This sequence of events (pattern 1) is exemplified in figure 3.

The second mode of defibrillation was usually observed during the successive delivery of shocks of subthreshold intensity. Each of these shocks, although ineffective in itself, resulted in coarsening of the fibrillation pattern for a short time, then in a return to the initial fibrillation configuration. At a certain point, usually following the delivery of 3 to 4 shocks of similar or progressively increasing intensities, the coarsening increased even further for approximately 0.5 sec before then converting into a coordinated rhythm. This pattern was particularly frequent when the distal electrode was located in the pulmonary artery. An example of this mode of defibrillation (pattern 2) is shown in figure 4.

In the third pattern, the typical ventricular fibrillation waveform was suddenly replaced following the countershock by regular, rapid and high amplitude complexes. This more stable new rhythm lasted up to 30 sec before reverting back to fibrillation. However, a second low-energy shock, delivered while this intermediate rhythm was present, was consistently able to restore sinus rhythm (fig. 5).

The review of the motion pictures identified six photographic sequences of catheter defibrillation suitable for analysis. This review confirmed the presence of more than one mode of defibrillation. Three photographic sequences showed a direct resumption of a coordinate rhythm following the

Figure 1
Bipolar No. 11 French defibrillating catheter containing two sets of inter-connected platinum rings 12.5 cm apart.

Figure 2
Panel A) Defibrillation position with distal electrode in the right ventricular apex and the proximal electrode in the superior vena cava. Panel B) Defibrillation position with distal electrode advanced into the pulmonary artery and the proximal electrode in the right ventricle.

Figure 3
Prompt resumption of a coordinated rhythm. In this example, a few ectopic ventricular beats are followed by sinus rhythm. 5 WS = 5 watt-sec countershock.
countershock. The three other sequences showed a violent whipping motion of the apex for approximately 0.5 sec before sinus rhythm resumed.

**Discussion**

Electrical shocks applied through an intracardiac catheter have recently been shown capable of consistently restoring normal sinus rhythm in fibrillating hearts, both in animals and man. From a purely electrophysiologic point of view, the feasibility of catheter defibrillation raises basic questions regarding its mechanism. It had previously been thought that catheter defibrillation would be ineffective in ventricular fibrillation.

The possibility exists that the various modes of defibrillation described in this study reflect differing electrophysiological mechanisms. Ventricular defibrillation is currently explained only in terms of complete depolarization of all myocardial fibers. While it is conceivable that such total depolarization of the ventricles does occur as a consequence of a high energy countershock delivered by transthoracic or direct paddles, it has not been shown that it represents a prerequisite for successful defibrillation.

On purely conceptual grounds, intraventricular catheter shocks delivering energies as low as 5 to 15 watt seconds are unable to consistently create and maintain a sufficiently large and strong electrical field to encompass and to depolarize the whole of the myocardial mass. On the other hand, these shocks certainly result in depolarizing a more or less substantial portion or portions of the ventricles. While the exact mechanism by which low energy catheter countershock restores an effective cardiac rhythm has yet to be determined, it may involve an electrical synchronization of a critical mass of the myocardium which in turn reduces and depolarizes the mass of the fibrillating fibers, no longer allowing the arrhythmia to be self-sustaining.

The relationship between the initiation, maintenance, and recovery from ventricular fibrillation and the amount of tissue mass involved was recognized many years ago. It has been observed that when the ventricular mass is large, ventricular fibrillation is easier to induce, more prone to persist, and less likely to spontaneously revert. Accordingly, ventricular fibrillation can be induced only with difficulty in small animals such as frogs, turtles, and cats, and if induced, spontaneous reversion is frequent. On the other hand, spontaneous reversion of ventricular fibrillation virtually never occurs in adult dogs, sheep, and man. Spontaneous defibrillation has never been observed during fibrillation studies in over 200 dogs of the body weight range described in the present experiments (Moore and Spear, unpublished data).

The concept that catheter defibrillation may act by an analogous mechanism affords an attractive interpretation of the various patterns of defibrillation observed in this study. Thus, the direct resumption of sinus rhythm noted in pattern 1 might correspond to a total or virtually total depolarization of the ventricles by the countershock. On the other hand, pattern 2, characterized by progressive coarsening of the fibrillatory wave due to subthreshold shocks, might reflect partial depolarization of the ventricles. When the myocardial mass required for maintenance of the fibrillating process is reduced below a critical level by the depolarizing effects of one or several catheter shocks, defibrillation is accomplished and an effective rhythm comes into play. Interestingly enough, a similar coarseness in the ventricular fibrillation waveform is present in the frog and turtle in which fibrillation is induced and maintained only with great difficulty.

It is more difficult to provide a satisfactory explanation of pattern 3 characterized by the high amplitude ventricular flutter-like complexes. This pattern may
merely be a higher degree of coarsening and increase in coordination of ventricular activation as characteristic of pattern 2, or may represent a new rhythm distinct from fibrillation. When the high amplitude flutter-like complexes of pattern 3 were present, another similar intensity subthreshold shock was consistently able to revert the heart to sinus rhythm.

References
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