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Abstract

A promising therapy for ventricular fibrillation--a life-threatening cardiac arrhythmia--is the implantation of an automatic defibrillator. A critical component of such a device is the system that detects the presence of ventricular fibrillation. Automatic systems for detecting ventricular fibrillation have been tested with arrhythmias produced by electric shocks in normal canine hearts, but have not been tested with spontaneous arrhythmias in hyperirritable hearts. We have developed an animal model to create arrhythmias without electrical stimulation and have used it to test our automatic defibrillator. This model permits evaluation of both reliability to diagnose ventricular fibrillation and reliability to reject other tachyarrhythmias.

Key words: animal testing; automatic defibrillator; strophanthidin-toxic hearts; tachyarrhythmias; ventricular fibrillation detection

Introduction

Ventricular fibrillation is a life-threatening cardiac arrhythmia which, if untreated, will most likely cause death of the patient in whom it occurs. Cobb and his coworkers (1975) identified a group of high risk patients for whom no effective medical therapy is currently available. Such patients are candidates for implantation of an automatic defibrillator (Mirowski et al. 1970; Schuder et al. 1970). A critical component of such a device is the system that detects the presence of ventricular fibrillation. If the detecting system were to diagnose the presence of ventricular fibrillation when it, in fact, is not present, the patient will be subjected to an unnecessary and perhaps harmful electric shock. On the other hand, if the detecting system failed to recognize ventricular fibrillation, then the patient would probably die.

Different approaches to the automatic detection of fibrillation are possible. Langer and his colleagues (1976) described a detecting system that diagnoses ventricular fibrillation based upon changes in the electrocardiogram. We have previously reported a detecting system that requires both the presence of fibrillation waves in the ECG and cessation of pumping in the ventricular mechanogram as criteria for fibrillation (Bourland et al. 1977). In this study we are reporting the

development of an animal model to test an automatic defibrillator, and the performance of its automatic detecting system.

Detecting system

Our detecting system acquires both electrical and mechanical signals from the ventricles via a pair of electrodes at the end of a right ventricular catheter. The spacing between the pair of electrodes is such that both of the sensing electrodes are totally contained within the right ventricle. Electrical activity of the right ventricle is monitored by detecting changes in the voltage between the electrodes; the mechanical activity is monitored by detecting changes in impedance between the electrodes. In diastole, the ventricles are relaxed and filled with blood and the impedance between the electrodes is minimal. In systole, the ventricles contract and eject blood, and the impedance between the electrodes increases. Thus, changes in impedance between the catheter electrodes in the right ventricle correlate with the changes in blood volume within the ventricle, that is, cardiac stroke volume.

Typical signals recorded from the catheter electrodes are shown in Fig. 1. Operation of the detecting system is illustrated in Fig. 2. The central element of the detecting system is an accumulator. The count in the accumulator represents the net number of waves in the ECG that qualify as waves of fibrillation. A wave in the ECG that qualifies as a wave of fibrillation incrementally increases the accumulator count; one which does not meet criteria for fibrillation decreases the accumulator count. Thus, during rhythms other than fibrillation, the accumulator contains zero; during ventricular fibrillation, the number in the accumulator increases.

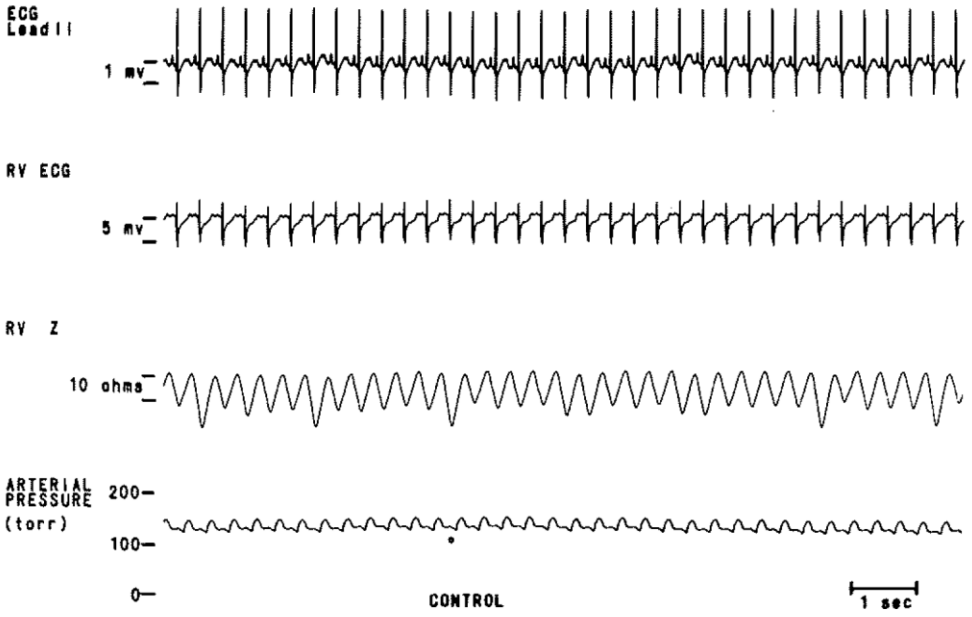

Fig. 1. Typical recordings of ventricular contractions. RV ECG = right ventricular electrocardiogram; RV Z = right ventricular impedance.

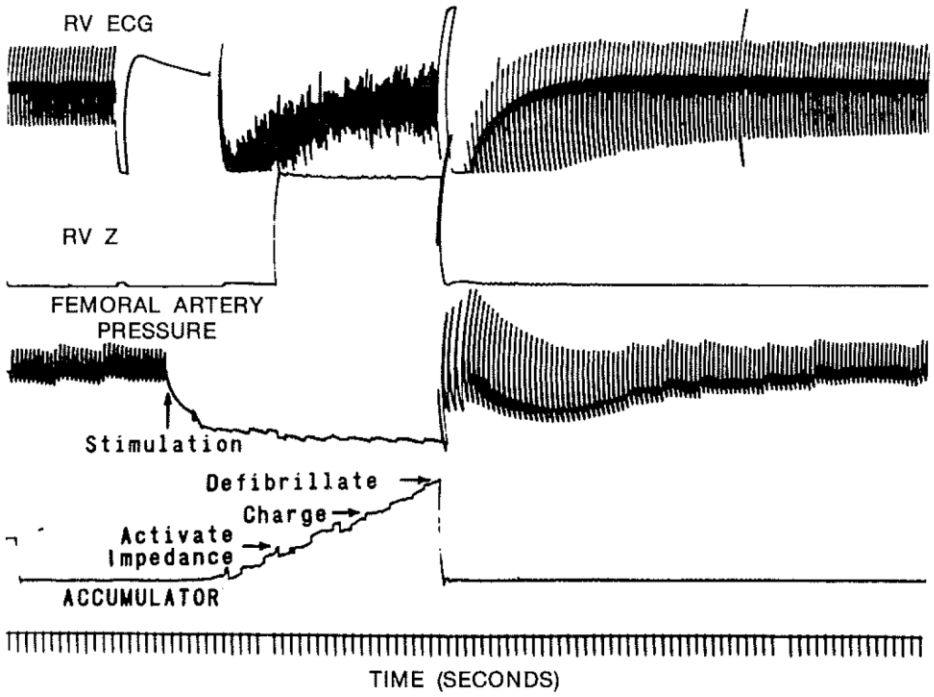

Fig. 2. Detection system operation with accumulator sensing fibrillation.

In order to conserve battery energy, the mechanical sensing circuitry is normally inactive. However, when the number in the accumulator reaches 30, the mechanical-sensing (impedance) circuitry is activated and the operating mode of the detecting system changes. Thereafter, a change in impedance that fulfills criteria for a ventricular contraction causes the next two events in the ECG to gradually decrease the count in the accumulator. Thus, effective pumping by the ventricles overrides the information contained in the ECG signal. When the number in the accumulator reaches 60, charging of the energy-storage capacitor is initiated. When the number in the accumulator is 99 and the energy storage capacitor is charged, a defibrillating shock is delivered and the accumulator is reset to 0. The defibrillating current passes between the pair of electrodes within the right ventricle and a second pair of electrodes located on the catheter 100 mm from to the catheter tip.

One additional operating condition should be mentioned. If the impedance-sensing circuitry is activated and the accumulator count decreases decrementally to 0, the impedance-sensing circuitry is inactivated.

Animal Model

Ventricular tachyarrhythmias were induced by progressive digitalis intoxication. Twelve mongrel dogs of both sexes with body weight from 9 to 15 kg were anesthetized with pentobarbital sodium administered intravenously and the trachea was intubated. With the right jugular vein exposed, the sensing/defibrillating catheter was passed into the right ventricle. The tip of the catheter was positioned at the apex of the right ventricle. A femoral artery was catheterized for recording arterial pressure, and a femoral vein was catheterized for drug administration.

Progressive digitalis intoxication was achieved by stepwise intravenous administration of the rapid-acting digitalis preparation, strophanthidin-K. An initial intravenous injection of 50 μg/kg was followed by injections of 10 μg/kg every 30 min until ectopic beats occurred at a rate of 20/min. Between the first few strophanthidin injections, epinephrine (5 μg/kg) was injected intermittently to increase the incidence of arrhythmias. The epinephrine precipitated short episodes of tachyarrhythmias when the level of strophanthidin was relatively low, and more severe arrhythmias as the level of strophanthidin was increased. Data recorded on analog magnetic tape included a lead II ECG, the right ventricular endocardial electrogram, right ventricular impedance changes, and femoral artery pressure.

Results

Progressive digitalis toxicity produced a wide range of ventricular tachyarrhythmias. As the level of strophanthidin was increased, the severity and duration of ventricular tachyarrhythmias increased. Each of the subjects demonstrated a spectrum of arrhythmias which, in general, ranged from an occasional premature beat to ventricular tachyarrhythmias that reverted to other rhythms, to ventricular tachyarrhythmias that degenerated to ventricular fibrillation. Typically, ventricular ectopic beats were first observed only in response to an epinephrine injection, and normal sinus rhythm resumed spontaneously as the epinephrine was metabolized. As the level of strophanthidin was increased, the arrhythmias became more severe and persisted longer. In some animals, more than 30 episodes of ventricular fibrillation were precipitated, successfully detected, and converted to another rhythm by the automatic defibrillator.

Of the arrhythmias produced, two types are most useful to the automatic defibrillator designer: very rapid ventricular tachycardias that are not ventricular fibrillation; and ventricular fibrillation. In general, when criteria for an automatic detecting system are changed to increase the probability of rejecting tachyarrhythmias that are not ventricular fibrillation, the probability of correctly diagnosing ventricular fibrillation may be decreased. The arrhythmias induced in our animal model permit the designer to assess changes in detector design, using for example, ROC analysis.

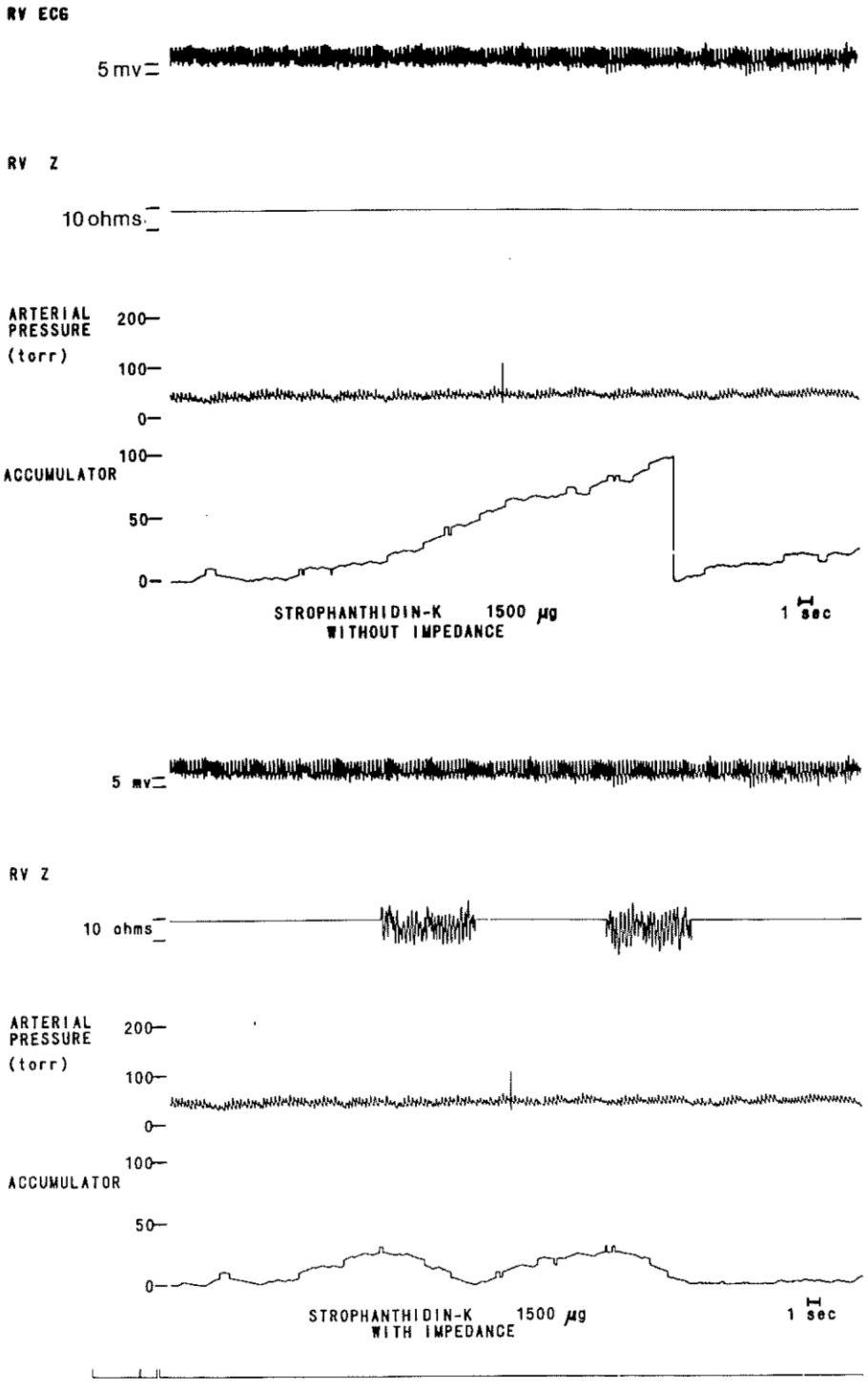

Fig. 3. Detection system recordings of ventricular contractions. 3a (top): impedance signal disconnected. 3b (bottom): impedance signal connected. Without impedance sensing there was a false positive detection of ventricular fibrillation.

The performance of our detecting system for a ventricular tachyarrhythmia other than ventricular fibrillation is shown in Fig. 3, a and b. Although the electrogram is very rapid and irregular, the record of arterial pressure shows that the left ventricle is pumping blood. Fig. 3a shows the result of using only the information contained in the endocardial electrogram from the right ventricle. Note that the impedance signal from the right ventricle had been disconnected from the detecting system, as shown in the right ventricular impedance (RV Z) record of Fig. 3a. The accumulator repeatedly increased incrementally, as shown in the bottom trace, indicating that the detecting system incorrectly interpreted the arrhythmia as ventricular fibrillation. If a detecting system using only the right ventricular endocardiagraphic signal were used, the automatic defibrillator would have delivered an inappropriate shock (at the time noted by the sudden return of the accumulator to zero in the right portion of Fig. 3a).

The same tachyarrhythmia is shown in Fig. 3b. However, both the electrical and mechanical signals were connected to the detecting system. When the accumulator reached a count of 30, the impedance-sensing circuitry was activated. During the time when the impedance-sensing circuitry was active, the accumulator decreased to zero. When the accumulator contained zero, the impedance-sensing circuitry was inactivated, and the sequence was repeated. Using both electrical and mechanical signals, the detecting system correctly distinguished the arrhythmia from ventricular fibrillation, and no shock was given.

Discussion

Mirowski and his coworkers (1978) have described an implantable fibrillator for testing automatic defibrillators. The device is implanted subcutaneously and, when a magnet is placed over the fibrillator, an alternating current is applied to the ventricles to induce fibrillation. Mirowski's animal model for testing automatic defibrillators does permit testing the reliability of a detecting system to diagnose ventricular fibrillation. However, it does not permit assessing the ability of the detecting system to reject ventricular tachyarrhythmias other than fibrillation. Conversely, our animal model permits evaluation of both aspects of defibrillator operation. Furthermore, our model permits evaluation of changes in detector design. This study also demonstrated that strophanthidin-toxic hearts can be defibrillated by a catheter-based automatic defibrillator. Typically, a dog experienced more than 20 episodes of fibrillation and automatic defibrillation. A considerable variety of arrhythmias other than ventricular fibrillation was recorded between fibrillation episodes. Our ventricular fibrillation detector correctly rejected virtually all of the tachyarrhythmias that were not fibrillation and correctly identified and treated almost all fibrillation episodes.

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