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Effects of myocardial infarction on catheter defibrillation threshold

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Abstract

Because the automatic implantable defibrillator may be used in patients having ischemic heart disease, it is important to know whether myocardial ischemia changes the threshold for ventricular defibrillation under experimental conditions simulating automatic internal defibrillation. We determined changes in ventricular defibrillation threshold following coronary occlusion, using an electrode catheter designed for use with an automatic implantable defibrillator. Acute myocardial ischemia was produced without thoracotomy in 10 dogs (experimental group) by embolization with a plastic bead injected via a catheter into the left coronary artery. A control group of 4 dogs had only saline injected into the artery. Defibrillation threshold was measured at 15-min intervals from 1 hour before embolization to 2 hours after embolization. In the control group, voltage, current, energy, and impedance were unchanged after injection of saline into the coronary artery, and India ink perfusion revealed no ischemic areas. In the experimental group post-embolization threshold current and energy were significantly higher than pre-embolization values: 0.47 vs 0.40 A/kg and 1.01 vs 0.80 J/kg, respectively ($p < 0.01$). The magnitude of the peak change in threshold current after embolization was positively correlated ($r = 0.79$) with the size of the ischemic zone, determined by weighing unstained areas from India ink perfusion. Defibrillation threshold for a catheter electrode configuration increases for at least 2 hours following onset of acute myocardial ischemia. This finding must be accounted for in the design and use of an automatic implantable defibrillator.

Keywords: automatic implantable defibrillator; automatic internal defibrillation; defibrillation threshold; myocardial infarction; myocardial ischemia

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Introduction

The automatic implantable defibrillator has been proposed for use in patients at high risk for ventricular fibrillation, and clinical testing has begun in patients who have survived repeated episodes of sudden death (Mirowski et al. 1980). However, patients with myocardial infarction have not been included, even though infarction is the most common cause of ventricular fibrillation in the general population. We previously reported that current and energy requirements for direct, open-chest ventricular defibrillation increase within the first hour following coronary artery occlusion in dogs (Tacker et al. 1974). This effect would increase the output requirements for an automatic implantable defibrillator. However, Ruffy and associates (1980) have reported no increase in defibrillation threshold associated with short periods of ischemia when they used a variety of intrathoracic electrodes designed for use with an automatic implantable defibrillator. Therefore, we conducted this study to determine changes in ventricular defibrillation threshold following acute coronary occlusion, using an electrode catheter designed for use with an automatic implantable defibrillator.

Methods and Materials

We produced experimental myocardial infarction by embolization of the left coronary artery without thoracotomy, using the procedure described by Chagrasulis and Downey (1977). Thoracotomy was avoided because anatomic alteration of the open chest is likely to cause redistribution of electric current flow and alteration of defibrillation threshold, as was demonstrated in preliminary studies. The left coronary artery was cannulated, and a 1.5 mm diameter plastic bead was injected to produce experimental myocardial infarction. The specially fabricated rigid cannula was advanced via a carotid artery into the aortic root and maneuvered into the left coronary ostium for this purpose.

Fourteen pentobarbital anesthetized dogs, weighing 15-28 kg (mean 21.0 kg), served as subjects. This anesthetic was chosen because it does not alter the ventricular defibrillation threshold (Babbs 1978). The catheter electrodes for defibrillation (Fig. 1) were inserted via the right jugular vein during fluoroscopy, and the tip of the catheter was wedged in the apex of the right ventricle. Current passed from the proximal pair of ring electrodes to the distal pair; total surface area of each pair was 250 mm². Permanent radiographs were taken to document proper catheter placement. Each animal's electrocardiogram (lead II) and femoral artery blood pressure were monitored to confirm ventricular fibrillation and defibrillation. The ECG preamplifier was a specially fabricated quick-recovery unit, which returned the ECG signal within 300-500 msec after application of a defibrillator shock. This device was designed to detect any instances of refribrillation that might have occurred within seconds after a successful shock. Gelled 10-cm-diameter disk electrodes were sutured to the chest wall for delivery of backup shocks when the test defibrillation shock failed.

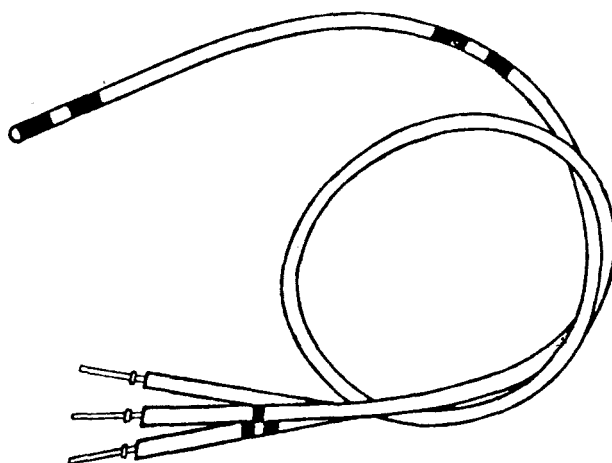


Figure 1. Catheter electrode used for defibrillation.

The procedure for measuring defibrillation threshold has been reported earlier (Bourland et al. 1978). An experimental trapezoidal waveform defibrillator (Bourland et al. 1977) was the pulse generator for test shocks. A capacitance of 125 μF and a shunt resistance of 40 Ω was selected to produce a 5-msec trapezoidal waveform of $65 \pm 2\%$ tilt. Fibrillation was produced by stimulation of the endocardium through the catheter electrode. The stimulus was a 5-sec train of 50-Hz, 2 msec duration rectangular wave pulses at 5-20 V intensity. A test shock from the defibrillator was delivered through the catheter electrodes 10 sec after onset of fibrillation. If the test shock produced defibrillation, a 10% lower peak current amplitude shock was selected for the next trial. Trials were repeated only after arterial blood pressure had returned to a stable level. If the test shock failed to defibrillate, a backup shock of known effectiveness was applied via the transthoracic electrodes from a standby damped sine wave defibrillator. After recovery, a 10% stronger shock was then tested. Ventricular defibrillation threshold was defined as the lowest peak current intensity that defibrillated the ventricles and that was no more than 10% greater than a shock which failed to defibrillate.

Two groups of animals were studied: an experimental group (n = 10 dogs) that received 1-4 intracoronary plastic bead emboli and a control group (n = 4 dogs) that received intracoronary saline only. The procedure for both groups was to monitor threshold at 15-min intervals for a period of 1 hour, inject the beads or saline into the left coronary artery, and monitor the defibrillation threshold at 15-min intervals for the next 2 hours.

At the end of each experiment, the animal's chest was opened and the heart perfused with dilute India ink (3% Pelikan #17 noir) in 0.9% saline to delineate the ischemic areas, according to the technique described by Abendschein et al (1978). Perfused areas of the heart were blackened by the India ink and were easily distinguished from non-perfused areas, which remained red-brown. This procedure was done to document the production of ischemia by the bead embolization technique, and is important because some dogs have sufficient collateral circulation to reduce ischemia, even though a bead is successfully

placed in a major coronary artery. After India ink perfusion, the hearts were removed from the thorax and sectioned longitudinally into 1-cm slices. Non-perfused ischemic areas were cut out and weighed, and the percentage of acutely ischemic ventricular tissue (by weight) was calculated. In this way defibrillation threshold change could be related to the size of the ischemic zone.

Results

After perfusion of the hearts, the non-inked, acutely ischemic areas were clearly demarcated from the blackened, perfused areas. The percentages of ventricular myocardium rendered acutely ischemic for the 10 experimental animals in the study are given in Table 1, together with ventricular mass.

Table 1. Ventricular myocardium after perfusion.

<i>Dog No.</i>	Mass (g)	Ischemic Tissue (%)
1	195	12
2	157	15
3	142	28
4	187	19
5	154	23
6	145	22
7	138	8
8	203	18
9	114	5
10	123	12

Notes: Mean ventricular mass (\pm SD) = 156 ± 30 g; mean percent ischemic tissue (\pm SD) = 16 ± 7 .

Fig. 2 illustrates relative changes in threshold current and energy produced by coronary artery embolization in the two groups. On the horizontal axes, time zero represents the time of embolization (or sham embolization in the control group). Negative time represents the pre-embolization baseline period. The vertical axes show relative changes in threshold, obtained by dividing individual threshold measurements by the mean of the pre-embolization threshold values. This procedure corrects for animal-to-animal variation in threshold levels, so that the changes for all the animals can be plotted together. Like threshold current, threshold energy increased after coronary artery embolization in all animals. Statistical analysis with Student's t- test showed the pooled postinfarction values for current and energy to be higher than the corresponding preinfarction values ($p < 0.01$). The mean threshold values for peak current rose from 0.40 ± 0.01 to 0.47 ± 0.01 A/kg body weight, and mean threshold energy rose from 0.80 ± 0.05 to 1.01 ± 0.05 J/kg body weight. Mean impedance (peak voltage/peak current) was stable at 107 ± 2 n. As shown in Fig. 2, peak current was unchanged in the control subjects after injection of saline into the coronary artery. Likewise, energy and impedance were unchanged in this group.

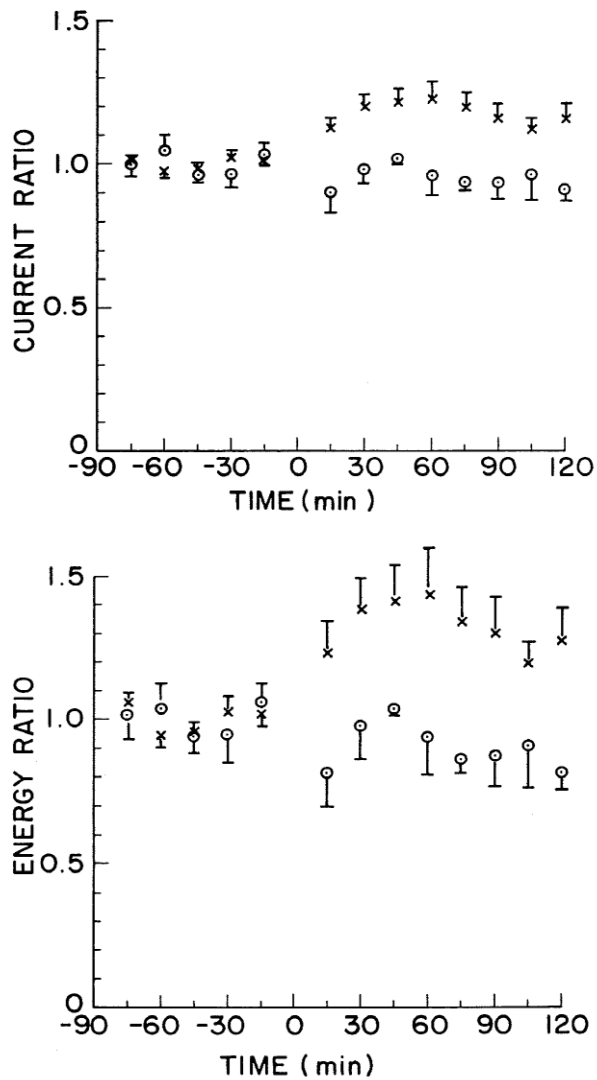


Figure 2. Relative changes in threshold current and energy after occlusion of the coronary circulation. Top: current ratio; bottom: energy ratio. Each X represents the mean value for 10 experimental subjects. Each circle represents the mean value for 4 control subjects. 1.0 on the vertical axis represents the mean threshold before embolization in each animal

The magnitude of the maximal peak change in threshold current after embolization was related to the size of the acutely ischemic zone (Fig. 3). Control animals (0% ischemia) and one animal that had only 5% ischemia showed less than 10% increase in threshold. Other animals with 8-28% ischemia showed a threshold current rise from 25 to 53%. The correlation coefficient between the variables in Fig. 3 is $r = 0.79$.

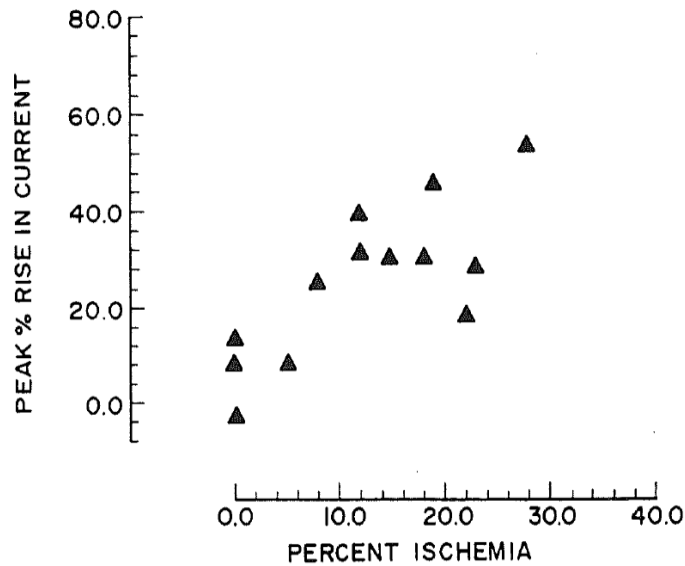


Figure 3. Relationship between maximum increase in peak current threshold and size of the infarcted region of the heart (expressed as percent of the ventricle which was ischemic)

Careful analysis of high-speed records from this series revealed no instance of defibrillation, as detected with the quick-recovery ECG preamplifier. That is, in no case was the heart shown to be defibrillated but the shock scored a failure because fibrillation began again very soon (0.5--5 sec) after the shock.

Discussion

This study reveals a significant increase in defibrillation threshold following the onset of acute myocardial ischemia. Maximum increase in threshold is reached about 1 hour after occlusion. A similar finding was previously reported by us for open-chest, direct-heart defibrillation (Tacker et al.). Ruffy and coworkers, using a pervenous bipolar endocardial electrode, did not observe elevation of defibrillation threshold with ischemia. However, they occluded the coronary artery for only 8 min, then reperfused the ischemic area. In their study, repeated trials were accomplished by re-occlusion. Apparently, the defibrillation threshold did not rise detectably during the first few minutes after occlusion, and did not continue to rise when the occluded artery was reopened within 8 min.

If the data of Ruffey and coworkers represent the clinical analogue of coronary artery spasm (brief periods of ischemia) and our data represent the clinical analogue of coronary artery occlusion (permanent ischemia), one would expect that fibrillation associated with brief, transient ischemia might respond to shocks of the same strength required for the normally perfused heart. To the contrary, if acute myocardial infarction is the precipitating agent for fibrillation, defibrillation may require increased shock strength only a few minutes after the infarction occurs. The observation that a larger infarct produces a greater increase in threshold is not surprising, but does suggest that the dose required to defibrillate may be directly related to infarct size.

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