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ABSTRACT

A transient, dose-dependent cardiac depression was produced by defibrillator shocks in an isolated, working canine heart preparation perfused with oxygenated arterial blood from a support dog. Accompanying this depression was an efflux of potassium (K^+), forced out of the myocardial cells by the passage of defibrillating current. The transient increase in extracellular K^+ concentration was recorded graphically in the venous outflow. It was found that 5-msec rectangular wave shocks, from three to ten times defibrillatory current threshold, released dose-related pulses of K^+. We conclude that because extracellular K^+ is a myocardial depressant, at least part of the myocardial depression after defibrillation is caused by the release of K^+ from the myocardial cells.

Key words: defibrillation, myocardial damage, toxicity, ventricular fibrillation, waveform

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INTRODUCTION

Previously, we reported that high-strength defibrillator shocks produce a transient decrease in myocardial contractile force [1, 2]. In those studies, a modified Langendorff isolated-heart preparation was used, and the coronary arteries of the isolated canine heart were perfused with oxygenated arterial blood from a second support dog (fig. 1). We found that, regardless of the defibrillator current waveform used, an electrical dose-dependent depression of myocardial contractile force occurred immediately following the shock. This depression was transient, noncumulative, and dose dependent.

All of the mechanisms underlying shock-induced myocardial depression are, as yet, not elucidated. However, transient, current-dependent myocardial depression can result from a sudden increase in extracellular potassium concentration [3]. In addition, an increase in coronary venous K^+ concentration has been reported following defibrillator-strength shocks [4, 5]. Accordingly, one explanation for shock-induced myocardial depression could be shock-induced K^+ efflux, a hypothesis that can now be tested because of the availability of K^+-sensitive detectors.

METHODS

To test the hypothesis that K+ efflux is one factor in myocardial depression after defibrillator shock, we employed the isolated-heart preparation shown in figure l. The donor and support dogs were anesthetized with intravenous sodium pentobarbital (30 mg/kg), and the donor dog was artificially respired during removal of the heart. The support dog maintained adequate respiration throughout the experiment without the need of ventilatory support. A K^+-sensitive electrode (Ionetics Inc., Costa Mesa, California), mounted in a flow through cell, was placed in the venous outflow line from the isolated heart. When the K^+ concentration increased, the voltage output of the K^+ sensor increased (fig. 2). Blood flow through the cell was determined by injecting a small amount of potassium chloride into the venous line a known distance upstream and recording the time required for the K^+ electrode to register the change in concentration. By multiplying the measured velocity by the cross-sectional area of the tubing, a flow value was obtained (approximately 250-300 ml/min). By recording the increase in K^+ concentration, micro-equivalent amounts of K^+ released from the heart could be determined.

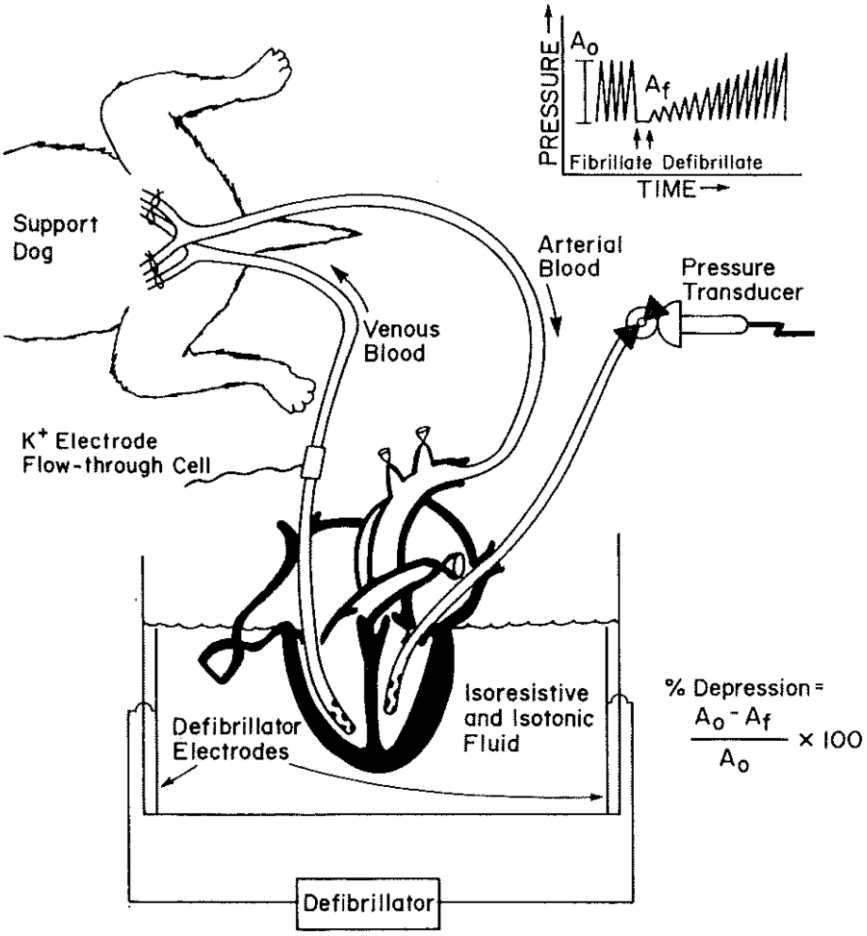

Figure 1. The perfused isolated canine heart preparation used in this study. The heart is perfused with arterial blood from a support dog, and the coronary venous blood is returned to the support dog. The K^+ electrode is placed in a flow through cell in the venous return line.

The isolated, blood perfused heart was suspended in a shock bath containing an isoresistive and isotonic volume conductor (mixture of glucose and NaCl solutions) at 37°C. The heart maintained a relatively constant spontaneous rate (approximately 120 beats/min) throughout the experiment. Rectangular waveform defibrillator shocks of 5-msec duration were delivered to the heart through the volume conductor via two parallel plate electrodes placed at either end of the shock bath. The defibrillating current and voltage were measured on a storage oscilloscope. Because the dimensions of the shock bath were known, current and energy densities could be calculated.

Defibrillation threshold, defined as the lowest current that would defibrillate, was first established with a resolution of 10% in current. In other words, a shock 10% less than threshold would not defibrillate. The threshold value was periodically checked throughout each experiment and was relatively constant (typically, 55 mA/cm^2). Then, defibrillator shocks of three to 12 times threshold current were delivered to the heart in a random order. The degree of myocardial depression was identified by the immediate decrease in left-ventricular isovolumic pressure following each shock. The inset in figure 1 shows the myocardial depression and the method of calculating percentage of depression. The study involved four isolated heart preparations.

Each suprathreshold shock (i.e., one above defibrillation threshold current) was followed by a transient depression that was dependent on current density, as described in our earlier studies [1, 2]. The depression was followed by a sudden increase in venous K^+, as shown in figure 2. There was a linear relationship between delivered current density and the amount of potassium released, as shown in figure 3, for which the correlation coefficient was 0.98.

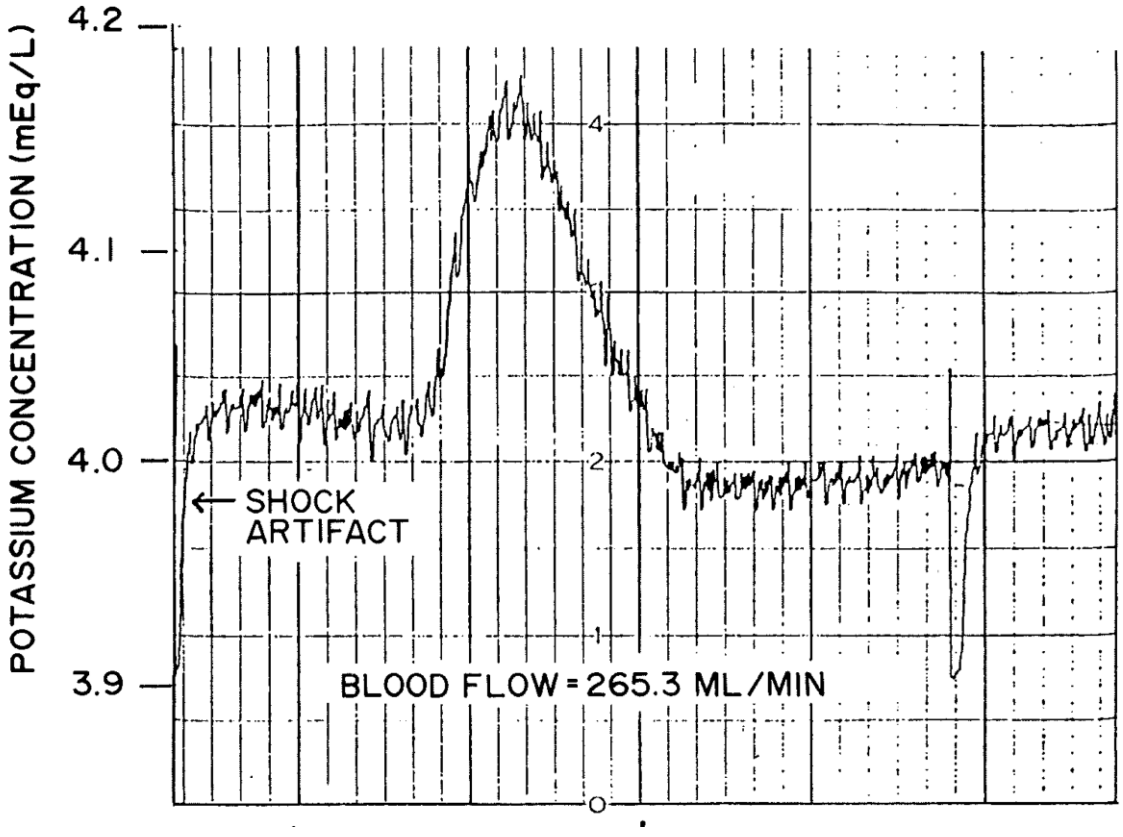

Figure 2. A record of the K^+ increase in the coronary venous blood following a 0.6-A/cm^2 , 5-msec rectangular wave defibrillator shock.

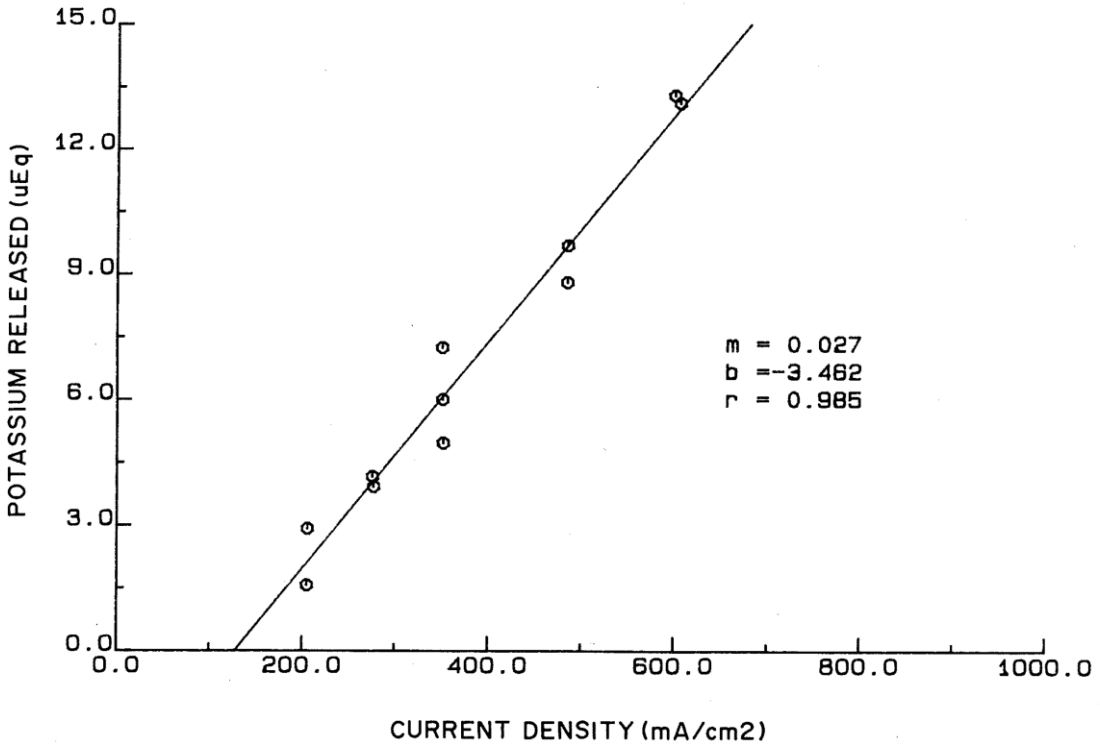

Figure 3. The relationship between potassium released from the heart and the defibrillating shock current. K^+ (mEq) = 0.027 (mA/cm^2) – 3.46, with a correlation coefficient of 0.985.

DISCUSSION

The amount of K^+ released was linearly related to the strength of the defibrillator shock. Extracellular potassium (K^+) is commonly known to depress myocardial contractility [3]. Thus, it is likely that at least part of the depression after defibrillator shock is caused by the efflux of K^+ from the myocardial cells.

Defibrillator strength voltage gradients have been shown to produce a transient dielectric breakdown of the myocardial cell membranes [6]. This phenomenon would allow relatively free movement of intracellular ions in proportion to the delivered current. Thus, high intensity shocks would be expected to move K^+ out of the myocardial cells and into the extracellular space in proportion to the delivered current and, thus, increase the extracellular K^+ concentration suddenly. The relatively high rate of blood flow through the isolated heart quickly washed this K^+ out of the myocardium before significant amounts could be actively transported back into the cells by the membrane-bound ion pumps.

The results obtained in this study are consistent with our hypothesis that post-defibrillation myocardial depression after defibrillation is in part caused by a sudden increase in extracellular K+ concentration produced by the defibrillating current passing through the heart. Clinically, this phenomenon may be responsible for some episodes of electromechanical dissociation after defibrillation and may be much greater in diseased, hypoxic hearts with low rates of coronary blood flow. In such cases, good cardiopulmonary resuscitation techniques may greatly improve the situation by washing out the excess K^+, and appropriate pharmacologic interventions could restore the contractility more quickly.

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