Left Ventricular Geometry Immediately Following Defibrillation-Strength Shocks

V Ramanathan, BK Hoffmeister*, AL de Jongh

Joint Graduate Program in Biomedical Engineering
The University of Memphis and University of Tennessee Health Science Center
*Department of Physics, Rhodes College, Memphis, USA

Abstract

A previous 2D ultrasound study suggested that there is relaxation of the myocardium after defibrillation. However, that 2D study could not measure activity occurring within the first 33 ms following the shock. Thus, the objective of our study is to determine the left ventricular (LV) geometry during this period. Biphasic defibrillation shocks were delivered in seven dogs. One-dimensional, short-axis ultrasound images of the LV cavity were acquired, the boundary of the anterior and posterior endocardial walls was extracted, and the distance between them computed from 32 ms before to 32 ms after the shock. The normalized mean pre- and post-shock slopes are 0.2 ± 2.2 and 3.3 ± 7.9 %/10 ms, respectively. The post-shock LV diameter slope is positive in the first 32 ms following both successful and unsuccessful defibrillation shocks ($p<0.05$). Therefore, our results confirm that the bulk of the myocardium is relaxing immediately after defibrillation shocks.

1. Introduction

A previous study by Malkin *et al.* [1] measured left ventricular (LV) geometry in canines after defibrillation using 2D ultrasound. The 2D study showed that the cross-sectional area of the LV cavity rapidly increased within approximately 200 ms after defibrillation, suggesting that there is relaxation of the myocardium after defibrillation. One limitation of the study is that the first 33 ms after the shock may not have been captured due to a sampling rate of 30 Hz. This time period may be critical for discriminating between shock-induced and excitation-induced relaxation.

The purpose of this study is to determine the LV diameter in the first 32 ms after the defibrillation shock using M-mode echocardiography (1D ultrasound). The one-dimensional approach offers a sampling rate of 250 Hz. Both the 1D and 2D studies provide a quantitative approach towards evaluating the mechanical reaction of the heart following defibrillation.

2. Methods

Seven mongrel dogs were anesthetized with intravenous pentobarbital (30 – 35 mg/kg initially) and then maintained with doses of 3 mg/kg/hr. Skeletal muscle paralysis was maintained by succinylcholine to prevent excessive motion artifact. The ECG (lead II), blood pressure (femoral artery) and the rectal temperature were continuously monitored. A hot water blanket was used whenever necessary to maintain body temperature.

Following ten episodes of ventricular fibrillation, defibrillation shocks were administered using truncated exponential biphasic waveforms. If the primary shock failed to defibrillate, backup shocks were administered but were not analyzed.

The ultrasound measurements were made with a Hewlett Packard Sonos 1000 ultrasonic imaging machine and M-mode data were recorded on a VHS tape recorder at a sampling rate of 250 Hz before the defibrillation pulse, during the defibrillation pulse and for several seconds following the pulse. The ultrasonic transducer was placed on the left thorax and the orientation for which the two-dimensional image showed well-defined endocardial walls with the M-mode line in the center of the left ventricular cavity was chosen.

The duration chosen for image analysis was 32 ms before each shock and 32 ms after the shock. The LV diameter is defined as the distance between the anterior and posterior endocardial walls, which were outlined by hand using Scion Image (Figure 1). For each episode, data points were normalized to the LV diameter immediately preceding the shock. The pre- and post-shock slopes of the LV diameter were determined using regression analysis.

3. Results

A total of 70 primary defibrillation shocks were recorded and 51 episodes were analyzed. All episodes for one animal were rejected due to poor image quality. Episodes were rejected due to poor contrast between the blood pool and the anterior and posterior endocardial walls (4 out of 70), due to lateral motion of the heart and shifting of the M-mode line outside the ventricular cavity (10 out of 70), or because no images were captured (5 out of 70). Figure 1 shows an example ultrasound image of a defibrillation episode. Figure 2 shows the time course of pre- and post-shock LV diameter. The overall mean pre- and post-shock slopes are 0.2 ± 2.2 and 3.3 ± 7.9 %/10 ms, respectively. Student's *t*-tests show that the post-shock LV diameter slope is positive (>0) in the first 32 ms following both successful and unsuccessful defibrillation shocks ($p<0.05$).

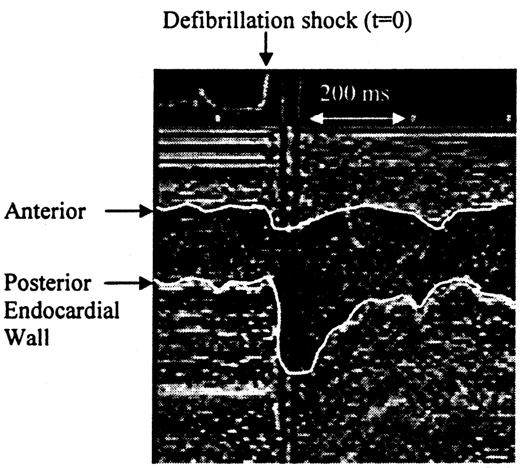

Figure 1. Example of a 1D ultrasound image of the LV cavity with the anterior and posterior endocardial wall tracings.

4. Discussion

The results show that the post-shock LV diameter slope is positive in the first 32 ms following both successful and unsuccessful defibrillation shocks ($p<0.05$). Therefore, our study suggests that the bulk of the myocardium is relaxing immediately after defibrillation shocks. Using the principles of excitation-contraction coupling, we hypothesize that this observed relaxation is a result of direct de-excitation of the bulk of the myocardium. Recent in-vitro studies have suggested de-excitation is a key mechanism for defibrillation [2], but are limited to epicardial measurements. A distinct advantage of the ultrasonic technique used here is that it provides non-invasive, in-vivo measurements that reflect the global activity of the myocardium.

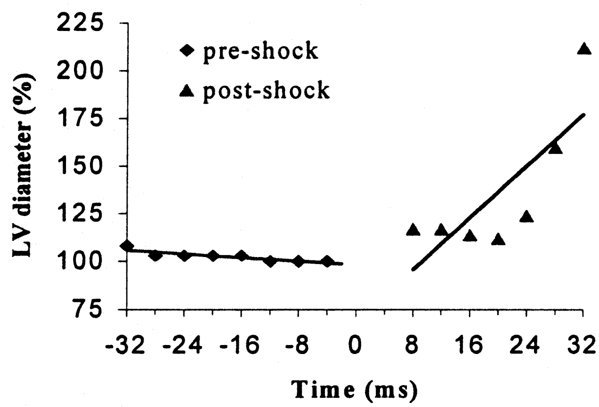

Figure 2. Typical time course of pre- and post-shock LV diameter, where t=0 represents the time of the defibrillation shock. Data points were normalized to the LV diameter immediately preceding the shock (t = – 4 ms).

Acknowledgements

The authors would like to thank Dr. Robert Malkin for his guidance during this study.

References

[1] Malkin RA, Sminth SR, Hoffmeister BK. Defibrillation and the geometry of the heart: a novel measurement with implications for defibrillation mechanisms. Physiol Meas 2001;22:309-21.

[2] Efimov IR, Gray RA, Roth BJ. Virtual electrodes and deexcitation: new insights into fibrillation induction and defibrillation. J Cardiovasc Electrophysiol 2000;11:339-53.

Address for correspondence.

Amy L. de Jongh
Biomedical Engineering
The University of Memphis
Memphis, TN 38152
adejongh@memphis.edu