ELECTROPORATION INDUCED BY INTERNAL DEFIBRILLATION SHOCKS IN NORMAL AND CHRONICALLY INFARCT RABBIT HEARTS

ACC Poster Contributions
Ernest N. Morial Convention Center, Hall F
Tuesday, April 05, 2011, 9:30 a.m.-10:45 a.m.

Session Title: Defibrillation Threshold Testing and Predictors of Shocks in ICD Recipients
Abstract Category: 29. Defibrillation/Implantable Antiarrhythmia Devices
Session-Poster Board Number: 1163-385

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Background: Implantable cardioverter defibrillators (ICDs) automatically detect arrhythmias and deliver lifesaving rescue shocks. However, these shocks can also damage cardiac tissue via electroporation, which disrupts the cell membrane. In this study, the spatial distribution and extent of electroporation were characterized in intact fibrillating rabbit hearts with and without a chronic myocardial infarction (MI).

Methods: The defibrillation threshold (DFT) for 8-ms monophasic truncated exponential shocks delivered by a coil electrode in the right ventricle was determined in 7 Langendorff-perfused MI hearts during dynamic pacing-induced sustained (> 20 s) ventricular fibrillation (VF). In 7 normal and 7 MI hearts, electroporation from a single 300-V monophasic truncated exponential shock at twice the DFT after 35-45 s of sustained VF was measured by membrane-impermeant propidium iodide (PI) uptake. One control and 1 MI heart had VF self-terminate before defibrillation and served as pseudo-shock controls. PI staining of the ventricles was quantified from fluorescence images of transverse sections, and histological studies were performed using Masson's Trichrome staining.

Results: The DFT was 161.4 ± 17.1 V. In all control and MI hearts which received a single shock, PI staining was concentrated in the shock electrode region. Between control and MI hearts, there was no difference in PI uptake in the shock electrode region or in the whole ventricles. However, compared to controls, MI hearts had a small but significant increase in PI staining in the left ventricular epicardial regions. Both pseudo-shock hearts showed minimal PI uptake. Additionally, PI staining between fibrillating hearts in this study and previously studied paced hearts was not significantly different.

Conclusion: The majority of electroporation was spatially associated with the active region of the shock electrode. An MI only increased electroporation in the epicardial regions of the left ventricle correlated with surviving myocytes in the infarct region. A short period of sustained VF before a single, successful defibrillation shock did not increase electroporation.