

MetroHealth Medical Center

RESEARCH DAY 2023

Abstract Submission Form

Poster Title: The cellular mechanisms of low voltage in atrial fibrillation.

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Category: Clinical Physiology

Atrial fibrillation (AFib) is the most common form of cardiac arrhythmia. The presence of low voltage zones (LVZs) in the atrium have been associated with the progression of AFib, and AFib outcomes can be improved when LVZs are targeted by ablation. However, the cause of LVZs is not well understood. This study hypothesizes that LVZs will be accompanied by changes in underlying cellular atrial action potential activity. To test this hypothesis, a tachycardia (rapid pacing) induced AFib swine model (n=6) plus healthy control animals (n=4) were used. In vivo low voltage zones were measured in the atria during sinus rhythm using a 3D CARTO mapping system, while ex vivo atrial conduction velocity (CV) and action potential duration (APD) were obtained using optical mapping techniques. After 4 weeks of rapid pacing, all the animals in the AFib group had acquired AFib. AFib animals registered a higher percentage of low voltage sites (11.4%) compared to controls (4.7%). Furthermore, the average CV was significantly slower in AFib animals (0.69 ± 0.16) vs Control (1.06 ± 0.12 , $p < 0.05$). Importantly, CV in regions near the LVZs (0.62 ± 0.08) was slower than regions far from the LVZs (0.81 ± 0.06 , $p < 0.05$). On the other hand, while APD was significantly longer for AFib (166.47 ± 18.33) vs Control (111.23 ± 17.48 , $p < 0.01$), there was no difference in ADP between areas near and far from LVZs. These results confirm our hypothesis that LVZs are accompanied by changes in atrial action potential activity. Understanding the relationship between LVZs and cellular electrophysiology could be key in improving AFib treatment outcomes.