Optical Mapping Of VF In Isolated Swine Hearts With Scars

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Ventricular fibrillation (VF) is the main cause of sudden cardiac death. We hypothesized that VF induced by large scars in an isolated porcine heart model could aid the understanding of VF in human insects associated with structural dysfunction. The explanted hearts were perfused with blood and Tyrode solution at 37°C, and optically imaged with a voltage-sensitive fluorescence dye (di-ANEPs excited at 530nm with 150W halogen lamp). The emitted signal was filtered (610nm) and recorded with high speed cameras (MiCAM02, BrainVison, Jp) at 0.7mm spatial resolution. No optical signals could be recorded from the core of chronic infarcts or RF lesions. A total of 10 hearts were used: 4 controls, 3 with lesions generated via RF ablation and 3 with chronic infaracts. We observed the propagation of the depolarization waves and analyzed the VF waveforms at the border zone (BZ) and normal myocardium. We analyzed the VF waves in the frequency domain by calculating the dominant frequency (DF) on select regions of interest using Matlab (Mathworks, Natick, MA). Our results showed that DF is smaller at the BZ compared to healthy tissue. Referenced to the average DF in the control hearts (10.07+/−0.54 Hz), the DF was slightly smaller in healthy myocardium of infarct hearts (i.e., 8.9+/−0.71Hz) and significantly smaller at the border zone (i.e., 6.03+/−0.66Hz).

We suggest that these differences are related to the heterogeneous restitution properties as well as the changes in tissue structure at the BZ. The BZ of chronic scars is comprised of a mixture of viable and necrotic fibers; whereas in the acute settings of RF lesions, inflammation and edema are present at the BZ without alteration of fiber directions.

Aim: Pregnancy is characterized by a hypothermic remodeling of the heart, but little is known about the role of hormonal regulation in this cardiac modification. Mineralocorticoid receptors (MRs) have been shown to mediate structural and functional remodeling of the heart in pathological conditions. Also, its agonists (aldosterone and cortisol) as well as cardiac ion channels, are significantly enhanced in pregnancy. Our aim is therefore to examine the possible role of MRs in cardiac myocyte adaptation during rat pregnancy. Methods: Pregnant rats were studied one day before parturition. One group of pregnant rats (Pcan) was treated with potassium canrenoate (20 mg/kg/day), a MRs antagonist, for the last seven days of pregnancy, and compared to normal pregnant rats (P). These groups were also compared to non-pregnant rats, treated (NPcan) or not treated (N) with mock echocardiography was performed for the whole heart study. Results: MR antagonism in Pcan induced a decrease of the systolic and diastolic dimensions of the heart, when compared to P. This result was corroborated by a lower cell volume in Pcan. Cell contractility was not modified in all groups, whereas K+ channel activity was the only one to be affected by MR modulation, leading to a modified responsiveness to energetic substrates lactate and pyruvate, naturally increased in the blood of P. Indeed, while cell contractility was raised in P, this effect was not observed in Pcan. Interestingly, in Pcan, ICa-L tend to increase in the same energetic condition when compared to ICa-L, with glucose only. Conclusions: Our data indicate that MRs are involved in the adaptation of cardiac myocytes to pregnancy at the structural, metabolic, as well as functional level.