CALCIUM CYCLING PROTEIN S100A1 DOES NOT RECOVER FOLLOWING CONTINUOUS-FLOW LEFT VENTRICULAR ASSIST DEVICE SUPPORT OF THE FAILING HUMAN HEART

ACC Oral Contributions
McCormick Place South, S404
Saturday, March 24, 2012, 9:00 a.m.-9:15 a.m.

Session Title: Pathogenic and Therapeutic Insights from Experimental Heart Failure Models
Abstract Category: 15. Heart Failure: Basic
Presentation Number: 906-7

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Background: Heart failure (HF) is characterized by abnormal regulation of Ca2+. LVAD support causes reversal of the HF phenotype, including normalization of Ca2+ cycling protein expression. S100A1 is a cardiac Ca2+ regulatory protein. Animal HF models have shown decreased S100A1, and over-expression of this protein enhances cardiac performance in animals as well as failing human cardiomyocytes. Studies verifying S100A1 dysregulation in large groups of human hearts are limited and the effects of LVAD support have not been addressed. We hypothesized that S100A1 is altered in human HF, and is influenced by the hemodynamic support of an LVAD.

Methods: We compared S100A1 levels in left ventricular tissue from 25 non-failing (NF) hearts of unmatched organ donors, 33 failing (F) hearts, and 22 continuous flow LVAD-supported failing hearts (F+LVAD), obtained at transplant.

Results: S100A1 protein expression was significantly lower in F hearts as compared to NF (P <0.001; Figure 1). S100A1 levels did not recover with LVAD.

Conclusions: Our data confirm down-regulation of S100A1 protein in human HF. Based on the lack of reversal of protein changes with continuous-flow LVAD support in our study, we further hypothesize that S100A1 may be a critical protein in the regulation of cardiac myocyte intracellular Ca2+ cycling and the lack of reversal may contribute to incomplete recovery of cardiac function following LVAD support. Therapeutic strategies designed to increase levels of S100A1 should be further pursued.