

ALTERATIONS IN THE EXPRESSION OF GENES RELATED TO CONTRACTILE FUNCTION AND HYPERTROPHY OF THE LEFT VENTRICLE IN CHRONICALLY PACED PATIENTS FROM THE RIGHT VENTRICULAR APEX (PRELIMINARY RESULTS)

ACC Poster Contributions
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Background: Long term asynchronous ventricular activation from right ventricular apex results in increased wall stress and hypertrophy of the left ventricle (LV), leading to reduced systolic and diastolic function. The purpose of this study is to assess in the peripheral blood alterations of the expression of genes related to LV contractile function and hypertrophy, after right ventricular apical pacing in patients with preserved LV systolic function.

Methods: We enrolled chronically paced patients who were divided into two categories based on the cumulative percentage of ventricular pacing post implant: individuals who were paced due to atrioventricular conduction disturbances and ventricular pacing exceeded 90% (group A) and controls who suffered sinus node dysfunction with preserved intrinsic atrioventricular conduction (group B). At the time of implantation and 3 months later, we evaluated in the peripheral blood concentrations of messenger ribonucleic acid (mRNA) of sarcoplasmic reticulum calcium ATPase (SERCA), and β -myosin heavy chain (β -MHC). We also estimated LV end-diastolic diameter, LV end-systolic diameter and LV ejection fraction echocardiographically.

Results: Up to now, we have collected data from 30 patients during a period of 3-months follow up. In group A (14 patients with QRS 142 ± 12 msec) at 3-months follow-up, mRNA levels of SERCA were decreased ($9,3\pm 1,49$ vs $4,04\pm 1,33$ $p=0,021$) and β -MHC mRNA levels were increased though not significantly ($62,12\pm 46,97$ vs 424 ± 245 $p=0,127$) while echocardiographic parameters remained unaltered. In controls (16 patients with QRS 85 ± 5 msec) all measured parameters showed no significant changes.

Conclusions: Permanent right ventricular apical pacing is associated with alterations, in the peripheral blood, in the expression of genes regulating LV function and hypertrophy. These findings are traceable, while at the same time LV function has not deteriorated.