

**Effect of Pacing Site on Left Ventricular Synchronization - Validation of a Novel Rat Model**  
*Mor, Michal<sup>1</sup>; Dror, Shani<sup>1</sup>; Tsadok, Yossi<sup>2</sup>; Bachner-Hinenzon, Noa<sup>2</sup>; Katz, Amos<sup>3</sup>; Etzion, Yoram<sup>1</sup>; Liel-Cohen, Noah<sup>4</sup>*

*<sup>1</sup>Soroka University Medical Center and Faculty of Health Sciences, Ben Gurion University of the Negev, Beer Sheva, Israel; <sup>2</sup>Technion-Israel Institute of Technology, Faculty of Biomedical Engineering, Haifa, Israel; <sup>3</sup>Barzilai Medical Center, Ashkelon, Israel; <sup>4</sup>Soroka University Medical Center, Beer Sheva, Israel*

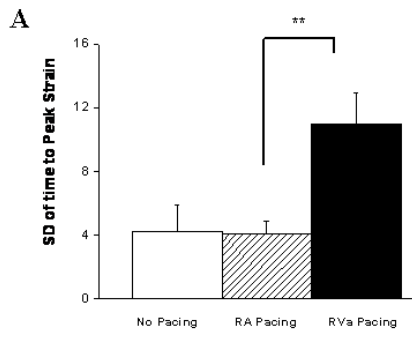
Background: Right ventricular (RV) pacing can cause abnormalities in left ventricular (LV) mechanics. On the other hand, biventricular pacing (CRT) can improve heart failure outcome by altering the LV electric and mechanical substrates. At the present, relevant pacing models utilize large animals, which are expensive and can not be manipulated genetically. Here, using novel methodology, we studied whether the small rodent heart can mimic pacing related findings in humans.

Methods: Rats were implanted with two miniature-bipolar-hook electrodes as follows: Group A (n=6) right atrium (RA) and RV apex (RVa). Group B (n=5) RVa and posterobasal aspect of the LV (LVpb). Electrodes were attached to a skin connector in the back. Following surgical recovery, two-dimensional transthoracic echocardiography was performed at baseline (no pacing) and during pacing through the various electrodes at a rate slightly higher than the spontaneous heart rate (400 bpm). Segmental 2D circumferential strain analysis was performed (short-axis at midventricle).

Results: Group A: Compared to baseline, RA pacing had no effect on LV synchrony as measured by standard deviation of segmental time to peak strain ( $4.22 \pm 1.66$  vs.  $4.05 \pm 0.79$ , for baseline and RA pacing, respectively,  $p=0.46$ ). In contrast, RVa pacing increased the observed dyssynchrony to  $11.0 \pm 1.94$ , ( $p = 0.003$  vs. RA pacing, Fig 1A). Group B: LVpb pacing reduced LV dyssynchrony by  $40.4 \pm 15.4$  % compared to RVa pacing ( $p = 0.04$ , Fig1B). Similar results were obtained for the standard deviation of peak segmental strain (Fig 1 C,D).

Conclusions: In rats, similarly to humans, LV synchrony is sensitive to pacing location. RVa pacing induces marked LV dyssynchrony. LVpb pacing is more favorable as it induces markedly less LV dsynchrony. Thus, rodent pacing appears to mimic important features seen in humans and large animals. This model has the potential to become a reliable tool for pacing related studies in small animals.

Group A (n=6)



Group B (n=5)

