A novel technology for heart failure that utilizes the respiratory effort to shift fluids from the lung and alleviate cardiac workloads; feasibility study in sheep

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Funding Acknowledgements: Type of funding sources: Private company. Main funding source(s): Levron Cardiovascular Ltd, Yokneam Ililt, Israel

Introduction: Hemodynamic congestion is governed by inflow from RV and drainage through LV. However, ventricle functions are affected by the intrathoracic/pleural pressure: Negative pleural pressure (PPL) increases RV outflow while decreasing LV outflow, and vice versa at high PPL. Cardiac decompensation is associated with an increase in the respiratory effort (the peak-to-peak changes in PPL). We suggest to utilize this respiratory effort to shift fluid out of the lung and alleviate hemodynamic congestion. It is done by pacing the heart when PPL is high and the LV stroke volume is larger than the RV stroke volume.

Purpose: To test the feasibility of the novel technology in sheep. To verify that it is possible to modify the distribution of the heart beat within the respiratory cycle (slightly modify the rhythm according to the PPL) with insignificant effect on mean heart rate.

Methods: The utility was validated in sheep (N=9), under spontaneous breathing before and after induction of acute infarction by balloon inflation in the mid-LAD artery. The device senses the intracardiac electrical activity and respiratory effort and paces the right atrium when the PPL is high. The R-R interval of the paced beats was shorter than the sinus R-R interval by less than 10%. The effects of PPL and device pacing on LV and RV stroke-volumes were derived from simultaneous measurements of RV and LV pressure-volume loops, utilizing two impedance catheters (CD-Leycom) within both ventricles.

Results: The respiratory effort had significant effects on RV and LV outputs before and after infarction. The stroke volumes of the two ventricles were in antiphase! At low PPL the RV output increased while the LV decreased by about 10%, and the opposite occurred at high PPL. The pacing increased cardiac beating probability at high PPL. Sequential pacing for one minute increased the systolic pressure by 5 mmHg while decreasing the pulmonary artery pressure. Based on the pressure-volume loops, each paced breath cycle increased the LV outflow by 1.14 ml and decreased RV outflow by 0.36 ml, yielding a shift of 1.5 ml from the lung. Interestingly, it was associated with a decrease in the sinus rate, which further decreased cardiac beating at low PPL and may represent a decrease in sympathetic tone.

Conclusions: Pacing only 1% of the ~20,000 daily breath-cycles can shift 300 ml from the lung per day! The technology is agnostic of the heart-failure etiology. Preclinical studies in a model of chronic heart failure are required to further validate the novel approach.