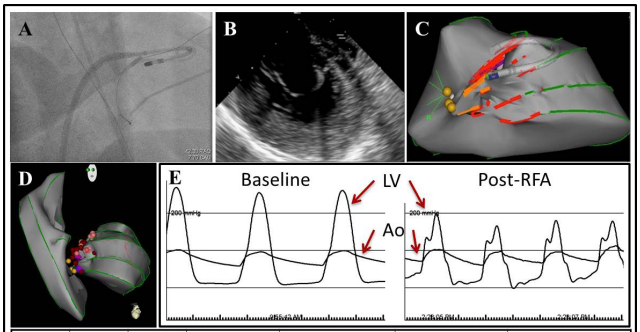

F Patient	Age	Gender	Type of Hypertrophy	Baseline Measurements		Non-invasive LVOT Gradient (mmHg)		Invasive LVOT Gradient (mmHg)	
				EF (%)	Septal Thickness (mm)	Baseline	Post-RFA	Baseline	Post-RFA
1	68	F	Asymmetric LVH	65	16	122	15	150	70
2	48	F	Asymmetric LVH	68	23	64	12	23	16
3	58	M	Concentric LVH	64	16	16	0	90	30
4	53	M	Asymmetric LVH	58	26	69	25	45	35
5	78	F	Asymmetric LVH	42	18	57	0	73	59

Panel A: Fluoroscopic view atrial transseptal access for LV septal ablation. Panel B: Intraprocedural ICE demonstrating ablator positioning. Panel C: 3D electroanatomic map with catheter positioned at the LV septum. Panel D: Final lesion set with RV and LV maps. Panel E: Baseline and post-RFA LVOT pressure recordings demonstrating decreased gradient. Panel F: Demographic and hemodynamic data for each patient

B-MP02-04

EARLY FEASIBILITY USING A NOVEL NEUROMODULATION DEVICE TO INCREASE INOTROPY

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Background: Acute decompensated heart failure (ADHF) is a leading cause of mortality and morbidity, and results in approximately one million hospital admissions annually in the United States. Symptom relief remains the current standard of care and new treatments are needed to address this condition. Cardionomic, Inc. (New Brighton, MN) is developing a system to increase left ventricular (LV) contractility via endovascular stimulation of cardiac autonomic nerves.
Objective: To investigate the feasibility of increasing LV contractility by electrical stimulation of cardiac autonomic nerves via a catheter-based endovascular approach.
Methods: An ongoing early feasibility study enrolled 29 HF subjects undergoing a catheterization procedure or ICD/CRT device implant. A Cardionomic or commercial catheter was delivered to the pulmonary artery via a jugular approach to stimulate cardiac autonomic nerves. Left ventricular pressure was measured using a Millar catheter and aortic pressure was measured using a fluid column. The maximum first derivative of LV pressure (max dP/dt) was used as a contractility measure. Refinement of the stimulation target was conducted in the first 15 subjects. Here we report on responses to stimulation in the second set of subjects (n=14).
Results: Of the 14 subjects, 1 was not studied. An increase in LV contractility of >5% as compared to a non-stimulation baseline was observed in 10/13 subjects. When evaluating maximum responses by subject, max dP/dt increased by an average of 29.9%, heart rate by 1.6%, and mean arterial pressure (MAP) by 16.6%. The average stimulation duration was 11.8 minutes. In 7 of these 10 subjects, stimulation was maintained for at least 10 minutes (mean 36.6 minutes). For these subjects, max dP/dt increased by an average of 27.6%, heart rate by 2.6% and MAP by 22.4%.
Conclusion: These feasibility study results show that stimulation of cardiac autonomic nerves can selectively increase cardiac contractility with minimal increases in heart rate. The effect can be maintained during the stimulation period and results in an increase in MAP. The improvement in hemodynamics observed with this therapy represents a step toward identifying new treatments for patients with ADHF.

B-MP02-05

CHRONIC INTERMITTENT LOW-LEVEL TRANSCUTANEOUS VAGUS NERVE STIMULATION REVERSES CARDIAC REMODELING IN A RAT MODEL OF HEART FAILURE WITH PRESERVED EJECTION FRACTION

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Background: Inflammation and cardiac fibrosis contribute to ventricular stiffness and diastolic dysfunction and play central roles in the development of heart failure with preserved ejection fraction (HFpEF). Studies showed that low-level transcutaneous vagus nerve stimulation (LLTS) significantly suppresses systemic inflammation and cardiac fibrosis.
Objective: This study aimed to investigate whether chronic intermittent LLTS could suppress cardiac fibrosis and reverse diastolic dysfunction in a rat model of HFpEF.
Methods: Forty-eight Dahl salt-sensitive (DS) rats at 7 weeks of age were randomized into the low salt (LS) group (0.3% NaCl diet, n=12), the high salt (HS) sham group (4% NaCl diet with sham LLTS, n=18) and the HS active group (4% NaCl diet with active LLTS, n=18). After 6 weeks of HS diet, LLTS (20Hz, 2mA) was delivered to bilateral auricular concha region (active LLTS) or auricular margin (sham LLTS) for 30 minutes daily over a 4-week period.
Results: After 6 weeks of HS diet, rats developed hypertension and left ventricular hypertrophy compared to LS rats (129±15 mmHg vs. 114±17 mmHg; p=0.03 and 2.3±0.2 mm vs. 2.0±0.1 mm; p=0.001, respectively). At the end of 4 weeks, chronic intermittent LLTS significantly attenuated the blood pressure elevation (SBP: 125 ± 22 mmHg vs. 157± 9 mmHg, DBP: 88±24 mmHg vs. 115±23 mmHg; both P<0.05). In addition, LLTS prevented the deterioration of diastolic function as assessed by the E/A ratio (1.50±0.12 vs. 1.67±0.29, P<0.05), E/e' ratio (8.13±1.65 vs. 11.08±3.02, P<0.05) and left ventricular diastolic strain (-24.19±4.81% vs. -19.78±3.18%, P<0.05) in the HS active group as compared to those in the HS sham group, without a change in left ventricular ejection fraction. Left ventricular fibrosis was decreased in the HS active group (2.5±1.2% vs. 4.0 ± 2.2%, P<0.05) compared to the HS sham group, to the levels seen in the LS group (2.3±1.5%).
Conclusion: Chronic intermittent LLTS significantly improves diastolic dysfunction, decreases blood pressure and reverses cardiac fibrosis in a rat model of HFpEF, suggesting that LLTS may be used clinically as a novel noninvasive neuromodulation therapy to treat patients with HFpEF. Further studies to examine the efficacy of this novel treatment in patients with HFpEF are warranted.