diastolic dysfunction, the corresponding BNP levels were lower following sildenafil therapy (193 ± 253 to 141 ± 192 pg/mL, p<0.001).

Echocardiographic Parameter observed	n	Before sildenafil	After sildenafil	P value
Left Atrial diameter (diastolic)	64	3.72 cm	3.83 cm	<0.05
Left Atrial Area	57	18.0 cm sq	19.1cm sq	<0.05
Left Ventricular Internal Diameter (diastolic)	72	3.89 cm	4.07 cm	<0.05
Left Ventricular Monoplane Volume (diastolic)	71	69.1 ml	76.4 ml	<0.05
Left Ventricular Ejection Fraction	76	58%	57%	NS
Right Ventricular Systolic Pressure	69	78 mm Hg	75 mm Hg	NS
Tricuspid Regurgitant Jet Velocity	66	421 cm/ second	415 cm/ second	NS

NS = Not Significant.

Conclusion: In patients with PAH, sildenafil therapy provided no significant reduction in estimated pulmonary artery systolic pressure but noticeable improvement in left-sided chamber dimensions and corresponding lower BNP levels, implying an improvement in preload delivery rather than directly reducing pulmonary artery pressures.

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Acute Effects of Biventricular Versus Right Ventricular Pacing on Left Ventricular Function and Twist Assessed by Magnetic Resonance Imaging in a Canine Post Myocardial Infarction Model of Cardiomyopathy

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The acute effects of cardiac resynchronization therapy on left ventricular (LV) function have not been fully elucidated. In order to experimentally assess the acute effects of resynchronization, we developed a canine model of chronic myocardial infarction (MI) with right ventricular pacing (RVP) to simulate left bundle branch block. We first ligated the left anterior descending coronary artery and all visible collateral vessels and implanted a biventricular pacing (BVP) system under open-chest conditions in six mongrel dogs. At one week post-MI, we ablated the His bundle and initiated RVP. At two weeks post-MI, the LV ejection fraction (EF) was uniformly <35% during RVP. At this point, we performed cardiac magnetic resonance (MR) imaging during both RVP and BVP and assessed LVEF and torsional rotation (twist). Twist is the normally counterclockwise systolic rotation of the apical portion of the LV that increases the efficiency of ejection. Twist recoil during diastole is thought to be an important mechanism of diastolic filling. Twist was estimated from tagged MR images as endocardial angular strain. During RVP, LVEF was .21 ±X (±SE). LVEF was .25 ±X during BVP, but this difference was not statistically significant. During RVP, anteroseptal twist was clockwise, opposite of the normal direction, and averaged +4 ±X° . BVP reduced dyssynchrony and on average restored the normal direction of twist, resulting in a mean value of -1±X°(p <.01 vs RVP). We conclude that in this model of MI with reduced LVEF, RVP causes reverse twist. Acutely, BVP does not significantly improve LVEF but normalizes twist direction. Thus, BVP may have beneficial acute effects even in the absence of significant changes in EF.

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WITHDRAWN

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The Determinants of Right Ventricular Failure in Patients Admitted with Acute Left Heart Failure

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Objectives: In this present study we sought to analyze the prevalence and determinants of right ventricular (RV) failure in patients admitted with acute left ventricular failure. **Background:** RV failure has been shown to be a strong predictor of poor outcome. It has been shown that pulmonary hypertension, which may lead to RV failure, is not related to the degree of LV systolic dysfunction, but is strongly associated with diastolic dysfunction, suggesting that the severity or the degree of diastolic dysfunction may correlate better with RV failure. This study aims to investigate this correlation. **Methods:** 120 consecutive patients admitted with left heart failure were retrospectively studied to evaluate the prevalence of right ventricular failure and the variables associated with it; RV failure was defined as RV dilatation with or without RV hypokinesis on transthoracic 2-dimentional echocardiography. Systolic pulmonary artery pressure (using tricuspid regurgitant velocity), and left ventricular systolic and diastolic function, significant valvular lesions , cardiac chambers sizes in addition to other laboratory values were quantified. **Statistical Analysis:** Patients were divided to 2 groups: Group1 with RV failure (#50)and Group 2 (#70)without evidence of RV failure. Results are presented as mean value ± SD or percentages. Groups' comparisons were performed using a standard *t* test. **Results:** 50 out of the 120 patients were found to have evidence of RV failure (prevalence: 42%). Patients in the RV failure group were more likely to be males(66% vs. 40% ,p:0.0047),and characterized by higher LV diastolic degree(2.2 ± 0.6 vs. 1.84 ± 0.7 ,p: 0.0070), higher pulmonary artery systolic pressure(57.8mmhg±15.3 vs. 50.14mmhg±12.1 , p:0028),larger LV systolic internal dimension(4 ± 1.2 vs. 3.48 ± 1, p:0.0159), in addition , there was a higher prevalence of right atrial enlargement (92% vs18% , 0.000048) and higher prevalence of tricuspid regurgitation(58% vs. 27.1%, p:0.0006) in this group. **Conclusions:** RV failure is frequent finding in patient who present with left ventricular failure and is strongly associated with the severity of left ventricular diastolic dysfunction.

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Differences in Right Ventricular Function between Ischemic and Nonischemic Cardiomyopathy

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Background: Previous studies have suggested that right ventricular (RV) dysfunction is more pronounced in patients with ischemic cardiomyopathy (ICM) than those with nonischemic cardiomyopathy (NICM). Our purpose was to use tissue Doppler echocardiography to compare RV function in patients with ICM and NICM. **Methods:** Study population consisted of 43 heart failure patients (mean age 57 ± 16; 32 males; 22 ICM and 21 NICM; LV ejection fraction 22 ± 5) who underwent color tissue Doppler imaging of the RV as well as right heart catheterization within 24 hours of each other. All patients had a clinical history of congestive heart failure and ejection fraction was < 35%. RV peak systolic myocardial velocity was measured at the tricuspid annulus and the basal and mid-ventricular segments of the RV free wall and averaged together to obtain a global measure of RV function (RV S_m). **Results:** ICM and NICM patients were not significantly different from each other in age, gender, New York Heart Association functional class, or LV ejection fraction. Almost all patients (86%) were in functional class III or IV. RV myocardial systolic velocities were significantly decreased in ICM compared to NICM at the tricuspid annulus, basal free wall, and mid-ventricular wall (see table). RV S_m, our composite measure of RV function, was significantly decreased in the ICM group compared to NICM. There were no differences between the ICM and NICM groups in mean pulmonary artery pressure, pulmonary vascular resistance, pulmonary capillary wedge pressure, or Fick cardiac output. **Conclusion:** RV dysfunction as determined by tissue Doppler echocardiography is more pronounced in ICM patients versus NICM. This finding is not attributable to differences in either pulmonary artery pressures or cardiac output between the 2 groups.

	ICM (n = 22)	NICM (n = 21)
Tricuspid annulus (cm/s)	5.5 ± 2.2	7.5 ± 1.9*
RV base (cm/s)	4.7 ± 2.1	6.3 ± 2.3†
RV mid (cm/s)	3.5 ± 1.9	5.5 ± 2.2*
RV Sm (cm/s)	4.5 ± 1.9	6.4 ± 2.1*

* p < 0.01 NICM versus ICM; † p < 0.05 NICM versus ICM.

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Progressive Decline in Circulating CNP with Aging Is Associated with Progressive Cardiac Fibrosis and Myocardial Impairment

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Background: Cardiac aging is associated with altered myocardial structure and function that may contribute to the development of HF. C-type natriuretic peptide (CNP) is of endothelial cell origin and represents the most potent anti-fibrotic peptide of the NP family. CNP activates the NP receptor-B (NPR-B),which is found in high abundance in cardiac fibroblasts. Further, selective cardiac knockout of NPR-B contributes to exaggerated cardiomyocyte hypertrophy in response to pressure overload. In addition, infusion of CNP suppresses post-MI induced cardiac fibrosis in a rodent model. The impact of aging on plasma CNP and associated left ventricular (LV) structure and function are undefined. **Objective:** We hypothesized that a decline in plasma CNP occurs with aging and is associated with an increase in LV fibrosis and altered LV function and structure. **Methods:** Studies were performed in 2,11 and 20 month old male Fischer rats (n=8/group). Echocardiography was used to assess LV structure and function. Left ventricles were harvested for analysis. Plasma CNP and BNP were measured. **Results:** Aging from 2 to 20 months (equivalent to human aging from childhood to the 6th decade) was associated with progressive reductions in plasma CNP. Specifically, there was a significant incremental decrease in plasma CNP in the 2 month (30 ± 3pg/ml) to the 11 month (21 ± 1pg/ml) to the 20 month groups (9 ± 1pg/ml). Significant and progressive LV fibrosis was observed with aging (9 to 15 to 21%,p<0.001). Importantly, LV fibrosis was inversely correlated with plasma CNP levels. In contrast, plasma BNP was slightly but significantly increased from the 2 month to 20 month groups (21 ± 2 to 26 ± 1pg/ml,p<0.05). Finally, the decrease in plasma CNP, seen from the 2 month to the 20 month groups, was also associated with a significant reduction in LV weight to body weight ratio (2.24 ± 0.02 to 1.79 ± 0.03,p< 0.001) and EF (88 ± 1 to 80 ± 1%,p< 0.001) and increases in LV end-diastolic chamber diamater (6.61 ± 0.09 to 7.48 ±