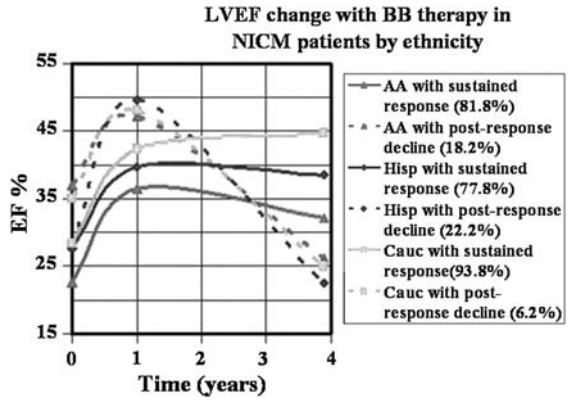

LVEF decline after initial response to BB between African American (AA), Hispanic and Caucasian patients with HF and non-ischemic cardiomyopathy (NICM). **Methods:** 22 AA, 18 Hispanic, and 16 Caucasian patients with LVEF ≤ 40% and NICM whose LVEF rose ≥ 5% after 1 year of BB were selected from the HF Clinic at AECOM. Post-response LVEF decline ≥ 5% to a final LVEF ≤ 35% was evaluated. LVEF was determined from the modified Simpson's Rule using 2D echocardiography. **Results:** Shown in the graph are AA, Hispanic, and Caucasian NICM patients with and without post-response LVEF decline. 35.7% of patients were treated with metoprolol and 64.3% with carvedilol. The mean nadir LVEF of the 16.1% of patients with post-response LVEF decline was 24.4 ± 3.0 at a mean interval of 3.9 ± 2.4 years after initial LVEF. No significant differences between ethnicities were observed in the baseline characteristics or the proportion of patients with post-response LVEF decline (p=0.429). **Conclusion:** A significant proportion of AA (18.2%), Hispanic (22.2%), and Caucasian (6.2%) patients with NICM whose LVEF rose in response to 1 year of BB experienced a subsequent LVEF decline. The proportions of patients with post-response decline between ethnic groups were not significantly different.

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Experiment with a New Drug (TAMERIT) of Treating MI Which Protects the Myocardium and Prevents Heart Failure

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Prevalence of a Myocardial Infarctionn(MI) in Russia, according to Institute of cardiology of A.L.Mjasnikova (2005), makes 3,9 % among men and 2,2 % among women that deduces it on one of the first places in the world. It is thus underlined that from MI in Russia 330 men and 154 women on 100 thousand population annually die which is about3,2 times the values in the USA. And about 20 − 25% of the patients who suffered MI end up with Heart failure in the fomr of Ischemic Cardio myopathy. These circumstances testify to a high urgency of the problem, in particular its treatments. As per the recommendations of the European Cardiological society and AHA (2000) and also the recommendations of Russian Cardiological associations, the treatment for MI is based on using Inhibitors of Thrombin, Anti-thrombotic agents (like aspirin, GCp IIb/IIIa receptor blockers along with Beta-blockers, Nirates and Calcium channel blockers. In this study on experimental animals we have created models of MI in white rats and have shown the protective effects of TAMERIT (A phthalhydrazine derivative) on the Myocardium and how it potentially could prevent Ischemic Cardiomyopathy. Tamerit is a potent Immunomodulator and it prevents the hyperactivity of the Macrophage and results in the Normal Natural Killer mechanism of the Neutrophils. We have demonstrated the Protective effects of Tamerit on the Myocardium. Rats which were not treated by Tamerit developed Transmural Infarctions and those which were treated developed only a streak of Sub-Epicardial Necrosis And in rats, besides the Complete Blood picture, we also investigated the activity of CPK, ASAT, LDH Fractions before and during the process of necrosis and apoptosis (in 1, 5 and 7 days).

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Initial Observations of Intravenous CD-NP, Chimeric Natriuretic Peptide, on Renal Functions in Chronic Heart Failure Patients

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Background: Nesiritide, a natriuretic peptide with arterial vasodilator property, has been associated with worsening of renal function. CD-NP is a novel chimeric natriuretic peptide engineered with venodilatory and renal protective properties. The effects of CD-NP in patients (pts) with chronic heart failure (HF) are unknown. Accordingly, the purpose of this study was to evaluate the effects of increasing doses of CD-NP on diuresis in HF pts. **Methods:** Chronic HF pts with systolic blood pressure ≥ 110 mmHg and EF < 40 % with signs and symptoms of HF (NYHA class 2/3) received escalating doses of CD-NP infusion for 24 hours in this open-label trial. Daily medications, including furosemide and vasoactive medications, were administered to pts on the day prior to and withheld on the day of CD-NP infusion. **Results:** Eighteen HF pts completed the CD-NP infusion (6 at 3 ng/kg/min, 6 at 10 ng/kg/min, and 6 at 20 ng/kg/min). The mean age was 56 ±13 years and 85% were men. CD-NP effect on blood pressure was dose dependent. The maximum tolerated dose was 20 ng/kg/min. Renal function was significantly improved following treatment with CD-NP (table). Improvement of renal function and filtration markers was consistent across the doses up to 20 ng/kg/min of CD-NP. CD-NP's effect on diuresis was observed for all doses. While the comparison is limited by the non-randomized temporal sequence, the volume of diuresis induced by CD-NP was comparable to that induced by physician-selected doses of furosemide over a 24 hour period. **Conclusions:** In chronic HF pts, 24-hour infusion of CD-NP statistically improved renal filtration markers. The extent of CD-NP induced diuresis was comparable to furosemide induced diuresis, without worsening renal function. Additional trials are needed to confirm these findings

Chronic HF Patients (n=16)	Baseline (furosemide day)	Combined CD-NP Doses (without furosemide)	P value
CrCL (mL/min, CockCroft-Gault)	119 ± 46	129 ± 48	P < 0.01
Creatinine (mg/dL, plasma)	1.09 ± 0.4	0.99 ± 0.4	P < 0.01
Cystatin (mg/dL, plasma)	1.10 ± 0.39	1.03 ± 0.35	P < 0.01
Absolute Urine Volume (mL, 24-hour urine collection)	2217 ± 754	2738 ± 1274	P = 0.14

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Nesiritide Worsens Renal Insufficiency in Right Ventricular Failure

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Background: Little literature exists regarding ADHF (Acute Decompensated Heart Failure) due to isolated right ventricular (RV) failure. These patients are particularly challenging due to the strong correlation between RV failure and renal insufficiency. Administration of diuretics in these patients often leads to renal vasoconstriction and secondary deleterious effects upon renal function. Nesiritide was shown to have beneficial effects on heart failure symptoms as well as cardio-renal hemodynamics such as reduction in mean wedge pressure, central venous pressure and systemic vascular resistance. The drug also helps to increase sodium and water excretion. Thus, we hypothesized that nesiritide would have a beneficial effect on renal function in a subset of patients with RV failure, renal insufficiency and ADHF. **Methods:** Accordingly, a retrospective study of 15 patients with RV failure and normal LV systolic function documented by trans-thoracic echocardiogram was undertaken to explore the use of nesiritide in RV failure and renal insufficiency. Between December 2003 and March 2009, 11 women and 4 men with right ventricular heart failure secondary to pulmonary hypertension, normal left ventricular ejection fraction and renal insufficiency (mean serum creatinine 1.6 ± 0.12 mg/dL) were admitted at Montefiore Medical Center for ADHF and treated with IV Nesiritide. Serum creatinine at baseline, 3 days, time-of-discharge, and one month post nesiritide infusion was assessed.

Serum creatinine levels with nesiritide infusion in RV failure

	Creatinine at baseline (mg/dL)	Creatinine at 3 days (mg/dL)	Creatinine at discharge (mg/dL)	Creatinine at 1 month (mg/dL)
Baseline	1.6 ± 0.12	1.82 ± 0.21	1.93±	2.07 ± 0.21
p-value (compared to baseline)		0.564	0.062	0.046*

Results: Baseline average serum creatinine was 1.6 ±0.12 mg/dL on admission while average creatinine at the one month time point was 2.07 ± 0.21 mg/dL. Patients with ADHF due to RV failure showed a significant increase in serum creatinine (p = 0.046) when treated with nesiritide. The mean nesiritide time administration was 5.6 ± 1.09 days. **Conclusions:** While our study is limited by its retrospective nature and small sample size, caution should be exercised when using nesiritide in patients with RV failure and renal failure.