associated with a higher prevalence of atherosclerosis. We evaluated the relationship of LVH to smoking.

We studied 82 untreated patients with essential hypertension. They were divided into smokers (26 men and 6 women), and non-smokers (23 men and 27 women). Their median age was 52 (range: 18 – 73) years (the two groups didn’t differ significantly). All patients had a full lipid profile, a blood count and a two-dimensionally directed M-mode echocardiography.

Both IVS and left ventricular PW of smokers were significantly thicker than those of non-smokers (11.0±1.5 vs 10.1±1.2mm; p<0.01 and 10.7±1.5 vs 9.9±1.2mm; p<0.05, respectively). There was no significant difference in blood pressure between smokers and non-smokers, either in systolic (SBP) or in diastolic blood pressure (DBP) (157±18 vs 161±23mmHg and 97±8 vs 99±12mmHg, respectively).

In addition, non-smokers had significantly higher high density lipoprotein (HDL) than smokers (49±13 vs 41±10mg/dl; p<0.05). There was no other significant difference in the rest lipid profile between the two groups of patients. Smokers had also significant higher hematocrit when compared with non-smokers hypertensives (45.9±3.7 vs 42.5±4.7%; p<0.01).

Conclusion: Our study suggests that smoking is associated with increased left ventricular thickness, independently of the blood pressure of untreated hypertensive patients.

Key Words: Smoking, Left Ventricular Hypertrophy, Essential Hypertension

Vascular remodeling and endothelial dysfunction seems to be associated with LV remodeling pattern, being more severe in concentric LVH.

Key Words: Left Ventricular Hypertrophy, Intima-Media Thickness, Endothelial Function

P-147
THE EFFECT OF MILDRONATE ON AUTONOMIC NERVOUS SYSTEM FUNCTION IN PATIENTS WITH HEART FAILURE
Daina Veita, Daina Bojare, Ite Skarda, Indulis Kukulis, Latvian Cardiological centre; Latvian institute of Cardiology, Riga, Latvia; PJSC Grindeks, Riga, Latvia, Latvia; PJSC Grindeks, Riga, Latvia; Latvia; Latvian Cardiological centre, Latvian institute of Cardiology, Riga, Latvia, Latvia.

Chronic congestive heart failure is connected with increased sympathetic nervous system activity and decreased parasympathetic nervous activity - that contributes to the progression of disease and increase of mortality. It is very important to search new drugs for improvement of sympatho-parasympathetic activity in heart failure. Mildronate has been synthesised at Latvian Institute of Organic Synthesis. It is metabolic drug that modulates individual stages of energy metabolism in myocardium. Mildronate belongs to the group of cytoprotectors/antihypoxants and ensures cellular protection and energy supply in conditions of ischemia and high stress.

Objective: To investigate the effect of Mildronate upon cardiac baroreflex function in heart failure.

Design and Methods: 60 pts. with stable heart failure/no changes in medical therapy within the last 2 months, aged 51-76 yr., II–III class (according to NYHA) were randomly assigned in double-blind fashion to receive either 1000 mg of Mildronate or placebo. At the beginning of study and after 12 weeks of treatment heart-beat- to- beat heart rate and finger mean arterial pressure were monitored non-invasively. Bradycardic and hypotensive reaction to carotid baroreceptors activation were studied, applying neck suction (-60 mmHg for 5s).

Results: Mildronate (dose 1000 mg/day) in addition to standard therapy is associated with no significant decrease of mean arterial pressure, diastolic blood pressure and heart rate. Systolic blood pressure (p<0.05) decreases and bradycardic and hypotensive reactions to carotid baroreceptors activation increase (p<0.01) significantly after Mildronate therapy.

Conclusions: 12-week treatment period with Mildronate significantly improves sympatho-parasympathetic balance in patients with stable heart failure.

Key Words: Baroreceptor Reflex, Heart Failure, Autonomic Nervous System

P-147A
ATRIAL NATRIURETIC PEPTIDE DOSE DEPENDENTLY INHIBITS CARDIAC REMODELING AFTER EXPOSURE TO PRESSURE OVERLOAD
Veronica Franco, Yiu Fai Chen, Ji An Feng, Fadi Hage, Suzanne Oparil, Gilbert Perry. Vascular Biology and Hypertension Program, Division of Cardiovascular Disease, University of Alabama at Birmingham, Birmingham, AL.

Background: We have previously shown that mice with homozygous deletion of the pro-atrial natriuretic peptide (ANP, Nppa−/−) gene exhibit left ventricular (LV) hypertrophy at baseline and exaggerated hypertrophy after pressure overload induced by transverse aortic constriction (TAC). Nppa−/− mice have increased expression of extracellular matrix molecules, i.e. metalloproteinase-2 and tissue inhibitor of metalloproteinase-3 and cardiac remodeling after TAC.

Hypothesis: A single copy of the pro-ANP gene (Nppa+/−) will not be adequate to protect heterozygous mice against exaggerated LV hy-
pertrophy resulting from pressure overload stress. Therefore, Nppa+/− mice will have exaggerated cardiac remodeling compared to wild type Nppa+/+ mice after pressure overload stress. Cardiac remodeling in Nppa+/− mice will be less marked than in Nppa+/− animals.

Methods: Nppa+/−, Nppa−/− and Nppa+/+ mice were subjected to TAC. Mice were fed a normal NaCl diet. One week after TAC, echocardiography was performed, hearts were removed and weighted.

Results: LV weight and LVEDD were compared by ANCOVA with body weight as a covariate. LVEF was compared by two-way ANOVA. Nppa+/− mice exhibit exaggerated LV hypertrophy compare to Nppa+/+ mice, but less than the Nppa−/− mice. Cardiac remodeling after pressure overload is negatively associated with ANP gene load since Nppa−/−, but not Nppa+/−, mice have signs of LV dysfunction and failure.

Conclusion: The heterozygote is a novel model for studying LV hypertrophy and the transition to failure during pressure overload stress. It is less abnormal than Nppa−/− at baseline and may more closely resemble the human condition.

Key Words: Atrial Natriuretic Peptide, Cardiac Remodeling, Cardiac Hypertrophy