collagen degradation (ICTP) in hypertensive patients with a high prevalence of left ventricular hypertrophy (LVH).

**Methods:** Forty-one patients (19 females) from the LIFE study aged 69 years (46-80) with ECG-determined LVH were examined in baseline after 14 days on placebo treatment. BAC and BAD were measured using a volume-oscillometric metric (Artcomp®, Critikon®). Atrial and brain natriuretic peptide (Nt-proANP and Nt-proBNP) were measured by immunonassay (Elecsys®, Roche Diagnostics®). Carboxyterminal telopeptide of type I procollagen (ICTP) was measured by immunoassay.

**Results:** BP was 164±28/89±16 mmHg and HR 69±12 bpm. BAC was 22±7.1 μl/mmHg/10 cm and BAD was 17±5.9 10^3 mmHg^-1 at a transmural pressure of 0 mmHg. Nt-proANP was 1406±642 pmol/l and Nt-proBNP was 65±10 pmol/l. ICTP was 3.8±1.7 μg/l. Nt-proBNP correlated to BAC and BAD (r = -0.32, p<0.05 and r = -0.25, ns). Nt-proANP correlated only insignificantly to BAC and BAD (r = -0.19 and r = -0.17, both ns). No relation was seen between the neurohormones and the arterial volume. ICTP correlated to BAC and BAD (r=0.25 and r=0.30, both p<0.05).

**Conclusion:** The negative correlation observed between BAC and Nt-proBNP might reflect the known association between neurohormones and wall stress in the left ventricle. The absent correlation between neurohormones and arterial volume could indicate a more important relation to arterial wall structure rather than arterial diameter at the relaxed state of the vessel as investigated in this study. The correlation between BAC, BAD and ICTP might represent a relation where the degradation of vascular collagen has an impact on arterial compliance.

Key Words: Arterial Compliance, Collagen, Neurohormones

**P-38 MP-37 CHANGES IN BRACHIAL ARTERY DISTENSIBILITY RELATED TO CV RISK FACTORS IN HEALTHY ADOLESCENTS**

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Autopsies studies in adolescents & young adults have demonstrated the presence of subclinical cardiovascular (CV) disease. In healthy children & adolescents many studies have examined CV risk factors but few have attempted to assess end organ compromise related to CV risk profile. Using the brachial artery as a surrogate of the vascular tree, this study measured brachial artery distensibility (BrachD) in healthy school-aged adolescents & young adults & explored the association of CV risk factors with BrachD. The study population consisted of 976 subjects 17.8 ± 1.8 years of age (range 13-22 years; 45% male; 53% Caucasian, 46% African-American; 95% completed puberty; all fasting blood sugars <100 mg/dl). Written informed consent was obtained followed by collection of demographic, anthropometric & laboratory data. After 5 minutes of rest, trained personnel obtained 3 measures of BP & brachial artery pressure curves with a Dynapulse Pathway instrument (PulseMetric, Inc, San Diego, CA). The curves were uploaded to the on-line automated system for calculation of BrachD via the technique of pulse wave form analysis. The calibrated pressure wave is incorporated into a physical model of the CV system assuming a straight tube brachial artery & T-tube aortic system. This instrument has been previously validated with proven reproducibility. BrachD was normally distributed with an average of 6.26 ± 1.67 mmHg^-1. As seen previously, females had greater distensibility (6.54 ± 1.75 vs 5.91 ± 1.49, p<0.0001). There were no differences by age or race. BrachD was lower in overweight (BMI>85%) subjects (5.66 ± 1.39 vs 6.56 ± 1.72, p<0.0001). Correlation analyses showed decreased BrachD in males & subjects with larger body size, greater SBP, Pulse Pressure (PP), fasting glucose & log of fasting insulin (p<0.0001). After adjusting for distending pressure (PP), significant correlations remained between BrachD & gender, adiposity, DBP & fasting glucose (p<0.05). Non-invasive measures of BrachD are normally distributed in a healthy, school aged population. However, lower levels of Brachial Artery Distensibility are found in subjects who are male, obese, & have greater DBP & insulin resistance. Therefore, adverse levels of CV risk factors affect vascular function even at a young age.

Key Words: Brachial Artery, Cardiovascular Risk Factors, Vascular Function

**P-39 LONG-TERM SILDENAFIL ADMINISTRATION IMPROVES AORTIC STIFFNESS IN PATIENTS WITH ERECTILE DYSFUNCTION OF VASCULAR ORIGIN**

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Aortic stiffness is an important factor for the performance of cardiovascular system and an important prognosticator of cardiovascular risk. Erectile dysfunction occurs in the setting of endothelial dysfunction and is often the first manifestation of atherosclerotic disease. We have previously shown that sildenafil, a phosphodiesterase type-5 inhibitor, has an acute beneficial effect on aortic stiffness. Whether sildenafil has a long-term beneficial effect on aortic elastic properties with chronic daily administration has not been determined.

The effects of a two-week long treatment with sildenafil on aortic stiffness were studied in 9 men (age 57.7±15.6 years) with vasculogenic erectile dysfunction, considered competent to have a safe sexual activity and take PDE-5 inhibitor therapy. The study was carried on two separate arms, one with sildenafil (100 mg) and one with placebo according to a randomized, placebo-controlled, double blind, cross-over design. All measurements were performed 24 hours after the last sildenafil intake. Carotid-femoral pulse wave velocity (PWV) was measured as an index of aortic stiffness using an automated, non-invasive device (Complior®).

Daily sildenafil intake led to a significant sustained decrease in PWV, denoting a decrease in aortic stiffness (ANOVA: P<0.05, figure). In contrast, no changes were noted in systolic and pulse pressure.

This study shows for the first time that chronic treatment with sildenafil has a favourable effect on aortic stiffness in patients with vasculogenic erectile dysfunction. This finding may have important implications in patients receiving sildenafil therapy for erectile dysfunction.

Key Words: Aortic Stiffness, Sildenafil