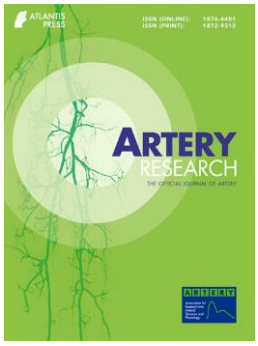

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P.032: GRADED ASSOCIATION BETWEEN LARGE ARTERY STIFFNESS AND ERECTILE DYSFUNCTION SEVERITY AND DURATION IN ESSENTIAL HYPERTENSIVE MEN

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the 3-year follow-up period 38 patients experienced MACE (acute myocardial infarction, coronary intervention, or cardiac death). A receiver operating characteristic curve demonstrated that the best cut-off point of a baPWV for predicting a MACE was 13.35 m/s. A univariate binary logistic regression model demonstrated that only the baPWV had a significant odds ratio for MACE: 2.30 (1.10-4.81), p=0.024. Other risk factors including BP and age (with the cut-off point at the lowest tercile - 49.0 years) were not significantly associated with prognosis.
Conclusions: baPWV is the only significant predictor of MACE for middle aged men with CAD.

P.031
CHRONIC COFFEE CONSUMPTION HAS A LESS POTENT EFFECT ON AORTIC STIFFNESS THAN CAFFEINE

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Purpose: Aortic stiffness is an independent marker of cardiovascular risk. Caffeine, which is the strongest compound of coffee, has been associated with an acute, unfavorable effect on aortic elastic properties. Whether there is a differential chronic effect of coffee and caffeine on aortic stiffness has not been defined.
Methods: The chronic effect of coffee and caffeine were studied in 9 healthy volunteers. The study was carried out on four separate arms: a) triple espresso, b) decaffeinated triple espresso, c) 240mg of caffeine alone (amount contained in a triple espresso) and d) placebo. Each treatment period of two weeks was separated by wash-out periods of one week. Carotid-femoral pulse wave velocity (PWV) was measured as an index of aortic stiffness.
Results: The effect of daily caffeine and coffee intake on PWV is described as response of each variable, where response is defined as net caffeine or coffee minus placebo and decaffeinated coffee respectively values at each time point. PWV was significantly increased with caffeine compared to placebo ($P<0.05$, responses of 0.40 m/sec at 14 days). Furthermore PWV was significantly increased with coffee compared to decaffeinated coffee ($P<0.05$, responses of 0.43 m/s at 7 days and 0.57 m/s at 14 days). Both caffeine and coffee increased PWV, however, the effect of caffeine was more pronounced ($P=0.07$, response of 0.31 m/sec at 14 days).
Conclusions: Both coffee and caffeine increase PWV, however caffeine intake leads to a more potent chronic response. These findings indicate that substances other caffeine may partially counterbalance the chronic unfavourable effects of caffeine on the cardiovascular system.

P.032
GRADED ASSOCIATION BETWEEN LARGE ARTERY STIFFNESS AND ERECTILE DYSFUNCTION SEVERITY AND DURATION IN ESSENTIAL HYPERTENSIVE MEN

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Purpose: Erectile dysfunction (ED) is very common in men with hypertension. Increased arterial stiffness and clinical evidence of ED are both associated with atherosclerosis process. We examined the possible correlations between large artery stiffening and ED severity in essential hypertensive patients.
Methods: Our population of 140 non-diabetic patients with vasculogenic ED and stage I to II essential hypertension was divided into three groups according to carotid-femoral pulse wave velocity (PWV) values, by means of an automated non-invasive device. Group A (PWV< 8.1 m/sec), group B (PWV =8.2-9.4 m/sec) and group C (PWV >9.5 m/sec). Pharmacologically stimulated peak systolic velocity (PSV) of cavernous arteries was used to assess penile vascular damage.
Results: Patients in group C (n=42) compared to subjects in group A (n=55) and B (n=43) had higher systolic BP ($P<0.01$ for all) and longer duration of essential hypertension ($P<0.05$ for all). Moreover, patients in group C had longer duration of ED (3.3 ± 1.7 vs 2.8 ± 1.5 vs 1.7 ± 1.2 years, respectively; $P<0.05$ for all) and lower PSV (29 ± 8 vs 32 ± 10 vs 36 ± 10 cm/s, respectively; $P<0.05$ for all). In the total population, PWV was correlated with PSV (r=-0.29, $P<0.01$), while PSV was associated with systolic BP (r=-0.18, $P<0.05$) and pulse pressure (r=-0.23, $P<0.05$). Analysis of covariance revealed that PSV and ED duration remained significantly different between groups after adjustment for confounding factors ($P<0.05$).
Conclusions: In men with ED and essential hypertension, there is an augmentation in penile inflow insufficiency and longer duration of ED throughout increasing PWV tertiles. These findings suggest that arterial stiffening is closely related to ED processes, in this setting.

P.033
ASSOCIATION BETWEEN ARTERIAL WAVE REFLECTIONS AND PENILE DOPPLER FINDINGS IN HYPERTENSIVE MEN WITH VASCULOGENIC ERECTILE DYSFUNCTION

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Purpose: Hypertension is the most common comorbidity in patients with erectile dysfunction (ED). Wave reflections (WR) are an important predictor of cardiovascular risk. The association of ED with WR in hypertensive patients has not been investigated.
Methods: 88 consecutive non diabetic treated hypertensive patients (61 ± 8yrs) affected by non-psychogenic and non-hormonal ED for more than 6 months were evaluated for penile vascular disease severity by penile Doppler ultrasound. Augmentation index (AIx) and augmented pressure (AP) were measured as an estimate of WR. Subendocardial-viability ratio (SVR) which is the ratio of diastolic pressure time interval/systolic pressure time interval was evaluated as a sensitive measurement of the adequacy of subendocardial muscle perfusion in response to myocardial oxygen demand.
Results: In univariate analysis, a negative correlation between AIx, AP and mean peak systolic velocity (PSV) of cavernous arteries (r=-0.33, $P<0.01$ and r=-0.24, $P<0.05$, respectively) and a positive correlation between SVR and PSV (r=0.28, $P<0.05$) was observed. Furthermore, in a multiple regression model, AIx was significantly associated with penile vascular disease severity ($P<0.01$) after controlling for age, heart rate, height, body-mass index, mean pressure, lipid profile, C-reactive protein, intensity of smoking (pack-years), antihypertensive agents and statines, (adjusted R2 of model: 0.42).
Conclusions: Our study shows that in hypertensive patients with ED, WR correlate significantly with increasing severity of penile vascular disease as measured by penile Doppler. This finding provides further insights into the pathophysiology of ED and may have implications for the cardiovascular risk in these patients.

P.034
THE INFLUENCE OF GENDER, CYCLOOXYGENASE-2 INHIBITION, AND HYPERGLYCEMIA ON VASCULAR FUNCTION IN UNCOMPLICATED TYPE 1 DIABETES MELLITUS

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Purpose: We have previously shown that young women vs. men with uncomplicated type 1 diabetes mellitus (DM-1) exhibit a deleterious renal vasoconstrictive response to clamped hyperglycemia. Since the cyclooxygenase-2 (COX2) system has important systemic hemodynamic effects in DM-1 and is activated by hyperglycemia, we hypothesized we would find a similar gender-based systemic vascular response to selective COX2 inhibition and accentuation during clamped hyperglycemia.
Methods: Outpatient ambulatory blood pressure monitoring and inpatient vascular function assessment (applanation tonometry and brachial artery reactivity) during clamped euglycemia and hyperglycemia were assessed before and after COX2 inhibition (celecoxib 200mg daily for 14 days).
Results: Before COX2 inhibition, women [n=9] vs. men [n=12] with DM-1 exhibited a lower ambulatory systolic BP (114 ± 3 vs. 124 ± 3mmHg, $p=0.029$), which was no longer present after COX2 inhibition. Radial augmentation index was higher before (euglycemia 11.5 ± 4.2 vs. 0.8 ± 3.1%, $p=0.048$; hyperglycemia 10.8 ± 4.5 vs. -3.1 ± 3.6%, $p=0.024$) and after COX2 inhibition (euglycemia 13.1 ± 2.3 vs. -1.3 ± 3.5%, $p=0.005$; hyperglycemia 9.1 ± 2.7 vs. 1.1 ± 4.0%%, $p=0.045$). No differences in pulse wave velocity were detected. Endothelial-dependent FMD responses were also not different, but endothelial-independent GTN response was higher before (euglycemia 14.0 ± 0.2 vs. 10.8 ± 1.0%, $p=0.045$; hyperglycemia 16.6 ± 2.3 vs. 11.5 ± 1.1%, $p=0.045$) and then no different after COX2 inhibition.
Conclusions: COX2 inhibition in women with DM-1 results in a loss of gender-based systolic BP protection. Increased augmentation index in women with