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### **P.032: CORRELATION OF AORTIC STIFFNESS WITH SEVERITY OF ERECTILE DYSFUNCTION**

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(left figure). Habitual and nonhabitual drinkers demonstrated similar changes with caffeine, whereas the effect of coffee (regular: middle figure; or decaffeinated: right figure) was more potent in nonhabitual compared to habitual drinkers. Pressures also increased, however the increase was more potent in nonhabitual drinkers after both regular ( $p < 0.05$ ) or decaffeinated ( $p < 0.01$ ) coffee intake.

**Conclusions:** Both coffee and caffeine increase WR, however drinking coffee leads to a more potent response in nonhabitual drinkers. These findings indicate that substances other than caffeine are partially responsible for the unfavourable effects of coffee on the cardiovascular system.

#### P.031

##### AORTIC STIFFNESS AND WAVE REFLECTIONS ARE ASSOCIATED WITH PENILE DOPPLER FINDINGS IN PATIENTS WITH VASCULOGENIC ERECTILE DYSFUNCTION

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**Background:** Erectile dysfunction (ED) has been reported as the first sign of a generalized vascular disease. Aortic stiffness and wave reflections are independent markers and prognosticators of cardiovascular risk. The association between ED and measures of aortic stiffness and wave reflections has not been investigated.

**Methods:** A total of 107 men with ED were evaluated for penile vascular disease severity by penile Doppler ultrasound: 40 men (aged  $61 \pm 9$  yrs) with coronary artery disease (CAD) and 67 men (aged  $59 \pm 11$  yrs) without CAD. Aortic stiffness was evaluated with carotid-femoral pulse wave velocity (PWV) and wave reflections with augmentation index (AIx) of the aortic pressure waveform using high-fidelity pulse wave analysis.

**Results:** Patients with CAD had decreased peak systolic velocity (PSV) ( $27$  vs  $34$  cm/s,  $p = 0.001$ ), and increased PWV ( $9.0$  vs  $8.4$  m/s,  $p < 0.05$ ) and AIx ( $30$  vs  $24\%$ ,  $p < 0.01$ ) compared with men without CAD. PSV was correlated with age ( $r = -0.24$ ,  $p < 0.05$ ), Framingham risk score ( $r = -0.27$ ,  $p < 0.05$ ), PWV ( $r = -0.31$ ,  $p = 0.001$ ) and AIx ( $r = -0.33$ ,  $p < 0.001$ ). In multivariate linear regression models adjusting for age, height, heart rate, mean pressure and cardiovascular risk factors (BMI, total cholesterol, HDL, logCRP, hypertension, diabetes and intensity of smoking), penile Doppler results were significantly associated with both AIx ( $\beta = -0.265$ ,  $p = 0.004$ ) and PWV ( $\beta = -0.250$ ,  $p = 0.009$ ).

**Conclusions:** Our study shows that aortic stiffness and wave reflections 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.

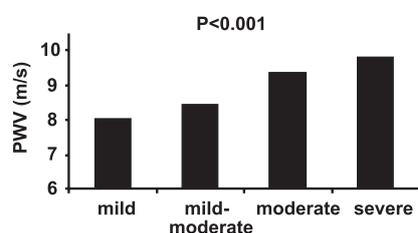
#### P.032

##### CORRELATION OF AORTIC STIFFNESS WITH SEVERITY OF ERECTILE DYSFUNCTION

N. Ioakeimidis\*, C. Vlachopoulos, K. Rokkas, C. Aggeli, D. Tsekoura, N. Alexopoulos, G. Latsios, C. Stefanadis. *1st Department of Cardiology, Athens Medical School, Hippokraton Hospital, Athens, Greece*

**Background:** Accumulating evidence suggests that erectile dysfunction (ED) may be an early manifestation of generalized vascular disease. Aortic stiffness is an independent marker and prognosticator of cardiovascular risk. The association of ED with aortic stiffness has not been defined.

**Methods:** A total of 164 men (mean age  $59 \pm 9$  yrs) affected by non-psychogenic and non-hormonal erectile dysfunction for more than 6 months were studied. All participants were invited to complete a 5-item form of the International Index of Erectile Function (IIEF-5) which is a validated and widely applied method for the evaluation of ED. ED was defined as mild (SHIM score 17-21), mild to moderate (11-16), moderate (8-10) and severe (7 or less). Carotid-femoral pulse wave velocity (PWV) was measured as an index of aortic stiffness using an automated non-invasive device (Complior®).



**Results:** There was a stepwise increase in PWV from mild ED, to mild-moderate and moderate ED and to severe ED ( $p < 0.001$ , figure). In univariate analysis, a negative correlation between PWV and IIEF-5 score was observed ( $r = -0.37$ ,  $p < 0.001$ ). Moreover, in separate backward elimination multiple regression model, PWV was significantly associated with IIEF-5 score ( $b = -0.223$ ,  $P = 0.006$ ,  $R^2 = 0.41$ ), after controlling for age, body-mass index, mean pressure, cholesterol, triglycerides, C-reactive protein, hypertension, diabetes, history of smoking, antihypertensive agents and statines.

**Conclusions:** ED is associated with impaired aortic elastic properties. This finding provides further evidence for the potential link between ED and cardiovascular risk.

#### P.033

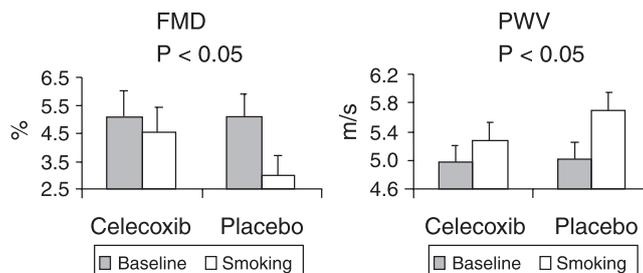
##### SELECTIVE CYCLOOXYGENASE-2 INHIBITION BY CELECOXIB ABROGATES THE ACUTE SMOKING-INDUCED VASCULAR DYSFUNCTION

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**Background:** The cardiovascular toxicity that is associated with cyclooxygenase-2 (COX-2) inhibitors is perhaps not a class effect, but may be rather limited to certain drugs in the class. Endothelial function and aortic stiffness are predictors of cardiovascular risk. The effect of celecoxib, a selective COX-2 inhibitor on acute smoking-induced vascular impairment is unknown.

**Methods:** We studied the effect of 200 mg of celecoxib in 12 healthy smokers (mean age 29.5 years) according to a randomized, double-blind, crossover fashion. Endothelial function and aortic stiffness were evaluated with flow-mediated dilatation (FMD) of the brachial artery and carotid-femoral pulse wave velocity (PWV) respectively. Measurements were done before celecoxib/placebo and immediately after a regular cigarette (tar 14 mg, nicotine 1 mg) that was smoked 3 hours after drug administration.

**Results:** Celecoxib blunted the smoking-induced increase in systolic BP ( $p < 0.05$ ), but not in diastolic BP ( $p = NS$ ). Celecoxib abrogated the smoking-related decrease in FMD (decrease by 2.1 vs 0.6%,  $p < 0.05$ , left figure). Moreover, the increase in PWV after smoking was significantly lower with celecoxib (increase by 0.69 vs 0.29 m/s,  $p < 0.05$ , right figure).



**Conclusion.** Selective COX-2 inhibition by celecoxib abolishes the endothelial dysfunction and aortic stiffening that is induced acutely by smoking. This finding provides further insights into the cardiovascular profile of this drug.

#### P.034

##### ERECTILE DYSFUNCTION IS RELATED TO ARTERIAL STIFFNESS AND MARKERS OF SYSTEMIC VASCULAR INFLAMMATION AND ENDOTHELIAL DYSFUNCTION IN PATIENTS WITH METABOLIC SYNDROME

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**Background:** Erectile dysfunction (ED) has been reported as the first sign of a generalized vascular disease. Arterial stiffness may be an early marker for vascular changes associated with metabolic syndrome (MetS). We evaluated associations between ED, arterial stiffness and markers of systemic vascular inflammation and endothelial dysfunction in patients with MetS.

**Methods:** Two groups of subjects with MetS were investigated: 39 men (mean age: 59 yrs) with ED of vascular origin and 30 men (mean age: 57 yrs) with normal erectile function. Aortic stiffness was evaluated with carotid-femoral pulse wave velocity (PWV) using high-fidelity pulse wave analysis. Plasma levels of interleukin 1 $\beta$  (IL-1 $\beta$ ), tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ) and soluble vascular cell and intercellular adhesion molecules (sVCAM-1, sICAM-1) were measured with ELISA.

**Results:** The mean erectile function score (IIEF-5) was 13 (range 6-20) in men with MetS and ED and 23 (range 22-25) in men with MetS and normal erectile function. ED patients had increased PWV compared to patients