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### **P1.14: PWV IMPROVEMENT IN PREVIOUSLY UNTREATED MILD HYPERTENSIVE PATIENTS AFTER 1 YEAR OF MONOTHERAPY**

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### P1.11 ARTERIAL STIFFNESS AND LEFT VENTRICULAR DIASTOLIC FUNCTION IN TREATED AND UNTREATED HYPERTENSIVES

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The study was aimed to compare arterial stiffness and echocardiographically determined parameters in subjects from general population with treated and untreated hypertension.

**Methods:** We recruited 303 (mean age, 46.9 years). Peripheral and central pulse pressure (pPP; cPP), augmentation index (pAI; cAI) and pulse wave velocity (PWV) were evaluated by means of an oscillometric sphygmomanometer and pulse wave analysis (SphygmoCor). Relative wall thickness (RWT), left atrial (LA) diameter, ascending aorta (AO) diameter, and ratio of early and late diastolic peak of transmitral flow velocities (E/A) as well as ratio of transmitral early filling velocity to tissue doppler early diastolic mitral annular velocity (E/E') were assessed by echocardiography.

**Results:** In the study group, there were 140 normotensives (NT), 61 untreated hypertensives (UTHT), and 102 treated hypertensives (THT). Parameters of interest significantly differed between these groups ( $p < 0.05$ ). In post-hoc analysis with Bonferroni correction, UTHT had significantly higher blood pressure and evaluated target organ damage parameters in comparison to normotensives participants. Despite higher ( $p < 0.05$ ) office (141.6/95.7 vs 135.1/85.9 (mmHg) and 24-h blood pressure (127.5/79.5 vs 121.2/72.6 mmHg) in UTHT, THT had significantly higher pAI, cAI, cPP, PWV, E/E', lower E/A and larger LA ( $p < 0.05$ ) in comparison to UTHT group.

**Conclusions:** Antihypertensive treatment was not associated with less pronounced target organ damage. This may reflect inadequate blood pressure control or too late initiation of antihypertensive therapy which result in progression of arterial and left ventricular stiffening. This also emphasizes the need of early diagnosis of elevated BP and early introduction of appropriate therapy.

### P1.12 TETRAHYDROBIOPTERIN (BH<sub>4</sub>) IMPROVES ENDOTHELIAL FUNCTION, BUT NOT AORTIC STIFFNESS IN PATIENTS WITH RHEUMATOID ARTHRITIS

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**Background:** Rheumatoid arthritis (RA) is a systemic inflammatory condition associated with increased cardiovascular risk. The aetiology is most likely multi-factorial, including endothelial dysfunction, caused by uncoupling of the endothelial nitric oxide synthase (eNOS). We hypothesised that oral tetrahydrobiopterin (BH<sub>4</sub>), an essential co-factor for eNOS, would lead to an improvement of endothelial function and subsequently, aortic stiffness.

**Methods:** This was a randomised, double-blinded crossover study, consisting of two separate regimens, 1: a single dose of BH<sub>4</sub> 400mg vs. placebo and 2: a one-week treatment with BH<sub>4</sub> 400mg OD vs. placebo. In study 1, aortic pulse wave velocity (aPWV), and flow mediated dilatation (FMD) were studied before and 3 hours after BH<sub>4</sub> supplementation and placebo. In study 2, FMD and aPWV were assessed four times, separated by a week.

**Results:** A single dose of BH<sub>4</sub>, but not placebo, improved endothelial dysfunction ( $+3.57 \pm 4.14$  vs.  $+0.05 \pm 1.17\%$ ,  $P = 0.03$ ;  $n = 18$ ). There was no change in aPWV following BH<sub>4</sub> or placebo ( $-0.13 \pm 0.58$  vs.  $-0.21 \pm 0.43$  m/s;  $P = 0.6$ ). One-week treatment with BH<sub>4</sub> improved endothelial function, whereas placebo did not ( $+3.69 \pm 4.90$  vs.  $+0.19 \pm 2.51\%$ ,  $P = 0.02$ ;  $n = 15$ ). There was no change in aPWV following BH<sub>4</sub> or placebo ( $-0.22 \pm 1.3$  vs.  $-0.25 \pm 0.51$  m/s,  $P = 0.4$ ) and no correlation between change in aPWV and FMD in either regime. **Conclusion:** Both acute and chronic BH<sub>4</sub> supplementation lead to an improvement of endothelial function, but did not reduce aortic stiffness. This suggests that there is no causality between endothelial function and aortic stiffness and that these conditions may just exist in parallel, both influenced by common risk factors, such as inflammation.

### P1.13 INTRACORONARY AND INTRAVENOUS ADMINISTRATION OF ADENOSINE ACHIEVE COMPARABLE MAXIMAL HYPEREMIA AND STENOSIS PRESSURE GRADIENT-FLOW VELOCITY RELATIONS

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**Background:** The aim of this study was to compare stenosis hemodynamics resulting from intravenous (iv) and intracoronary (ic) adenosine administration.

**Methods:** In 12 vessels with 25-57% DS (10 patients,  $61 \pm 8$  years), aortic pressure, distal coronary pressure and flow velocity (v) were simultaneously measured during the hyperemic response to either ic injection (40µg bolus) or iv adenosine infusion (140µg/kg/min). Cycle-averaged stenosis pressure gradient (ΔP) and flow velocity were obtained to derive ΔP-v relations from baseline to maximal velocity. For each lesion, we defined v<sub>1</sub> and v<sub>2</sub> as the lowest and highest common flow velocity for ic and iv-derived ΔP-v relations. The equivalence of both adenosine administrations was assessed by the difference in ΔP at v<sub>1</sub> and v<sub>2</sub>.

**Results:** Maximal flow velocity was  $56 \pm 19$  cm/s for ic and  $51 \pm 15$  cm/s for iv,  $p = \text{NS}$ . The stenosis ΔP-v relations largely overlapped (95% of the velocity range for ic injections; 85% of the flow velocity range for iv infusions). Common flow velocities ranged from v<sub>1</sub> =  $18 \pm 5$  cm/s to v<sub>2</sub> =  $50 \pm 15$  cm/s. The difference in pressure gradient (ivΔP - icΔP) at v<sub>1</sub> was  $0.2 \pm 0.7$  mmHg and  $0.8 \pm 3.2$  mmHg at v<sub>2</sub> ( $p = \text{NS}$ ), with no trend for differences in ΔP with increasing velocity.

**Conclusion:** Stenosis ΔP-v relations are not affected by the mode of adenosine administration and comparable hyperemia can be achieved. Ic adenosine injections are preferable, since they are faster and easier to perform and iv infusion tends to induce systemic hemodynamic variability.

### P1.14 PWV IMPROVEMENT IN PREVIOUSLY UNTREATED MILD HYPERTENSIVE PATIENTS AFTER 1 YEAR OF MONOTHERAPY

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**Objective:** Arterial stiffness is a measure of organ damage but procedures to destiffen arteries are still elusive. Our study describes the 1 year change in pulse wave velocity (PWV) in new diagnosed previously untreated, hypertensive patients.

**Patients and methods:** Longitudinal study including 427 consecutive, never-treated patients with suspected hypertension. After standard clinical assessment, including pulse wave analysis and PWV (Sphygmocor®, AtcorMedical), 231 showed elevated office and ambulatory blood pressure (BP) and received mono-therapy treatment accordingly. Clinical assessment was repeated after a median of 1.1 years in the whole cohort. PWV was adjusted to BP.

**Results:** 103 patients were female (44,6%), mean age was  $48 \pm 12$  years. The hypertensive diagnosed group tend to be older (50 vs. 46 years,  $p < 0.001$ ) and had higher PWV even after mean BP adjustment ( $8.6 \pm 2.0$  vs.  $7.9 \pm 1.6$  m/s,  $p < 0.001$ ), higher baseline office, ambulatory and central BP (145/86, 136/86 and 138/89 mmHg vs. 131/78, 123/79 and 124/83 respectively,  $p < 0.001$ ). After 1 year of treatment, BP was significantly improved in the hypertensive group (follow-up office BP  $128 \pm 13$  /  $75 \pm 9$  mmHg,  $p < 0.001$ ). The reduction of central and peripheral systolic BP was of the same magnitude ( $-16 \pm 1,2$  vs.  $-17 \pm 1,1$  mmHg,  $p = \text{ns}$ ). PWV was significantly reduced even after BP adjustment ( $\Delta = 0,3$  vs.  $0,05$  m/s,  $p < 0,001$ ) but remained higher than in the non-hypertensive group. There was no differential effect in PWV reduction depending on antihypertensive class.

**Conclusions:** Blood pressure reduction in newly diagnosed stage 1 hypertensive patients improves arterial stiffness within a year of treatment confirming that rapid tight controlled of BP is important even mild hypertensive.

### P1.15 MODULATING EFFECT OF TARGET PRESSURE ACHIEVEMENT ON PULSE WAVE VELOCITY IN HYPERTENSIVE PATIENTS

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**Aim:** Carotid-femoral pulse wave velocity (PWV) is a strong independent predictor of cardiovascular morbidity and mortality. The aim of the study was to evaluate treatment-induced changes in PWV in hypertensive subjects treated to target clinic BP (CBP).

**Methods:** Pts with grade I-II arterial hypertension were treated to target CBP  $< 140/90$  mmHg with combination of RAAS-inhibitors and amlodipine for 1 yr. Baseline BP was  $163,4 \pm 8,1/100,9 \pm 4,2$  mmHg; achieved BP  $123,7 \pm 9,7/76,8 \pm 6,7$  mmHg. Central BP and PWV were measured before treatment and after 8mo of target CBP achievement and maintenance.

**Results:** 47 pts (20 men, age  $58,9 \pm 9,0$  yrs; 4 smokers; 6 diabetics) achieved and maintained target CBP. In 11 (23%) pts PWV decreased by  $\geq 1$  m/s from baseline (G1), in 15 (32%) pts - unchanged (G2), in 21 (45%) - increased by