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

W. Wojciechowska, K. Stolarz-Skrzypek, A. Olszanecka, L. Klima, J. Gasowski, T. Grodzicki, D. Czarnecka
Jagiellonian University, Krakow, Poland

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 (PP; CPp), augmentation index (pA; AI) and pulse wave velocity (PWV) were evaluated by means of an oscillometric sphygmomanometer and pulse wave analysis (Sphygmogor). Left aortic (LAD) diameter, measuring aortic AI (AD) diameter, and ratio of early and late diastolic peak of transmittal flow velocities (E/A) as well as ratio of transmittal early filling 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 (UHTH), and 102 treated hypertensives (HTH). Parameters of interest significantly differed between these groups (p < 0.05). In post-hoc analysis with Bonferroni correction, UHTH 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 UHTH, THHT had significantly higher pA; cAI, CPP, CPWV, E/E', lower E/A and larger LA (p < 0.05) in comparison to UHTH 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 (BH4) IMPROVES ENDOTHELIAL FUNCTION, BUT NOT AORTIC STIFFNESS IN PATIENTS WITH RHEUMATOID ARTHRITIS

University of Cambridge, Cambridge, UK

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 (BH4), an essential co-factor for eNOS, would lead to an improvement in endothelial function and subsequently, aortic stiffness.

Methods: This was a randomised, double-blinded crossover study, consisting of two separate regimes, 1: a single dose of BH4 400mg vs. placebo and 2: a one-week treatment with BH4, 400mg OD vs. placebo. In study 1, aortic pulse wave velocity (aPWV), and flow mediated dilatation (FMD) were studied before and 3 hours after BH4 supplementation and placebo. In study 2, FMD and aPWV were assessed four times, separated by a week.

Results: A single dose of BH4, but not placebo, improved endothelial function (ΔP < 0.05). One-week treatment with BH4 improved endothelial function, whereas placebo did not (ΔP < 0.001; ΔP vs. placebo 0.2; ΔP vs. placebo 0.0). There was no change in aPWV following BH4 or placebo (ΔP = 0.1; ΔP vs. placebo 0.0; ΔP vs. placebo 0.0). One-week treatment with BH4, improved endothelial function, whereas placebo did not (ΔP < 0.001; ΔP vs. placebo 0.2; ΔP vs. placebo 0.0). There was no change in aPWV following BH4 or placebo (ΔP = 0.1; ΔP vs. placebo 0.0; ΔP vs. placebo 0.0). One-week treatment with BH4, improved endothelial function, whereas placebo did not (ΔP < 0.001; ΔP vs. placebo 0.2; ΔP vs. placebo 0.0). There was no change in aPWV following BH4 or placebo (ΔP = 0.1; ΔP vs. placebo 0.0; ΔP vs. placebo 0.0). One-week treatment with BH4, improved endothelial function, whereas placebo did not (ΔP < 0.001; ΔP vs. placebo 0.2; ΔP vs. placebo 0.0). There was no change in aPWV following BH4 or placebo (ΔP = 0.1; ΔP vs. placebo 0.0; ΔP vs. placebo 0.0). One-week treatment with BH4, improved endothelial function, whereas placebo did not (ΔP < 0.001; ΔP vs. placebo 0.2; ΔP vs. placebo 0.0). There was no change in aPWV following BH4 or placebo (ΔP = 0.1; ΔP vs. placebo 0.0; ΔP vs. placebo 0.0).

Conclusion: Both acute and chronic BH4 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 HYPERTENSION AND STENOSIS PRESSURE GRADIENT-FLOW VELOCITY RELATIONS

L. Casadonte a, K. Marques b, J. Spaan a, M. Siebes a
aAcademic Medical Center, Amsterdam, The Netherlands
bVU Medical Center, Amsterdam, The Netherlands

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 ± 8 years), aortic pressure, distal coronary pressure and flow velocity (v) were simultaneously measured during the hyperemic response to either ic injection (40ug bolus) or iv infusion of adenosine. Time-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 v1 and v2 as the lowest and highest common flow velocity for iv and ic derived ΔP/v relations. The equivalence of both adenosine administrations was assessed by the difference in ΔP at v1 and v2.

Results: Maximal flow velocity was 56 ± 19 cm/s for ic and 51 ± 15 cm/s for iv, p = 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 v1 = 18 ± 5 cm/s to v2 = 50 ± 15 cm/s. The difference in pressure gradient (ΔP-ΔP) at v1 was 0.2 ± 0.7 mmHg and 0.8 ± 3.2 mmHg at v2 (p = 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. iv 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

E. Rodilla a, s, X. Millasseau d, M. Escriva c, I. Garcia b, J. Costa b, J. Pascual a, X
aHospital de Sagunto, Sagunto, Spain
bUniversidad de Valencia, Valencia, Spain
cCardenal Herrera, Castellon, Spain
dPulse Wave Consulting, Saint Leu la Foret, France

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 newly 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±12years. The hypertensive diagnosed group tend to be older (50 vs. 46years, p<0.001) and had higher BP even after mean BP adjustment (8.6±2.0 vs. 7.9±1.6mmHg, 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 128/73 / 75/9mmHg, p<0.001). The reduction of central and peripheric BP was of the same magnitude (16±1.2 vs -17±1.1mmHg, p<0.001). PWV was significantly reduced even after BP adjustment (Δ = 0.3 vs 0.05mmHg, 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 VELOCITY WAVE IMPROVEMENT IN HYPERTENSIVE PATIENTS

E. Troitskaya, Y. Kotovskaya, Z. Kobalava
Peoples'Friendship University of Russia, Moscow, Russia

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 amiodipine for 1 yr. Baseline BP was 163±8/77±4 mmHg; achieved BP 123±7/9/7/76/8±6 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±10,0 yrs;4 smokers;6 diabetics) acquired and maintained target CBP. In 11 (23%) pts PWV decreased by 11±13% from baseline(G1), in 15 (32%) pts - unchanged(G2), in 21 (45%) - increased by