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Results: Brachial BP was similar between atorvastatin and placebo groups. Carotid systolic BP was slightly lower in the atorvastatin group but there was a statistically significant interaction between lipid-lowering and antihypertensive regimen; carotid SBP was lower in people randomized to atorvastatin + amlodipine-based therapy (placebo + atenolol = 130.6±2.4mmHg; atorvastatin + atenolol = 132.1±2.3mmHg; placebo + amlodipine = 131.0±2.8 mmHg; atorvastatin + amlodipine = 122.5±2.3 mmHg; Interaction $p = 0.04$; comparison placebo + amlodipine vs. atorvastatin + amlodipine $p < 0.01$).

Conclusions: The combination of atorvastatin with amlodipine-based antihypertensive treatment lowers central BP. This effect may contribute to the reduced incidence of cardiovascular events seen in people receiving this combination in ASCOT-LLA.

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P1.03

DIFFERENCES IN THE LATE SYSTOLIC SHOULDER PRESSURE (SBP₂) OF THE RADIAL ARTERY PRESSURE WAVEFORM BY ANTIHYPERTENSIVE REGIMEN IN THE ANGLO SCANDINAVIAN CARDIAC OUTCOMES TRIAL (ASCOT)

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Previous work using a transfer function to estimate central systolic blood pressure (SBP) from the pressure waveform in the radial artery reported that, central SBP was lower in the amlodipine-based regimen compared with the atenolol-based regimen in the Anglo Scandinavian Cardiac Outcomes Trial (ASCOT) ¹. However use of a transfer function to estimate central BP has been criticised and more recently Munir et al.,² have proposed that the late systolic shoulder (pSBP₂) may be a direct estimate of central SBP. We compared pSBP₂ between patients randomized to the amlodipine-based and atenolol-based regimens in a substudy of ASCOT.

229 patients participated in the substudy. Applanation tonometry was performed at the right radial artery using a Millar tonometer. Waveforms were ensemble averaged and calibrated to brachial artery pressure. All data are means (SD). Brachial BP did not differ significantly (137.5 (12.2)/79.6 (7.4) vs. 142.1 (15.3)/80.5 (9.2) mmHg; NS), but pSBP₂ was significantly lower in the amlodipine-based regimen (112.4 (10.4) vs. 119.3 (13.1) ; $p < 0.01$). pSBP₂ occurred earlier in the amlodipine-based group (415.8 (53.0) vs. 453.5 (51.6) ms; $p < 0.01$) probably due to the lower heart rate in the atenolol-based regimen. Lower pSBP₂ in people randomized to the amlodipine-based regimen in ASCOT are in keeping with previous findings using a transfer function applied to the radial pressure waveform and direct measurements in the carotid artery and suggest that pSBP₂ may be a useful indicator of central SBP.

1. Williams et al., *Circulation* 2006; 113:1213-25

2. Munir et al., *Hypertension*. 2008;51:112-8.

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P1.04

THE INFLUENCE OF QUINAPRIL ON ARTERIAL STIFFNESS, BLOOD VISCOSITY AND ARTERIAL SHEAR STRESS IN PATIENTS WITH ESSENTIAL ARTERIAL HYPERTENSION

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Aim: to analyze the influence of quinapril on aortic PWV, whole blood viscosity (WBV), shear stress in the ascending aorta (AA) and common carotid artery (CCA), prometalo proteinase-1 (proMMP-1) and its tissue inhibitor (TIMP-1) plasma concentration in patients with essential arterial hypertension (HT).

Methods: 55 patients, (mean age 55,8 ± 13,8 yrs.) with HT 1 and 2 gr. were treated with quinapril in stepwise increased doses from 10 to 40 mg/d till BP goal <140/90 mmHg was achieved. At baseline and then after 3 and 6 months of treatment PWV, WBV, proMMP-1 and TIMP-1 were determined. Shear stress in AA and CCA was calculated from WBV, internal vessel diameter and blood flow velocity (Vmax) measured ultrasonographically.

Results: After 6 months of treatment by quinapril we observed decrease of BP (155.6/92.0 mmHg vs. 135.9/82.9 mmHg, $p < 0.001$), PWV (10.35 m/s vs. 9.64 ms, $p < 0.001$), WBV (5.14 cP vs. 4.86 cP, $p < 0.05$) and TIMP-1 (111.0 ng/ml vs. 94.1 ng/ml, $p < 0.001$) and increase of Vmax in AA (127.1 cm/s vs. 131.3 cm/s, $p < 0.05$, Vmax in CCA (69.9 cm/s vs. 78.4 cm/s, $p < 0.05$) and shear stress in

CCA (22.2 dyne/cm² vs. 24.7 dyne/cm², $p < 0.05$). Significant positive correlation was observed for PWV and TIMP-1, and negative correlations for PWV and Vmax in AA, PWV and Vmax in CCA, as well as PWV and shear stress in CCA.

Conclusions: Quinapril reduces arterial stiffness by inhibition of collagen metabolism. This effect is mediated by influence on arterial shear stress.

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P1.05

INFLUENCE OF FELODIPIN ON BLOOD PRESSURE AND ARTERIAL PROPERTIES IN OLDER HYPERTENSIVE PATIENTS

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Pulse wave velocity (PWV) and endothelial function are prognostic factors in arterial hypertension. Modification of them, apart from blood pressure (BP) lowering seems to be important in the evaluation of antihypertensive drugs. The AIM of this study was to prove otherwise while assessing the direct effect of felodipin (Felodip, Teva) on arterial properties in patients (more than 55 years old) with mild, moderate and severe hypertension.

Materials and methods: 30 hypertensive patients (mean age 63,98±6,46 years, 22 male, 8 female) received felodipin in individual titrated doses 2,5-10mg (mean dose 7,96 mg) daily for 3 months. The examination comprised routine tests, ECG, blood glucose, total cholesterol, triglycerides. The assessment of arterial stiffness was done by way of measuring brachial-ankle pulse wave velocity (baPWV). Endothelial function was calculated based on flow-mediated dilatation (FMD) parameters.

Results: The treatment produced a significant reduction in systolic (-30.4mmHg) and diastolic BP (-15.2mmHg). Significant decrease of baPWV (by 7.0%) and increase of FMD (by 21.5%) was observed. There was an insignificant rise in the levels of cholesterol, triglycerides, glucose. Felodipin has been well tolerated in most patients.

Conclusion: These results demonstrate that felodipin increases arterial distensibility. This effect of felodipin should be attributed to BP lowering and endothelial function improvement.

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P1.06

THE EFFECT OF SPINAL ANESTHESIA ON BLOOD PRESSURE AND AUGMENTATION INDEX

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Objectives: Parameters characterizing arterial stiffness - augmentation index (Alx) and pulse wave velocity (PWV) - are autonomous and independent cardiovascular risk factors. In this study we examined if the spinal anesthesia has considerable effects on the peripheral and central blood pressure, augmentation pressure, Alx and PWV.

Methods: The measurements were carried out using SphygmoCor (AtCor Medical, Australia) device. Spinal anesthesia was performed in all patients because of surgery, due to varicosity of lower extremity. Spinal anesthesia was performed uniformly by 3.2 milliliters of bupivacaine (0.5 %) via the L3 interstice. One liter of crystalloid infusion was administered before the procedure. Patients were examined before anesthesia, and after enrollment of sensory analgesia and motor block.

Results: 29 patients (10 males and 19 females, aged: 53.7±12.9 years) were included in the study. As a result of spinal anesthesia not only the systolic (143.4±20.4 vs. 119.5±16.4 mmHg, $p < 0.0001$) and diastolic peripheral (83.2±11.3 vs. 68.3±10.7 mmHg, $p < 0.0001$), but also the systolic (132.6±18.3 vs. 105.2±14.8 mmHg, $p < 0.0001$) diastolic (85.8±12.1 vs. 69.3±11.2, $p < 0.0001$) central BP, mean pressure (105.8±13.9 vs. 84.7±13.1, $p < 0.0001$) and pulse pressure (60.2±13.9 vs. 51.2±10.8, $p < 0.02$) decreased. We also observed a significant difference in the augmentation pressure (14.6±7.9 vs. 6.1±3.8 mmHg, $p < 0.001$), and AlxHR75 (26.8±6.9 vs. 15.1±10.9%, $p < 0.001$), respectively. There were no significant difference in PWV (9.3±3.7 vs. 9.1±3.4 m/s, $p = 0.8$).

Conclusion: As a result of spinal anesthesia and so regional sympathetic nervous block not only the peripheral and central BP, but also the Alx decreased significantly, while PWV remained unchanged.

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