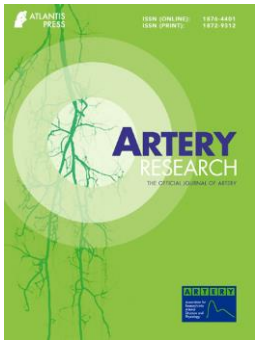

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P120: DETERMINANTS OF BRACHIAL-ANKLE PULSE WAVE VELOCITY

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Background and Aim: Low density lipoprotein cholesterol (LDL-C) is a primary risk factor for atherosclerosis, but it is also associated with elevated blood pressure (BP) and future development of hypertension. We examined the relationship between LDL-C and haemodynamic variables in normotensive and never-treated hypertensive subjects.
Methods: Altogether 615 volunteers (19—72 years) without lipid- and BP-lowering medication were recruited. Supine haemodynamics were recorded using continuous radial pulse wave analysis, whole-body impedance cardiography, and single channel electrocardiogram. The haemodynamic relations of LDL-C were examined using linear regression analyses with age, sex, body mass index (BMI) (or height and weight as appropriate), smoking status, alcohol use, and plasma C-reactive protein, sodium, uric acid, high density lipoprotein cholesterol (HDL-C), triglycerides, estimated glomerular filtration rate, and quantitative insulin sensitivity check index as the other included variables.
Results: The mean (SD) characteristics of the subjects were: age 45 (12) years, BMI 27 (4) kg/m^2, office BP 141/89 (21/13) mmHg, creatinine 74 (14) μmol/l, total cholesterol 5.2 (1.0), LDL-C 3.1 (0.6), triglycerides 1.2 (0.8), and HDL-C 1.6 (0.4) mmol/l. LDL-C was an independent explanatory factor for aortic systolic and diastolic BP, aortic pulse pressure, augmentation index, pulse wave velocity (PWV), and systemic vascular resistance index ($p \leq 0.013$ for all). When central BP was included in the model for PWV, LDL-C was no more an explanatory factor for PWV.
Conclusions: LDL-C is independently associated with BP via systemic vascular resistance and wave reflection. These results suggest that LDL-C may play a role in the pathogenesis of primary hypertension.

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DETERMINANTS OF BRACHIAL-ANKLE PULSE WAVE VELOCITY

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It has been proven that aortic pulse wave velocity (aoPWV) the measure of arterial stiffness is a strong and independent predictor of both cardiovascular events and all-cause mortality. Beyond the "gold standard" cfPWV brachial-ankle PWV (baPVW) measurement has been accepted for assessing arterial stiffness and endorsed into the position paper of Artery Society as a recommended method. The aim of this study was to define the determinants of baPVW.
Patients and Methods: baPVW and ABI was measured with BOSO-ABI system in 188 consecutive adults (98 male, 91 female) at risk of or with manifest CV disease (mean age: 58 years). This oscillometric device is capable to measure blood pressure on both upper and lower extremities simultaneously with four cuff to assess ABI and baPVW, as well.
Results: baPVW was significantly correlated with age but this correlation was not as strong as it can be observed with aortic PVW ($R = 0.172$). Linear backward regression analysis confirmed that age and brachial systolic blood pressure proved to be the main determinants of baPVW.
Conclusions: baPVW is a suitable complimentary method for assessing arterial stiffness which can provide useful information regarding not only aortic stiffness but the peripherial arteries.

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ASSOCIATION OF CARDIORESPIRATORY FITNESS WITH ARTERIAL STIFFNESS AND PERIPHERAL AND CENTRAL BLOOD PRESSURE IN RESISTANT HYPERTENSION PATIENTS

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Background: The relationship between arterial stiffness, blood pressure (BP) and cardiorespiratory fitness (CRF) has been studied in healthy populations and cardiovascular patients, since all of which proved to be independent predictors of all-cause mortality. We aimed to investigate the association of CRF with arterial stiffness and peripheral and central hemodynamics, in patients with resistant hypertension (RT).
Methods: In this cross-sectional study, 30 patients (13 men, 17 women; age, 57.7 ± 8.1 years; weight, 79.2 ± 11.6 kg; body mass index, 29.7 ± 4.0 kg/m^2) with resistant hypertension were recruited in the Hospital Infante D. Pedro (Aveiro) and Hospital Pedro Hispano (Matosinhos). Outcome measures included CRF (VO2peak), peripheral and central BP, and carotid-femoral pulse wave velocity (cf-PWV). Correlation analysis was conducted to assess the association between variables.
Results: A significant negative correlation was found between VO2peak (33.7 ± 6.2 mLO_2/kg/min) and central (141.0 ± 21.3 mmHg; $r = -0.395$, $p = 0.031$) and peripheral systolic BP (148.4 ± 21.3 mmHg; $r = -0.363$, $p = 0.049$). VO2peak was also correlated with pulse pressure (PP) amplification ratio (1.2 ± 0.1 mmHg; $r = 0.361$, $p = 0.050$). The association with central systolic BP ($r = -0.403$, $p = 0.035$) and PP amplification ratio ($r = 0.408$, $p = 0.033$) remained significant after adjusted for age. VO2peak showed no correlation with PWV (9.4 ± 2.9 m/s; $r = -0.075$, $p = 0.694$) and peripheral (88.1 ± 12.2 mmHg; $r = -0.138$ $p = 0.467$) and central diastolic BP (88.5 ± 12.7 mmHg; $r = -0.133$, $p = 0.483$).
Conclusion: This study confirms the inverse relationship between CRF and central systolic BP and PP amplification ratio in RH patients, regardless of age.

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THE ARTERIAL STIFFNESS DYNAMICS UNDER THE EFFECT OF ROSUVASTATIN ADDED TO DIFFERENT COMBINATIONS OF ANTIHYPERTENSIVE DRUGS

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We studied the influence of rosuvastatin adding to lisinopril/amlodipine or lisinopril/ hydrochlorothiazide fixed combinations on blood pressure (BP) and arterial stiffness dynamics in hypertensive patients of high or very high cardiovascular risk.
Methods: 60 patients (36 men and 24 women aged 52.1 ± 7.3) with uncontrolled high or very high cardiovascular risk hypertension (HTN) were randomized into two groups. Group 1 ($n = 30$) received a fixed combination of lisinopril/amlodipine 10/5 mg/day. Group 2 consisted of 30 patients who received a fixed combination of lisinopril/hydrochlorothiazide 10-20/12.5 mg/day. The rosuvastatin 20mg/day was added in the both groups. The office BP, central (aortic) BP, augmentation index (AIx), carotid-femoral and carotid-radial pulse wave velocity (PWV) dynamics was evaluated during 24-week follow-up period.
Results: Baseline clinical characteristics did not differ in the groups. The office BP decreased in both groups from $173.3 \pm 20.2/104.4 \pm 14.0$ to $131.2 \pm 10.4/83.5 \pm 7.8$ mmHg ($p < 0.001$) in the 1-st group and from $168.6 \pm 23.6/103.6 \pm 15.6$ to $135.6 \pm 15.1/87.3 \pm 11.5$ mmHg ($p < 0.001$) in the 2nd one. The extent of office BP did not differ. Howeverthe degree of central systolic BP reduction was more prominent in the 2nd group ($10.5 \pm 6,8$ and 6.5 ± 7.8 mmHg, respectively). The extent of AIx decline did not differ. Carotid-femoral PWV equally decreased in both groups (from 9.5 ± 1.7 to 8.8 ± 1.8; $p = 0.043$ and from 8.9 ± 1.2 to 8.1 ± 1.4 m/s; $p = 0.001$,