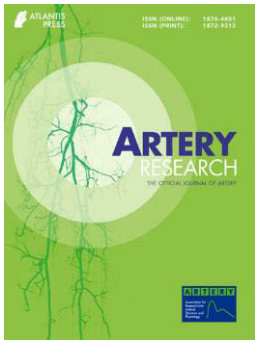

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P122: THE ARTERIAL STIFFNESS DYNAMICS UNDER THE EFFECT OF ROSUVASTATIN ADDED TO DIFFERENT COMBINATIONS OF ANTIHYPERTENSIVE DRUGS

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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.

P120
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.

P121
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.

P122
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; $p = 0.001$,

respectively). Carotid-radial PWV reliably declined only in the 1st group (from 9.5 ± 1.8 to 8.8 ± 1.1 m/s; p = 0.034).
Conclusion: Addition of rosuvastatin to a fixed lisinopril/amlodipine combination has proved to be more effective than lisinopril/hydrochlorothiazide plus rosuvastatin combination in terms of impact on central aortic systolic BP and carotid-radial PWV.

P123
UNATTENDED AND ATTENDED BP VALUES AND VASCULARE AND CARDIAC ORGAN DAMAGE

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It has been suggested that measurement of "unattended" blood pressure values may provide advantages over conventional BP measurement; some hypertension guidelines now suggest this approach as the preferred one for measuring office BP. Data on the relationship between unattended BP and cardiovascular events are less solid as compared to those obtained with attended BP; only few studies suggested that unattended BP might be more strictly correlated with hypertensive target organ damage than "attended" BP.
Aim: to evaluate the relationship between "attended" or "unattended" BP values and target organ damage in 261 subjects attending the outpatient clinic of an ESH-Excellence-Centre.BP values were measured by the physician with an automated oscillometric device (OmronHEM9000Ai, mean of 3 measurements),after 5 minutes of rest;thereafter,the patient was left alone and unattended BP was measured automatically after 5 minutes(3 measurements at 1 minute interval).
Results: mean age was 61 ± 16yrs, BMI 26.1 ± 4.2,60% female, 88% hypertensives (64% treated). Systolic unattended BP was lower as compared to attended SBP (130.1 ± 15.7 vs 138.6 ± 17.2 mmHg).Left ventricular mass index(LVMI) was similarly correlated with unattended and attended SBP(r = 0.132 and r = 0.133, p < 0.05,respectively). LVMI was similarly correlated with unattended and attended pulse pressure(PP)(r = 0.277 and r = 0.299, p < 0.05,respectively).Carotid IMT was significantly and similarly correlated with both attended and unattended BP values(CBMaxIMT:r = 0.172 and r = 0.153 for attended and unattended SBP, p < 0.05 and: r = 0.459 and r = 0.436 for attended and unattended PP, p<0.001).The differences between correlations were not statistically significant.
Conclusion: Measurement of BP "unattended" or "unattended" provides different values, being unattended BP lower as compared to attended BP. Our results suggest that attended and unattended BP values are similarly related with cardiac and vascular hypertensive target organ damage.

P124
CENTRAL BLOOD PRESSURE MEASUREMENT: PARADIGM SHIFT

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Introduction: It is estimated that currently 17 million deaths annually in the world occur due to cardiovascular disease (CV), about one third of all deaths. 9.4 million are related to arterial hypertension (HA). The use of methods that allow the early identification of structural and functional cardiovascular alterations can improve the strategy of treatment and control of these patients.
Description: LSO, 65 years old, female, white. Ringing in the ear and headache. Hypertension for 18 years and panic syndrome using Candesartan 8 mg, Fluoxetine 20 mg and Alprazolan 0.5 mg. In 06-2016, presenting blood pressure (BP):172 x 104 mmHg. Candesartan was elevated to 16 mg, initiating Rosuvastatin 10mg (C-reactive protein:16 and LDL-C:142), targeted improvement of lifestyle habits. ABPM 2 weeks after normal. Returned on 10-2017 with tachycardia and dizziness. She stopped Fluoxetine and Rosuvastatin. BP:178 x 84 mmHg. Reintroduced Fluoxetine and Rosuvastatin with new normal ABPM. Returned in 02-2018 with feeling of death, uneasiness and palpitations. BP:138 x 78 mmHg and normal ECG, in regular use of the medications. Accomplished non - invasive central blood pressure measurement (Mobil O'Graph) with arterial stiffness elevation, central AP:143 mmHg and augmentation index (AI):50 was performed. Felodipine -2.5 mg was started even with the new normal ABPM. 4 months later new measures with central BP:128 and AI = 33-table 1.
Conclusion: The treatment of HA depends on the choice of the drug and early onset with reduction of BP and CV outcomes 3,4. The central BP has greater relevance in the reduction of BP and cardiovascular outcomes than the peripheral BP 5,6. Keywords: Hypertension; Central Blood Pressure; Arterial Stiffness.

Table 1

EXAMES	06/2016	10/2017	02/2018	06/2018
SODIUM	142mg/dl	142mg/dl		
POTASSIUM	4,2 mg/dl	3,9 mg/dl		
UREA	39 mg/dl	36 mg/dl		
Creatine	1,25 mg/dl	1,09 mg/dl		
TSH	1,97	2,2		
C-reactive protein	16	0		
HEMOGRAM	NORMAL	NORMAL		
GLYCEMIA	71 mg/dl	94 mg/dl		
GLYCADA HEMOGLOBIN	6%	5%		
URIC ACID	3,9 mg/dl	3,5 mg/dl		
C. TOTAL	216 mg/dl	222 mg/dl		
LDL cholesterol	142 mg/dl	148 mg/dl		
ELECTROCARDIOGRAM	NORMAL	NORMAL	NORMAL	
DOPPLER OF CAROTIDAS	NORMAL			
ECHOCARDIOGRAM	NORMAL	NORMAL		
Central Blood Pressure			VOP=10, AI=50, PC=142, PP=155x92	VOP=9,5, AI=33, PC=128, PP=136x98
ABPM	Vigília:125x77 Sono:113X71	Vigília:125x77 Sono:113X71		

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P125
ESTIMATION OF MEAN ARTERIAL PRESSURE IN NON-INVASIVE STUDIES

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Background: Mean arterial pressure (MAP) is required for many hemodynamic calculations. Most automated devices do not report MAP and a form factor (FF) is used to estimate it from systolic and diastolic blood pressure (BP). The appropriate choice of FF in the brachial artery is controversial: 0.33 is the traditional value, but invasive measurements report a value of 0.4. [1] Non-invasive studies have provided some support for FF = 0.4 but have usually not measured MAP directly, nor used brachial blood pressure waveforms, or accounted for BP measurement errors. We addressed these issues in a sample of white Europeans drawn from the Southall and Brent Revisited study.
Methods: BP was measured using a Pulsecor device (USCOM). Form factors (FFosc and FFwave) were calculated as (MAP-diastolic BP)/(systolic BP-diastolic BP) using MAPosc calculated by oscillometry and MAPwave calculated as the waveform mean respectively.
Results: Data are mean ± SD of 527 observations (Table 1). FFosc was lower than FFwave and use of FF = 0.4 (MAP0.4) overestimated MAPosc. Allowing for measurement errors based on [2-3] gave estimates of MAPwave that were more similar to MAPosc.
Conclusions: Measurement errors confound estimation of MAP using FF. Measurement errors vary substantially between devices [4] precluding a single FF for all studies. Non-invasive MAP should be estimated by oscillometric methods.