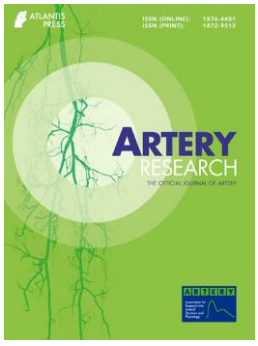

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P25: 24-HOUR AORTIC AMBULATORY BLOOD PRESSURE IS BETTER ASSOCIATED WITH COMMON CAROTID ARTERY HYPERTROPHY THAN 24-HOUR BRACHIAL PRESSURE – THE SAFAR STUDY

Antonios Argyris, Evaggelia Aissopou, Efthymia Nasothymiou, Theodoros Papaioannou, Jacques Blacher, Michel Safar, Petros Sfikakis, Athanase Protogerou

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removal) to aortic mean and diastolic BP. For MoG, central pressure was derived through standard systolic-diastolic calibration (MoGC1) as well as mean-diastolic calibration (MoGC2).
Results: Mean±SD differences between device and intra-arterial BP are presented in the Table. There was moderate correlation between device and intra-arterial brachial systolic BP ($R = 0.58$ XCEL, $R = 0.47$ MoG, $P < 0.01$) and central systolic BP ($R = 0.69$ XCEL, $R = 0.64$ MoGC1, $R = 0.43$ MoGC2, $P < 0.01$). Intra-arterial central-to-brachial pulse amplification factor was 1.17 ± 0.16 (range 0.88 to 1.55), but there was no correlation between device and intra-arterial amplification ($R = 0.07$ XCEL, $R = 0.07$ MoGC1, $R = 0.19$ MoGC2, $P > 0.18$). Results in sub-groups $\geq$13 and <13 years were similar.
Conclusion: Both oscillometric devices overestimated brachial and central systolic/pulse BP, exceeding the validation criteria of 5 ± 8 mmHg, and there was no correlation between intra-arterial and device-derived central-to-brachial pulse amplification. Diastolic BP was acceptable.

Table: Mean±SD of the difference (mmHg) between device and intra-arterial measurements.

	Systolic	Diastolic	Pulse
Brachial XCEL	11.2 ± 8.9	-1.7 ± 6.0	13.0 ± 10.1
Brachial MoG	12.9 ± 11.7	-4.7 ± 5.4	17.9 ± 11.4
Central XCEL	8.8 ± 6.6	-0.7 ± 6.2	9.0 ± 7.7
Central MoGC1	7.7 ± 10.3	-3.1 ± 6.1	10.6 ± 11.6
Central MoGC2	22.3 ± 14.3	-3.2 ± 6.6	25.4 ± 15.0

P25
24-HOUR AORTIC AMBULATORY BLOOD PRESSURE IS BETTER ASSOCIATED WITH COMMON CAROTID ARTERY HYPERTROPHY THAN 24-HOUR BRACHIAL PRESSURE — THE SAFAR STUDY

Antonios Argyris [1], Evaggelia Aissopou [1], Efthymia Nasothymiou [1], Theodoros Papaioannou [2], Jacques Blacher [3], Michel Safar [3], Petros Sfikakis [4], Athanase Protogerou [1]
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Objective: Evidence suggests the superiority of office aortic pressure over brachial on the evaluation of vascular damage and prognosis of cardiovascular disease (CVD); 24-hour ambulatory blood pressure monitoring (ABPM) is regarded the optimal method for assessing blood pressure (BP) profile. The non-invasive 24-hour aortic ABPM is feasible and superior to 24-hour brachial regarding the association with left ventricular hypertrophy and diastolic dysfunction. The aim of our study was to examine the association of 24-hour aortic and brachial ABPM with common carotid artery (CCA) hypertrophy.
Methods: Consecutive subjects referred for CVD risk assessment underwent 24-hour aortic and brachial ABPM using a validated oscillometric brachial cuff-based devise (Mobil-O-Graph). CCA hypertrophy was assessed by high-resolution ultrasound (assessment of intima media thickness - IMT).
Results: 497 subjects (aged 54 ± 13 years, 57% men, 80% hypertensives) were examined. Using Hotelling's-Williams test it was shown that 24-hour aortic BP was significantly better correlated with IMT as compared with brachial BP (r: 0,254 vs. r: 0,202 for right IMT, r: 0,244 vs. r: 0,207 for left IMT, $p < 0,05$). Multivariate analysis (adjusted for possible confounders) revealed superiority of 24-hour aortic BP regarding the association with IMT as well as carotid hypertrophy. Last, in ROC analysis, aortic BP had a higher discriminatory ability compared to brachial for the detection of carotid hypertrophy (AUC: 0,707 vs. 0,656 for right carotid artery hypertrophy, AUC: 0,636 vs. 0,602 for left carotid artery hypertrophy, $p < 0,05$).
Conclusions: Non-invasively assessed 24-hour aortic pressure is more strongly associated with CCA IMT and provides a higher discriminatory ability for the detection of CCA hypertrophy.

P27
INVASIVE CENTRAL PULSE PRESSURE IS RELATED TO AORTIC ROOT DILATATION

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Background: Aortic root dilation is an established risk factor for aortic dissection. Despite the relations between aortic root remodeling, carotid-femoral pulse wave velocity (cfPWV) and aortic blood pressure have been advocated by several clinical studies and is supported by physical law, invasive data are lacking. We aimed to investigate the relationship between aortic root remodeling, invasively-measured central blood pressure and cfPWV in patients referred for invasive hemodynamic evaluation for suspected coronary disease.
Methods: In 71 patients aortic pulse pressure (aoPP) was measured in the proximal aorta with a calibrated fluid-filled pressure catheter. Before entering the hemodynamic room all patients underwent 2D echocardiographic quantification of aortic root diameter and measurement of cfPWV. Aortic root diameter was then expressed into z-score following age, sex and height adjusted reference values (1).
Results: Mean age was 67 ± 10 years and 76.1% of patients were men. Invasive aortic systolic pressure was 146 ± 23 mmHg, diastolic pressure was 78 ± 13 mmHg, and aoPP was 68 ± 21 mmHg. Aortic Z-score was -0.32 ± 1.7, while CfPWV was 9.8±3 m/s. While Log10cfPWV and aoPP showed a positive relation ($r=0.426$, $p<0.01$) while aoPP and aortic Z-score were inversely associated ($r = -0.271$, $p = 0.02$). In a multivariable linear regression analysis, Z-score and Log_{10}cfPWV were statistically-significant independent predictors of aoPP ($p = 0.01$ and $p < 0.01$, respectively) after adjustment for age, sex, BSA, heart rate, invasive MBP, and stroke volume.
Conclusions: In a population referred to invasive coronary hemodynamic evaluation for suspected coronary disease, aortic root remodeling and aortic stiffness were independently associated with a lower aoPP.

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P29
MECHANISMS OF VASCULAR ENDOTHELIAL GROWTH FACTOR INHIBITION INDUCED HYPERTENSION

Kaisa Maki-Petaja [1], Adam McGeoch [2], Lucy Yang [1], Annette Hubsch [1], Carmel McEniery [1], Fraz Mir [1], Parag Gajendragadkar [1], Nicola Ramenatte [3], Gayathri Anandappa [3], Christoph Brune [4], Yoeri Boink [4], Carola Bibiane-Schonlieb [5], Paul Meyer [6], Simon Bond [7], Ian Wilkinson [1], Duncan Jodrell [8], Joseph Cheriyan [9]
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Introduction: Drugs targeting Vascular Endothelial Growth Factor (VEGF) signaling pathway are approved therapies for cancer. Unfortunately, VEGF