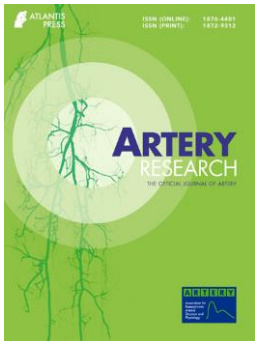

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P7.10: PRE-TREATMENT AORTIC PULSE PRESSURE AS A POSSIBLE PREDICTOR OF FUTURE VISIT-TO-VISIT SYSTOLIC BLOOD PRESSURE VARIABILITY

E. Troitskaya, Y. Kotovskaya, Z. Kobalava

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Methods: Successful AM of brachial and central BP was done with oscillometric BPLab VASOTENS system (OOO Petr Telegin, Russia) in 84 untreated hypertensive subjects (55,8±9,6 years, male 36,9 %). Gender differences of central BP and AIx were evaluated in patients <55 (14 men, 23 women), 55-60 (7 men, 16 women), 61-70 years (10 men, 14 women). Differences were considered significant if $p<0,05$.
Results: Several findings were consistent among all age groups. First, men had slightly higher levels of brachial and aortic SBP than women: respectively, <55 years brachial SBP day/night 144±14/134±20 vs 137±16/128±16, 55-60 years 147±18/146±21 vs 139±16/132±22, 61-70 years 141±23/135±32 vs 137±14/129±17 mmHg. Second, nocturnal decline of brachial and aortic SBP was greater in women than in men: respectively, <55 years aortic SBP day/night 128±15/121±15 vs 132±14/132±14, 55-60 years 130±15/124±21 vs 134±17/137±19, 61-70 years 128±14/122±16 vs 131±22/127±29 mmHg. Third, women had a higher AIx@HR75 in all time periods but night increase AIx was significantly more evident in men: respectively, AIx (day-night)/day (%) <55 years -44±12 in men vs -22±7,0 in women, 55-60 years -38±18 vs -20±16, respectively, 61-70 years -45±19 vs -19±10, respectively ($p<0,05$ for all ages).
Conclusion: Gender differences in characteristics of AIx and its diurnal variation were identified and should be considered when analyzing the results of AM of central BP

P7.8
SYSTOLIC PRESSURE AMPLIFICATION IN CHILDREN

L. Milne [a], L. Keehn [a], M. Sinha [a,b], P. Chowienczyk [a]
[a]*St Thomas' Hospital, King's College London, London, UK*
[b]*Evelina London Children's Hospital, Guy's and St. Thomas' NHS Trust, London, UK*

Objective: The aim of our study was to measure systolic pressure wave amplification (brachial minus central systolic blood pressure) in children and to assess anthropometric measures correlated with amplification in children with and without chronic kidney disease (CKD).
Design and Methods: In a prospective single centre study 150 children (103 with non-dialysis CKD, 23 with hypertension) aged 12.9 ± 2.9 years (81 boys) were recruited from the paediatric nephrology and hypertension out-patient clinics at the Evelina London Children's Hospital. Peripheral blood pressure was measured manually, in triplicate, by aneroid sphygmomanometer. Central blood pressure and carotid-femoral pulse wave velocity (cfPWV) were measured in triplicate using the SphygmoCor system. Renal function was determined by estimated GFR (eGFR) using the Schwartz formula.
Results: Mean amplification (mean ± SD) was 18.8 ± 6.4 mmHg. Systolic pressure amplification was significantly different across age groups of children 5-10 years (16.3 ± 4.8 mmHg), 10-15 years (18.2 ± 5.2 mmHg) and 15-18 years (21.0 ± 7.3 mmHg) ($p = 0.003$). In univariate analysis amplification was correlated with age, height, weight, BMI, and eGFR. It was not correlated with gender, ethnicity or cfPWV. In multivariate linear regression analysis including age, gender, height, BMI, and eGFR, amplification was independently associated with height, BMI and eGFR ($\beta=0.213$, $P=0.012$; $\beta=0.264$, $P=0.002$; $\beta=0.206$, $P=0.006$, respectively, model adjusted $R2=0.203$).
Conclusions: Amplification is greater in adolescents (15-18 years) than in younger children (5-10 years) and is independently associated with BMI, height, and renal function.

P7.9
ELEVATED ARTERIAL STIFFNESS PRECEDES DEVELOPMENT OF HYPERTENSION IN NEVER TREATED PREHYPERTENSIVE PATIENTS

E. Rodilla [a,c], S. Millasseau [d], M. Escriva [a], J. Garcia [a], J. Costa [a], J. Pascual [a,b]
[a]*Hospital de Sagunto, Sagunto, Spain*
[b]*Universidad de Valencia, Valencia, Spain*
[c]*Universidad Cardenal Herrera, Castellon, Spain*
[d]*Pulse Wave Consulting, Saint Leu la foret, Spain*

Objective: Arterial stiffness and arterial blood pressure (BP) share similar natural history. To which extent the first causes the latter is unknown. Our study describes the development of pulse wave velocity (PWV) and BP in untreated, prehypertensive patients after one year of follow-up.
Patients and methods: Longitudinal study including 427 consecutive, never-treated patients with suspected hypertension. After standard clinical assessment, including PWV (Sphygmocor®, AtcorMedical), 125 showed normal office and ambulatory BP. According to the median baseline PWV , patients were divided into 2 groups: PWV+ and PWV-. After a median of 1.1 years, clinical assessment was repeated. PWV was adjusted to BP.
Results: 76 patients were female (60.8%), mean age was 47 years, baseline office and ambulatory BP were 126/76 and 120/77 mmHg, respectively. The PWV- group were significantly younger (40 vs. 52years, p<001), but systolic, diastolic and mean office BP did not differ compared to the PWV+ group. Only ambulatory SBP was significantly different(118 vs. 121, $p<0.03$). At follow-up, office systolic and MBP were significantly higher in the PWV+ group (129 vs. 123, $p<0.01$; 97 vs. 93, $p<0.02$, respectively), difference in ambulatory SBP remained significant (118 vs. 123; $p <0.02$). Follow-up PWV did not change in the PWV- group (adjusted PWV 6.9 vs. 7.0; p=ns), whereas PWV significantly improved in the PWV+ group (adjusted PWV 9.1 vs. 8.7; $p<0.01$).
Conclusions: Measurement of PWV in untreated prehypertensive patients allows to predict development of higher BP values whithin a year, suggesting that arterial stiffness may play a causal role for hypertension.

P7.10
PRE-TREATMENT AORTIC PULSE PRESSURE AS A POSSIBLE PREDICTOR OF FUTURE VISIT-TO-VISIT SYSTOLIC BLOOD PRESSURE VARIABILITY

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

Background: Visit-to-visit systolic blood pressure variability (SBPV) in treated hypertensive patients may have impact on prognosis and it stimulates searching for its predictors. The aim of the study was to evaluate SBPV and its predictors in patients with controlled AH.
Methods: 52 pts(20 men, age58,9±9,0yrs;4 smokers;6 diabetics) with uncomplicated AH were treated to target BP<140/90mmHg with combination of RAAS-inhibitor and amlodipine for 1yr. Baseline BP was 163,4±8,1/100,9±4,2mmHg; achieved-123,7±9,7/76,8±6,7mmHg. SBPV was calculated as SD for 5 visits during 8 months after target BP achievement. Central BP and pulse wave velocity (PWV) were measured before treatment initiation and at the end of the study. $p<0,05$ was considered significant.
Results: SBPV after achievement of target BP varied from 1,79 mmHg to 16,79 mmHg (tertile I < 5,38; II 5,38 - 7,78; III > 7,78 mmHg). The groups were similar by age (I 56,6±8,94, II 59,4±9, III 60,7±9,1 yrs, $p>0,05$), gender, metabolic risk factors, baseline and achieved BP. Higher SBPV was associated with higher baseline central PP: for tertI 47,2±10,6, tertII 55,6±11, tertIII 51,1±11,5 mmHg ($p<0,05$ vs tertI). Number of patients with baseline central PP>50 mmHg was higher in the tertII(76,5%vs50% in tertII and 41%in tertI,Pearson's $\chi^2=2,1$;$p<0,05$) No significant difference was found for Aix@75HR(23,3±11,2 vs 23,2±13,4 vs 25,5±9,1% for corresponding tertiles,$p>0,05$) and PWV(13±1,6 vs14,2±2,2 vs12,9±1,8 m/s). No correlation was found between SBPV and any other characteristics, including baseline central PP.
Conclusion: Association between baseline central PP and SBPV after achievement of target BP confirms the role of arterial stiffness as predictor of BP variability.

P7.11
TESTOSTERONE AND CARDIOVASCULAR PERFORMANCE; THE IMPACT OF THE ANDROGEN DEFICIENCY IN ARTERIAL-VENTRICULAR COUPLING AND VASCULAR STIFFNESS DOCUMENTED BY 3D ECHOCARDIOGRPHY

A. Angelis, N. Ioakimidis, C. Vlachopoulos, K. Aznaouridis, M. Abdelrasoul, K. Aggeli
University of Athens, Athens, Greece

Purpose: Arterial-ventricular coupling (EA/ELV) constitutes an important determinant of cardiovascular performance and cardiac energetic. There is increasing evidence of testosterone influence to cardiovascular health. Aim of this work is to investigate the impact of testosterone deficiency (TD) on arterial-ventricular coupling, documented by the accuracy of the three dimensional echocardiography.
Methods: 76 men (56±11 years) underwent cardiac ultrasound and carotid-femoral pulse wave velocity (PWVc-f) evaluation. Based on systolic blood pressure (SBP), end systolic blood pressure was defined (ESP=0,9XSBP) and 3D- echo evaluation of ejection fraction (EF) and left ventricular outflow tract area (CSALVOT) were performed. Consequently left ventricular stroke volume (SV=VTI x CSA), end-diastolic (EDV) and end-systolic (ESV) volumes were estimated, followed by LV elastance (ELV=ESP/ESV-V0), arterial elastance (EA=ESP/SV) and arterial-ventricular coupling (EA/ELV) calculation. Testosterone deficiency considered when the total testosterone (TT) < 3.4 ng/m.