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

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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 Alx 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 Alx@HR75 in all time periods but night increase Alx was significantly more evident in men: respectively, Alx (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 Alx 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

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**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  $R^2 = 0.203$ ).

**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

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**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. 52 years,  $p < 0.001$ ), 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 within 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

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**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, age 58.9±9.0 yrs; 4 smokers; 6 diabetics) with uncomplicated AH were treated to target BP < 140/90 mmHg with combination of RAAS-inhibitor and amlodipine for 1 yr. Baseline BP was 163.4±8.1/100.9±4.2 mmHg; achieved-123.7±9.7/76.8±6.7 mmHg. 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% vs 50% in tertI 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 vs 14.2±2.2 vs 12.9±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 ECHOCARDIOGRAPHY

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**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/ml.

**Results:** Compared to normal TT levels, TD patients (n=19) were older ( $59 \pm 8$  vs  $52 \pm 10$  years,  $P < 0.05$ ) with higher BMI ( $28.6 \pm 4.0$  kg/m<sup>2</sup> vs  $27.0 \pm 4$  kg/m<sup>2</sup>,  $P < 0.05$ ). They had lower EF, SV and inversely, higher EA/ELV compared to subjects with normal TT. TD was also associated to a higher mitral E/E' and PWV-f. The association remained significant in multivariate analysis after adjustment for age and cardiovascular risk factors.

**Conclusion:** Testosterone deficiency associates to an unfavorable LV performance as well to central arterial stiffness, with an adverse outcome on cardiac energetic. This information adds clinical value on hormone lower level, in both cardiovascular risk assessment and stratification of future preventive strategies.

#### P7.12 CIRCULATING VASCULAR GROWTH FACTORS AND AORTIC INDICES IN GHANAIS WITH DIABETES AND HYPERTENSION

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**Objectives:** Impaired angiogenesis may be one mechanism linking large artery stiffness to organ damage. We investigated the relationship between arterial stiffness and regulators of angiogenesis as circulating vascular growth factors: vascular endothelial growth factor (VEGF), angiopoietin (Ang)-1, Ang-2, which together with endogenous VEGF induces proliferation and the sprouting of new blood vessels, in Ghanaians with type 2 diabetes (T2DM) and hypertension (HTN).

**Methods:** 63 T2DM plus HTN patients, 44 patients with T2DM only, 54 patients with HTN only and 39 subjects without T2DM nor HTN were included in the study. Aortic pulse wave velocity (PWVao) and aortic systolic pressure (SBPao), augmentation index (Aix) and aortic pulse pressure (PPao) were measured with Tensiomed's Arteriograph. Fasting blood samples were measured for blood glucose, lipid profile, Ang-1, Ang-2 & VEGF.

**Results:** T2DM plus HTN patients had higher levels of Ang-1 ( $44.3$  vs  $36.1$  and  $36.3$  ng/ml;  $p = 0.004$ ) & Ang-2 ( $875.65$  vs  $764.4$  and  $710.35$  pg/ml;  $p = 0.009$ ) than T2DM only and HTN only patients respectively. Ang-2 levels were positively associated with PWVao ( $r = 0.17$ ,  $p = 0.03$ ), SBPao ( $r = 0.28$ ,  $p < 0.01$ ), and Aix ( $r = 0.22$ ,  $p < 0.01$ ). When all the vascular growth factors were forced into multiple regression analysis, adjusting for age, BMI, systolic BP and fasting glucose, only Ang-2 emerged significantly related to PWVao ( $\beta = 0.027$ ,  $p = 0.02$ ), SBPao ( $\beta = 0.54$ ,  $p < 0.01$ ), Aix ( $\beta = 0.3$ ,  $p < 0.01$ ).

**Conclusion:** Vascular growth factors were related to arterial stiffness indices, Ang-2 independently, in Ghanaians, and higher in patients with both diabetes and hypertension than with either condition alone.

#### P7.13 DOES CAROTID ARTERY APPLANATION TONOMOMETRY CAUSE BAROREFLEX ACTIVATION?

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**Introduction:** Carotid artery applanation tonometry is widely used to assess carotid-femoral pulse wave velocity and the local carotid artery pressure waveform. However, the substantial pressure applied locally to the carotid artery with applanation tonometry, might evoke a baroreceptor response. This response would lead to changes in heart rate (HR) and blood pressure waveforms, influencing the intended measurements. In this study, we assessed whether carotid applanation tonometry has an influence on HR.

**Methods:** In 22 hypertensive subjects, HR was assessed during carotid as well as femoral applanation tonometry by continuous finger pulse waveform recording (Nexfin). Subjects were in supine position. Both carotid and femoral acquisitions were measured in alternation and in triplicate. Median averaging over the three measurements was used to obtain a subject's median HR during carotid as well as femoral tonometry.

**Results:** HR during carotid tonometry and femoral tonometry was  $64.0 \pm 9.3$  bpm and  $64.6 \pm 9.0$  bpm, respectively. Difference (carotid-femoral) was  $-0.7 \pm 2.4$  bpm ( $p = 0.198$ , two-sided t-test, 95% CI:  $[-1.7, 0.4]$  bpm). Given

a power (1- $\beta$ ) of 0.8 and  $\alpha = 0.05$ , our study was powered to statistically detect a 1.4bpm HR difference.

**Conclusion:** We conclude that carotid artery tonometry influences HR by at most 1.4bpm, which appears clinically insignificant.

#### P8.1 FEASIBILITY OF 24-HOUR CENTRAL BLOOD PRESSURE MEASUREMENTS—THE ISAR HEMODIALYSIS STUDY

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**Background:** Calculation of central blood pressure (BP) values using oscillometric systems at brachial level obtain feasible office values, but also allow a 24-hour determination of 24-hour central BP. We were interested in the feasibility of the determination of 24-hour central BP measurements in end-stage renal disease patients.

**Methods:** In the ISAR hemodialysis study 556 chronic hemodialysis patients were investigated. 24-hour central BP was measured using the mobil-o-graph (IEM, Germany). Measurement started after a short dialysis interval prior to dialysis and lasted for 24-hours. In a preliminary analysis we describe the results of the first 327 patients with respect to feasibility of central BP measurements.

**Results:** The mean age of the patients was  $65.0 \pm 15.1$  years. 224 patients were male (69%), 103 patients were female (31%). Out of these 327 patients 16.948 measurements were performed, reflecting an average of 52 measurements per patient. The mean number of measurements was  $>70\%$  for the whole cohort. Out of the 16.948 measurements 13.069 measurements had a "high quality" and 3.879 had an "acceptable" quality" reflecting a ratio of 3.4. In younger patients  $<40$  years more "high quality" measurements were present (ratio 9.8). This ratio reduced with age (2.6).

**Discussion:** We examined the feasibility of 24-hour central BP measurement in chronic hemodialysis patients. With  $>70\%$  performed central BP measurement throughout the 24-hours period this method offers acceptable results for further investigation. The role of different qualities of the determined central measurement needs further investigation especially whether quality of measurements plays a role in the prediction of cardiovascular events.

#### P8.2 PROGRESSION OF CAROTID ARTERY REMODELING AND STIFFNESS IN HYPERTENSIVE PATIENTS WITH OR WITHOUT DIABETES MELLITUS: A COHORT PROSPECTIVE STUDY

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**Aim:** To evaluate the progression over time of carotid and aortic stiffness and carotid remodeling in hypertensive patients, according to the presence of diabetes mellitus.

**Methods:** In this prospective observational study, 124 hypertensive patients (32 with Type 2 diabetes -HTDM-, 92 without -HT-) were evaluated at Visit 0 (V0) and after a  $3.4 \pm 1.0$ -year follow-up (V1). Carotid-femoral pulse wave velocity (PWV), carotid intima-media thickness (cIMT), carotid stiffness (CS) and circumferential wall stress (CWS) were assessed.

**Results:** In HT BP was unchanged, due to increased antihypertensive drugs ( $1.3 \pm 1$  to  $1.7 \pm 0.8$ ,  $p = 0.001$ ); in HTDM there was a decrease in DBP ( $82.5 \pm 9.1$  to  $76.2 \pm 8.6$  mmHg,  $p = 0.006$ ) and an improvement of metabolic control (blood glucose  $168 \pm 45$  to  $147 \pm 31$  mg/dl; LDL  $104 \pm 34$  to  $82 \pm 24$  mg/dl,  $p < 0.05$ ). At V0 PWV, cIMT and CS were significantly higher in HTDM than in HT ( $10.9 \pm 2.1$  vs  $8.6 \pm 1.5$  m/s,  $p < 0.0001$ ;  $808 \pm 125$  vs  $731 \pm 151$   $\mu$ m,  $p = 0.01$ ;  $7.23 \pm 1.25$  vs  $6.69 \pm 1.21$  m/s,  $p = 0.03$  respectively). These variables were unchanged during follow-up. Conversely carotid diameter, which was similar in the two groups at V0, increased in HT ( $7.47 \pm 1.11$  to  $7.8 \pm 0.8$  mm,  $p = 0.01$ ) but not in HTDM ( $7.7 \pm 0.9$  to  $7.7 \pm 0.8$  mm,  $p = 0.83$ ) as well as CWS (HT  $55 \pm 12$  to  $59 \pm 17$  kPa,  $p = 0.03$ ; HTDM  $52 \pm 13$  to  $54 \pm 21$  kPa,  $p = 0.53$ ).

**Conclusions:** In a cohort of hypertensive patients, followed-up for about 3 years and treated according to routine clinical practice, aortic and carotid stiffness, as well as cIMT did not change over time. Interestingly, in non-diabetic hypertensive patients there was a progression of carotid artery