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P11.30: HERITABILITY OF CENTRAL BLOOD PRESSURE AND PULSE PRESSURE – A TWIN STUDY

A.D. Tarnoki, D.L. Tarnoki, M.A. Stazi, E. Medda, R. Cotichini, L. Nistico, P. Lucatelli, E. Boatta, C. Zini, F. Fanelli, C. Baracchini, G. Meneghetti, G. Schillaci, G. Jermendy, J. Osztoivits, A. Lannert, A.A. Molnar, L. Littvay, Z. Garami, V. Berczi

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presented greater values 12.18 ± 2.51 vs 9.84 ± 1.75 in CT vs 9.71 ± 1.9 m/sec in TT genotype $p=0.04$, with ascending trend for the rest of the parameters ($p=NS$), but only ascending trends (without statistical significance) were registered in men.

Conclusion: In the present study, the presence of the CC homozygote status was associated with the increase of arterial rigidity.

P11.30

HERITABILITY OF CENTRAL BLOOD PRESSURE AND PULSE PRESSURE – A TWIN STUDY

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Objective: Central blood pressure (SBP_{ao}), peripheral and aortic pulse pressure (PP, PP_{ao}) are powerful predictor of cardiovascular events. No comprehensive twin study has investigated their heritabilities.

Methods: 389 Italian, Hungarian and American twin pairs (230 monozygotic and 159 dizygotic) underwent oscillometric arterial stiffness investigation (TensioMed Arteriograph, TensioMed Ltd., Budapest) to measure brachial and aortic augmentation index (Aix_{bra}, Aix_{ao}), pulse wave velocity on aorta (PWV_{ao}) and SBP_{ao}. MPLUS Version6 statistical software was used.

Results: Age, sex and country-adjusted heritability of SBP_{ao}, PP and PP_{ao} indicated 45.5% (95% confidence interval /CI/, 10.5 to 60.0%), 46.6% (95% CI, 29.8 to 58.0%), and 39.9% (95% CI, 1.4 to 53.9%). Unshared environmental effects accounted for the largest part of variance, respectively. Model fit was normal. Bivariate saturated model showed high and significant correlations between SBP_{ao}, PP_{ao} and arterial stiffness measures ($r=0.588$, $p<0.001$ between SBP_{ao} and Aix_{bra}; $r=0.587$, $p<0.001$ between SBP_{ao} and Aix_{ao}; $r=0.475$, $p<0.001$ between SBP_{ao} and PWV_{ao}; $r=0.582$, $p<0.001$ between PP_{ao} and Aix_{bra}; $r=0.581$, $p<0.001$ between PP_{ao} and Aix_{ao}; $r=0.456$, $p<0.001$ between PP_{ao} and PWV_{ao}). Non-significant correlations were estimated for PP and Aix ($r=-0.077$, $p=0.057$ between PP and Aix_{bra}; $r=-0.078$, $p=0.055$ between PP and Aix_{ao}; $r=0.083$, $p<0.05$ between PP and PWV_{ao}).

Conclusions: SBP_{ao}, PP and PP_{ao} are moderately heritable. High significant correlations were estimated between SBP_{ao}, PP_{ao} and arterial stiffness, suggesting a genetic background.

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P11.31

CLINICAL AND VASCULAR PARAMETERS CORRELATED WITH AUGMENTATION PRESSURE IN A BRAZILIAN HYPERTENSIVE POPULATION

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Background: Augmentation pressure (AP) has been considered an absolute index that represents vascular stiffness.

Objective: To evaluate clinical and vascular parameters in a Brazilian population presenting hypertension and increased augmentation pressure.

Methods: A cross-sectional study was carried out to evaluate hypertensive patients, both genders, aged 30-75 years. Cardiovascular risk was estimated using SCORE by gender, age, systolic blood pressure, total cholesterol and smoking status. Carotid-femoral pulse wave velocity (cfPWV) was measured by Complior SP, aortic pressures and AP were obtained using SphygmoCor device, and intima-media thickness (IMT) was measured by carotid ultrasonography.

Results: Subjects ($n=129$) were divided into two groups according to AP median (16mmHg). Individuals with increased AP were older (59 vs 51 years, $p<0.001$) and presented higher SCORE (4,0 vs 2,5%, $p<0.05$), pulse pressure (66 vs 48mmHg, $p<0.001$), time of hypertension (16 vs 8 years, $p<0.001$), total cholesterol (216 vs 193mg/dl, $p<0.01$), cfPWV (10.9 vs 9.8m/s, $p<0.01$), carotid intima-media thickness (0.87 vs 0.67mm, $p<0.05$), and lower estimated glomerular filtration rate (74 vs 84ml/min, $p<0.01$). All these variables were correlated with AP, but in a multiple linear regression, time of hypertension was the only parameter associated with AP.

Conclusion: Many clinical variables may contribute to an increased AP in hypertensive patients, and time of diagnosis seems to be important suggesting that intensive and early antihypertensive treatment could smooth the progress of patient's vascular status.

P12 – Techniques and Mechanisms 2

P12.01

MEASURING AORTIC DISTENSIBILITY WITH CMR USING CENTRAL PRESSURES ESTIMATED IN THE MAGNET: COMPARISON WITH CAROTID AND PERIPHERAL PRESSURES

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Objective: Evaluate the feasibility of local aortic distensibility measurement using central pressure estimation in the magnet, simultaneous to aortic imaging with cardiovascular magnetic resonance (CMR).

Methods: We studied 49 asymptomatic subjects (26 men, age 44 ± 18 years). Ascending aortic strain was determined by CMR using automated segmentation of SSFP cine acquisitions. Central pressures were estimated as: 1) carotid pressures using tonometry measured immediately after CMR; 2) estimated from brachial cuff pressure using Vicorder™ acquired simultaneously with aortic cine imaging in the magnet. Central pressures were used to calculate aortic distensibility defined as aortic strain over central pulse pressure (AAD-carotid using carotid pressure and AAD-vicorder using Vicorder) and the carotid augmentation index (Aix). Carotid-femoral pulse wave velocity (cfPWV) was measured using tonometry.

Results: Average \pm SD systolic brachial, carotid and Vicorder pressures were respectively: 114 ± 13 , 105 ± 13 , 106 ± 14 mmHg. We found a strong linear relationship between AAD-carotid and AAD-vicorder ($\beta=0.89$, $R^2=0.91$, $p<0.001$). The mean distensibility difference between the two methods was: -1.1 ± 12 mmHg and variability 0.9%. Distensibilities measured using brachial pressures were higher than using either central pressures (Table).

The correlations with age, Aix and cfPWV obtained using AAD-vicorder (respectively: $r=-0.82$, $r=-0.62$; $r=0.61$; $p<0.001$) were significantly higher than using AAD-carotid ($r=-0.79$, $r=-0.50$, $r=-0.58$; $p<0.001$).

Conclusions: Aortic distensibility may be measured by CMR using central pressures measured in the magnet, simultaneously with cine acquisitions. Resulting distensibilities are closely related to those using carotid pressures measured by tonometry outside the magnet and achieve higher correlation with age and markers of global aortic stiffness such as Aix and cfPWV.

Table: Average ascending aortic distensibilities according to central pressure measurement technique and age group

Distensibilities, $kPa^{-1} \cdot 10^{-3}$	Age < 50 years n=26	Age \geq 50 years n=23
AD peripheral (Brachial)	65 \pm 29	24 \pm 13
AAD central Carotid	80 \pm 34	31 \pm 17
AAD central Vicorder	83 \pm 37	30 \pm 18