P4.5: CHARACTERISTICS AND DETERMINANTS OF THE SUBLINGUAL MICROCIRCULATION IN A FLEMISH POPULATION

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Aortic stiffness is an independent predictor of adverse CV outcomes and elevated in COPD. However, the influence of gender on aortic stiffness in COPD has not been established. We hypothesized that males with COPD would have greater aortic stiffness than females.

**Methods:** As part of the ARCADE study, we assessed 500 patients with COPD confirmed by spirometry and 150 comparators. Aortic pulse wave velocity (PWV) was evaluated using the sphygonCor device. Other assessments included body composition, blood pressure, heart rate, number of exacerbations, smoking history and C-reactive protein and fibrinogen.

**Results:** Patients and comparators were similar in age, BMI and gender. Males with COPD (23%) had greater aortic PWV mean (SD)10.2 (2.7) than females,9.5 (2.4), p = 0.003. However, they were similar in age, FEV1%, BMI, peripheral and central blood pressure indices and heart rate, number of exacerbations, smoking history and inflammatory biomarkers, p > 0.05. The difference remained after controlling for age and peripheral mean arterial pressure (Adjusted R2 = 0.26, F = 6.15, p = 0.014). The gender difference was not evident in the comparator group.

**Conclusion:** Males with COPD had greater aortic stiffness compared to the females, independent of traditional cardiovascular risk factors. The increased aortic stiffness may explain the high incidence of fatal and non-fatal cardiac events in the male patients, which may offer a therapeutic target.


**P4.4**

**DOPPLER INDEXES OF LEFT VENTRICULAR SYSTOLIC AND DIASTOLIC FLOWS AND CENTRAL PULSE PRESSURE IN RELATION TO RENAL RESISTIVE INDEX IN A GENERAL POPULATION**

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**Background:** The cardio-renal interaction occurs via hemodynamic and humoral factors. Non-invasive assessment of renal hemodynamics is currently possible by assessment of renal resistive index (RRI) derived from intrarenal Doppler arterial waveforms. So far, only limited information is available regarding the relationship between RRI and cardiac hemodynamics in the general population. We investigated these associations in randomly recruited subjects from a Flemish population.

**Methods:** In 171 participants (48.5% women; mean age, 52.2 years), using conventional pulsed wave Doppler, we measured RRI (mean, 0.60) and left ventricular outflow tract (LVOT) and transmitral (E and A) blood flow peak velocities and its velocity time integrals (VTI). Using carotid applanation tonometry, we measured central pulse pressure (cPP) and arterial stiffness indexes such as augmentation pressure and carotid-femoral pulse wave velocity. cPP was lower in Whites than non-Whites (1.46 vs. 1.57µg/dL) and in women than men with no significant sex difference in PP and HT prevalence (P = 0.11). BPb was lower in Whites than non-Whites (1.46 vs. 1.57µg/dL). In multivariable analyses of all participants, BPb doubling was associated with higher (P < 0.0007) SBP, DBP and MAP (0.76 [CI, 0.38-1.13]); 0.43 [0.18-0.68]; and 0.54 [0.29-0.79]mmHg, respectively) and higher HT prevalence (44.7 vs. 36.8%). SBP, DBP and MAP (123.3 vs. 125.5; 68.9 vs. 71.2; and 87.1 vs. 89.3mmHg) were lower in women than men with no significant sex difference in PP and HT prevalence (P = 0.11). Associations with BPb were nonsignificant (P = 0.09) for SBP in women, but for DBP and MAP in non-Whites. Among men, SBP increased with BPb (P = 0.06) with effect sizes associated with BPb doubling ranging from -0.65mmHg in Whites to -1.61mmHg in Blacks. SBP and PP, interactions of ethnicity and sex with PPb were all significant (P = 0.027). In conclusion, small and inconsistent effect sizes in the associations of BP with BPb likely exclude current environmental lead exposure as a major HT cause in the US.

**P4.6**

**PULSATIVE AND STEADY BLOOD PRESSURE COMPONENTS IN RELATION TO ENVIRONMENTAL LEAD EXPOSURE IN THE NATIONAL HEALTH AND NUTRITION EXAMINATION SURVEY 2003-2010**

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In view of the declining environmental lead exposure in the US, we analyzed the National Health and Nutrition Examination Survey (2003-2010) for association of pulsatile and steady BP components and hypertension (HT) with blood lead (BPb). The 12,725 participants included 21.2% Blacks, 20.5% Hispanics, 58.4% Whites, and 48.7% women. Blacks compared with non-Blacks had higher SBP, DBP, and mean arterial pressure (MAP) (126.5 vs. 123.9; 71.9 vs. 69.6; and 90.1 vs. 87.7mmHg, respectively) and higher HT prevalence (44.7 vs. 36.8%). SBP, DBP and MAP (123.3 vs. 125.5; 68.9 vs. 71.2; and 87.1 vs. 89.3mmHg) were lower in women than men with no significant sex difference in PP and HT prevalence (P = 0.11). BPb was lower in Whites than non-Whites (1.46 vs. 1.57µg/dL) and in women than men (1.25 vs. 1.80µg/dL). In the associations of BPb doubling with BPb, DBP, and MAP (0.76 [CI, 0.38-1.13]; 0.43 [0.18-0.68]; and 0.54 [0.29-0.79]mmHg, respectively) with no change in SPBR associated with a 1-

**Methods:** In 281 subjects randomly recruited in a Flemish population, we measured SPBR using GlycoCheck software. SPBR is the distance between the median red blood cell column width and the estimated outer edge of the red cell in cell perfused lumina. We standardized SBPb to medians of haematocrit and density of perfused capillaries. In 42 participants, we computed repeatability coefficients (RC) expressing bias as percentage of maximal biological variation. We searched for significant (P < 0.05) coefficients of SPBR using stepwise regression.

**Results:** In 281 subjects (mean age, 51.2y; 53.0% women), SPBR averaged 1.80µm. RCs for intra- and inter-observer variability were ±53.4%. Of 14 potential covariates, only age and mean arterial pressure (MAP) and use of diuretics correlated with SPBR (p = 0.049). Changes in SPBR associated with a 1-SD increments in age (+16.6y) and MAP (+11.3mmHg) were -58.2mm and -35.5mm, while SPBR was 97.7mm wider in diuretic users. Disregarding 140 patients with albuminuria, hypertension, diabetes, and cardiovascular disease, the 5th and 95th percentiles of SPBR across age (-30y to +50y) ranged from 1.54µm to 1.43µm and from 2.52µm to 2.28µm, respectively.

**Conclusion:** SPBR variability is high probably because of physiological factors, because imaging is software controlled. The inverse association of SPBR with age and MAP might reflect a defense mechanism in the presence of these cardiovascular risk factors.

**P4.7**

**RELATIONSHIP OF DIFFERENT CARDIOVASCULAR TISSUE BIOMARKERS WITH ESTABLISHED RISK FACTORS AND FRAMINGHAM RISK SCORE IN MIDDLE-AGE SUBJECTS WITHOUT CARDIOVASCULAR EVENTS WITH ESTABLISHED RISK FACTORS AND FRAMINGHAM RISK SCORE IN MIDDLE-AGE SUBJECTS WITHOUT CARDIOVASCULAR EVENTS**

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The relations between emerging biomarkers of preclinical CV disease and established risk algorithms are not well defined. Aim: this study evaluated the relationships of various tissue CV biomarkers with Framingham risk score (FRS) and its individual determinants.

**Methods:** In 435 subjects without previous cardiovascular events (287 males, mean age 58.11, 56% diabetics (DM), 48% treated for hypertension (HBP), 51% with dyslipidemic treatment, 27% smokers), we measured radio-frequency immunosorbent assay (QAS) and QAS, carotid intima-media thickness (IMT), wave speed (WS) and local pulse pressure (Pp), carotid-femoral pulse wave velocity (PWV; Compilor), LV mass index (LVMI) and relative wall thickness (RWT).

**Results:** In 281 subjects randomly recruited in a Flemish population, we measured SPBR using GlycoCheck software. SPBR is the distance between the median red blood cell column width and the estimated outer edge of the red cell in cell perfused lumina. We standardized SBPb to medians of haematocrit and density of perfused capillaries. In 42 participants, we computed repeatability coefficients (RC) expressing bias as percentage of maximal biological variation. We searched for significant (P < 0.05) coefficients of SPBR using stepwise regression.

**Results:** In 281 subjects (mean age, 51.2y; 53.0% women), SPBR averaged 1.80µm. RCs for intra- and inter-observer variability were ±53.4%. Of 14 potential covariates, only age and mean arterial pressure (MAP) and use of diuretics correlated with SPBR (p = 0.049). Changes in SPBR associated with a 1-SD increments in age (+16.6y) and MAP (+11.3mmHg) were -58.2mm and -35.5mm, while SPBR was 97.7mm wider in diuretic users. Disregarding 140 patients with albuminuria, hypertension, diabetes, and cardiovascular disease, the 5th and 95th percentiles of SPBR across age (-30y to +50y) ranged from 1.54µm to 1.43µm and from 2.52µm to 2.28µm, respectively.

**Conclusion:** SPBR variability is high probably because of physiological factors, because imaging is software controlled. The inverse association of SPBR with age and MAP might reflect a defense mechanism in the presence of these cardiovascular risk factors.