P4.6: PULSATILE 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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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 sphygmocor 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 (225) 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, 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 = 26%).

**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 males, 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 of interest. We investigated the 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 central pulse pressure (C20)

0.027). In conclusion, small and inconsistent effect sizes in the associations of BP with BPb lead to reflect a defense mechanism in the presence of these cardiovascular risk factors.

**P4.6**

**PULSATILE 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 BPb, DBP, and mean arterial pressure (MAP) (126.5 vs. 123.9; 71.9 vs. 69.6; and 90.1 vs. 87.7 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. 89.3 mmHg) 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. 1.57 μg/dL) and in women than men (1.25 vs. 1.80 μg/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) with no change in PP (P = 0.063) or the odds of HT (P = 0.11). Associations with BPb were nonsignificant (P > 0.09) for SBP in women and for DBP and MAP in non-Whites. Among men, SBP increased with BPb (P = 0.060) with effect sizes associated with BPb doubled ranging from 0.65μmHg in Whites to 1.61μmHg in Blacks. For SBP and PP, interactions of ethnicity and sex with BPb 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.

**P4.7**

**RELATIONSHIP OF DIFFERENT CARDIOVASCULAR TISSUE BIOMARKERS 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 based (QIMT® and QAS®, Esaote) carotid intima-media thickness (IMT), wave speed (WS) and local pulse pressure (pPP), carotid-femoral pulse wave velocity (PWV; Compilo), LV mass index (LVMI) and relative wall thickness (RWT).

**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 blood cell perfused lumina. We standardized SPBR 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) correlated of SPBR using stepwise regression.

**Results:** In 281 subjects (mean age, 51.2y; 53.0% women), SPBR averaged 1.80μm. RRs for intra- and inter-observer variability were 0.53-0.4%. Of 14 potential covariables, 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 -58.2nm and -35.5nm, while SPBR was 97.7nm 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 1.25μm 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.

**References:**