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### **P20: AUGMENTATION INDEX ASSOCIATES WITH IMPAIRED EARLY VENTRICULAR EJECTION**

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**Conclusions:** Changing from seated to supine position imparts a BP change across the carotid-femoral arterial path, the majority of the effect being hydrostatic. Measuring cfPWV in these two stable BP positions allows calculation of the BP dependency of cfPWV.

#### P146

##### METHODOLOGICAL ASPECTS AND DETERMINANTS OF HYPEREMIA-MEDIATED SLOWING IN PULSE WAVE VELOCITY: A GENERAL POPULATION STUDY

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**Background:** Recent studies proposed that deceleration in pulse wave velocity (PWV) following hyperemia might reflect arterial distensibility and endothelial function. We therefore investigated methodological aspects and clinical determinants of newly proposed indexes of such flow-mediated slowing (FMS) in a community-based sample.

**Methods:** In 71 subjects (60.3 ± 13.7 years; 50.7% women), we continuously assessed brachial-radial pulse wave velocity (PWV) using Vicorder<sup>®</sup> equipment at rest and after 3 or 5 minutes suprasystolic occlusion to induce reactive hyperemia. We calculated the relative change ( $\Delta$ ) in PWV per 30s post-occlusion intervals. We performed stepwise regression analyses to assess determinants of the PWV response.

**Results:** The decline in PWV during hyperemia was significantly stronger after 5 minutes of occlusion as compared to 3 minutes (effect sizes for 0–180s intervals: –3.58% to –0.1%;  $P \leq 0.0019$ ). PWV declined significantly less with higher age during the 0–90s post-occlusion intervals (+1.61 to +3.99%;  $P \leq 0.023$ ). On the other hand, we observed that, after 120s of hyperemic response,  $\Delta$ PWV remained significantly lower in smokers (–4.28% to –5.37%) and subjects with high mean arterial pressure (–2.14% to –2.23%) and low pulse pressure (+2.06% to +2.07%;  $P \leq 0.046$  for all). Hence, compared to non-smoking normotensives, subjects with cardiovascular risk factors exhibited a delayed age-adjusted recovery of PWV after 5 minutes of occlusion ( $P \leq 0.039$ ).

**Conclusions:** Our findings confirm an occlusion time of 5 minutes for assessment of endothelial function by FMS. Whereas early FMS response might deteriorate with ageing, cardiovascular risk factors such as smoking and hypertension might impair the late recovery of PWV following reactive hyperemia.

#### Poster Session II – Clinical Aspects P18

##### THE ASSOCIATION BETWEEN METABOLIC SYNDROME COMPONENTS, ARTERIAL MARKERS OF EARLY ATHEROSCLEROSIS AND LEFT VENTRICULAR DIASTOLIC DYSFUNCTION

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**Background:** The aim of the study was to evaluate the relationship between MetS components and arterial stiffness in concert with left ventricular diastolic dysfunction (LVDD) in patients with high risk of cardiovascular disease.

**Methods:** A study was carried among 436 subjects (aged 53.8 ± 6, 37.2% men) without overt atherosclerotic disease and systolic LV dysfunction. The average of observations was 4.4 years. According to the MetS components (pathologically increased waist circumference – W, increased triglyceride – T, increased fasting plasma glucose – G, low high-density lipoprotein level-H, arterial hypertension – B) patients were divided into the metabolic phenotypes. Arterial stiffness parameters (carotid to femoral pulse wave velocity (cfPWV), aortic augmentation index (AlxHR75)) were assessed by applanation tonometry. Cardioankle vascular index (CAVI) was calculated using the VaSera VS-1000. Impaired relaxation was described as E/A < 1.0 and E/e' mean < 13. Participants were considered as having pseudonormal/restrictive LVDD if the E/e' mean ratio was  $\geq 13$ . In case of E/A > 1.0 and

e' septal  $\geq 8$ cm/s and e' lateral  $\geq 10$ cm/s diastolic function was interpreted as normal.

**Results:** Most of study subjects had LVDD at the first visit (n = 358, n = 171 with relaxation abnormalities and n = 187 with pseudonormalisation). In presented cohort the most common metabolic phenotypes were: WTGHB (n = 70), WGB (n = 66), WTGB (n = 61), WTB (n = 46), WTHB (n = 30), WGHB (n = 27). During the observation period we found significant changes of LV diastolic function distribution between metabolic phenotypes ( $p < 0.001$ ). All patients with WGHB phenotype at first visit had LVDD comparing with other groups. We found significant differences of arterial markers between first and follow up visits- in women (cfPWV 8.70 vs 8.94m/s,  $p < 0.001$ ), in man (CAVI 8.05 vs 8.45,  $p < 0.001$ ) and in whol cohort (AlxHR75 23.1 vs 24.1,  $p > 0.001$ ).

**Conclusion:** Metabolic phenotype is closely associated with the development of LVDD. Some metabolic phenotypes promote early arterial aging.

#### P19

##### INFLUENCE OF BRACHIAL ARTERY STIFFNESS ON FLOW-MEDIATED DILATATION IN HEALTHY YOUNG AND OLDER POPULATIONS

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**Background:** Increased brachial artery (BA) stiffness has previously been shown to affect the magnitude of FMD response in patients with high cardiovascular risk. However, it is unclear whether increased BA stiffness explains the diminished FMD response typically observed in a healthy older population. We determined whether BA stiffness would be greater in the older than the young population, and whether it would influence FMD responses in the former.

**Methods:** Data from 33 young (YNG: 27.5 ± 4.9yrs) and 33 older (OLD: 64.9 ± 3.6yrs) individuals were analysed. FMD was assessed with reactive hyperaemia using Ultrasound Advanced Open Platform (ULA-OP). All acquired raw data were post-processed using custom-designed software to obtain parameters of WSR and diameter. BA stiffness was calculated from BA systolic and diastolic diameters with simultaneous contra-lateral BA blood pressure measurements, and was expressed as pulse wave velocity (PWV) and  $\beta$ -stiffness index.

**Results:** Both PWV [YNG: 9.5(8.7–10.3) vs OLD: 9.4(8.6–10.2) m/s] and  $\beta$ -stiffness index [YNG: 17.5(14.7–20.2) vs OLD: 16.7(13.9–19.4) au] were similar between populations. In YNG, there was no association between BA stiffness parameters and diameter changes obtained during FMD and nitroglycerin-mediated dilatation assessments. The association was also absent in OLD during either assessment.

**Conclusions:** These results demonstrate that BA stiffness is not increased in the healthy older population compared to the young counterpart. Furthermore, there is no association between BA stiffness parameters and the FMD response in either population, suggesting that BA stiffness may not influence BA vasodilatory response in healthy adults.

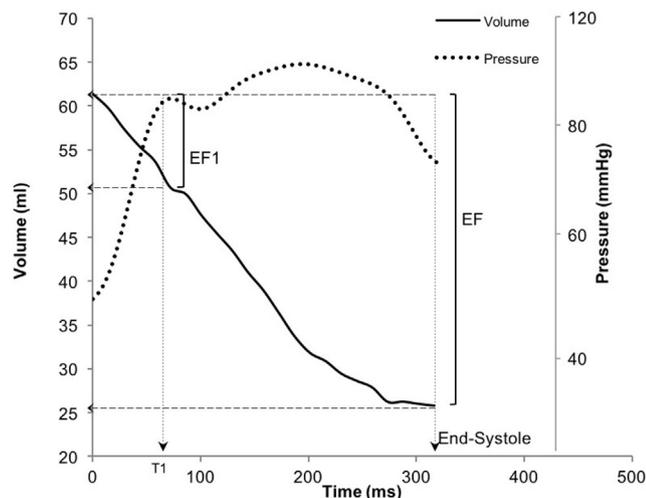
#### P20

##### AUGMENTATION INDEX ASSOCIATES WITH IMPAIRED EARLY VENTRICULAR EJECTION

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**Background:** Previously regarded as a measure of pressure wave reflection, central augmentation index (cAI) may be influenced by the pattern of early ventricular ejection. We examined the relationship of cAI to first-phase ejection-fraction (EF1), a measure of ventricular ejection up to the time of the first systolic peak in central pressure in patients with a wide range of cardiac and arterial phenotypes.

**Methods:** Carotid pressure, obtained by tonometry calibrated from peripheral mean and diastolic blood pressure, was used to calculate augmentation pressure (difference between the second and first systolic peaks of the aortic waveform) and index. Time-resolved LV volumes were obtained by 2D echocardiography. EF1 was defined as the fraction of LV volume ejected from the start of systole to the time of the first systolic peak (T1) on the carotid pressure waveform (Figure 1). Aortic arch to abdominal aorta pulse wave velocity (aPWV) was measured by pulsed wave Doppler.



**Results:** We studied 127 subjects, including healthy subjects ( $n = 44$ , aged  $51.5 \pm 13.6$  years) and patients with hypertension ( $n = 52$ ,  $53.6 \pm 12.9$ ), severe aortic stenosis (AS,  $n = 10$ ,  $73.5 \pm 9.6$ ) and Hypertrophic Obstructive Cardiomyopathy (HOCM,  $n = 21$ ,  $54.2 \pm 12.7$ ). Ejection-fraction ( $58.7 \pm 5.3\%$ ) was preserved in all subjects. There was a graded inverse relationship between EF1 and cAI across different disease groups (healthy:  $EF1 = 21.0 \pm 1.3\%$ ,  $cAI = 22.6 \pm 2.5\%$ ; hypertension:  $EF1 = 17.4 \pm 1.0\%$ ,  $cAI = 31.7 \pm 1.5\%$ ; AS:  $EF1 = 15.9 \pm 2.7\%$ ,  $cAI = 36.0 \pm 3.8\%$ ; HOCM:  $EF1 = 23.7 \pm 1.3\%$ ,  $cAI = -1.4 \pm 4.2\%$ ). In a multiple linear regression model, cAI was negatively associated with EF1 independent of age, gender, mean arterial pressure, aPWV and disease group (standardized regression coefficient  $\beta = -0.422$ ,  $p = 0.003$ ).

**Conclusion:** In patients with preserved EF, an impairment of early ejection is associated with greater augmentation pressure.

## P21

### REDUCTION OF CARDIAC PRE-LOAD HAS ANTITHETICAL EFFECTS ON BLOOD PRESSURE AND ARTERIAL STIFFNESS: IS BLOOD PRESSURE THE MAIN DETERMINANT OF WITHIN-SUBJECT VARIATION IN PULSE WAVE VELOCITY?

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**Objective:** Blood pressure (BP) is considered the most important determinant of within-subject variation in pulse wave velocity (PWV) and the possibility of altering arterial stiffness independently of BP is still a matter of debate. When investigating acute effects of a reduction in cardiac pre-load, we hypothesised that this would decrease BP and PWV.

**Design and methods:** Hypertensive patients (mean  $\pm$  SD age  $44 \pm 14$  years,  $n = 45$ ) had brachial BP measurements (OMRON), central BP recorded by radial pulse wave analysis (SphygmoCor) and estimation of aortic PWV (aoPWV) by trans-thoracic echocardiography. Carotid-femoral PWV (cfPWV)

was also evaluated by SphygmoCor in  $n = 17$ . Measurements were performed before and after ( $>5$  minutes) supra-diastolic, sub-systolic pressure inflation of thigh cuffs in order to decrease venous return from the lower limbs. Evaluation of inferior vena cava (IVC) diameter was used to assess pre-load.

**Results:** Leg cuff-inflation was effective in reducing cardiac pre-load (change in IVC diameter (mean  $\pm$  SE) from  $1.6 \pm 0.4$  cm to  $1.3 \pm 0.4$  cm,  $p < 0.01$ ) and decreased both brachial and central SBP ( $-3 \pm 0.9$  mmHg and  $-3.6 \pm 1.2$  mmHg respectively, both  $p < 0.05$ ) while change in DBP ( $0.8 \pm 0.9$  mmHg) and heart rate ( $-0.1 \pm 0.6$  bpm) were not significant. By contrast, aoPWV increased by  $0.8 \pm 0.35$  m/s ( $p < 0.01$ ) and cfPWV by  $1.05 \pm 0.33$  m/s ( $p = 0.014$ ).

**Conclusions:** Contrary to our hypothesis, acute reduction of cardiac pre-load significantly decreased BP but had an opposite effect on PWV. This could be mediated by an increase in sympathetic tone triggered by reduction in circulating blood volume; sympathetic tone might affect PWV independently of BP.

## P22

### CIRCADIAN VARIATIONS IN THE CARDIOVASCULAR SYSTEM

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**Background:** Comprehensive information on 24-hour profiles of pulsatile as well as steady-state hemodynamics in humans is not available yet.

**Methods:** In 693 healthy individuals (352 men) free from antihypertensive drugs, we performed 24-hour blood pressure (BP) monitoring with a validated oscillometric brachial cuff (mobilograph, i.e.m., Stolberg, Germany). Brachial waveforms were acquired and processed with ARCSolver algorithms to derive information on central pressures, wave reflections, stroke volume and systemic vascular resistance. Nighttime/daytime difference (N/D) was defined as nighttime (01.00–06.00) minus daytime (09.00–21.00) values / daytime values. Patients were categorized as young (Y: 15–40 years;  $n = 187$ ), middle-aged (M: 41–70 years;  $n = 446$ ), and old (O: 71–94 years;  $n = 60$ ).

**Results:** Averaged 24-hour brachial BP was 123/78 (Y), 127/82 (M), and 126/74 (O) mm Hg. N/D for brachial SBP was  $-13\%$  (Y),  $-12\%$  (M), and  $-5\%$  (O). N/D for heart rate was  $-20\%$  (Y),  $-17\%$  (M), and  $-15\%$  (O). N/D for central SBP was less pronounced:  $-4\%$  (Y),  $-6\%$  (M), and  $-0\%$  (O).

Brachial pulse pressure (PP) displayed small circadian variations, whereas central PP was higher at nighttime: N/D was 25% (Y), 14% (M), and 17% (O). Consequently, PP amplification was higher at daytime (N/D was  $-21\%$  (Y),  $-16\%$  (M), and  $-12\%$  (O)), and was related to heart rate, age, and gender. Measures of wave reflections were higher at nighttime, with N/D related to age, heart rate, mean pressure, systemic vascular resistance and stroke volume.

**Conclusion:** The circadian profiles we provide may serve as reference for cardiovascular diseases and drug studies.