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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[®] equipment at rest and after 3 or 5 minutes suprasystolic occlusion to induce reactive hyperemia. We calculated the relative change (Δ) 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, Δ 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 ≥ 13 . In case of E/A > 1.0 and

e' septal ≥ 8 cm/s and e' lateral ≥ 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 β -stiffness index.

Results: Both PWV [YNG: 9.5(8.7–10.3) vs OLD: 9.4(8.6–10.2) m/s] and β -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.