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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Methods: Carotid pressure, obtained by tonometry calibrated from periph-eral 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 (Figure1). 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 ± 13.6 years) and patients with hypertension (n = 52, 53.6 ± 12.9), severe aortic stenosis (AS, n = 10, 73.5 ± 9.6) and Hypertrophic Obstructive Cardiomyopathy (HOCM, n = 21, 54.2 ± 12.7). Ejection-fraction (58.7 ± 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 ± 1.3%, cAI = 22.6 ± 2.5%; hypertension: EF1 = 17.4 ± 1.0%, cAI = 31.7 ± 1.5%; AS: EF1 = 15.9 ± 2.7%, cAI = 36.0 ± 3.8%; HOCM: EF1 = 23.7 ± 1.3, cAI = -1.4 ± 4.2%). In a multiple linear regression model, cAI was negatively associated with EF1 independent of age, gender, mean arterial pressure, pulse pressure and disease group (standardized regression coefficient β = -0.422, p = 0.003).

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

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 126/70 (O), and 127/82 (M), and 126/70 (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.