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P22: CIRCADIAN VARIATIONS IN THE CARDIOVASCULAR SYSTEM

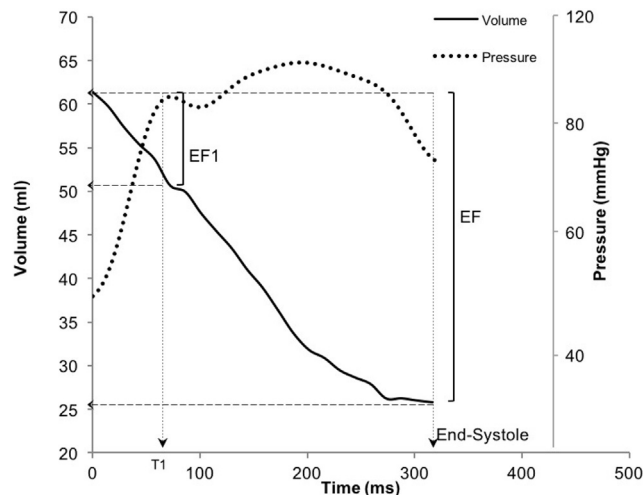
Thomas Weber, Siegfried Wasserheurer, Arno Schmidt-Trucksäss, Enrique Rodilla Sala, Piotr Jankowski, Maria Lorenza Muisan, Cristina Giannatasio, Ian Wilkinson, Joerg Kellermair, Bernhard Hametner, Jose Maria Pascual, Robert Zweiker, Danuta Czarnecka, Anna

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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 ± 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 ($58.7 \pm 5.3\%$) 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.

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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?

Luca Faconti¹, Bushra Farukh², Andrii Boguslavskyi², Phil Chowieniczky²

¹Department of Clinical Pharmacology, King's College London, UK

²King's College London British Heart Foundation, Department of Clinical Pharmacology, St Thomas' Hospital, London, UK

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 ± 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 ± 0.4 cm to 1.3 ± 0.4 cm, $p < 0.01$) and decreased both brachial and central SBP (-3 ± 0.9 mmHg and -3.6 ± 1.2 mmHg respectively, both $p < 0.05$) while change in DBP (0.8 ± 0.9 mmHg) and heart rate (-0.1 ± 0.6 bpm) were not significant. By contrast, aoPWV increased by 0.8 ± 0.35 m/s ($p < 0.01$) and cfPWV by 1.05 ± 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

Thomas Weber¹, Siegfried Wasserheuer², Arno Schmidt-Trucksäss³, Enrique Rodilla Sala⁴, Piotr Jankowski⁵, Maria Lorenza Muisan⁶, Cristina Giannatasio⁷, Ian Wilkinson⁸, Joerg Kellermair⁹, Bernhard Hametner², Jose Maria Pascual⁴, Robert Zweiker¹⁰, Danuta Czarnecka⁵, Anna Paini⁶, Massimo Salvetti⁶, Alessandro Maloberti⁷, Peter Fitscha¹¹, Kai Mortensen¹², Elisabeth Wagner¹, Carmel McEniery⁸, Cornelia Ablasser¹⁰

¹Cardiology Department, Klinikum Wels-Grieskirchen, Austria

²Center for Health & Bioresources, Department of Health and Environment, Austrian Institute of Technology, Vienna, Austria

³Department of Sport, Exercise and Health, Division Sport and Exercise Medicine, University of Basel, Switzerland

⁴Department of Medicina Interna, Hospital de Sagunto, Universidad Cardenal Herrera-CEU, CEU Universities, Valencia, Spain

⁵1st Department of Cardiology, Interventional Electrophysiology and Hypertension, Jagiellonian University, Medical College, Krakow, Poland

⁶Department of Clinical and Experimental Sciences, University of Brescia, Italy

⁷Cardiology IV, 'A. De Gasperi' Department, ASTT Ospedale Niguarda Ca' Granda, School of Medicine and Surgery Department, Milano-Bicocca University, Milan, Italy

⁸Division of Experimental Medicine and Immunotherapeutics, University of Cambridge, UK

⁹Cardiology Department, Kepler University Clinic, Linz, Austria

¹⁰Cardiology Department, Medical University Graz, Austria

¹¹Cardiologist, Vienna, Austria

¹²Universitätsklinikum Schleswig-Holstein, Campus Lübeck, Germany

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.