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### 8.7: CHANGES IN CARDIAC FUNCTION BUT NOT STRUCTURE IN HEALTHY SUBJECTS WITH PREMATURE VASCULAR AGEING

Oscar Mac Ananey, Vincent Maher

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### 8.5 HEMODYNAMICS DURING INTRA- AND INTERDIALYTIC PERIODS DEPEND ON ULTRAFILTRATION VOLUME

Christopher Mayer<sup>1</sup>, Stephan Geilert<sup>2</sup>, Julia Matschkal<sup>2</sup>, Uwe Heemann<sup>2</sup>, Marcus Baumann<sup>2</sup>, Christoph Schmaderer<sup>2</sup>  
<sup>1</sup>AIT Austrian Institute of Technology GmbH, Vienna, Austria  
<sup>2</sup>Technical University of Munich, Germany

**Introduction:** Parameters of arterial stiffness are independent cardiovascular risk factors for end-stage renal disease patients. Significant changes of these parameters between intra- and interdialytic periods have been reported previously [1]. The aim of this cross-sectional study is to describe the influence of the ultrafiltration volume on hemodynamic parameters.

**Methods:** All measurements were obtained with the Mobil-O-Graph 24h PWA (I.E.M. GmbH, Germany) within the ISAR hemodialysis study. Measurement started before the midweek dialysis session and lasted for 24-hours. 348 patients (238 male / 110 female 65 ± 18 years) were included. Intra- and interdialytic parameters were averaged and compared for three subgroups (ultrafiltration volume (UFV) ≤ 500 ml (N = 50) 500 < UFV < 2000 ml (N = 159) UFV > 2000 ml (N = 139)) and all subjects.

**Results:** The results for all patients support the findings of Karpetas et al. [1] (see Table). Beyond [1], the results underpin the differences between subgroups for intra- and interdialytic periods. Furthermore, there are significant differences between intra- and interdialytic periods depending on the ultrafiltration volume (see Table). Exemplarily, there is a significant rise in the augmentation index (26.0 vs. 28.5%, p < 0.05) for UFV > 2000 ml and for central pulse pressure (39.6 vs. 43.4 mmHg and 36.0 vs. 38.3, p < 0.05) for UFV ≤ 2000 ml opposed to non-significance for the other subgroups.

**Conclusions:** Our findings support the hypothesis that hemodynamic parameters depend on ultrafiltration volume. Further studies should investigate their prognostic value considering the ultrafiltration volume.

compliance, but its effect on peripheral arterial compliance (pC) is controversial. We aimed to test the hypotheses that aerobic training augments pC at rest and during different autonomic nervous system provocations (ANSP) in young healthy men.

We enrolled 44 males, 19-24 years old (22 trained, VO<sub>2</sub>max = 48 ml/kg/min – group A, 22 sedentary controls, VO<sub>2</sub>max = 30 ml/kg/min – group B). VO<sub>2</sub>max was determined using cycloergometry (QuarkCPET, Cosmed). On the testing day, ECG, arterial blood pressure (Finapres, Ohmeda) and finger artery compliance at rest, 3 minutes during 0.1 Hz breathing and 3 min during mental stress were measured. A noninvasive method was used to determine compliance index (CI), calculated as an average of the pressure dependant compliance curve in the range of arterial pressures from 97 to 105 mmHg.

Our results revealed elevated CI in group A compared to group B (4.18 ± 0.38 and 1.28 ± 0.25, p = 0.004) at rest and no significant differences in CI between groups during ANSP (1.34 ± 0.20 at 0.1 Hz breathing and 0.82 ± 0.18 during mental stress in group A compared to 1.09 ± 0.21, p = 0.06, and 0.60 ± 0.12, p = 0.08 in group B). A statistically significant positive linear correlation existed between CI and VO<sub>2</sub>max (P < 0.001) at rest in group A, however, no correlation was found at both ANSP.

Regular aerobic training increases pC in healthy young subjects at rest, but not during 0.1 Hz breathing or mental stress. Our findings indicate that peripheral and not central autonomic mechanisms govern pC in young healthy males.

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	UFV ≤ 500 ml		500 < UFV ≤ 2000 ml		UFV > 2000 ml		All	
	In	Out	In	Out	In	Out	In	Out
pSBP (mmHg)	128.9	131.0	123.7	122.3	124.5	123.0	124.8	123.8
pDBP (mmHg)	75.9	74.5	74.9	72.3	75.6	73.1	75.3	* 72.9
pPP (mmHg)	53.0	56.5	48.8	50.0	478.8	49.9	49.4	50.9
HR (bpm)	69.4	70.2	68.1	70.8	71.8	73.7	69.8	* 71.9
cSBP (mmHg)	116.9	119.3	112.4	111.9	113.4	112.6	113.4	113.2
cDBP (mmHg)	77.3	75.9	76.3	73.6	77.2	74.6	76.8	* 74.3
cPP (mmHg)	39.6	*	43.4	38.3	36.2	38.0	36.6	** 38.9
Alx (%)	29.7	31.8	29.5	30.2	26.0	*	28.5	* 29.8
Alx75 (%)	26.4	28.8	25.5	27.7	24.0	**	27.6	** 27.8
PWV (m/s)	10.03	10.14	9.88	9.85	9.08	9.07	9.58	9.58

**Table:** Averaged hemodynamic parameters for intra- and interdialytic periods (In vs. Out) for different subgroups based on ultrafiltration volume (UF) and all subjects. Abbreviations: peripheral diastolic blood pressure (pDBP), peripheral systolic blood pressure (pSBP), peripheral pulse pressure (pPP), heart rate (HR), central diastolic blood pressure (cDBP), central systolic blood pressure (cSBP), peripheral pulse pressure (pPP), augmentation index (Alx, Alx75) and pulse wave velocity (PWV) \*/\*\* marks a significant difference between intra- and interdialytic periods (p < 0.05 and p < 0.01, respectively).

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### 8.6 AEROBIC FITNESS LEVEL AND PERIPHERAL ARTERIAL COMPLIANCE – THE ROLE OF AUTONOMIC NERVOUS SYSTEM TONE

Nejka Potocnik, Ziva Melik, Ksenija Cankar, Martin Strucl  
 Medical Faculty, Institute of Physiology, University of Ljubljana, Slovenia

Physical activity has beneficial effects on prevention of cardiovascular disease. Aerobic fitness is associated with higher central arterial

### 8.7 CHANGES IN CARDIAC FUNCTION BUT NOT STRUCTURE IN HEALTHY SUBJECTS WITH PREMATURE VASCULAR AGEING

Oscar Mac Ananey<sup>1</sup>, Vincent Maher<sup>2</sup>

<sup>1</sup>School of Biological Science, Dublin Institute of Technology, Dublin, Ireland

<sup>2</sup>Department of Cardiology, Tallaght Hospital, Dublin, Ireland

**Purpose:** Changes in myocardial and arterial wall properties/function are consistently reported in patients with established cardiovascular disease<sup>1</sup>. However, few studies have reported these changes in early subclinical disease. The aim of the present study was to examine cardiac and vascular changes in early subclinical disease and to determine whether these changes occur in parallel.

**Methods:** For this study, 98 healthy lifelong never smokers were recruited. Subjects were categorised as having normal (Norm, n=71) or abnormal (High, n=27) arterial stiffness (carotid-femoral pulse wave velocity, PWV Vicorder, Skidmore, UK) for their age and blood pressure. M-mode Doppler echocardiography (Vivid 7 Dimension, GE, USA) was used to assess heart structure (interventricular septal thickness, IVSd left ventricular internal diameter, LVIDd left ventricular posterior wall thickness, LVPW left ventricular mass, LV Mass) and function (left ventricular isovolumetric relaxation time, LV IVRT mitral valve early/late filling velocity, MV E/A).

**Results:** No differences in age ( $39+/-9$  v  $39+/-9$  years P 0.87) or BMI ( $24.85+/-3.29$  v  $25.75+/-3.68$  kg.m<sup>2</sup> P 0.24) were observed. No differences in IVSD ( $0.86+/-0.15$  v  $0.85+/-0.18$  cm P 0.64), LVIDd ( $4.98+/-0.55$  v  $4.96+/-0.42$  cm P 0.95), LVPWd ( $0.81+/-0.17$  v  $0.90+/-0.21$  cm P 0.05) or LV Mass ( $168.86+/-56.85$  v  $182.61+/-61.70$  g P 0.43) were observed. However, MV E/A ( $1.85+/-0.51$  v  $1.48+/-0.51$  P 0.0004), but not LV IVRT ( $0.09+/-0.02$  v  $0.09+/-0.01$  P 0.25), was different.

**Conclusions:** Changes in cardiac function are observed before alterations in cardiac structure in healthy subjects with premature vascular stiffening.

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### 8.8

#### SYMPATHETIC VASOCONSTRICTOR RESPONSE TO LOWER BODY NEGATIVE PRESSURE IN YOUNG OBESE ADULTS: THE PRELIMINARY FINDING

Kanokwan Bunsawat, Georgios Grigoriadis, Bo Fernhall, Tracy Baynard  
University of Illinois at Chicago, Chicago, USA

**Background:** Elevations in muscle sympathetic nerve activity (MSNA) and sympathetic vasoconstrictor responsiveness to sympathoexcitation are associated with increased cardiovascular risks, which affect hemodynamics, and have been reported in obese adults with metabolic syndrome (1-3). It remains unclear whether this observation may also be present in young metabolically healthy obese adults.

**Purpose:** To compare sympathetic vasoconstrictor and hemodynamic responsiveness to lower body negative pressure (LBNP, -20 mmHg) in young normal-weight (NW) vs. obese (OB) adults.

**Method:** Eleven NW (female=6;  $25\pm 2$  yrs;  $22.4\pm 0.6$  kg/m<sup>2</sup>) and 13 OB adults (female=6;  $27\pm 1$  yrs;  $32.7\pm 0.6$  kg/m<sup>2</sup>) underwent 2-min of LBNP in the supine position. Ultrasonography [brachial diameter, forearm blood flow (FBF), forearm vascular conductance (FVC)], MSNA [burst frequency, total MSNA, sympathetic vascular transduction], and beat-to-beat hemodynamics [heart rate (HR), mean arterial pressure (MAP), total peripheral resistance (TPR), cardiac output (CO), stroke volume (SV), systemic compliance (SC)] were reported. FBF and FVC were normalized to lean forearm mass, and TPR, CO, SV, and SC to body surface area.

**Results:** Baseline MAP was lower in OB (P<0.05). In response to LBNP, normalized FBF, FVC, SV, CO, and SC decreased whereas TPR increased similarly in both groups (P<0.05). Brachial diameter and HR did not change in both groups. MAP decreased similarly by ~2-4 mmHg, but the values were lower in the OB group (P<0.05). Burst frequency, total MSNA, and sympathetic vascular transduction increased similarly in both groups (P<0.05).

**Conclusion:** Young metabolically healthy obese adults did not exhibit altered sympathetic vasoconstrictor responsiveness under resting condition.

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### 8.9

#### REDUCTION IN MYOCARDIAL WALL STRESS AND DELAYED MYOCARDIAL RELAXATION DURING EXERCISE

Haotian Gu, Xiaoli Zhang, Benyu Jiang, Sally Brett, Phil Chowienczyk  
King's College London, UK

**Introduction:** Myocardial wall stress (MWS) is thought to be the mechanical stimulus to ventricular hypertrophy (1,2). The objective of this study was to examine the effect of exercise on time-varying MWS (3).

**Methods:** Twelve subjects, aged  $42.0 \pm 16.8$  (mean  $\pm$  SD) years, systolic blood pressure (BP) ( $128 \pm 11$  mmHg), were studied before and during peak bicycle exercise (85% of target heart rate). We estimated MWS from 3D transthoracic echocardiographic imaging of the left ventricle (LV) and LV pressure was derived from carotid tonometry during systole. Carotid pressure calibrated by mean and diastolic BP was used to calculate time-varying LV wall stress from endocardial and epicardial volumes obtained from Philips 3DQ analysis package. Time of onset relaxation (TOR) was defined as percentage of time to peak wall stress to ejection duration.

**Results:** There was a significant reduction in peak and mean MWS during exercise (rest  $435.3\pm 25.3$  VS exercise  $385.9\pm 22.5$ ,  $p=0.001$  and  $387.3\pm 24.2$  VS  $368.7\pm 19.6$  kdynes/cm<sup>2</sup>,  $p=0.016$ ), despite significant increase in systolic BP ( $128\pm 3$  VS  $210\pm 6$  mmHg,  $p<0.001$ ). LV end-diastolic volume (EDV) ( $119.3\pm 9.4$  VS  $95.2\pm 7.8$  ml,  $p<0.001$ ) and volume at time of peak MWS ( $86.5\pm 7.0$  VS  $68.3\pm 6.3$  ml,  $p=0.001$ ) were reduced significantly during exercise, but TOR was delayed ( $24.5\pm 1.2$  VS  $31.0\pm 1.6\%$ ,  $p=0.003$ ).

**Conclusion:** Peak and mean MWS were reduced during peak exercise as a result of reduction in pre-load, despite of significant increase in systolic BP. But there was evidence of delayed myocardial relaxation during exercise.

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### 8.10

#### BRACHIAL ARTERY FLOW-MEDIATED DILATATION: DIFFERENT PATTERNS OF WALL SHEAR RATE INCREASE DURING REACTIVE HYPERAEMIA

Kunihiko Aizawa<sup>1</sup>, Sara Sbragi<sup>2</sup>, Alessandro Ramalli<sup>3</sup>, Piero Tortoli<sup>3</sup>, Francesco Casanova<sup>1</sup>, Carmela Morizzo<sup>2</sup>, Clare Thorn<sup>1</sup>, Angela Shore<sup>1</sup>, Phillip Gates<sup>1</sup>, Carlo Palombo<sup>2</sup>

<sup>1</sup>Diabetes and Vascular Medicine Research Centre, NIHR Exeter Clinical Research Facility, University of Exeter Medical School, Exeter, UK

<sup>2</sup>Department of Surgical, Medical, Molecular Pathology and Critical Care Medicine, University of Pisa, Italy

<sup>3</sup>Department of Information Engineering, University of Florence, Italy

**Background:** Wall shear rate (WSR) is considered an important stimulus for flow-mediated dilatation (FMD). However, its estimation by conventional ultrasound is challenging due to inherent difficulties of velocity estimation near the arterial wall. To evaluate how WSR influences brachial artery FMD, we used a prototype Doppler ultrasound system which provides simultaneous estimates of WSR at near and far walls and continuous arterial diameter tracking.

**Methods:** Data from 33 young healthy individuals ( $27.5\pm 4.9$  yrs, 19F) were analysed. FMD was assessed with a conventional reactive hyperaemia technique using Ultrasound Advanced Open Platform (ULA-OP). All acquired raw data were post-processed using custom-designed software to obtain WSR and diameter parameters.

**Results:** Baseline diameter and FMD were  $3.29\pm 0.45$  mm and  $6.54\pm 3.54$  %, respectively. During hyperaemia, we observed two distinct patterns of increased WSR: monophasic (MOP, n=15 fast increase reaching peak WSR at once) and biphasic (BIP, n=18 fast followed by slow increase before reaching peak WSR). In BIP, peak WSR ( $657\pm 153$  sec<sup>-1</sup> vs  $522\pm 132$  sec<sup>-1</sup>) and WSR area under the curve until peak dilation ( $20398\pm 6265$  au vs  $13530\pm 5592$  au) were significantly greater than in MOP (both  $p<0.05$ ). Absolute diameter increase was significantly greater in BIP ( $0.24\pm 0.10$  mm) than in MOP ( $0.15\pm 0.09$  mm,  $p<0.05$ ). Percentage diameter increase tended to be greater in BIP ( $7.6\pm 3.3$  %) than MOP ( $5.3\pm 3.5$  %,  $p=0.08$ ).

**Conclusions:** These results demonstrate that there are distinct WSR increase patterns during hyperaemia, and that these patterns are associated with differences in the magnitude of hyperaemic WSR. Our observations suggest that