



# **Artery Research**

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# P6.07: AORTIC STIFFNESS IS AN INDEPENDENT PREDICTOR OF NEW ONSET ATRIAL FIBRILLATION IN CHRONIC HEART FAILURE PATIENTS WITH REDUCED SYSTOLIC FUNCTION

S. Bonapace, A. Rossi, M.A. Cicoira, G. Arcaro, F. Valbusa, E. Barbieri, C. Vassanelli

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significantly higher PWV values (11.5 $\pm$ 2.7 and 11.4 $\pm$ 3 m/sec respectively) than patients with normal heart geometry (10.2 $\pm$ 2.6 m/sec) (p  $\leq$  0.001 for both). **Conclusions:** In EH patients arterial stiffness is associated with the degree of cardiac damage. This may reflect a common pathway leading to these alterations caused by hypertension in different but tightly related organs such as heart and arteries.

#### P6.06

## SYSTEMIC ARTERIAL PROPERTIES DURING NORMAL PREGNANCIES IN HEALTHY WOMEN

M. E. Estensen <sup>1,5</sup>, E. W. Remme <sup>2</sup>, A. Swillens <sup>3</sup>, T. Henriksen <sup>4</sup>,

O. A. Smiseth <sup>5</sup>, L. Gullestad <sup>5</sup>, P. Segers <sup>3</sup>, S. Aakhus <sup>5</sup>

<sup>1</sup>National Resource Centre for Women's Health, Oslo University Hospital, Rikshospitalet, Oslo, Norway

<sup>2</sup>Institute for Surgical Research, Oslo University Hospital, Rikshospitalet, Oslo, Norway

<sup>3</sup>IBiTech, Ghent University, Ghent, Belgium

<sup>4</sup>Department of Obstetrics, Oslo University Hospital, Rikshospitalet, Oslo, Norway

<sup>5</sup>Department of Cardiology, Oslo University Hospital, Rikshospitalet, Oslo, Norway

**Purpose:** During normal pregnancy (NP), the cardiovascular system adapts to the metabolic needs of mother and foetus with increased cardiac output (CO) and reduced vascular resistance. In NP blood pressure is not increased despite an increase of CO due to vasodilation of the resistance vessels. It is unknown whether this is also modulated by changes in the properties of the large arteries. **Methods:** 65 (33±1 years) with NP were investigated at gestational weeks 14-16, 22-24, 36 and 6 months postpartum (PP). Aortic root pressure and flow were obtained by calibrated right subclavian artery pulse trace, and aortic annular Doppler flow recordings. Systemic arterial properties were described by total arterial compliance(C), arterial elastance (Ea), characteristic impedance ( $Z_0$ ), and peripheral arterial resistance (R). Wave reflection was assessed as the ratio of the magnitude of the backward (Pb) to forward (Pf) pressure and flow data and methods based on the 2-element windkessel model.

**Results:** (Table) During NP, CO increased due to increased heart rate and stroke volume, whereas, blood pressures were lower as compared to 6 months PP. R was significantly reduced accompanied by a marginally lowered Ea and  $Z_0$ , whereas C was unchanged. The forward and backward pressure wave-amplitudes were significantly reduced, and reflection magnitude trended lower in mid to late pregnancy.

**Conclusions:** During NP profound alterations of systemic hemodynamics occur, with increased cardiac output and reduced blood pressures, where the latter is related mainly to reduction in peripheral arterial resistance.

 $({\rm LV})$  systolic function, The causes of its high incidence and prevalence in CHF are only partially understood.

**Aim**: To analyze the hemodynamic determinants of incident atrial fibrillation in CHF patients. Particulary, whether indexes of arterial stiffness might identify patients at risk for new onset AF.

Methods: 77 patients (mean age  $62.8\pm9.3$  years, ejection fraction 34.5 $\pm$ 8.3%, male 80%) were enrolled. All patients underwent a complete echocardiographic-Doppler evaluation. Aortic-pulse wave velocity (PWV) was determined by Doppler flow recordings as previously reported. Effective arterial elastance (Ea) was estimated as end-systolic pressure/ stroke volume (SV). Total arterial compliance (SAC) was calculated as SV/PP. Results: 15 (19.5%) patients developed AF during the follow up. There were no differences in age, SBP, DBP, PP, LV-EF, left atrial volume, plasma neurohormones and procollagens. Those with AF had higher aortic-PWV  $(7.0\pm2.5 \text{ vs } 5.3\pm1.9 \text{ m/sec}, p = 0.004)$ , higher Ea  $(1.68\pm0.46 \text{ vs})$ 1.35±0.47 mmHg/mL, p=0.03), lower SAC (1.57±0.55 vs 1.96±0.53 mL/ mmHg, p=0.02), higher time difference between pulmonary vein Ar-wave and mitral A-wave duration (PV-Ar-A) ( $43.5\pm44.2$  vs  $10\pm34.7$  ms, p=0.02) and mitral E-wave velocity (0.76 $\pm$ 0.23 vs 0.59 $\pm$ 0.24 m/s, p=0.02). In bivariate logistic regression models aortic-PWV predicted always independently incident AF:

Variables	OR	95% CI	p-value
Aortic-PWV	1.4	1.07-1.94	0.01
SAC	0.3	0.07-0.96	0.04
Aortic-PWV	1.5	1.09-1.97	0.01
Ea	2.9	0.95-8.9	0.06
Aortic-PWV	1.9	1.2-3.0	0.003
PV-Ar-A	1.04	1.0-1.07	0.01

 ${\bf Conclusion:}$  Aortic stiffness independently predicts incident AF in CHF with impaired LV-systolic function.

## Experimental Studies P7.01

### EFFECTS OF OMEGA 3 SUPPLEMENTATION ON ARTERIAL STIFFNESS IN RAT EXPERIMENTAL MENOPAUSE

P. Losurdo, E. Panizon, M. Jevnikar, L. Macaluso, B. Fabris, B. Toffoli, M. Bardelli, F. Fischetti, G. Biolo, M. Zanetti, S. Mazzucco,

G. Gortan Capellari, R. Carretta

Clinical Depart. of Medical, Technological, Translational Sciences of Trieste University, Trieste, Italy

	14-16 weeks	22-24 weeks	36 weeks	6 months PP	ANOVA P
Mean arterial pressure (mmHg)	82.8 ± 6.6	80.0 ± 6.2#	84.8 ± 7.1#¤	$88.0\pm7.3^{\tt m}$	<0.001
Heart rate (min <sup>-1</sup> )	71 ± 7	$74\pm8$ #	$77 \pm 10$ #¤	66 ± 7 #¤§	<0.001
Cardiac output (l min <sup>-1</sup> )	$\textbf{6.1} \pm \textbf{1.1}$	$\textbf{6.1} \pm \textbf{1.1}$	$\textbf{5.8} \pm \textbf{1.0}$	4.9 ± 0.9 #¤§	<0.001
R (mmHg ml <sup>-1</sup> s <sup>-1</sup> )	$\textbf{0.85} \pm \textbf{0.18}$	$\textbf{0.81} \pm \textbf{0.16}$	$0.92\pm0.23^{\tt m}$	$1.10 \pm 0.29$ #¤§	<0.001
Z <sub>0</sub> (10 <sup>3</sup> -mmHg ml <sup>-1</sup> s <sup>-1</sup> )	$43 \pm 18$	$51\pm36$	$45 \pm 23$	55 ± 21	0.168
C WK (ml mmHg <sup>-1</sup> )	$\textbf{1.45} \pm \textbf{0.33}$	$\textbf{1.56} \pm \textbf{0.45}$	$\textbf{1.55} \pm \textbf{0.46}$	$\textbf{1.40} \pm \textbf{0.45}$	0.220
C PPM (ml mmHg <sup>-1</sup> )	$\textbf{1.25} \pm \textbf{0.26}$	$\textbf{1.23} \pm \textbf{0.28}$	$\textbf{1.22} \pm \textbf{0.33}$	$\textbf{1.14} \pm \textbf{0.30}$	0.454
Ea (mmHg ml <sup>-1</sup> )	$\textbf{1.0} \pm \textbf{0.25}$	$\textbf{1.02} \pm \textbf{0.24}$	$1.19\pm0.28^{\tt m}$	$\textbf{1.27} \pm \textbf{0.28}\text{\#}\text{x}$	<0.001
Amplitude Forward Wave (Pf; mmHg)	$31\pm 6$	$30\pm9$	$28 \pm 5$ #	$30\pm6$	0.013
Amplitude Backward Wave (Pb; mmHg)	$20\pm3$	$17 \pm 4$ #	$17 \pm 4$ #	$19 \pm 4$ §	<0.001
Reflection magnitude (Pb/Pf)	$\textbf{0.64} \pm \textbf{0.13}$	$\textbf{0.59} \pm \textbf{0.15}$	$\textbf{0.61} \pm \textbf{0.13}$	$\textbf{0.64} \pm \textbf{0.13}$	0.051

Mean  $\pm$  SD. p< 0.05 vs #14-16 w, ¤22-24 w, §36 w. WK = C obtained using windkessel model fit; PPM= pulse pressure method.

#### P6.07

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S. Bonapace  $^1,$  A. Rossi  $^2,$  M. A. Cicoira  $^2,$  G. Arcaro  $^3,$  F. Valbusa  $^3,$  E. Barbieri  $^1,$  C. Vassanelli  $^2$ 

<sup>1</sup>Division of Cardiology, Sacro Cuore Hospital, Negrar-Verona, Italy

<sup>2</sup>Division of Cardiology, University of Verona, Verona, Italy

<sup>3</sup>Division of Medicine, sacro Cuore Hospital, Negrar-Verona, Italy

Background: Atrial fibrillation (AF) is the commonest supraventricular arrhythmia in chronic heart failure (CHF) with impaired left ventricular

Aim: To investigate the effects of omega-3  $(\Omega 3)$  dietary supplementation on large artery mechanics and vascular oxidative stress (VOS), in a rat model of surgical menopause.

Materials and methods: Thirty, 6-months-old, Wistar-Kyoto rats were equally divided into 3 groups: a) control: sham surgery - normal diet (CTRL)-, b) ovariectomy, - normal diet (OVX) -and c) ovariectomy, +  $\Omega$ 3 supplementation (0,8g/Kg/day - daily gavages administration-) (OVX+ $\Omega$ 3). Two months after surgery, carotid-femoral Pulse Wave Velocity (c-f PWV) and arterial pressure were directly measured, by aortic and femoral catheter. VOS was assessed by superoxide anion generation, in aorta rings, (SOD-inhibitable cytochrome C reduction assay). Erythrocytes membrane  $\Omega$ 3 index was measured by gas-chromatography.