



## **Artery Research**

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## P.013: BLOOD PRESSURE, ARTERIAL RIGIDITY AND ALCOHOL INTAKE IN A POPULATION SAMPLE\*

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**To cite this article**: O. Mayer, J. Filipovský, M. Dolejšová, L. Handl (2007) P.013: BLOOD PRESSURE, ARTERIAL RIGIDITY AND ALCOHOL INTAKE IN A POPULATION SAMPLE<sup>★</sup>, Artery Research 1:2, 55–55, DOI: https://doi.org/10.1016/j.artres.2007.07.070

To link to this article: https://doi.org/10.1016/j.artres.2007.07.070

Published online: 21 December 2019

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including hypertrophy, vascular inflammation, myocyte necrosis and fibrosis. This study set out to evaluate whether spironolactone had a beneficial effect on arterial stiffness (in comparison to bendrofluthiazide) and to what extent this could be predicted by the aldosterone renin ratio (ARR).

Methods: This is a substudy of a double-blind, randomised, crossover, trial in hypertensive subjects with either a high ARR (> 750 and a plasma aldosterone > 250pmol/l), or low ARR (< 300 and a plasma renin activity <10ng/ml/h). Each group underwent 12 weeks treatment with spironolactone 50mg OD and bendroflumethiazide (BFZ) 2.5mg OD in random order, separated by a 2-week washout. Brachial pulse wave velocity (Br-PWV) and pulse wave analysis for central blood pressure, augmentation index (Alx) and time of reflection (Tr) measurements using Sphygmocor technique was conducted at the end of each treatment.

**Result:** 98 subjects (59 high and 39 low ARR) completed the sub-study. Reduction in central SBP was significantly greater after spironolactone compared with BFZ for both the high and low ARR groups (Delta ( $\Delta$ ) -3.39 mmHg P< 0.035) with no difference between the groups in relative response to spironolactone and BFZ. There were no differences in Br-PWV, Alx or Tr between treatments or the ARR groups (P> 0.05 for all).

**Conclusion:** The result of this study suggests that the benefit of spironolactone on blood pressure is not influenced by the prevailing state of activation of the RAAS. There was no convincing evidence that mineralocorticoid antagonism had a beneficial effect on arterial stiffness independent of blood pressure lowering.

## P.010 RENAL ARTERY STENOSIS AND ITS DIAGNOSTICS IN TYPE 2 DIABETIC PATIENTS

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Aims: The purpose of this study was to evaluate sensitivity and safety of several techniques for renal artery stenosis (RAS) detection in subjects with type 2 diabetes and coexistent hypertension.

Materials and methods: We studied 157 patients. All of patients underwent duplex sonography (DS). Patients with RAS detected by DS underwent magnetic resonance angiography (MRA) or multislice computed tomography (MSCT) of the renal arteries. We used 1.5 T MR scanner and 16 slices MSCT. Results: We found that DS detected RAS in 58 (36.3%; 28.7% unilateral, 7.6% bilateral) type 2 diabetics (specially in smoking males) with myocardial dysfunction and rheological abnormalities. 10 patients with glomerular filtration rate (GFR) <60 ml/min but >30 ml/min underwent MRA of renal arteries and abdominal aorta with bolus injection of 20 ml gadolinium (Gd) based contrast agent. The diagnosis was confirmed in 8 cases, (6 subjects had unilateral RAS, 2 subjects had bilateral RAS) (sensitivity 80%). 28 patients with GFR >60 ml/min underwent MSCT of renal arteries and abdominal aorta with bolus injection of 50 ml "iso-osmolar" non-ionic contrast agent. The diagnosis was confirmed in 22 cases (16 subjects had unilateral RAS, 6 subjects had bilateral RAS) (sensitivity 78,6%). Serum creatinine, was recorded for three consecutive days after procedures. There was no change from the baseline in both groups.

**Conclusions:** DS is a valid routine method of investigation of diabetics at risk for RAS. MRA and MSCT are safe methods of RAS verification.

P.012
THE ASSOCIATION BETWEEN FREE THYROXINE, AORTIC RIGIDITY AND GENETIC POLYMORPHISM OF ANGIOTENSIN II TYPE 1 RECEPTOR IN A POPUL ATION SAMPLE\*

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**Background:** Thyroid hormones showed direct proliferative properties on cardiovascular system also by modulating the expression of renin-angiotensin axis. The aim of our study was to establish, whether interaction between mutation of angiotensin II, type 1 receptor (AGTR1) and mild changes in free thyroxine (fT4) may influence the aortic rigidity.

**Methods:** 249 euthyroid subjects (m121, f128, mean age  $48.03\pm0.70$ ) was selected from population based-study. Aortic pulse wave velocity (PWV) was measured using Sphygmocor, A1166C mutation of AGTR1 by PCR. The

sample was stratified according to fT4 quintiles for optimal ( $2^{nd}$  to  $4^{th}$  quintile), low-normal ( $1^{st}$  quintile) and high-normal ( $5^{th}$  quintile) fT4 strata. **Results:** [mean $\pm$ SEM; optimal vs. low-normal ( $p_1$ ) or high-normal ( $p_2$ ), \*adjusted for age and gender]

fT4 strata:	optimal	low-normal	$p_1$	high-normal	$p_2$		
A1166C mutation absent:							
n	72	32		32			
age	$\textbf{47.7} \pm \textbf{1.29}$	$\textbf{49.0} \pm \textbf{2.17}$	0.45	$\textbf{51.34} \pm \textbf{1.91}$	0.10		
systolic blood	$\textbf{122.6} \pm \textbf{1.72}$	$\textbf{128.4} \pm \textbf{3.40}$	0.12	$\textbf{130.6} \pm \textbf{3.21}$	0.07*		
pressure							
diastolic blood	$\textbf{79.2} \pm \textbf{1.06}$	$\textbf{81.4} \pm \textbf{1.93}$	0.43*	$\textbf{81.5} \pm \textbf{1.57}$	0.50*		
pressure							
aortic PWV	$\textbf{7.80} \pm \textbf{0.24}$	$\textbf{7.87} \pm \textbf{0.41}$	0.40*	$\textbf{8.06} \pm \textbf{0.75}$	0.57*		
[m/sec]							
A1166C mutat. present:							
n	75	23	0.17	15	0.26		
age	$\textbf{46.0} \pm \textbf{1.31}$	$\textbf{49.6} \pm \textbf{1.98}$	0.33*	$\textbf{49.4} \pm \textbf{2.68}$	0.09*		
systolic blood	$\textbf{123.5} \pm \textbf{1.82}$	$\textbf{128.7} \pm \textbf{3.19}$	0.34*	$\textbf{133.5} \pm \textbf{4.27}$	0.89*		
pressure							
diastolic blood	$\textbf{79.3} \pm \textbf{1.14}$	$\textbf{82.4} \pm \textbf{2.50}$	0.88*	$\textbf{81.3} \pm \textbf{2.85}$	< 0.004		
pressure							
aortic PWV	$\textbf{7.26} \pm \textbf{0.20}$	$\textbf{7.46} \pm \textbf{0.42}$		$\textbf{8.63} \pm \textbf{0.72}$			
[m/sec]							
age systolic blood pressure diastolic blood pressure aortic PWV [m/sec] A1166C mutat. p age systolic blood pressure diastolic blood pressure diastolic blood pressure aortic PWV	$47.7 \pm 1.29$ $122.6 \pm 1.72$ $79.2 \pm 1.06$ $7.80 \pm 0.24$ present: $75$ $46.0 \pm 1.31$ $123.5 \pm 1.82$ $79.3 \pm 1.14$	$49.0 \pm 2.17$ $128.4 \pm 3.40$ $81.4 \pm 1.93$ $7.87 \pm 0.41$ $23$ $49.6 \pm 1.98$ $128.7 \pm 3.19$ $82.4 \pm 2.50$	0.12 0.43* 0.40* 0.17 0.33* 0.34*	$51.34 \pm 1.91$ $130.6 \pm 3.21$ $81.5 \pm 1.57$ $8.06 \pm 0.75$ $15$ $49.4 \pm 2.68$ $133.5 \pm 4.27$ $81.3 \pm 2.85$	0.0 0.9 0.9 0.0 0.0		

**Conclusion:** In our sample of general population we found, that high-normal fT4 was associated with increased aPWV, however significantly expressed only in patient with A1166C mutation of AGTR1.

☆ (supported by grant IGA9333-3, Ministry of Health CZ).

## P.013 BLOOD PRESSURE, ARTERIAL RIGIDITY AND ALCOHOL INTAKE IN A POPULATION SAMPLE $^{\star}$

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**Objectives:** Increased alcohol intake impaires the control of hypertension in treated patients. The aim of this study was to evaluate the relation between weekly alcohol intake, blood pressure and arterial stiffness.

**Methods:** 257 subjects (m126, f131 mean age  $48.04 \pm 0.66$ ) were selected from population- based postMONICA study. Arterial stiffness was measured using Sphygmocor device as aortic pulse wave velocity (APWV) and radial augmentation index (RAIx), the one-week alcohol intake was ascertained by diet recall.

Results: The sample was divided according to reported weekly intake of pure alcohol into 3 categories: none or minimal intake (0-42g), mild drinkers (43-154 g) and moderate to heavy drinkers (> 154g). Among these categories, significant increasing trend in blood pressure and aortic rigidity were found (see table). These trends remained significant even after adjustment for age, current smoking, body mass index, antihypertensive treatment, LDL and glyceamia as potentially confounding factors of hypertension and/or increased arterial rigidity.

weekly alcohol intake:	0-42g	43-154 g	>154 g	p for trend
N	131	72	54	-
age [years]	$\textbf{47.9} \pm \textbf{0.99}$	$\textbf{48.3} \pm \textbf{1.30}$	$\textbf{48.1} \pm \textbf{1.45}$	0.83
gender [% of males]	21.4	65.3	94.4	< 0.0001
body mass index	$\textbf{26.1} \pm \textbf{0.36}$	$\textbf{26.9} \pm \textbf{0.45}$	$\textbf{27.2} \pm \textbf{0.56}$	0.14
[kg/m2]				
systolic BP [mmHg]	$\textbf{122.8} \pm \textbf{1.46}$	$\textbf{128.0} \pm \textbf{2.03}$	$\textbf{130.3} \pm \textbf{1.77}$	< 0.003
diastolic BP [mmHg]	$\textbf{78.2} \pm \textbf{0.88}$	$\textbf{82.0} \pm \textbf{1.43}$	$\textbf{83.1} \pm \textbf{1.28}$	< 0.03
antihypertensives [%]	18.3	23.6	13.0	0.31
APWV [m/sec]	$\textbf{7.36} \pm \textbf{0.18}$	$\textbf{7.44} \pm \textbf{0.24}$	$\textbf{8.24} \pm \textbf{0.46}$	< 0.02
RAIx [%]	$\textbf{73.8} \pm \textbf{1.89}$	$\textbf{71.2} \pm \textbf{2.51}$	$\textbf{66.9} \pm \textbf{2.99}$	0.35

**Conclusion:** Moderate to heavy drinkers showed in our general population sample gradually higher blood pressure, probably because of increased aortic stiffness.

☆ (supported by Internal Grant Agency, Ministry of Health CZ, grant No 9333-3).