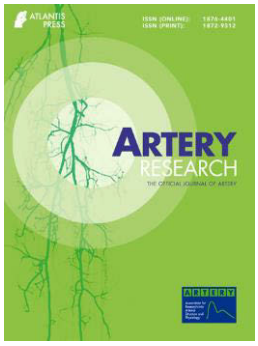


Artery Research

ISSN (Online): 1876-4401 ISSN (Print): 1872-9312
Journal Home Page: https://www.atlantis-press.com/journals/artres

P89: UPRIGHT POSTURE ENHANCES THE UNFAVOURABLE INFLUENCES OF BISOPROLOL ON CENTRAL BLOOD PRESSURE IN HYPERTENSIVE MIDDLE AGED MEN: A DOUBLE-BLINDED RANDOMIZED PLACEBO-CONTROLLED CROSS-OVER STUDY

Ilkka Pörsti, Lauri Suojanen, Antti Haring, Antti Tikkakoski, Arttu Eräranta, Heini Huhtala, Mika Kähönen, Kari Kivistö, Jukka Mustonen

To cite this article: Ilkka Pörsti, Lauri Suojanen, Antti Haring, Antti Tikkakoski, Arttu Eräranta, Heini Huhtala, Mika Kähönen, Kari Kivistö, Jukka Mustonen (2017) P89: UPRIGHT POSTURE ENHANCES THE UNFAVOURABLE INFLUENCES OF BISOPROLOL ON CENTRAL BLOOD PRESSURE IN HYPERTENSIVE MIDDLE AGED MEN: A DOUBLE-BLINDED RANDOMIZED PLACEBO-CONTROLLED CROSS-OVER STUDY, Artery Research 20:C, 86–87, DOI: https://doi.org/10.1016/j.artres.2017.10.126

To link to this article: https://doi.org/10.1016/j.artres.2017.10.126

Published online: 7 December 2019

[3]*Swinburne University, Melbourne, Australia*
[4]*University of Western Australia, Perth, Australia*
[5]*Australian Catholic University, Melbourne, Australia*

Background: Compared to regular active breaks, prolonged uninterrupted sitting amplifies postprandial glucose and insulin in overweight/obese adults with and without type 2 diabetes; and impairs lower limb endothelial function (a predictor of cardiovascular disease) in healthy adults. However, the effects of prolonged sitting on endothelial function in those at heightened risk of cardiometabolic disease have not been investigated.
Methods: Overweight/obese (BMI $>$ 25kg/m^2) adults (35–75y) completed two laboratory-based conditions in a random order: (i) 5h prolonged uninterrupted sitting (SIT); and (ii) 5h sitting interrupted with 3min of light-intensity simple resistance activities every 30min (SRA). Femoral artery endothelial function (flow mediated dilation; FMD) and shear rate was assessed at baseline, 1, 2 and 5h. Hourly plasma was collected for glucose, insulin and endothelin-1 measurement. Muscle sympathetic nervous activity (MSNA) was measured at 5h.
2**Preliminary results:** In the current sample (n = 7), SIT impaired FMD (-2.6 ± 0.9%; $p < 0.05$) and shear rate iAUC (-39 ± 14%; $p < 0.05$), compared to SRA. There was an increase in glucose (40 ± 28%; $p = 0.18$) and insulin (46 ± 25%; $p = 0.16$) iAUC, and mean endothelin-1 plasma concentration (0.28 ± 0.09 pg/ml; $p < 0.05$) in SIT, compared to SRA. MSNA (n = 4) was reduced in SIT, compared to SRA (-4 ± 1 bursts/min; $p < 0.05$). Testing and analysis (n = 20 participants) is expected to be complete by August, 2017.
Conclusions: These findings are consistent with a potential mechanistic link between sitting- induced endothelial dysfunction, vasoconstriction and insulin resistance, via reduced delivery of glucose and insulin to nutritive vascular beds in muscle. Endothelial dysfunction associated with prolonged sitting may be related to reduced shear rate, and impaired MSNA.

P176
ARTERIAL STIFFNESS AS A PART OF A GENERAL ABNORMALITY OF THE FIBROUS HEALING PROCESS

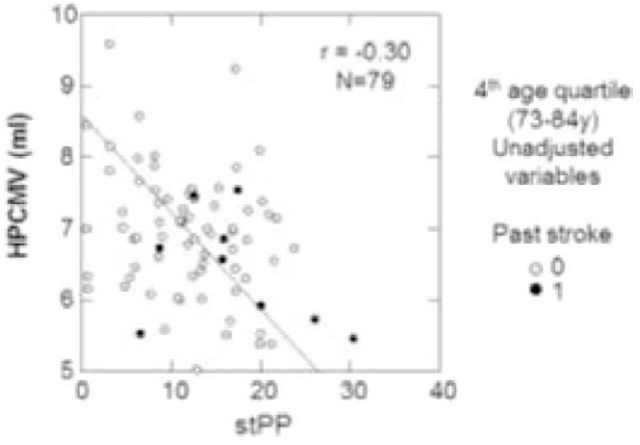
Pedro Forcada [1], Miguel Sangiovanni [2], Alejandra Camino [2], Wanda Pampinella [2], Oscar Montaña [2]
[1]*Cemic, Buenos Aires And Dim Prevencion Cardiovascular Buenos Aires, Argentina*
[2]*Dim Prevencion Cardiovascular, Buenos Aires, Argentina*

Environment
There seems to be a common abnormality of the healing process in several diseases as COPD, liver esteatosis, and arterial stiffening.
Objective: To asses the association of frequency between liver esteatosis and aortic stiffness by means of c-f PWV in patients included in a CV prevention programme.
Methods: 43 patients underwent a simultaneous evaluation including antropometry, biochemistry, cardiac, vascular and abdominal ultrasonography.
Results: Mean age was 53,4 $\pm$ 11 y.o, 67% male, SBP 137 $\pm$ 17, DBP 87 $\pm$ 10 PP 50,3 $\pm$ 11 mmHg, BMi 29 $\pm$ 4. CVRF: HTN 74%, DLP 69%, DBT 7%, TBQ 28%, OBS 42%, OVWT 28%, SED 71%.CV Drugs: 63%.
Nine (21%) presented abnormal PWV and esteatosis and 17 (39,5%) none of them. Whereas 4 (9,5%) presented abnormal PWV with normal liver and 13 (30%) the opposite. (Fisher NS, Cochran's $<$.05). More information about LV mass and atherosclerotic burden is presented.
Conclusion: In a group of p. in a Primary CV prevention programme there is a trend to a significative association between the presence or not of liver esteatosis and aortic stiffness.
A wider investigation of fibrosis and the healing process in different tissues should be considered as a future research target.

P177
ASSOCIATIONS OF AMBULATORY PULSE PRESSURE COMPONENTS WITH HIPPOCAMPAL VOLUME, WHITE MATTER HYPERINTENSITIES AND BRAIN INFARCTS

Benjamin Gavish [1], Therese Tillin [2], Alun D. Hughes [2], Nishi Chaturvedi [2]
[1]*Yazmonit Ltd., POB 606, Israel*
[2]*Institute of Cardiovascular Science, Faculty of Population Health Sciences, University College London, London, UK*

Background: Arterial stiffness is blood pressure (BP) dependent. Using 24-hour ambulatory BP monitoring (24hABPM) pulse pressure (PP) can be split into an 'elastic' part (elPP: 'diastolic stiffness'), and a 'stiffening' part (stPP: 'stiffness change during systole'). We investigated associations of elPP, stPP and PP with brain MRI measures.
Methods: A community-based sample of 542 individuals (59 ± 6y) with 24hABPM and brain MRI, including hippocampal volume (HPCMV), severity of White Matter Hyperintensities (WMH_SVR), and number of brain infarcts (N_INFRCT). 'High'/'low' (HI/LO) marked variables status (by medians).
Results: elPP and stPP were weakly correlated (r = 0.15); stPP-to-PP ratio was 0.21 ± 0.08. Adjusted HPCMV calculated at age quartiles for the HI_PP cohort correlated better with values from HI_stPP than from HI_elPP. For HI_PP and HP_stPP HPCMV reduction between age quartiles 1&4 was similar, but 20% larger than for HI_elPP. In hypertensives at highest age quartile HPCMV correlated negatively with stPP ($P < 0.05$: adjusted for age, sex and diabetes), but not with PP and elPP.
Adjusted WMH_SVR was greater in HI_elPP, HI_PP and HI_elPP comparing diabetics with non-diabetics by 0.38($P = 0.001$), 0.29($P = 0.008$) and 0.13 ($P = 0.25$), respectively.
In hypertensives N_INFRCT was greater in past-stroke than no-stroke cohorts in HI&LO elPP, stPP and PP subgroups by 1.96&0.63, 1.48&1.26, and 1.53&1.18 ($P < 0.0001$ for all).



Conclusion: The association of elastic and stiffening components calculated from ambulatory PP differ for different MRI brain measures and may provide a practical tool for associating arterial properties with brain-related pathological changes. Associations with PP may be mainly explained by its relatively-small stiffening component during systole.

Poster Session II — Interventions P89
UPRIGHT POSTURE ENHANCES THE UNFAVOURABLE INFLUENCES OF BISOPROLOL ON CENTRAL BLOOD PRESSURE IN HYPERTENSIVE MIDDLE AGED MEN: A DOUBLE-BLINDED RANDOMIZED PLACEBO-CONTROLLED CROSS-OVER STUDY

Ilkka Pörsti [1], Lauri Suojanen [1], Antti Haring [1], Antti Tikkakoski [1], Arttu Eräranta [1], Heini Huhtala [2], Mika Kähönen [1], Kari Kivistö [1], Jukka Mustonen [1]
[1]*Faculty of Medicine and Life Sciences, University of Tampere, Finland*
[2]Faculty of Social Sciences, University of Tampere, Finland

Objective: Treatment with beta-blockers is characterised by inferior reduction of central versus peripheral blood pressure. We examined changes in central and peripheral blood pressure, cardiac function, and vascular resistance during beta-blockade.
Methods: Haemodynamics were investigated after 3 weeks of bisoprolol treatment (5 mg/d) in a double-blinded, randomized, placebo-controlled cross-over trial in never-treated 16 Caucasian males with grade I-II primary hypertension using continuous tonometric pulse wave analysis and whole-body impedance cardiography.
Results: Bisoprolol decreased blood pressure in the aorta ($\sim$8/10 mmHg) and radial artery ($\sim$10/9 mmHg), reduced heart rate and left cardiac

work, and increased subendocardial viability index in supine and upright positions ($p < 0.01$ for all). Stroke volume was increased in the supine ($\sim$11 ml, $p < 0.01$) but not in the upright position, while upright ($\sim$1 l/min, $p < 0.01$) but not supine cardiac output was significantly reduced. Upright increase in systemic vascular resistance was amplified after bisoprolol ($p < 0.05$). Pulse pressure amplification was reduced especially in the upright position (supine reduction 10%, upright reduction 20%). Aortic augmentation index, augmentation pressure and pulse pressure were not changed in the supine position, but were increased in the upright position (from 7 to 20%, 3 to 7 mmHg, 28 to 35 mmHg, respectively, $p < 0.01$ for all).

Conclusions: Bisoprolol decreased central and peripheral blood pressure in male subjects with grade I to grade II hypertension, but central blood pressure was reduced less efficiently than peripheral blood pressure. Importantly, the harmful influences of bisoprolol on central pulse pressure and pressure wave reflection were especially observed in the upright position.

P90
POSITIVE EFFECTS OF ANTIHYPERTENSIVE TREATMENT ON AORTIC STIFFNESS IN THE GENERAL POPULATION

Markéta Mateřánková
Internal Department II, Faculty of Medicine in Pilsen, Charles University, Czech Republic

Aortic stiffness is strongly related to age and mean arterial pressure (MAP). We investigated whether antihypertensive treatment modulates the association of the aortic pulse wave velocity (PWV) with age and with MAP in the general population. In the Czech post-MONICA, we measured the PWV in 735 subjects (mean age 61.2 ± 7.8 years, 54.1% women, 44.3% on antihypertensive medication). We used a linear regression model to assess the effect of treatment on the PWV.

The independent covariates in our analysis included sex, age, MAP, body mass index, plasma glucose, low-density lipoprotein cholesterol, smoking and observer. The patients receiving treatment were older (64.1 ± 6.7 vs. 58.9 ± 7.8 years), had higher systolic blood pressure (135.9 ± 16.2 vs. 130.1 ± 16.5 mm Hg) and had higher pulse wave velocity (9.1 ± 2.2 vs. 8.2 ± 2.1 m s 1; P for all o0.0001) than untreated subjects.

After adjustment for MAP, the use of treatment modified the association between age and the PWV (regression equations, treated patients 9.68—0.009 age vs. untreated subjects 6.98 þ 0.020 age, difference of regression slopes, F $^{1}/_{4}$ 11.2; P $^{1}/_{4}$ 0.0009). In analyses adjusted for age, treatment was associated with a smaller increase of the PWV with MAP (treated patients 9.63—0.006 MAP vs. untreated subjects 7.18 þ 0.010 MAP, F $^{1}/_{4}$ 10.70; P $^{1}/_{4}$ 0.0001). These results were driven primarily by subjects whose blood pressure was below 140/90 mm Hg.

In the cross-sectional analysis from a random sample of the general population, antihypertensive treatment was associated with a less steep increase in the PWV with age and the mean arterial pressure.

P91
GREEN TEA EXTRACT REDUCES LIPID PROFILE, PERCENTAGE OF AORTIC AUGMENTATION INDEX AND INCREASES SOLUBLE RAGE CONCENTRATIONS IN NORMOTENSIVE PATIENTS WITH TYPE 2 DIABETES MELLITUS: A RANDOMIZED, DOUBLE-BLINDED, AND PLACEBO-CONTROLLED TRIAL

Fernando Grover Páez [1,2,3], Patricia Quezada Fernandez [4,5], Mariana Rodriguez de la Cerda [4], David Cardona Müller [2,6,7], Jhonatan Trujillo Quiroz [4,5], Walter Trujillo Rangel [5,4], Marycruz Barocio Pantoja [4,5]
[1]*University of Guadalajara, Physiology Department of the University of Guadalajara, Mexico*
[2]*Experimental Therapeutic and Clinic Institute, University of Guadalajara, Mexico*
[3]*National System of Researchers Grade 1, National Council of Science and Technology (CONACYT), Mexico*
[4]*Experimental Therapeutic and Clinic Institute, Physiology Department of the University of Guadalajara, Mexico*
[5]*PNPC, National Council of Science and Technology (CONACYT), Mexico*
[6]*National System of Researchers, National Council of science and technology (CONACYT), Mexico*
[7]*National Council of Cardiology, Mexico*

Background: Type 2 diabetes mellitus is associated with premature atherosclerosis and arterial stiffening by an accumulation of advanced glycation end-products in vessel wall (1). Green tea polyphenols are considered a cardioprotective substance and may be used as an adjuvant for diabetes treatment, because its ability to stimulates the soluble RAGE secretion (2). There is no clinical evidence of the effect of green tea extract administration on metabolic parameters, arterial stiffness and the soluble RAGE expression.

Material and Methods: A double-blind, placebo-controlled, randomized clinical trial in normotensive patients with type 2 diabetes mellitus was conducted to identify the effect of green tea extract on arterial stiffness, metabolic and anthropometric parameters and on soluble RAGE (sRAGE) with the S100A1 ligand.

Results: We included 20 subjects, there was no difference between groups at baseline. There was a decrease in the green tea extract group on aortic augmentation index (21.12 ± 8.9 to 18.07 ± 9.7, $p = 0.045$), total cholesterol (203.9 ± 37.6 to 176.9 ± 25.9 mg/dl, $p = 0.019$) triglycerides (202.6 ± 146.9 to 123.2 ± 64.8 mg/dl, $p = 0.023$) and an increase in sRAGE (1358.5 ± 390.0 to 1281.1 ± 369.7 $p = 0.052$).

Table 1. Effect of 12 weeks of Green tea extract intervention or placebo on circulating parameters.

	GTE		PLACEBO		P
	Basal n = 10	Final n = 10	Basal n = 10	Final n = 10	
Fasting Glucose, mg/dl	169.9 ± 92.3	123.9 ± 69.8	168.1 ± 49.7	171.3 ± 39.8	0.089
Creatinin mg/dl	0.75 ± 0.2	0.81 ± 0.14	0.7 ± 0.2	0.78 ± 0.14	0.853
Total Cholesterol mg/dl	203.9 ± 37.6	176.9 ± 25.9	187.9 ± 44.6	216.1 ± 48.2	0.019*
Triglycerids, mg/dl	202.6 ± 146.3	123.9 ± 64.8	159.9 ± 57.0	184.3 ± 93.9	0.023*
HDLc, mg/dl	47.9 ± 7.8	44.9 ± 5.2	48 ± 8.9	46.9 ± 10.2	0.529
LDLC. mg/dl	123 ± 32.8	109.4 ± 25.1	92.3 ± 30.2	111.2 ± 53.3	0.436
TGO, U/ml	25.6 ± 10.1	25.3 ± 7.08	40.7 ± 13.8	44.4 ± 26.8	0.971
TGP, U/ml	23.8 ± 13.6	28.9 ± 11.9	35.4 ± 14.5	44.7 ± 25.4	0.912
TFG, naL/min	119.9 ± 56.3	101.8 ± 23.9	120.6 ± 50.2	102.3 ± 22.7	0.739

Values are arithmetic means ± SE except for mean differences between groups, which have been adjusted for baseline values. Between-group P values reflect the between-group comparison change-scores from Man Whitney U statistic methodology. *Significant ($p < 0.05$) within-group change.

Conclusions: Green tea extract reduces lipid levels, percentage of aortic augmentation index and increases soluble RAGE concentrations in normotensive patients with Type 2 Diabetes.

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