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

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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 > 25 kg/m²) adults (25–75y) completed two laboratory-based conditions in a random order: (i) Sh prolonged uninterrupted sitting (SIT); and (ii) Sh 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 Sh.

Preliminary results: In the current sample (n = 7), SIT impaired FMD (−2.6 ± 0.9%; p < 0.05) and shear rate IAUIC (−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) IAUIC, 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 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_INFRACT). ‘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, diabetes, and obesity), 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_INFRACT was greater in past-stroke than no-stroke cohorts in HILLO 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.
work, and increased subendocardial viability index in supine and upright positions (p < 0.01 for all). Stroke volume was increased in the supine (≈11 ml; p < 0.01) but not in the upright position, while upright (≈17 ml; 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 ANTHYPERTENSIVE TREATMENT ON AORTIC STIFFNESS IN THE GENERAL POPULATION
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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 ± 1.1 m/s; 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 vs. untreated patients 9.63 ± 2.1 m s⁻¹; P for all <0.0001 for all).

Conclusions: Antihypertensive treatment was associated with a less steep increase in the PWV with age and the mean arterial pressure.

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

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

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.

9.68–0.009 age vs. untreated subjects 6.98 ± 0.020 age, difference of regression slopes, F 1/411.2; 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 ± 0.010 MAP, F 1/410.70; 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.

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

References