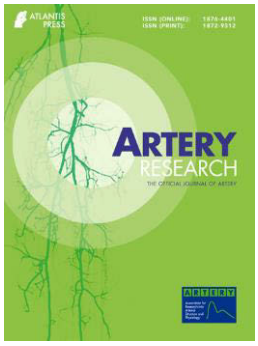

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P177: ASSOCIATIONS OF AMBULATORY PULSE PRESSURE COMPONENTS WITH HIPPOCAMPAL VOLUME, WHITE MATTER HYPERINTENSITIES AND BRAIN INFARCTS

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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 $>$ 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

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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

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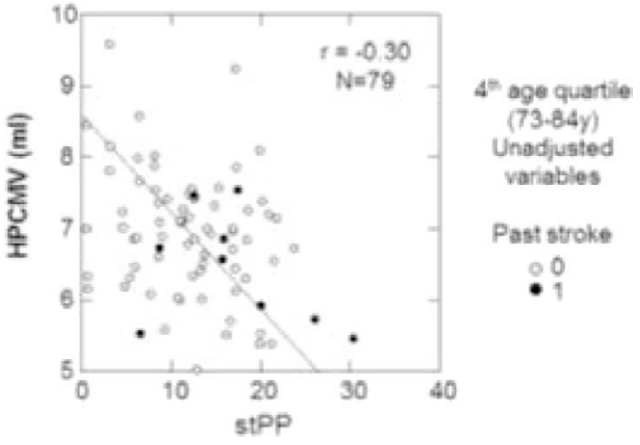
Background: Arterial stiffness is blood pressure (BP) dependent. Using 24-hour ambulatory BP monitoring (24hABPM) pulse pressure (PP) can be split into an 'elastic' part (elPP: 'diastolic stiffness'), and a 'stiffening' part (stPP: 'stiffness change during systole'). We investigated associations of elPP, 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

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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