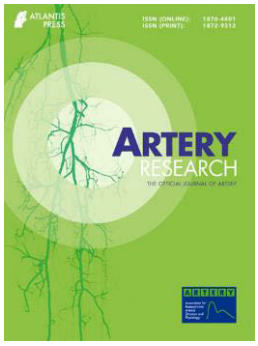

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P159: ASSOCIATION BETWEEN PULSE WAVE VELOCITY AND APNEA-HYOPPNEA INDEX IN PATIENTS WITH TYPE 2 DIABETES AND OBSTRUCTIVE SLEEP APNEA

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correlated with FMD ($r = 0.828$; $p = 0.011$; rspearman $= 0.738$; $p = 0.037$). No indices of BP variability correlated with cIMT or cDC.
Conclusions: BP variability, in particular ARV, shows a correlation with systemic but not local vascular stiffness in a sample of obese children, suggesting a relation between daily BP variability and arterial elastic properties. Further studies, especially perspective ones, are needed to clarify the pathophysiological significance of these relations.

P159
ASSOCIATION BETWEEN PULSE WAVE VELOCITY AND APNEA-HYOPPNEA INDEX IN PATIENTS WITH TYPE 2 DIABETES AND OBSTRUCTIVE SLEEP APNEA

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Introduction: Obstructive sleep apnea (OSA) is associated with increased cardiovascular (CV) risk. OSA is highly prevalent among patients with type-2 diabetes (T2D).
Patients with T2D have increased risk of cardiovascular events, and have an increased aortic stiffness.
Continuous Positive Airway Pressure (CPAP) treatment reduces severity of OSA, but whether it reduces CV risk remains unclear. One randomized trial with CPAP intervention and pulse wave velocity (PWV) as endpoint has shown a significant reduction in PWV after four months, in non-diabetic patients. The effect on patients with diabetes remains unknown.
Aim: Investigate the effects of CPAP treatment on PWV in patients with T2D and newly diagnosed OSA. Furthermore, investigate the relationship between PWV and severity of OSA.
Method: A randomized, controlled, multicenter study. 70 patients with T2D and newly diagnosed OSA randomized to: CPAP treatment or a control group. Data will be collected at baseline, 4 and 12 weeks. PWV was measured using SphygmoCor (AtCor Medical, Sydney, Australia) and AHI measured using ApneaLink (ResMed, Poway, CA, USA). Relationship between PWV and AHI was evaluated at baseline.
Results: Baseline data from the first 21 patients showed mean age 63 years ($\pm$8.1), mean systolic blood pressure (BP) was 134 ($\pm$12.5) mmHg, mean AHI was 30.2 ($\pm$12.4) and mean PWV was 11.6 ($\pm$1.9) m/s.
AHI was associated with PWV in multivariate analysis with adjustment for age and systolic BP, beta-coefficient 0.08, $p = 0.029$.
Conclusion: At baseline PWV and AHI were correlated. Progression of the study will reveal if CPAP treatment can lower PWV in this cohort.

P160
VASCULAR ABNORMALITIES AND HAEMODYNAMIC PATTERN IN OBESE YOUNG ADULTS

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Background: Obesity is linked to a higher prevalence of risk factors, metabolic and inflammatory pathways conducting to increased vascular disease and CV risk.
Objective: To assess vascular disarrangements and haemodynamic patterns in obese young subjects (O) compared with matched lean (L) controls, using non invasive methods.
Methods: From the database of our Non Invasive Vascular Lab with 3964 first evaluated patients, we performed a case control study with 363 sujects, 268 obese and 95 lean, age and sex matched controls. We measured IMT, Plaque analysis, PWV, Endothelial Function (EF) and arterial stiffness (CAP and Aix) (AS) using an oscillometric device (Arteriograph, Tensiomed. Hungary®) and non invasive haemodynamic evaluation using impedance cardiography (Z Logic Exxer®).
Results: Age (O 42.5 + 5; L 43.5 + 11) and sex % (O 80.6%; L 78%) were matched. BMI (O 33.5 + 3.3 L 25 + 1.1Kg/m^2), waist (O110.4 + 7.5; L 91.2 + 6.1cm) and BP (SBP O 139.8 + 16.8; L119 + 8.8 and DBP O 89 + 3.9; L 74.3 + 8 mmHg) were higher in O ($p < 0.001$).CV Risk Factors in O: HTN 68% DLP 59.7% SMKG 24.2% DBT2 7.8% SED 72.4%.The % of abnormalities in IMT (O/L : 65.8/25.3%), Plaques (75.6/38.9%), EF (57.5/33.7%) and PWV (41.4/17.9%) were higher in O ($p < 0.001$). Central and Peripheral PP were higher in O but not Aix. CI was significantly lower and PVRI and Thoracic Fluid content higher in O.
Conclusion: Young obese patients present a higher prevalence of vascular disarrangements either structural and functional and a haemodynamic pattern of high peripheral resistance with volume expansion that may explain the role of this condition as a CV risk factor.

P161
ROLES OF ANGIOPOIETINS 1 AND 2 ON ARTERIAL FUNCTION DURING A TREATMENT TRIAL IN PEOPLE WITH OR AT RISK OF DIABETES

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Background/ Objective: Vascular growth factors angiopoietin-1 (Ang1) and -2 (Ang2) regulate vascular permeability and inflammation, Ang2 likely as Ang1's selective antagonist. Their role before or in type 2 diabetes (pre-&T2D) is unknown. We hypothesised that higher circulating Ang1 and lower Ang2 (=lower Ang2/1 ratios) would be linked to increased arterial stiffness *and its change* over the trial, independent of blood pressure (BP).
Methods: ELISA assays were performed from 60 participants with all time-points of plasma samples from 'VaSera', a trial of single-centre, double-blind, parallel, randomised, controlled, 2x2 factorial design. Interventions were spironolactone and beetroot juice, a NO^{3-} donor, with doxazosin and placebo juice respectively to control for BP change (Δ) over the trial. Vascular measurements were aortic pulse wave velocity (aPWV), cardiac-ankle pulse wave velocity (CAVI), analysed by multiple regression adjusted for baseline BP and ΔBP over 6 months.
Results: Baseline Ang1 was positively while higher baseline Ang2 was negatively associated with baseline aoPWV at ($ß = 0.37$, $p = 0.01$; $ß = -0.27$, $p = 0.047$, respectively), independent of BP, BMI and DM status, so baseline $r = -0.45$ for the Ang2:1 ratio with aPWV, and $r = 0.39$ for ΔaPWV over the trial. Higher baseline Ang1 independently predicted decreased aoPWV over 6 months ($ß = -0.44$m/sec per ng/ml, $p = 0.006$). Angiopoietin concentrations were not associated with CAVI or BP.
Conclusions: Angiopoietins were related to baseline aPWV, independently of BP, and to ΔaPWV over the trial, also independent of BP change, but were unrelated to CAVI or BP. Monitoring and manipulating Angiopoietins may help arterial health in pre-&T2DM.

Poster Session II — Special Populations
P182
ARTERIES IN PATIENTS WITH HUNTINGTON'S DISEASE

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Background: Huntington's disease (HD) is a neurodegenerative disorder leading to the progressive death of neurons in various brain regions. Although it is a disease of the central nervous system (CNS), mortality surveys indicate that heart disease is one of the mayor causes of death in HD patients. The mechanisms of cardiac pathophysiology of the disease remain unknown. It might be a consequence of altered activity of autonomic nervous system as part of the CNS.