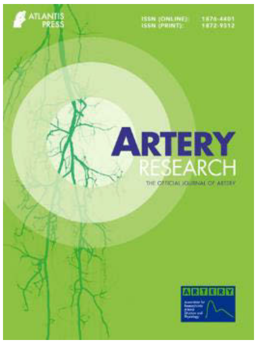

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P186: IMPACT OF OBESITY ON VASCULAR STRUCTURE AND FUNCTION IN INDIVIDUALS WITH MULTIPLE SCLEROSIS

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(CAVI) and heart-ankle pulse wave velocity (haPWV) using Vasera 1500N. Circulating levels of leptin, adiponectin, insulin and C-reactive protein (CRP) were measured by ELISA.
Results: Compared to non-SLE controls, SLE patients had higher levels of CAVI (7.3 ± 1.1 vs 6.1 ± 1, $p < 0.001$), haPWV (7.7 ± 1.3 vs 6.5 ± 0.8 m/s, $p = <0.001$), insulin [76.8 (45.9—184.8) vs 39.8 (22.9—86.3) pmol/ml, $p = 0.007$], leptin [856.1 (364.8—1509.3) vs 426.7 (426.8 (84.7—1178.7) ng/ml, $p = 0.039$], adiponectin [1.1 (0.8—2.3) vs 1.6 (1.3—2.6) ng/ml, $p = 0.039$] and CRP [1.6 (0.8—2.2) vs 0.9 (0.6—1.2) mg/ml, $p = 0.021$]. In a partial correlation analysis with adjustment for age and BMI, CAVI was associated with leptin ($r = 0.21$, $p = 0.031$), CRP ($r = 2.9$, $p < 0.001$) and insulin ($r = 0.18$, $p = 0.04$), but not adiponectin ($r = -0.15$, $p = 0.068$).
Conclusion: In our study population, SLE patients have higher arterial stiffness, associated with low-grade inflammation and deranged circulating adipokine levels.

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P186
IMPACT OF OBESITY ON VASCULAR STRUCTURE AND FUNCTION IN INDIVIDUALS WITH MULTIPLE SCLEROSIS

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Background: Cardiovascular disease is a leading cause of disease progression and death in multiple sclerosis (MS). Obesity has a negative impact on vascular structure and function, but whether this contributes to worse vascular function similarly in individuals with MS and controls is unknown.
Aim: To investigate the impact of obesity on vascular function and structure in a group with MS.
Methods: In a sample of $n = 133$ participants (MS: $n = 89$, control $n = 44$), height and weight were measured to calculate BMI. After a 10 minute rest in the supine position, resting heart rate (HR) and brachial blood pressure (BP) were collected. Augmentation index (AIX), HR normalized AIX (AIX@HR75) and pulse wave velocity (PWV) and subendocardial viability ratio (SEVR) were measured with applanation tonometry.
Carotid intima-media thickness (IMT) and beta-stiffness (beta) were measured with carotid ultrasound, and Forearm Blood Flow (FBF Baseline, Peak and Area Under the Curve (AUC)) was measured with strain gauge plethysmography. Data were analyzed with multiple linear regression analyses with group, sex, BMI and GroupxBMI as independent variables.
Results: Higher BMI correlated with higher HR and PWV in both groups. In the MS group however, a higher BMI was also correlated with worse outcomes on the SEVR, FBF Baseline, Peak and AUC.

Outcome variables	Standardized beta[1]				Adjusted R^2
	Group	Sex	BMI	Grp x BMI	
HR rest	1.081*	0.005	0.520*	−0.944	0.11
AIX	−0.019	−0.523*	0.040	0.045	0.26
AIX@HR75	0.341	−0.536*	0.235	−0.270	0.32
SEVR	−1.292*	0.175*	−0.565*	1.090*	0.22
PWVc	0.001	−0.003	0.321*	0.120	0.12
PWVc/MAP	0.324	−0.126	0.278	−0.122	0.11
IMT	0.715	0.164	0.385	−0.511	0.12
FBF Baseline	0.432	0.070	0.326*	−1.090*	0.33
FBF Peak	0.580	0.318*	0.152	−1.035*	0.35
FBF AUC	0.746	0.230*	0.316	−1.174*	0.21

[1]Group (0 = control, 1 = multiple sclerosis), Sex (1 = Female, 2 = Male).
* ($p < 0.05$).

Conclusions: Having a higher BMI contributes even more to a worse vascular profile in MS patients than in controls, suggesting that reducing overweight and obesity in the MS population will benefit their vascular structure and function.

P187
IN SEVERE AORTIC STENOSIS, DECREASED SYSTEMIC VASCULAR RESISTANCE IS ASSOCIATED WITH A LARGER, THICKER WALLED VENTRICLE EXCEPT FOR THE SEPTUM

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Background: The ventricle in aortic stenosis (AS) is influenced by both valvular and vascular factors. The importance of afterload on left ventricular (LV) remodeling is not completely understood. Traditional imaging techniques which rely on geometric assumptions may not assess regional remodeling accurately.
Aim: To understand the influence of systemic vascular resistance (SVR), systemic arterial compliance (SAC), valvulo-arterial impedance (Zva) on LV remodeling using a cardiac atlas technique.
Methods: 109 patients with symptomatic severe AS awaiting surgical valve replacement (age 69 ± 10y, 60% male, aortic valve area 0.7 ± 0.3cm^2, mean gradient 48 ± 15 mmHg) underwent comprehensive clinical, echocardiographic and cardiovascular magnetic resonance (CMR) examinations. SVR, SAC and Zva were calculated as previously published (1). CMR LV short axis steady-state free precession cine images were segmented and co-registered using a cardiac atlas technique (2). Data were extracted and analysed using mass-univariate 3D regression modeling adjusted for age, sex, and height and accounting for multiple testing, presented as standardized β.
Results: Lower SVR correlated with increased wall thickness and larger cavity volume. SVR related changes were more prominent in the lateral wall (β −0.3 to −0.6, $p = 0.04$), with no discernable influence on the septum (Figure 1). With lower SVR, LV cavity enlargement was directed away from the septum (β −0.17 to −0.56, $p = 0.002$). There was no influence of SAC or Zva on 3D parameters.

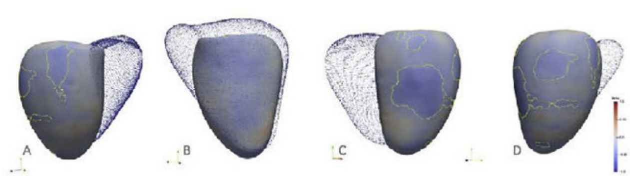

Figure 1 Mass-univariate 3D regression maps relating wall thickness and systemic vascular resistance over the left ventricle, adjusted forage, sex and height. Yellow line encloses areas with p values < 0.05. There is a negative correlation between SVR and wall thickness, sparing the septum (B). SVR = (80 * Mean Arterial Pressure (MAP))/Cardiac Output.

Conclusion: In severe AS there is an association between lower SVR and a larger, thicker walled ventricle except for the septum. The use of a cardiac atlas in aortic stenosis may offer new insights into regional LV remodeling.

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