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PO-12: RELATIONS BETWEEN AORTIC STIFFNESS AND LEFT VENTRICULAR MECHANICAL FUNCTION

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	DS (N = 13)	Control (N = 14)
carSBP (mmHg)	133 ± 22	126 ± 14
carDBP (mmHg)	74 ± 8	75 ± 9
HR (bpm)	62.5 ± 11.9	64.3 ± 13.1
Circumferential Strain ‡	9.94 ± 3.37	7.48 ± 2.56
R-CAVI ‡	4.86 ± 0.83	5.84 ± 0.68
L-CAVI ‡	4.97 ± 0.95	5.81 ± 0.68
β-Stiffness	5.24 ± 1.40	5.65 ± 1.99
Ep	72.38 ± 20.84	76.29 ± 20.48

‡ Significant group difference. Mean ± SD, Significance level, p < 0.05

PO-11

MULTIPLE SCLEROSIS PATIENTS EXPERIENCE MORE DECREMENTS IN CAROTID ARTERY FUNCTIONAL PROPERTIES WITH AGING THAN AGE-MATCHED PEERS

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Introduction: Peak prevalence of multiple sclerosis (MS) is approaching 60 years of age, suggesting an aging patient population compared to past reports. Aging is independently associated with increased cardiovascular disease risk. Additionally, arterial function is compromised with aging. Carotid artery stiffness serves as a non-invasive method to quantify aspects of arterial function. As MS patients increase their average lifespan, it is unclear if they may experience differential changes in aspects of carotid artery function compared to their healthy age-matched peers.

Objective: To compare carotid artery structure and function between young and older subjects with and without MS.

Methods: After 10 minutes of supine rest, 120 subjects (MS=89, Control=31) underwent applanation tonometry and ultrasonography of the carotid artery. Subjects were classified as young or older (<50 and ≥50 years, respectively).

Results: See table below. In those with MS, carotid artery pulse pressure (PP), carotid intima media thickness (IMT), beta stiffness, and elastic modulus were higher, and arterial compliance was lower, in the older group compared to young subjects, whereas no differences were detected between young and older subjects in the control group.

Conclusion: These data show that older subjects with MS exhibit more structural and functional alterations in carotid artery indices than older controls compared to their young counterparts. This highlights the importance of increased efforts to explore early interventions to preserve arterial function in those with MS.

	Control (n=31)		MS (n=89)	
	Young (n=15)	Older (n=16)	Young (n=44)	Older (n=45)
Carotid SBP (mmHg)	113.5 ± 3.7	116 ± 3.0	103.2 ± 1.6	112.0 ± 3.6
Carotid DBP (mmHg)	74.8 ± 2.2	76.8 ± 2.2	70.3 ± 1.2	74.1 ± 1.7
Carotid MAP (mmHg)	90.1 ± 2.5	91.9 ± 2.4	84.0 ± 1.3	90.3 ± 1.9
Carotid PP (mmHg)	38.7 ± 2.2	39.2 ± 2.4	32.9 ± 0.9	40.1 ± 1.4*
Carotid IMT (mm)	0.41 ± 0.02	0.48 ± 0.02	0.48 ± 0.01	0.61 ± 0.02*
Beta Stiffness (AU)	6.30 ± 0.46	7.38 ± 0.56	6.02 ± 0.30	8.68 ± 0.40*
Elastic Modulus (kPa)	77.31 ± 6.68	93.67 ± 7.32	68.76 ± 3.57	109.27 ± 5.56*
Arterial Compliance (mm ² /kPa)	1.11 ± 0.10	0.87 ± 0.05	1.17 ± 0.67	0.81 ± 0.05*

Mean ± SEM. *Significant difference between Young and Older groups.

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PO-12

RELATIONS BETWEEN AORTIC STIFFNESS AND LEFT VENTRICULAR MECHANICAL FUNCTION

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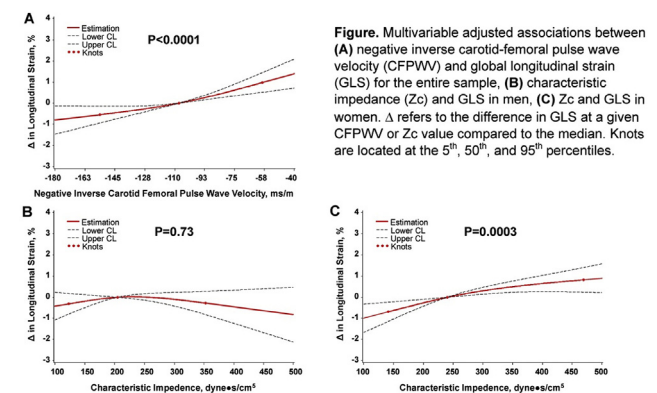
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Objectives: Left ventricular contraction produces longitudinal strain in the proximal aorta. As a result, aortic stiffening may impair optimal mechanical ventricular-vascular coupling and left ventricular (LV) systolic function, particularly in the long axis. LV global longitudinal strain (GLS) has recently emerged as a sensitive measure of early cardiac dysfunction. In this study, we investigated the relation between aortic stiffness and GLS in a large community-based sample.

Methods: In 2516 participants (age 39-90 years, 57% women) of the Framingham Offspring and Omni cohorts, free of cardiovascular disease, we performed tonometry to measure aortic stiffness and echocardiography to assess cardiac function. Aortic stiffness was evaluated as carotid-femoral pulse wave velocity (CFPWV) and as characteristic impedance (Zc), and GLS was calculated using speckle tracking-based measurements.

Results: In multivariable analyses adjusting for age, sex, height, systolic blood pressure, augmentation index, LV structure, and additional cardiovascular disease risk factors, increased CFPWV ($\beta \pm SE$: 0.122 ± 0.030 SD strain per SD CFPWV, P < 0.0001) and Zc (0.091 ± 0.029 SD/SD, P = 0.002) were both associated with worse (less negative) GLS. We observed effect modification by sex of the relation between Zc and GLS (P = 0.004); in sex-stratified multivariable analyses, the relation between greater Zc and worse GLS persisted in women (0.145 ± 0.040, P = 0.0003) but not in men (P = 0.73).

Conclusion: Higher aortic stiffness was associated with worse GLS after adjusting for hemodynamic variables. Parallel reductions in LV long axis shortening and proximal aortic longitudinal strain in individuals with a stiffened proximal aorta may represent a manifestation of abnormal direct mechanical ventricular-vascular coupling.



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