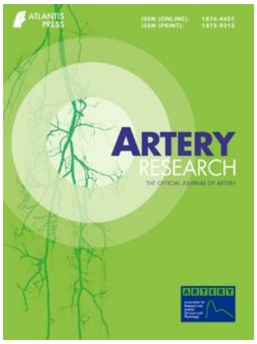

Artery Research

ISSN (Online): 1876-4401 ISSN (Print): 1872-9312
Journal Home Page: https://www.atlantis-press.com/journals/artres

OR-02: EFFECT OF ACUTE ISOKINETIC RESISTANCE EXERCISE ON SYSTEMIC ARTERIAL HEMODYNAMICS AND CEREBRAL BLOOD FLOW DYNAMICS: IS THERE A MISMATCH?

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To cite this article: A. Rosenberg, S.O. Wee, E. Schroeder, K. Bunsawat, G. Grigoriadis, B. Fernhall, T. Baynard (2016) OR-02: EFFECT OF ACUTE ISOKINETIC RESISTANCE EXERCISE ON SYSTEMIC ARTERIAL HEMODYNAMICS AND CEREBRAL BLOOD FLOW DYNAMICS: IS THERE A MISMATCH?, Artery Research 16:C, 101–102, DOI: https://doi.org/10.1016/j.artres.2016.08.005

To link to this article: https://doi.org/10.1016/j.artres.2016.08.005

Published online: 7 December 2019

Artery Research (2016) 16, 101–103

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/artres

North American Artery 2016 Annual Meeting — Oral

OR-02

EFFECT OF ACUTE ISOKINETIC RESISTANCE EXERCISE ON SYSTEMIC ARTERIAL HEMODYNAMICS AND CEREBRAL BLOOD FLOW DYNAMICS: IS THERE A MISMATCH?

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Resistance exercise (RE) is currently recommended for most adults and is important for reducing risk factors for cardiovascular and metabolic diseases, and improving quality of life. Despite functional and musculoskeletal benefits, high-intensity RE has been shown to acutely increase arterial stiffness and blood pressure, with reduced cerebral blood flow velocity and greater flow pulsatility in the cerebral circulation, which may be detrimental to cerebral microvasculature.

Objective: The purpose of this study was to investigate the effects of an acute bout of RE on hemodynamics and cerebral vascular responses in recreationally active, young adults.

Methods: Fifteen healthy adults aged 18-35 years (~26 years, male=7) performed RE, which consisted of 3 sets of 10 repetitions of isokinetic concentric/concentric unilateral knee flexion/extension. All measurements were obtained at baseline and post-exercise (1,5,30-minutes). Beat-to-beat heart rate (HR), brachial blood pressure (bSBP, bDBP, bMAP), cardiac output (CO), stroke volume (SV), total vascular resistance (TVR) and end-tidal CO2 were collected. Cerebral vascular blood flow velocity (CBFv) was measured by Transcranial Doppler technology. Central blood pressures (cSBP, cDBP, cMAP), and central pulse wave velocity (PWV) were obtained using an automated ambulatory blood pressure monitor. Carotid artery beta-stiffness index was measured by ultrasonography.

Results: Mean CBFv increased at 1-minute post ($p<0.01$), but decreased below baseline values post 5-minute ($p<0.001$). In contrast, CBFv pulsatility increased following RE and remained significantly elevated at 5-minute post ($p<0.001$). TVR decreased post-RE ($p<0.001$), and returned back to baseline at post 30-minute (See Table). PWV increased 1-minute post RE ($p<0.001$), returning to baseline values at 5-minutes. There were no increases in beta-stiffness index.

Conclusion: RE increased aortic stiffness, mean CBFv and CBFv pulsatility. Despite an increase in CO at 5-minute, mean CBFv drops below baseline values and CBFv pulsatility continued to rise further above baseline. This temporary disruption in cerebral autoregulation may impact brain health.

Variables	Baseline	1-minute	5-minute	30-minute
Heart Rate (bpm) *	68 ± 9 ^{abc}	89 ± 11 ^{bc}	78 ± 11	76 ± 9
CO (L/min) *	5.2 ± 1.0 ^{ab}	8.1 ± 1.5 ^{bc}	6.3 ± 1.2 **c**	5.5 ± 1.1
SV (ml/min) *	76.0 ± 17.5 ^{a}	90.0 ± 20.0 ^{bc}	80.1 ± 16.8 **c**	72.2 ± 17.0
bSBP (mmHg) *	122 ± 10 ^{a}	139 ± 12 ^{bc}	124 ± 11	123 ± 8
bDBP (mmHg) *	73 ± 7 ^{a}	77 ± 8 ^{b}	72 ± 6	75 ± 5
bMAP (mmHg) *	93 ± 8 ^{a}	102 ± 9 ^{bc}	93 ± 7	95 ± 5
TVR (mmHg*min / L)	18 ± 3 ^{ab}	13± 2 ^{bc}	15 ± 2 **c**	18 ± 3
cSBP (mmHg) *	108 ± 11 ^{a}	120 ± 11 ^{bc}	113 ± 8	109 ± 9
cDBP (mmHg) *	79 ± 9 ^{a}	87 ± 8 ^{bc}	81 ± 6	81 ± 8
cMAP (mmHg) *	97 ± 9 ^{a}	107 ± 8 ^{bc}	100 ± 7	98 ± 7
PWV (m/s) *	5.2 ± 0.5 ^{a}	5.6 ± 0.5 ^{bc}	5.3 ± 0.5	5.2 ± 0.4
Carotid Max Diameter (mm) *	6.90 ± 0.54 ^{abc}	6.61± 0.54	6.62± 0.54	6.70 ± 0.55
Carotid Min Diameter (mm) *	6.39 ± 0.50 ^{ab}	6.04 ± 0.54	6.10 ± 0.50	6.23± 0.53
Beta-Stiffness Index	6.3 ±1.6	5.9 ± 1.5	6.1 ± 1.2	6.4 ± 1.3
CBFv Mean (cm/s) *	59 ± 15 ^{ab}	70 ± 23 ^{bc}	55 ± 13 **c**	58 ± 13
CBFv Pulsatility Index *	0.86 ± 0.09 ^{b}	0.97 ± 0.19 ^{c}	1.02 ± 0.12 **c**	0.84 ± 0.11
End-Tidal CO2 *	1.95 ± 0.44 ^{ac}	2.23 ± 0.55 ^{bc}	1.82 ± 0.47	1.75 ± 0.53

All Data are mean ± SD, * Exercise effect, $p<0.05$. **a** significantly different from 1 min, **b** significantly different from 5 min. **c** significantly different from 30 min, $p<0.05$.

OR-04
DECREASED AORTIC INERTANCE INCREASES SUSCEPTIBILITY OF LATE-SYSTOLIC LEFT VENTRICULAR EJECTION TO ARTERIAL WAVE REFLECTIONS

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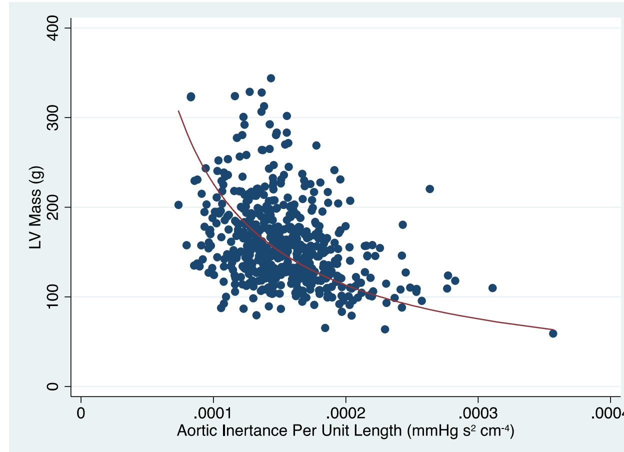
Background: Left ventricular (LV) afterload patterns consisting of late-systolic loading has been linked to LV remodeling and fibrosis in a number of studies. The contributions from arterial wave reflections (WR) has therefore garnered much interest. Aortic dilation may facilitate the adverse effects of WRs through its effect on aortic inertance. Decreased aortic inertance from aortic dilation is particularly important in late-systole, when the LV-aortic pressure gradient generally reverses and ejection decelerates until time of aortic valve closure.
Hypothesis: Decreased aortic inertance from aortic dilation is associated with LV hypertrophy.
Methods: We measured carotid-femoral pulse wave velocity (PWV; a measure of arterial stiffness) and LV mass (LVM) with SSFP-MRI in 409 subjects (mean age = 61 years). Aortic geometry was measured using SSFP-MRI, with a novel 3D aortic analyzer (Medical Imaging Applications, Coralville, Iowa). We computed compliance and inertance from PWV and geometric measurements. Reflection magnitude (RM) was calculated from pressure-flow analysis of calibrated carotid tonometry and aortic flow (PC-MRI).
Results: A non-linear relationship between inertance and LVM was found, with a more pronounced slope at lower inertance values (Figure). After log-transformation of LVM and adjusting for age, height, weight, sex, and area compliance of the thoracic aorta, decreased aortic inertance was independently associated with increased LVM (standardized β=-0.382; P<0.001). Aortic inertance was the strongest predictor of LVM in this model, whereas area compliance was not predictive. There was significant interaction between inertance and RM (P=0.029) such that the negative relationship between inertance and LVM was stronger for greater RM.
Conclusions: Reduced inertance from aortic dilation is independently associated with LV hypertrophy. This is consistent with the principle that reduced inertance diminishes the buffer between pressure gradient transients and aortic flow. In late-systole, augmentation of the negative LV-aortic pressure gradient by WRs imposes a greater deceleration force on LV ejection.



OR-05
SEX DIFFERENCES IN VASCULAR STRUCTURE AND FUNCTION IN INDIVIDUALS WITH MULTIPLE SCLEROSIS AND HEALTHY CONTROLS

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Objectives: Cardiovascular disease is a leading cause of death in multiple sclerosis (MS), and recent data showed that subclinical markers of atherosclerosis are higher in MS as well. Prevalence of MS in men is much lower than in women, but their prognosis is much worse. Men with MS also have higher rates of hypertension and diabetes than women with MS. Whether vascular function and structure differs in men than in women with MS, and whether potential sex differences are similar to those in healthy controls, is unknown.
Aim: To compare vascular function and structure between men and women in a group with MS and in healthy controls.
Methods: After a 10 minute rest in the supine position, resting heart rate (HR) and brachial blood pressure (BP) were collected. Augmentation index

	Control		MS		p-values factors #		
	Female (n=21)	Male (n=18)	Female (n=52)	Male (n=18)	Group	Sex	Inter-action
Age	49 ± 10	41 ± 9	48 ± 12	48 ± 13	0.228	0.126	0.109
Height (cm)	164 ± 6	177 ± 5	163 ± 7	179 ± 6	0.867	**<0.001****	0.264
Weight (kg)	69 ± 10	89 ± 13	73 ± 14	88 ± 17	0.679	**<0.001****	0.431
BMI	26 ± 4	28 ± 5	28 ± 6	28 ± 6	0.579	0.218	0.243
HR rest	59 ± 9	60 ± 12	65 ± 8	66 ± 12	**0.004****	0.582	0.739
SBP rest	120 ± 12	128 ± 8	119 ± 16	125 ± 12	0.440	**0.015***	0.672
DBP rest	76 ± 9	76 ± 11	72 ± 10	77 ± 8	0.429	0.269	0.341
MAP rest	91 ± 10	94 ± 10	88 ± 11	93 ± 9	0.410	0.081	0.688
AIX	31 ± 10	10 ± 15	27 ± 12	17 ± 12	0.510	**<0.001****	**0.038***
AIX@HR75	23 ± 8	3 ± 16	22 ± 11	13 ± 9	0.074	**<0.001****	**0.018***
PWVc	6 ± 1	7 ± 1	7 ± 2	7 ± 2	0.058	0.695	0.675
PWVc/MAP	0.07 ± 0.01	0.07 ± 0.01	0.08 ± 0.02	0.08 ± 0.02	**0.013***	0.525	0.445
IMT	0.45 ± 0.08	0.51 ± 0.11	0.53 ± 0.12	0.6 ± 0.13	**0.001****	**0.010***	0.985
Beta	7.04 ± 2.21	6.64 ± 2.04	7.25 ± 2.03	8.07 ± 3.57	0.104	0.675	0.227
FBF Baseline	3.1 ± 1.3	3.7 ± 1	1.9 ± 0.9	2 ± 0.9	**<0.001****	0.099	0.203
FBF Peak	20.6 ± 7.1	27.2 ± 7	15.6 ± 5.8	20.5 ± 6.6	**<0.001****	**<0.001****	0.533
FBF AUC	70 ± 23.3	94 ± 27.7	58 ± 22.2	68 ± 26.6	**<0.001****	**0.001****	0.160

two-way independent ANOVA with Group, Sex and Group*Sex as factors.
* p<0.05.
**p<0.01.