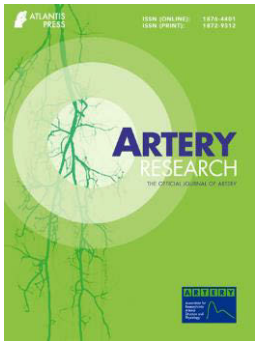

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5.4: EFFECT OF ACUTE RESISTANCE EXERCISE ON ARTERIAL HEMODYNAMICS AND CEREBRAL BLOOD FLOW DYNAMICS: DOES SEX MATTER?

Alexander Rosenberg, Tommy Wee, Elizabeth Schroeder, Kanokwan Bunsawat, Georgios Grigoriadis, Garett Griffith, Bo Fernhall, Tracy Baynard

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5.2
REDUCED SUBLINGUAL ENDOTHELIAL GLYCOCALYX IN TYPE 1 DIABETICS WITH DIABETIC NEPHROPATHY

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Background: Glycocalyx is a glycoprotein layer protecting the capillary endothelium. An impaired glycocalyx may precede the development of microvascular complications in diabetes. Capillaroscopy is a new method to estimate the dimensions of the glycocalyx by measuring the perfused boundary region (PBR). We evaluated the glycocalyx thickness in type 1 diabetic patients with different levels of historical and current albuminuria.
Methods: Cross-sectional study including 77 type 1 diabetics stratified by history of normoalbuminuria (<30 mg/g; n = 26), microalbuminuria (30–299 mg/g; n = 27) and macroalbuminuria (>300 mg/g; n = 24).
Glycocalyx thickness was assessed by 5 measurements with the GlycoCheck® device, a non-invasive hand-held microscope generating video recordings of the sublingual capillaries. Endothelial glycocalyx thickness was estimated from the PBR in capillaries with a diameter range of 5–25 μm. Higher PBR indicates smaller glycocalyx width. Urinary albumin-to-creatinine ratio (UACR) was measured in 3 morning samples.
Results: In normo-, micro-, and macroalbuminurics PBR was (mean ± SD) 2.30 ± 0.22 μm, 2.32 ± 0.25 μm, and 2.49 ± 0.35 μm, respectively. Differences between normo- and macroalbuminurics and micro- and macroalbuminurics were significant ($p < 0.05$) in an unadjusted model and remained significant after adjustment for age, sex, HbA_{1c}, diabetes duration and systolic blood pressure. In pooled (n = 77) multivariate linear regression, higher level of current UACR was associated with a higher PBR ($p = 0.0007$).
Conclusion: In type 1 diabetics with a history of macroalbuminuria, measurements with the non-invasive GlycoCheck® device revealed significantly higher PBR, suggesting an impaired glycocalyx, compared to patients with normo- or microalbuminuria.
Moreover, higher current level of albuminuria was associated with higher PBR.

5.3
HIGH FIT OLDER ADULTS MAINTAIN A SIMILAR ENDOTHELIAL RESPONSE TO ACUTE INFLAMMATION AS YOUNGER ADULTS

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Inflammation is associated with an increased risk of cardiovascular events and reduced endothelial function. Higher cardiorespiratory fitness is associated with lower risk of cardiovascular events and improved vascular function. Whether fitness plays a role during acute inflammation is unknown.
Purpose: Evaluate the role of fitness in the endothelial response to acute inflammation in younger (YA) and older (OA) adults.
Methods: Acute inflammation was induced using influenza vaccine in 23 YA (12 male, 26 ± 4 yrs, 24.0 ± 3.5 kg/m^2) and 60 OA (20 male, 65 ± 5 yrs, 27.8 ± 5.0 kg/m^2). Blood pressure, flow-mediated dilation (FMD) and inflammatory markers were measured before and 24-hours post-vaccination. VO_2peak was measured via a treadmill test. A VO_2peak greater than age- and sex-associated 50th percentile according to the American College of Sports Medicine was defined as Fit.
Results: Fit OA reduced FMD more than low fit OA ($p = 0.02$) 24 h post-vaccination. High and low fit YA similarly decreased FMD at 24 h ($p = 0.66$). YA and high fit OA had a similar reduction (relative %) in FMD (pz0.05). Regression analyses indicated no association between VO_2peak and change in FMD ($\beta = -0.01$, $p = 0.98$) in YA, but a significant association existed in OA ($\beta = -0.36$, $p = 0.04$) after adjusting for age, sex, BMI and baseline FMD.

	YA-high fit	YA-low fit	OA-high fit	OA-low fit
n	14	9	16	43
VO_2Max, ml/kg/min	48.4 (5.6)*	35.6 (4.9)	32.3 (4.2)*	21.9 (4.3)
Baseline SBP	113 (13)	115 (13)	125 (14)	125 (14)
Baseline PP	47 (8)	47 (11)	54 (10)	53 (11)
Baseline FMD, %	12.4 (2.7)	11.6 (5.5)	7.5 (3.9)*	5.4 (2.5)
Baseline CRP, mg/1	1.1 (0.7)	0.6 (0.6)	2.4 (3.3)	2.5 (2.2)
Baseline IL-6, pg/ml	1.0 (0.7)	0.8 (0.5)	1.7 (1.4)	1.6 (1.1)
Change SBP, mmHg	−3 (6)	2 (8)	−2 (9)	−2 (11)
Change PP, mmHg	2 (6)	5 (7)	0 (7)	0 (8)
Change FMD, %	−3.2 (4.1)	−2.3 (5.5)	−3.7 (3.8)*	−0.2 (3.5)
Change CRP, mg/l	1.0 (1.5)	0.9 (0.8)	1.1 (2.2)	0.9 (1.4)
Change IL, pg/ml	1.0 (1.5)	1.6 (2.3)	0.8 (2.6)	0.4 (1.0)

FMD: flow mediated dilation, CRP: C-reactive protein, IL-6: interleukin-6, SBP: systolic blood pressure, PP: pulse pressure
*P < 00.5 vs. low-fit of same age category

Conclusion: In OA, higher fitness is associated with a greater decrease in endothelial function during acute inflammation; high fit OA had a similar endothelial response compared to YA. This suggests intact reactivity of the vasculature to inflammatory stress in high fit OA, which may indicate a healthier vessel versus low fit OA.

5.4
EFFECT OF ACUTE RESISTANCE EXERCISE ON ARTERIAL HEMODYNAMICS AND CEREBRAL BLOOD FLOW DYNAMICS: DOES SEX MATTER?

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High-intensity resistance exercise (RE) acutely increases arterial stiffness and blood pressure (BP), coupled with reduced cerebral blood flow velocity (CBFv) and greater flow pulsatility in the cerebral circulation, which may be detrimental to cerebral microvasculature. Because females have different CV control mechanisms, it is important to assess potential sex differences in cerebrovascular responses to acute RE.
Purpose: To examine the effect of sex on hemodynamics and cerebral vasculature following acute RE.
Methods: Men (n = 18, 27 yrs, BMI = 24.2) and women (n = 14, 25 yrs, BMI = 23.8) performed RE (3 × 10, isokinetic knee flexion/extension). Measurements were obtained at baseline and post-exercise (1, 5, 30-minute). Beat-to-beat heart rate (HR), brachial BP, cardiac output (CO), stroke volume and end-tidal CO_2 were collected. CBFv was measured by transcranial Doppler, carotid BP by applanation tonometry and central pulse wave velocity (PWV) by an automated ambulatory BP monitor.
Results: Table 1. CBFv pulsatility increased following RE at 1-minute post ($p < 0.05$) in men and was elevated above baseline 5-minute post-exercise ($p < 0.05$) in both groups (Figure 1). Mean CBFv increased 1-min post-exercise and decreased below baseline 5-minute post-exercise ($p < 0.05$) in both sexes. PWV increased 1-minute post-exercise ($p < 0.05$) in both groups.

Table 1 All Data are mean ± SD; *Exercise effect, $p < 0.05$. †Group effect. $p < 0.05$. ‡Interaction effect. $p < 0.05$. a Significantly different from 1-minute, b Significantly different from 5-minute. c Significantly different from 30-minute. $p < 0.05$). Brachial systolic BP (bSBP), brachial diastolic BP (bDBP), brachial mean BP (bMAP), Carotid systolic BP (cSBP),carotid diastolic BP (cDBP), cardiac output (CO), stroke volume (SV) and central pulse wave velocity (PWV), cerebral blood flow velocity (CBFv).

Variables		Baseline	1-minute	5-minute	30-minute
Heart Rate(bpm)*†	Men	63 ± 9	82 ± 9	71 ± 10	70 ± 8
	Women	70 ± 7	93 ± 13	79 ± 10	76 ± 9
CO (L/min) *	Men	4.9 ± 0.7	7.9 ± 1.3	6.1 ± 1.3	5.2 ± 0.9
	Women	5.4 ± 1.0	8.2 ± 1.4	6.3 ± 1.0	5.7 ± 0.8
SV (ml/min)*‡	Men	77 ± 16[ab]	95 ± 17[bc]	85 ± 17[c]	72 ± 13
	Women	80 ± 17[a]	91 ± 20[bc]	81 ± 14	76 ± 16
bSBP (mmHg)*	Men	124 ± 10	140 ± 12	127 ± 10	127 ± 9
	Women	124 ± 9	136 ± 15	122 ± 13	123 ± 9
bDBP (mmHg)*	Men	72 ± 8	74 ± 7	72 ± 7	76 ± 6
	Women	73 ± 5	76 ± 9	71 ± 7	73 ± 5
bMAP (mmHg)*	Men	92 ± 8	100 ± 8	93 ± 8	96 ± 7
	Women	95 ± 6	101 ± 11	93 ± 8	95 ± 6
cSBP (mmHg)*	Men	120 ± 12	129 ± 18	125 ± 13	126 ± 10
	Women	1 22 ± 12	124 ± 13	119 ± 18	123 ± 8
cDBP (mmHg)*	Men	75 ± 7	74 ± 7	75 ± 7	79 ± 6
	Women	74 ± 6	73 ± 6	75 ± 7	74 ± 5
cMAP (mniHg)*	Men	93 ± 8	94 ± 8	94 ± 7	97 ± 7
	Women	94 ± 6	93 ± 8	95 ± 9	96 ± 5
PWV (m/s)*	Men	5.2 ± 0.5	5.6 ± 0.5	5.4 ± 0.5	5.2 ± 0.3
	Women	5.0 ± 0.4	5.3 ± 0.5	5.1 ± 0.3	5.0 ± 0.3
CBFv Mean (cm/s)*†	Men	55.8 ± 7.6	63.9 ± 9.6	51.4 ± 6.9	53.7 ± 7.9
	Women	69.8 ± 14.4	81.0 ± 23.1	63.7 ± 12.9	65.8 ± 12.0
CBFv Pulsatility Index*†‡	Men	0.91 ± 0.12[ab]	1.10 ± 0.16*	1.13 ± 0.17[c]	0.89 ± 0.13
	Women	0.81 ± 0.09[b]	0.90 ± 0.18	0.95 ± 0.13[c]	0.81 ± 0.11
End-Tidal CO_2 (%)*	Men	4.86 ± 0.48	5.72 ± 0.66	4.64 ± 0.51	4.51 ± 0.58
	Women	4.41 ± 0.60	5.44 ± 0.65	4.26 ± 0.39	4.24 ± 0.50

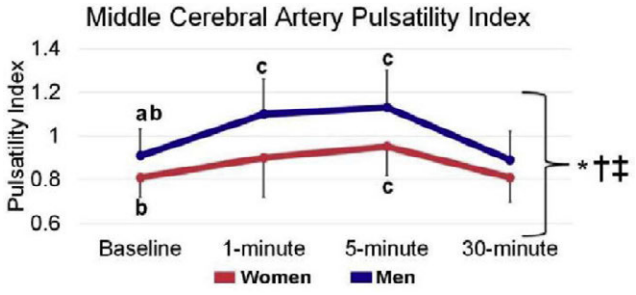

Figure 1 All Data are mean ± SD, *Exercise effect, $p < 0.05$. †Group effect, $p < 0.05$. ‡Interaction effect, $p < 0.05$. a Significantly different from 1-minute, b Significantly different from 5-minute. c Significantly different from 30-minute, $p < 0.05$).

Conclusion: Men increased CBFv pulsatility at 1-minute post-RE compared to women, demonstrating a sex difference in cerebral vascular reactivity. RE increased central arterial stiffness, mean CBFv, HR, and BP similarly for both sexes. CO was also elevated at 5-minute, but CBFv dropped below baseline and pulsatility continued to rise above baseline. This temporary disruption in cerebral autoregulation may impact brain health in both sexes.

5.5
IMPACT OF PULMONARY ENDARTERECTOMY ON PULMONARY ARTERIAL WAVE PROPAGATION AND RESERVOIR FUNCTION

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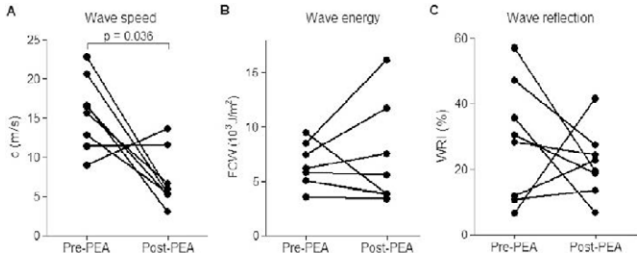
Background: Recent studies have demonstrated distinctive arterial wave characteristics in patients with chronic thromboembolic pulmonary hypertension (CTEPH)[1]. Therefore, we aimed to assess the impact of pulmonary endarterectomy (PEA) on pulmonary arterial wave propagation and reservoir function in CTEPH patients.

Methods: Right heart catheterization was performed using a pressure and Doppler flow sensor tipped guidewire to obtain simultaneous pressure and flow velocity measurements in the pulmonary artery in eight CTEPH patients before and 3 months after PEA. Wave intensity and reservoir-excess pressure analyses[2] were subsequently applied to the acquired data and the diastolic pressure decay time was estimated.

Results: Following PEA, mean pulmonary pressure (49 ± 10 mmHg versus 32 ± 13 mmHg), pulmonary vascular resistance (PVR) and wave speed, i.e. local arterial stiffness, significantly improved. However, there were no significant changes in arterial wave energy and wave reflection index (29.3 % [11.4—41.4 %] versus 21.2 % [16.2 — 25.9 %] post-PEA), even in patients with normalized pulmonary pressure. The RC-time (product of PVR and compliance) decreased post-PEA. Furthermore, the reservoir pressure related to arterial compliance, excess pressure caused by arterial waves and asymptotic pressure at which the flow would cease significantly decreased post-PEA and the changes were associated with improved right ventricular afterload, function and size.



Conclusion: Large wave reflection persisted post-PEA indicating lack of normalization of vascular impedance mismatch. Decreased RC-time suggests structural damage to the pulmonary vasculature. Wave intensity and reservoir-excess pressure analysis may be used as an additional assessment of the hemodynamic outcomes following PEA.