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PO-02: EFFECTS OF ACUTE INDUCED INFLAMMATION ON PRESSURE WAVEFORMS: DOES AGE MATTER?

K. Bunsawat, A.D. Lan, R.M. Kappus, S.M. Ranadive, H. Yan, Sang-Ouk Wee, S. Phillips, T. Baynard, J. Woods, R. Motl, B. Fernhall

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NAA Poster Presentation Abstracts

PO-01

LONGITUDINAL AND CIRCUMFERENTIAL STRAIN OF THE PROXIMAL AORTA

V. Bell ^a, W. A. Mitchell ^a, S. Sigurðsson ^b, J. J. Westenberg ^c, J. D. Gotlib ^a, A. Torjesen ^a, T. Aspelund ^{b,d}, L. J. Launer ^e, A. de Roos ^c, V. Gudnason ^{b,d}, T. B. Harris ^e, G. F. Mitchell ^a

^aCardiovascular Engineering, Inc., Norwood, MA, USA

^bIcelandic Heart Association, Kopavogur, Iceland

^cLeiden University Medical Center, Leiden, The Netherlands

^dUniversity of Iceland, Reykjavik, Iceland

^eNational Institute on Aging, National Institutes of Health, Bethesda, MD, USA

Objectives: Proximal aortic stiffness increases with age and contributes to pathogenesis of wide pulse pressure and epidemic proportions of isolated systolic hypertension, which is difficult to control. Elucidation of factors that contribute to abnormal mechanical properties of the proximal aorta may facilitate development of more effective interventions. During systole there is substantial aortic long axis displacement and longitudinal strain, which we hypothesize causes overestimation of ascending aortic stiffness calculated from circumferential strain.

Methods: We performed magnetic resonance imaging in 375 participants (72 to 94 years of age, 204 women) in the Age, Gene/Environment Susceptibility-Reykjavik Study and measured circumferential and longitudinal strain along the aortic arch. Local pulse wave velocity (PWV) was calculated from circumferential strain and central pulse pressure using the Bramwell-Hill equation.

Results: Observed circumferential area strain was lower (geometric mean [95% confidence interval], 7.7 [7.3, 8.1] vs. 12.7 [12.2, 13.2]%, $P < 0.001$) and PWV was higher (11.0 [10.7, 11.3] vs. 8.5 [8.3, 8.8] m/s, $P < 0.001$) in the proximal ascending versus proximal descending thoracic aorta. In contrast, peak flow was similar at the two locations (39 [38, 40] vs. 39 [38, 40] cm/s, $P = 0.78$), which was inconsistent with observed differences in strain and PWV. When ascending aortic circumferential strain was corrected for longitudinal strain (7.8 ± 2.6%), PWV was comparable in the ascending and descending aorta (8.3 [8.2, 8.5] vs. 8.5 [8.3, 8.8] m/s, $P = 0.074$), consistent with comparable flow velocities.

Conclusion: Longitudinal strain represents a substantial and previously ignored component of proximal aortic volume storage that should be considered in order to avoid misclassification of ascending aortic stiffness.

PO-02

EFFECTS OF ACUTE INDUCED INFLAMMATION ON PRESSURE WAVEFORMS: DOES AGE MATTER?

K. Bunsawat ^a, A. D. Lan ^b, R. M. Kappus ^a, S. M. Ranadive ^c, H. Yan ^d, Sang-Ouk Wee ^a, S. Phillips ^a, T. Baynard ^a, J. Woods ^d, R. Motl ^d, B. Fernhall ^a

^aDepartment of Kinesiology, Nutrition, and Rehabilitation, University of Illinois at Chicago, Chicago, IL, USA

^bDepartment of Health and Human Physiology, University of Iowa, Iowa City, IA, USA

^cDepartment of Anesthesiology, Mayo Clinic, Rochester, MN, USA

^dDepartment of Kinesiology, East Carolina University, Greenville, NC, USA

The Augmentation index (Alx) is a strong independent predictor of atherosclerosis. Aging is characterized by increased Alx and low grade

PO-02. Table 1

Variables		Baseline	Post 24-hr	Post 48-h	Time	Age	Interaction
aPP (mmHg)	YA	30 ± 6	32 ± 6	33 ± 6	0.730	<0.001	0.001
	OA	43 ± 10 [#]	41 ± 10 [#]	39 ± 9 ^{#,*}			
Alx	YA	7.80 ± 11.26	3.05 ± 11.14	4.60 ± 11.54	0.085	<0.001	0.185
	OA	34.52 ± 10.45 [#]	34.40 ± 13.09	32.56 ± 8.33			
Alx@75	YA	-1.40 ± 12.3	-4.35 ± 11.13	-5.05 ± 10.61	0.132	<0.001	0.434
	OA	28.24 ± 9.89 [#]	28.56 ± 10.09	26.68 ± 7.37			
cPWV	YA	5.38 ± 0.76	5.48 ± 0.90	5.50 ± 1.05	0.681	<0.001	0.885
	OA	7.61 ± 1.76 [#]	8.04 ± 1.97	7.69 ± 1.33			
FPH (mmHg)	YA	27 ± 5	29 ± 5	29 ± 5	0.682	1.812	0.010
	OA	28 ± 5	25 ± 4 ^{#,*}	25 ± 5			
RPH (mmHg)	YA	13 ± 4	13 ± 4	14 ± 3	0.002	<0.001	<0.001
	OA	22 ± 6 [#]	19 ± 6 ^{#,*}	18 ± 5 ^{#,*}			
IL-6 (ng/mL)	YA	0.89 ± 0.57	2.29 ± 2.14 [*]	1.14 ± 1.33	<0.001	0.001	0.001
	OA	2.18 ± 1.47 [#]	2.60 ± 1.85	2.23 ± 1.46 [#]			
CRP (mg/L)	YA	1.03 ± 1.01	1.28 ± 0.95	1.62 ± 1.19	<0.001	0.002	0.492
	OA	2.60 ± 2.19 [#]	3.94 ± 3.76	4.38 ± 4.13			

Data are mean ± SD. BMI, body mass index; VO_{2peak}, peak oxygen consumption; aPP, aortic pulse pressure; Alx, augmentation index; Alx@75, augmentation index corrected for heart rate 75 bpm; cPWV, central pulse wave velocity; FPH, forward pulse height; RPH, reflected pulse height; IL-6, interleukin-6; CRP, C-reactive protein.

*Significantly different than baseline ($P < 0.05$).

[#]Significantly different than young adults ($P < 0.05$).

inflammation. However, the effect of induced systemic inflammation on Alx is unclear.

Purpose: To investigate the effect of acute induced inflammation on wave reflection using wave separation analysis (WSA) in young (YA) vs. old adults (OA) pre-vaccination and 24-hr and 48-hr post vaccination.

Methods: Subjects were 22 YA (female=14; age 25±4 yrs; BMI 23.3±3.0 kg/m²) and 26 OA (female=17; age 63±6 yrs; BMI 29.6±6.3 kg/m²). Alx was assessed using applanation tonometry and followed by wave separation analysis (SphygmoCor, AtCor Medical). CRP and IL-6 were measured using ELISA assays.

Results: Compared to YA, OA had higher baseline aortic pulse pressure (aPP), Alx, Alx@75, central pulse wave velocity (cPWV), reflected wave pressure (RPH), IL-6, and CRP (P<0.05). Alx, Alx@75, and cPWV did not change from baseline, but were higher in OA at all time points (P<0.05). aPP, Forward wave pressure (FPH) and RPH decreased from baseline in OA (P<0.05), but did not change in YA. IL-6 increased from baseline at post 24-hr in YA, but not in OA (P<0.05).

Conclusions: Although acute induced inflammation did not change indices of central arterial stiffness in OA, WSA revealed that FPH and RPH decreased in OA, concomitant with an aPP reduction. It appears that induced inflammation has a greater effect on arterial function and aPP in OA possibly due to greater effects of inflammation on peripheral vasodilatation in this group.

**PO-03
SEX DIFFERENCES IN STIFFNESS PARAMETERS FOLLOWING MAXIMAL EXERCISE**

R. M. Kappus ^a, S. M. Ranadive ^b, H. Yan ^c, A. D. Lane ^d, J. A. Woods ^e, K. R. Wilund ^e, B. Fernhall ^a

^aUniversity of Illinois at Chicago, Chicago, IL, USA

^bMayo Clinic, Rochester, MN, USA

^cEast Carolina University, Greenville, NC, USA

^dUniversity of Iowa, Iowa City, IA, USA

^eUniversity of Illinois at Urbana Champaign, Urbana, IL, USA

Objectives: The sex differences found in cardiovascular disease risk and progression are well established. These discrepancies are potentially attributed to the cardioprotective effect of estrogen or sex specific differences in fitness. There may also be sex differences in the cardiovascular responses to exercise, which could underlie this disease risk. We investigated arterial stiffness parameters at rest and following maximal exercise in untrained males and females.

Methods: Eighty-three young (mean age=25 years), healthy males (n= 39) and females (n=44) underwent measures of vascular stiffness at rest and

both 15 and 30 minutes (po15, po30) following maximal exercise. The exercise stimulus was an acute progressive maximal exercise bout on a cycle ergometer.

Results: Females had significantly lower pressures (carotid, aortic and brachial) at all time points compared to males, with no heart rate differences. Arterial compliance (AC) and Elastic Modulus (Ep) changed similarly between sexes, with a decreased compliance at po15, returning to baseline values at po30. Males had significantly elevated central stiffness (cPWV) at both rest and po15 compared to females, but significantly decreased at po30 to match values of the females. The significance in cPWV between sexes remained after controlling for aortic MAP.

Conclusions: Females have a less stiff resting arterial profile compared to males. However, with maximal exercise, males altered their arterial profile to eliminate any significant differences between females in stiffness indices. This suggests that a maximal bout of exercise is an appropriate stimulus for evaluating stress induced sex differences in arterial stiffness.

**PO-04
CORRELATIONS BETWEEN ARTERIAL STIFFNESS/CENTRAL HEMODYNAMICS AND SERUM CARDIAC TROPONIN T AND NATRIURETIC PEPTIDE LEVELS**

Hirofumi Tomiyama, Kazutaka Kimura, Chisamatsumoto, Kazuki Shiina, Akira Yamashina
Tokyo Medical University, Tokyo, Japan

Objective: Elevated serum levels of cardiac troponin T (cTnT) and N-terminal fragment of B-type natriuretic peptide (NT-proBNP), and also increased arterial stiffness/abnormal central hemodynamics are well-known risk factors for future cardiovascular events. The present study was conducted to clarify which of the two - the serum level of cTnT or that of NT-proBNP - might be more closely associated with the arterial stiffness/central hemodynamics.

Methods and results: In 2374 male employees of a company (46 ± 9 years old), the following parameters were measured: second peak of the radial systolic pressure waveform (SBP2), radial augmentation index (rAI), PP2 (SBP2 minus the diastolic blood pressure), brachial-ankle pulse wave velocity (baPWV), and serum levels of cTnT and NT-pro BNP. After adjustments for confounding variables, binary logistic regression analyses demonstrated that baPWV was associated with a significant odds ratio for serum NT-proBNP ≥125 pg/mL (1.690; 95% confidence interval = 1.136–2.514, p = 0.002) and rAI was associated with a significant odds ratio for serum NT-proBNP ≥55 pg/mL (1.205; 95% confidence interval = 1.012–1.435, p = 0.036). The baPWV, rAI, SP2 and PP2 were not associated with significant odds ratios for elevated serum cTnT levels (≥0.014 ng/mL and ≥0.010 ng/mL).

Conclusions: Increased arterial stiffness/abnormal central hemodynamics may be associated with elevated serum NT-proBNP levels, rather than with minimally elevated serum cTnT levels. This difference may be one of the plausible explanations for the independency of the predictive values of the two serum markers for future cardiovascular events.

**PO-05
BUFFERING OF CAROTID ARTERY PRESSURE AND FLOW PULSATILITY DURING COGNITIVE ENGAGEMENT IN HEALTHY ADULTS**

Kevin S. Heffernan ^a, Nicole L. Spartano ^a, Jacqueline A. Augustine ^a, Wesley K. Lefferts ^a, William E. Hughes ^a, Gary F. Mitchell ^d, Randall S. Jorgensen ^b, Brooks B. Gump ^c

PO-03. Table 1 Descriptive statistics.

	Total (n=83)	Males (n=39)	Females (n=44)
Age (yr)	25.3 ± 0.8	25.2 ± 1.5	24.9 ± 0.7
BMI (kg/m ²)	27.2 ± 0.8	26.7 ± 0.7	27.6 ± 1.3
Height (cm) *	170.2 ± 1.1	178.2 ± 0.9	163.2 ± 1.1
Weight (kg) *	79.1 ± 2.4	84.9 ± 2.4	74.1 ± 4.0
VO2peak (ml/kg/min) *	33.1 ± 0.9	38.4 ± 1.3	28.3 ± 0.9

PO-03. Table 2 Pressure and stiffness response before and following maximal exercise.

	Males			Females		
	Rest	Post15	Post30	Rest	Post15	Post30
bMAP (mmHg)	90±1*	92±2*	89±1* #	85±1	84±2	84±1
aorMAP (mmHg)	87±1*	90±2*	88±1*	84±1	82±1	82±1
HR (bpm)	63±2	86±2 \$	79±2 \$#	65±1	83±2 \$	78±2 \$#
cPWV (m/s)	6.12±0.17*	6.21±0.15*	5.84±0.17\$	5.50±0.16	5.34±0.14	5.56±0.15
Ep (kPa)	72.51±3.42*	81.09±4.52	71.51±3.93	61.33±3.12	71.41±4.13\$	66.14±3.49
AC (mm ² /kPa)	1.14±0.07*	0.97±0.07\$	1.12±0.06#	1.31±0.06	1.10±0.06\$	1.22±0.06#
B-Stiffness	5.81±0.27	6.23±0.33	5.71±0.32	5.24±0.24	6.10±0.30\$	5.71±0.29

*p<0.05 between sexes.
\$ sig diff from rest.
sig diff from po15.