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PO-24: SEX DIFFERENCES IN HEMODYNAMIC RESPONSES FOLLOWING ACUTE INFLAMMATION: WAVE SEPARATION ANALYSIS

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the differences in markers of vascular function between Hispanics (HS), Caucasians (CA), and African Americans (AA). This study sought to assess the differences in vascular function at the endothelial cell level between these racial groups.

Methods: Three human umbilical vein endothelial cell (HUVEC) lines from different donors with HS, CC, and AA backgrounds were used. All cells were grown until confluent before cell medium and cell lysate was harvested. The cell medium was collected for the measurement of Interleukin 6 (IL-6) in an ELISA assay kit. The harvested cell lysate was used for western blotting for the measurement of Endothelial Nitric Oxide Synthase (eNOS), Phosphorylated Endothelial Nitric Oxide Synthase (p-eNOS), and Endothelin Converting Enzyme (ECE).

Results: The expression of eNOS in both the CC and HS cell lines was significantly lower when compared to the AA cell lines ($p \leq 0.001$). p-eNOS expression was significantly higher in the HS cell lines compared to both the AA and the CA cell lines ($p \leq 0.001$). The p-eNOS to eNOS ratio was significantly lower in both the AA ($p \leq 0.03$) and CA ($p \leq 0.001$) cell lines compared to the HS cell lines. ECE expression was significantly higher in the HS cell lines compared to the AA cell lines ($p \leq 0.001$). IL-6 levels were significantly higher in the CA and HS cell lines compared to the AA cell lines ($p \leq 0.001$).

Conclusions: Differences in endothelial cell biology that could affect function were evident among cell lines of different racial origin.

PO-22

HIGHER AORTIC STIFFNESS AND CAROTID SYSTOLIC AND PULSE PRESSURE ARE SELECTIVELY ASSOCIATED WITH LOWER WHITE MATTER INTEGRITY IN THE GENU AND FRONTAL CORTEX IN OLDER HEALTHY ADULTS

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Introduction: Previous studies have demonstrated an association between higher aortic stiffness and central pulse pressure (PP) with lower brain white matter structural integrity (WMI) and neuropsychological functioning in older adults. However, it is unknown if aortic stiffness and central PP are associated with lower WMI in select brain regions or if they relate to cognitive abilities that decline with age such as processing speed.

Objectives: We hypothesized that greater aortic stiffness and carotid PP would be associated with lower regional WMI and slower processing speed.

Methods and results: In younger ($n=12$, age 23.2 ± 2.3 yrs) and older ($n=7$, 67.7 ± 2.7 yrs) healthy adults, aortic stiffness (carotid-femoral pulse wave velocity, cFPWV) and carotid blood pressure (BP) were determined non-invasively using applanation tonometry and brachial cuff BP (Cardiovascular Engineering, Inc.). Fractional anisotropy (FA) (3T MRI, Siemens) assessed from diffusion imaging measured WMI. The association between vascular variables and FA was determined using voxel-wise and region-of-interest (ROI) analyses. Letter and pattern comparison assessed processing speed.

Results: In the entire cohort, cFPWV (adjusted for age, mean BP) and carotid and brachial PP (adjusted for age) were not correlated with WMI in any brain regions using voxel-wise or ROI. Among older adults using ROI, cFPWV (adjusted for mean BP) was correlated with genu corpus callosum ($r = -0.90$, $p < 0.05$) and frontal ($r = -0.77$, $p < 0.05$) FA values and corroborated in voxel-wise analyses. Carotid, but not brachial systolic BP or PP, was negatively correlated with genu and superior frontal gyrus and medial prefrontal cortex FA values ($p < 0.05$) using voxel-wise analysis. cFPWV, but not FA in the genu or frontal ROIs, was correlated with processing speed ($p < 0.05$) in older adults.

Conclusion: Preliminary results suggest that greater aortic stiffness is selectively associated with lower WMI in the genu and frontal cortex, and slower processing speed in older adults.

PO-23

DEPENDENCY OF ARTERIAL STIFFNESS INDICATORS ON ACUTE BLOOD VOLUME CHANGES

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Increased arterial stiffness is associated with greater risk for cardiovascular disease. It is unknown if indicators of stiffness are dependent on acute changes in cardiovascular conditions (such as altered central blood volume).

Objectives: To examine if arterial stiffness indicators change with acute reductions in stroke volume (SV) within normal physiological variability.

Methods: Seven young healthy volunteers (4M, 3F) were recruited to participate in this study. To acutely alter blood volume, subjects were sealed from their waist down into a lower body negative pressure (LBNP) box and a vacuum was used to create a pressure gradient of 30mmHg. Heart rate (HR) was continuously monitored and SV was obtained with Doppler ultrasound. Aortic and femoral artery velocity profiles were obtained with Doppler ultrasound to determine central pulse wave transit time (cPWTT). cPWTT was calculated by subtracting the time between the peak of the R-wave and the foot of the aortic velocity profile from the time between the peak of the R-wave and the foot of the femoral velocity profile. Common carotid distensibility (cDa) was determined with simultaneous tonometry to determine pulse pressure (PPcar) and ultrasound imaging to determine diastolic and systolic diameters ($cDa = \text{systolic area} - \text{diastolic area} / \text{PPcar} - \text{carotid diastolic area}$).

Results: The increase in HR from baseline to LBNP was not significant while SV was significantly lower at LBNP ($45 \pm 13 \text{ mL/beat}$) compared to baseline ($69 \pm 11 \text{ mL/beat}$; $p = 0.002$). PPcar was lower at LBNP ($43 \pm 6 \text{ mmHg}$) compared to baseline ($48 \pm 5 \text{ mmHg}$; $p = 0.007$). While cDa was significantly decreased (Baseline = $0.00732 \pm 0.00186 \text{ mmHg}^{-1}$ vs. LBNP = $0.00592 \pm 0.00219 \text{ mmHg}^{-1}$; $p = 0.033$), cPWTT tended to get faster with LBNP (baseline = $95 \pm 17 \text{ sec}$ vs. LBNP = $87 \pm 13 \text{ sec}$; $p = 0.089$).

Conclusions: The arterial stiffness indicators, cDa and cPWTT, might be affected by acute changes in central blood volume and cardiac SV within normal physiological variations.

PO-24

SEX DIFFERENCES IN HEMODYNAMIC RESPONSES FOLLOWING ACUTE INFLAMMATION: WAVE SEPARATION ANALYSIS

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Acute inflammation temporarily increases risk of cardiovascular events and alters hemodynamics. However, it is unknown whether acute inflammation differentially affects blood pressure and pulse wave characteristics, including forward or reflected pressure waves, in males versus females.

Objectives: The purpose of this study was to investigate the potential sex differences in the response to acute inflammation in blood pressure and pulse wave characteristics, measured with wave separation analysis.

Methods: 63 adults (29 males, 34 females) participated in the study. Participants received an influenza vaccine to induce acute inflammation. Central blood pressure and pulse waves were measured using tonometry and separated into forward and reflected waves, at baseline, 24hr post, and 48 hr post-vaccination. 2×3 repeated measure Analysis of Variance (ANOVA) was performed to investigate sex differences in acute inflammation.

PO-24. Table 1

	Brachial DBP(mmHg)†			Aortic DBP(mmHg)†			Aortic MAP(mmHg)†			Forward wave pressure(mmHg)			Reflected wave pressure(mmHg)		
	Baseline	24 hr Post	48 hr post	Baseline	24 hr Post	48 hr post	Baseline	24 hr Post	48 hr post	Baseline	24 hr Post	48 hr post	Baseline	24 hr Post	48 hr post
Male	69±9	67±9	69±9	70±9	68±9	69±9	87±10	84±9	85±10	29±5	29±5	28±6	19±7	17±7	18±6
Female	66±8*	63±7	64±7	66±8*	64±7	64±7	82±11*	79±8	79±9	27±5	27±5	26±5	17±7	15±5	15±4

* Different from other time point, $p < 0.05$.

† Sex difference. Significant at $p < 0.05$.

Results: (See table) There were significant sex differences in brachial SBP, brachial DBP, aortic DBP and aortic MAP with higher values in males. ($p < 0.05$). However, there were no statistically significant sex differences in wave separation variables or aortic SBP during acute inflammation, but acute inflammation decrease brachial DBP, aortic SBP, and aortic MAP in all subjects combined, and reflected pulse pressure approached a decline in the entire cohort ($p = 0.06$).

Conclusions: The results suggest that blood pressure, forward and reflected pulse wave pressure exhibited similar responses in males and females during acute inflammation.

PO-25

LEFT VENTRICULAR END-SYSTOLIC ELASTANCE (ECAVI) ESTIMATED WITH CAVI

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Objective: Left ventricular end-systolic elastance (Ecavi) was estimated using the parameters measured for calculating cardio-ankle vascular index (CAVI).

Methods: Participants comprised 4,954 healthy individuals (2,679 males, 2,275 females) who visited the health examination center at Fukui-ken Saiseikai Hospital between July 2007 and November 2013. Left ventricular-arterial coupling (Ees/Ea) was obtained from end-systolic arterial pressure (Pes), end-diastolic arterial pressure (Pd), pre-ejection period (PEP) and ejection time (ET), all of which were obtained as parameters measured on a vascular screening system (VaSera VS-1500N; Fukuda Denshi, Tokyo, Japan) based on the non-invasive method described by Hayashi et al.⁽¹⁾. Mean arterial pressure (Pm) was assumed to be equal to Pes for the calculation of Ees/Ea⁽²⁾ in this study. Ees/Ea was assumed as the balance of stiffness between the end-systolic left ventricle and aorta. Left ventricular end-systolic elastance estimated with CAVI was defined as $CAVI \times Ees/Ea$.

Results: The population showed the same results as the healthy group recruited in the user's manual of the vascular screening system (Fig. 1); namely, normal range of CAVI was between 6.3 and 8.7, CAVI was higher in males than in females, and CAVI was slightly increased in the high aged group. Mean and standard deviation of Ecavi were 9.3 and 4.5, respectively, in all age groups, and in both males and females (Fig. 2).

Conclusion: The original left ventricular end-systolic elastance (Ees) could be estimated as Ecavi, representing $CAVI \times Ees/Ea$, using a non-invasive vascular screening system.

References:

1. Hayashi K. et al., *Anesthesiology*, 2000;92:1769-76.

2. Takaku A. et al., *Proc of 11th Annual Meeting Tokai-Hokuriku Branch JSA*, 2013;11:Q01-1.

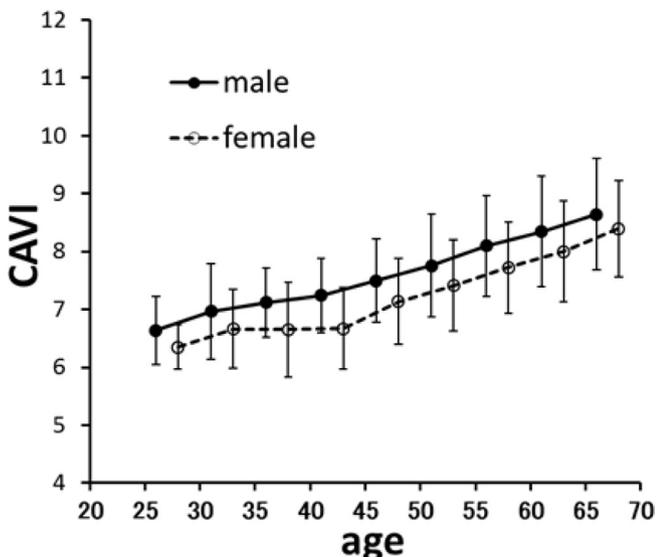


Figure. 1 Average of CAVI in Healthy Group.

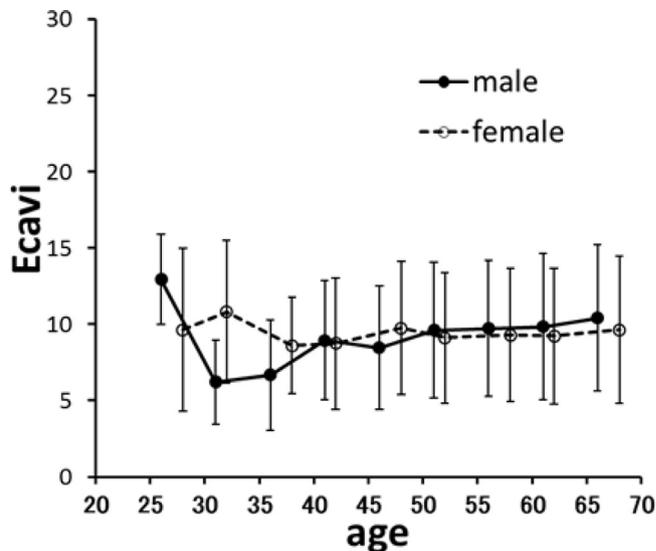


Figure. 2 Average of Ecavi in Healthy Group.

PO-26

THE IMPLICATIONS OF POOR SLEEP QUALITY ON ARTERIAL HEALTH IN PERSONS WITH MULTIPLE SCLEROSIS

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Objective: Multiple sclerosis (MS) is a chronic, autoimmune disease that is associated with increased risk of cardiovascular disease (CVD) when compared to the general population. Approximately 47% of patients with MS have reported poor sleep quality. Evidence supports an association between poor sleep and increased CVD risk. Augmentation index (AIx) is a marker of arterial health. The purpose was to examine the association between sleep quality and arterial health in patients with MS.

Methods: Thirty two patients with MS (Age: Mean \pm SD = 47.6 ± 10.6 yrs) and 32 matched controls (47.6 ± 11.3 yrs) were administered the Pittsburgh Sleep Quality Index (PSQI) to assess self-reported sleep quality. Subjects having a global score >5 were classified as "poor sleepers." Applanation tonometry was performed on the radial artery to obtain arterial pressure waveforms.

Results: Twenty MS subjects and 7 control subjects were classified as "poor sleepers." Statistical analysis confirmed that "poor sleep" was associated with higher AIx (16.2 ± 2.3 vs 23.7 ± 2.9 , $p < 0.05$) regardless of having MS. Among those with MS, AIx was significantly higher in the subjects who reported poor sleep quality when compared with those who reported good sleep quality (15.7 ± 3.8 vs 27.1 ± 3.0 , $p < 0.05$).

Conclusions: Poor sleep quality has a negative effect on arterial health overall and in those with MS. Additionally, those with MS who report poor sleep quality have an amplified negative arterial outcome compared to patients with MS with good sleep quality and healthy controls.

PO-27

HIGHER CENTRAL AUGMENTATION PRESSURE/INDEX IS ASSOCIATED WITH TENSION-TYPE HEADACHE BUT NOT MIGRAINE IN MIDDLE-AGED/ OLDER OBESE HUMANS

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