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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PO-25
LEFT VENTRICULAR END-SYSTOLIC ELASTANCE (ECAVI) ESTIMATED WITH CAVI

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Objective: Left ventricular end-systolic elastance (E cav i) 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 Sai-seikai Hospital between July 2007 and November 2013. Left ventricular-arterial coupling (Ees/Ea) was obtained from end-systolic arterial pressure (P es), end-diastolic arterial pressure (P d), 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).

Results: Participants comprised 4,954 healthy individuals (2,679 males, 2,275 females) who visited the health examination center at Fukui-ken Sai-seikai 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 = Ees/Ea.

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

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 (Alx) 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 ± 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 Alx (16.2 ± 2.3 vs 23.7 ± 2.9, p < 0.00) regardless of having MS. Among those with MS, Alx 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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Objective: To examine the association between sleep quality and arterial health in patients with MS.

Methods: Thirty two patients with MS (Age: Mean ± 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 Alx (16.2 ± 2.3 vs 23.7 ± 2.9, p < 0.00) regardless of having MS. Among those with MS, Alx 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.
**Objective**: Obesity is associated with a five-fold increased risk of developing chronic daily headache, especially chronic migraine. Migraine attacks are more frequent and more severe among obese migraineurs and they improve with weight loss; however, the underlying mechanisms are unknown. Given that elevated aortic stiffness and central pulse pressure are associated with cerebral microvascular dysfunction/damage, we hypothesized that obese middle-aged/older adults with history of migraine would demonstrate higher aortic stiffness, central blood pressure (BP) and augmentation index (AI)/pressure (AP) compared with those without a history of migraine.

**Methods**: Middle-aged/older obese adults who were stratified (via detailed survey and physical exam by a neurologist) by presence of migraine (n=39; age 54 ± 6 yrs, BMI 38 ± 6 kg/m², 67% female), tension-type headache (n=25; age 57 ± 6 yrs, BMI 37 ± 4 kg/m², 72% female) or no headache of any type (n=29; age 54 ± 7 yrs, BMI 37.5 ± 5.4 kg/m², 48% female) had aortic stiffness (carotid-femoral pulse wave velocity, CFPWV), brachial and central BP, and central AI and AP assessed by applanation tonometry (SphygmCor).

**Results**: Obese adults with tension-type headache, but not migraine (P=0.29), demonstrated higher AI (25.4 ± 9.6 vs. 17.8 ± 6.9%, P=0.02) and AP (11.7 ± 9.6 vs. 6.8 ± 6.9 mmHg, P=0.01) compared with no headache controls, but no difference in CFPWV between the 3 groups (P=0.47). After adjusting for age, mean BP, female sex, weight, height, and antihypertensive medication, higher AP (β=2.95, p=0.04) and AI (β=4.41, P=0.07) remained associated with greater frequency of tension-type headache.

**Conclusions**: Higher central AI and AP, but not aortic stiffness, is associated with tension-type headache but not migraine in obese middle-aged/older adults. Whether excessive penetration of pulsatile pressure into cerebral microcirculation contributes to the development of tension-type or migraine headache in obesity requires further study.

**PO-28**

**CHANGES IN CEREBROVASCULAR PULSATILITY DURING AEROBIC EXERCISE ARE UNRELATED TO BRACHIAL-ANKLE PULSE WAVE VELOCITY IN CHRONIC STROKE**

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Arterial stiffness contributes to increased cerebral hemodynamic pulsatility and independently predicts negative outcomes post-stroke. Exercise can contribute to recovery after stroke; yet it is unclear whether arterial stiffness influences acute cerebrovascular responses to exercise. One study in healthy young men showed high-intensity resistance exercise increased stiffness and stiffness pulsatility up to 30 minutes post-exercise without affecting cerebral hemodynamics (1). The influence during acute aerobic exercise, however, is unknown.

**Objectives**: To investigate the association of arterial stiffness with changes in pulse pressure (PP) and middle cerebral artery pulsatility index (PI) during aerobic exercise in chronic stroke adults. We hypothesized that resting brachial-ankle pulse wave velocity (baPWV) would be associated with greater exercise-related increases in PP and PI.

**Methods**: Participants were recruited to 3 to 12 months post-stroke. baPWV was quantified using applanation tonometry. A symptom-limited cardopulmonary assessment determined peak aerobic fitness (VO2peak). In a subsequent session, participants cycled on a recumbent ergometer for 20 minutes at 60% heart rate reserve. Cerebral blood flow velocity was measured using transcranial ultrasound. Arterial blood pressure was measured using finger-cuff photoplethysmography.

**Results**: Preliminary results from 9 men and 2 women are reported (age: 68.9 ± 15 years; VO2peak: 25 ± 9 mL/kg/min; baPWV: 12.0 ± 2 m/s). At rest, baPWV was not correlated with PP or PI (P=0.6). During exercise, PP and PI increased 22.1% and 44.2%, respectively (P=0.001). A non-significant association was noted between ΔPI and ΔPP (r=0.8, P=0.096). Resting baPWV was unrelated to ΔPP (r=0.42, P=0.228) or ΔPI (r=0.04, P=0.932).

**Conclusions**: baPWV, an index of stiffness influenced by central and peripheral vasculature, was unrelated to blood pressure or cerebrovascular pulsatility in this small cohort. Change in cerebral blood flow pulsatility during moderate intensity exercise appears to be independent of systemic arterial stiffness, although a larger sample is still necessary.

**References**