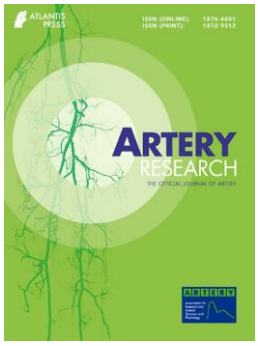

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# PO-27: HIGHER CENTRAL AUGMENTATION PRESSURE/INDEX IS ASSOCIATED WITH TENSION-TYPE HEADACHE BUT NOT MIGRAINE IN MIDDLE-AGED/OLDER OBESE HUMANS

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**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 × 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 × 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.

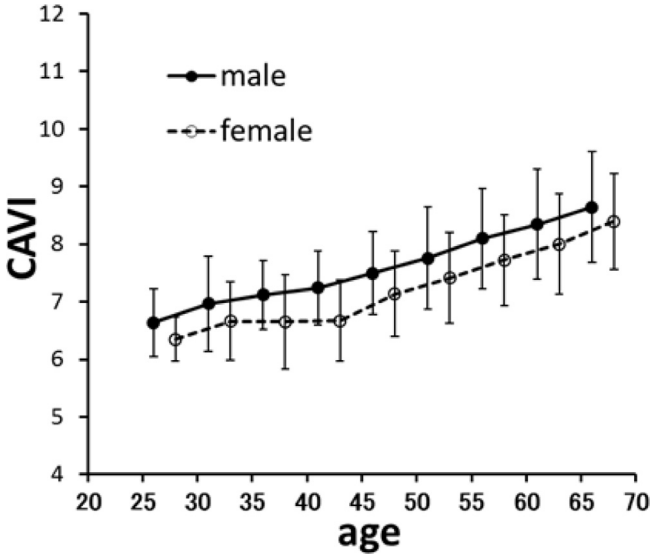


**Figure. 1** Average of CAVI in Healthy Group.

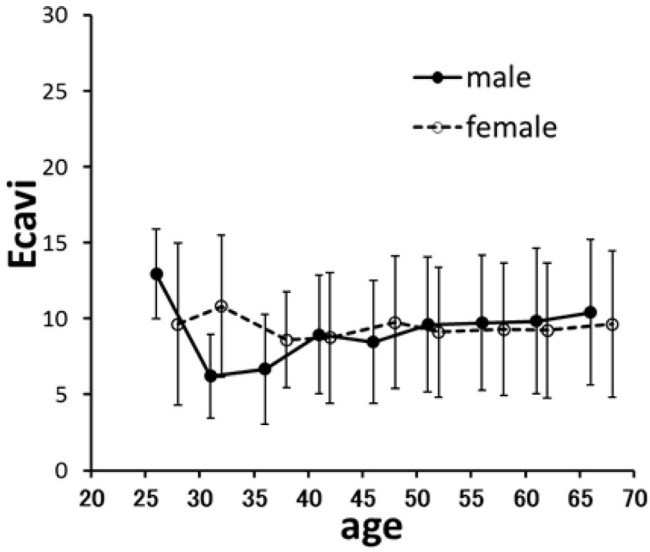


**Figure. 2** Average of Ecavi in Healthy Group.

## 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 ± 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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**Objectives**: 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 ± 8 yrs, BMI 38 ± 6 kg/m$^2$, 67% female), tension-type headache (n=25; age 57 ± 6 yrs, BMI 37 ± 4 kg/m$^2$, 72% female) or no headache of any type (n=29; age 54 ± 7 yrs, BMI 37± 5, 37± 5 kg/m$^2$, 48% female) had aortic stiffness (carotid-femoral pulse wave velocity, CFPWV), brachial and central BP, and central AI and AP assessed by applanation tonometry (SphygmoCor).
**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 towards 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 pressure 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 3 to 12 months post-stroke. BaPWV was quantified using applanation tonometry. A symptom-limited cardiopulmonary assessment determined peak aerobic fitness ($VO_{2peak}$). 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 years; $VO_{2peak}$: 19±5 mL/kg/min; baPWV: 12.0±2.0 m/s). At rest, baPWV was not correlated with PP or PI ($p>0.6$). During exercise, PP and PI increased 22±11% and 44±21%, respectively ($p\leq0.001$). A non-significant association was noted between ΔPI and ΔPP (r=0.68, $p=0.096$). 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
1. Lefferts WK *et al.* (2014) *Front. Physiol.* 5: 101.

## PO-29
## CREATION OF A FIXED CENTRAL ARTERIAL-VENOUS ANASTOMOSIS ON ARTERIAL STIFFNESS AND CENTRAL HAEMODYNAMICS: A TREATMENT FOR HYPERTENSION TARGETING THE PHYSICAL PROPERTIES OF THE ARTERIAL VASCULATURE

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**Introduction**: Current device based treatments for resistant hypertension target selective modification of the somatic, sympathetic, or parasympathetic nervous systems. The influence of the respective nervous systems on vascular stiffness and haemodynamics is unclear, and there is little data on the effect of current devices nor pharmaco therapy on arterial stiffness often associated with resistant hypertension.
A novel device technology (ROX Coupler, San Clemente, CA) has been developed that causes an immediate, significant and sustained reduction of blood pressure by exploiting the mechanical effects of creation of a low resistance, high compliance venous segment to the central arterial tree. The Coupler creates a 4 mm diameter AV anastomosis between the iliac artery and vein
To date no data exist on the effect of AV fistula placement on central haemodynamics and arterial stiffness. We present data on central pressure, and aortic pulse wave velocity (aPWV) from a 63yr old woman before and 4 months after AV fistula formation using the ROX Coupler device.
**Methods**: Peripheral blood pressure, central haemodynamics and carotid femoral pulse wave velocity (c-f PWV) were assessed (SphygmoCor AtCor Medical) before and 4 months after insertion of the ROX Coupler. Results are tabulated in (Table 1).
**Results**:

| | PRE AV Fistula | POST AV Fistula |
|---|---|---|
| Peripheral SBP mmHg | 184 | 172 |
| Central SBP mmHg | 172 | 158 |
| Peripheral DBP mmHg | 102 | 84 |
| Central DBP mmHg | 102 | 84 |
| Aix % | 34% | 27% |
| HR b/m | 66 | 68 |
| C-f PWV m/s | 15.2 | 13.7 |
| Peripheral MAP mmHg | 130 | 113 |
| Peripheral PP mmHg | 82 | 88 |
| Central PP mmHg | 70 | 74 |

aPWV decreased by 1.5 m/s from 15.2 to 13.7 m/s and MAP decreased by 17mmHg. Given that a 10mmHg reduction in MAP would produce an approximate reduction in aPWV of 0.5 m/s it would appear that the reduction in aPWV was in part blood pressure independent.
**Conclusions**: Insertion of the ROX Coupler was shown to produce a large reduction in aPWV which may not all be blood pressure dependent. These findings suggest that a mechanical solution to reduced arterial compliance may result in safe and effective lowering blood pressure, and address a mechanism of persistent hypertension unapproached by current therapy. Haemodynamic measurements in larger numbers of patients undergoing ROX Coupler insertion will be necessary to confirm this physiology and better appreciate its potential role in the prevention and treatment of the cardiovascular complications of hypertension.

## PO-30
## CAROTID STRAIN DOES NOT EXPLAIN SEX DIFFERENCES IN BLOOD PRESSURE

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**Objective**: Women have a lower incidence of cardiovascular morbidity and mortality prior to menopause when compared to age-matched men.