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

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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/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, 37 ± 5 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 (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≤0.001). 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

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PO-29

CREATION OF A FIXED CENTRAL ARTERIAL-VEIN 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 pharmacotherapy 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.

Specifically, on the vascular level, carotid arterial stiffness increases with age in both sexes, but with greater changes in older postmenopausal women. Arterial stiffness is a well-established predictor of future risk of CVD, and 2-dimensional ultrasound imaging of vascular deformation (Strain) using speckle tracking directly characterizes the elastic properties of the carotid arterial wall. The purpose of this study was to determine if sex differences exist for strain in the common carotid artery.

Methods: Twenty-eight healthy men and women (12/16; Range = 19-77 yrs) had 2-dimensional ultrasound images of the carotid artery taken. These images were obtained using an optimal circumferential view, and carotid strain (CS) and CS time to peak (TPK) were analyzed via speckle tracking software. Women were tested in the early follicular phase of menstrual cycle if they were premenopausal. Brachial (bSBP, bDBP, bMAP) and carotid (carSBP, carDBP, carMAP) pressure measurements were obtained in the supine position at rest using applanation tonometry.

Results: Females exhibited significantly lower resting blood pressure (bSBP, bDBP, bMAP, carMAP), radial displacement (RD) TPK, and CS TPK ($p < 0.05$). Strain and strain-rate were not different between sexes, even after controlling for age.

Conclusion: Women had lower resting blood pressures and a greater CS and RD TPK parameters compared to males. However, there were no sex differences in CS measurements. Therefore, elastic properties of the carotid artery do not account for the pressure difference demonstrated between sexes.

Variables	Males (n=12)	Females (n=16)
Age (yrs)	46 ± 21	46 ± 19
BMI (kg/m ²)	27.2 ± 4.3	25.3 ± 4.3
bSBP (mmHg)*	125 ± 12	111 ± 14
bDBP (mmHg)*	72 ± 7	66 ± 8
bMAP (mmHg)*	90 ± 8	81 ± 9
carSBP (mmHg)	117 ± 14	105 ± 19
carDBP (mmHg)*	73 ± 7	67 ± 8
carMAP (mmHg)*	92 ± 9	83 ± 10
Beta stiffness index	7.3 ± 3.3	6.8 ± 3.7
CS (PK%)	5.24 ± 3.22	5.58 ± 3.78
CS time to peak (ms)*	314 ± 36	356 ± 61
CS rate (PK 1/s)	0.43 ± .25	0.42 ± .28
CS rate TPK (ms)	170 ± 18	181 ± 25
Radial displacement (PK mm)	0.19 ± .10	0.18 ± 0.10
Radial displac. TPK (ms)*	313 ± 35	358 ± 61
Radial velocity (cm/s)	0.17 ± 0.08	0.15 ± 0.09
Radial velocity TPK (ms)	170 ± 18	181 ± 24

All Data are mean ± SD.

*Gender difference, $p < 0.05$.