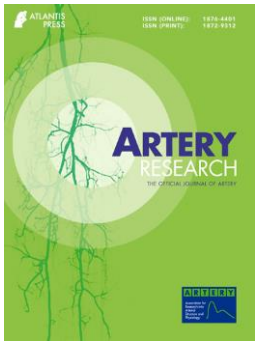

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# PO-05: BUFFERING OF CAROTID ARTERY PRESSURE AND FLOW PULSATILITY DURING COGNITIVE ENGAGEMENT IN HEALTHY ADULTS

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inflammation. However, the effect of induced systemic inflammation on AIx 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/m$^2$) and 26 OA (female=17; age 63±6 yrs; BMI 29.6±6.3 kg/m$^2$). AIx 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), AIx, AIx@75, central pulse wave velocity (cPWV), reflected wave pressure (RPH), IL-6, and CRP ($P<0.05$). AIx, AIx@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

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

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**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 $\geq$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 $\geq$55 pg/mL (1.205; 95% confidence interval = 1.012−1.435, p = 0.036). The baPWV, rAI, SP2 and PP2 were not associated with significant odds ratios for elevated serum cTnT levels ($\geq$0.014 ng/mL and $\geq$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 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.

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The matching of vascular supply to neuronal metabolic demand during cognitive engagement is known as neurovascular coupling (NVC). Excessive hemodynamic pulsatility may have a detrimental effect on neural function and affect NVC. Arterial stiffness is a prominent determinant of pulsatility in the brain.[1, 2]

**Objectives**: We explored changes in arterial stiffness and cerebrovascular hemodynamic pulsatility *during* cognitive engagement in healthy adults.

**Methods**: Twenty-seven adults (age 39 ± 3 yrs, BMI 24±1 kg/m$^2$) underwent Doppler ultrasonography and applanation tonometry of the common carotid artery (CCA) to derive 1) CCA elastic modulus (Ep) and β-stiffness index; 2) CCA flow pulsatility index (PI); 3) CCA pulse pressure (PP), and 4) CCA augmentation index (AIx). Transcranial Doppler was used to assess cerebral PI at the middle cerebral artery (MCA). All measures were made simultaneously at rest and during a 4-minute Stroop task.

**Results**: CCA PI was reduced ($p<0.05$) while MCA PI was unchanged ($p>0.05$) during Stroop. Brachial PP increased during Stroop ($p<0.05$) while CCA PP was unchanged ($p>0.05$). Similarly, CCA Ep ($p>0.05$) and β-stiffness ($p>0.05$) were unchanged. CCA AIx increased ($p<0.05$).

**Conclusion**: Carotid pressure pulsatility and cerebral flow pulsatility is unaltered while carotid flow pulsatility is reduced during cognitive engagement. Carotid stiffness does not change suggesting that factors other than the elastic properties of the vessel moderate cerebrovascular pulsatility during cognitive engagement.

**Table 1** Hemodynamic and vascular parameters at rest and during Stroop task.

| Variable | Baseline | Stroop | *P*—value |
|---|---|---|---|
| Brachial pulse pressure, mmHg | 43 ± 1 | 46 ± 1 | 0.002 |
| Carotid pulse pressure, mmHg | 36 ± 1 | 35 ± 1 | 0.324 |
| Carotid β-stiffness, aU | 4.4 ± 0.4 | 4.2 ± 0.3 | 0.224 |
| Carotid Ep, kPa | 54.5 ± 5.5 | 53.8 ± 4.9 | 0.670 |
| Carotid pressure AIx, % | 1 ± 4 | 13 ± 4 | 0.001 |
| Carotid distension AIx, % | 4 ± 2 | 8 ± 2 | 0.001 |
| Carotid mean diameter, mm | 5.62 ± 0.13 | 5.74 ± 0.13 | 0.010 |
| Carotid pulsatility index | 1.75 ± 0.06 | 1.57 ± 0.06 | 0.016 |
| Cerebral pulsatility index | 0.75 ± 0.02 | 0.75 ± 0.01 | 0.841 |

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## PO-06
## EFFECTS OF SYSTEMIC NIACIN INFUSION ON SYMPATHETIC ACTIVITY, ARTERIAL STIFFNESS AND AORTIC WAVE REFLECTION: A PILOT STUDY

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**Objective**: Free fatty acids (FFA) may increase sympathetic activity and arterial stiffness. Niacin decreases FFA, however, little is known regarding the direct effects of niacin on sympathetic activity and arterial stiffness. We tested the hypothesis that niacin would decrease arterial stiffness, central aortic blood pressures, indices of aortic wave reflection, and muscle sympathetic nerve activity (MSNA).

**Methods**: High-fidelity radial arterial pressure waveforms and carotid-femoral pulse wave velocity (c-f PWV) were measured noninvasively by applanation tonometry before and during intravenous infusion of niacin (2.8 mg/min) at t=60, 90, 120 and 150 minutes in 5 healthy adults (2M/3F; aged 29±9 years). FFA (HPLC), MSNA (microneurography), arterial blood pressure (brachial arterial catheter) and heart rate (HR, ECG) were measured before and during niacin.

**Results**: While niacin produced a 75% reduction in FFA, contrary to our hypothesis, MSNA increased by 28-56% over all time points. After 60 minutes of niacin infusion, augmentation index (AIx) corrected for HR (AIx@75bpm) increased compared to baseline (Table 1; $P<0.05$). Repeated measures ANOVA also revealed trends for a main effect of niacin over time for AIx (P=0.12), augmented pressure (AP; P=0.18), and c-f PWV (P=0.13) (Table 1). When only comparing changes between baseline and t=60 (via paired t-test), both AIx and AP were both significantly increased ($P<0.05$).

**Conclusions**: Our preliminary results in a small group of subjects suggest that although IV niacin dramatically reduces FFA, it causes increases in MSNA and aortic wave reflection (AIx@75bpm). Inclusion of more subjects is needed to statistically confirm the strong trends for increased indices of wave reflection and arterial stiffness with niacin. Additionally, further studies are warranted to determine if chronic oral niacin therapy exerts similar effects.

**Table 1** Hemodynamic and vascular measurements before and during niacin.

| | | Baseline | 60 min | 90 min | 120 min | 150 min |
|---|---|---|---|---|---|---|
| HR (bpm) | | 73±6 | 71±5 | 69±7 | 71±8 | 76±9 |
| BSP (mmHg) | | 122±4 | 128±5 | 126±7 | 126±6 | 126±6 |
| BDP (mmHg) | 76±2 | 79±3 | 79±3 | 77±3 | | 78±3 |
| BPP (mmHg) | 47±2 | 49±3 | 48±4 | 48±4 | | 48±4 |
| ASP (mmHg) | 105±4 | 115±6 | 112±8 | 110±6 | | 110±6 |
| ADP (mmHg) | 77±3 | 80±4 | 80±3 | 79±3 | | 79±3 |
| APP (mmHg) | 28±1 | 34±3 | 32±5 | 31±4 | | 30±5 |
| PPA (%) | 165±6 | 146±8 | 154±10 | 159±9 | | 165±10 |
| AIx (%) | 6.9±2.1 | 18.6±4.7 | 14.5±5.2 | 12.3±4.4 | | 8.6±5.6 |
| AIx@75bpm (%) | 5.0±3.3 | 15.8±4.8* | 10.9±3.4 | 9.8±2.7 | | 8.6±2.5 |
| AP (mmHg) | 1.9±0.6 | 6.9±2.0 | 5.6±2.7 | 4.6±2.2 | | 3.7±2.6 |
| c-f PWV (cm/s) | 7.1±0.4 | 7.8±0.8 | 7.8±1.0 | 7.3±0.6 | | 7.2±0.5 |

Data are mean±SE; N=5;
*$P<0.05$; BSP, brachial systolic pressure; BDP, brachial diastolic pressure; BPP, brachial pulse pressure; ASP, aortic systolic pressure; ADP, aortic diastolic pressure; APP, aortic pulse pressure; PPA, pulse pressure amplification.

## PO-07
## RACIAL DIFFERENCES IN CIRCULATING csRAGE AND ALTERNATIVELY SPLICED esRAGE IN HEALTHY ADOLESCENTS: RELATION WITH AORTIC STIFFNESS

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**Background**: Binding of ligands to the receptor for advanced glycation end products (RAGE) triggers pro-inflammatory/oxidant signaling in the vascular wall. Increased circulating soluble forms of RAGE (sRAGE) are associated with decreased vascular risk and may be protective by acting as a decoy to prevent ligand binding to full-length RAGE. Sheddases, such as matrix metalloproteinase-9 (MMP 9) proteolytically cleave cell surface receptors including RAGE, forming cleaved soluble RAGE (csRAGE). However, sRAGE also includes endogenous secretory RAGE (esRAGE), an isoform of RAGE without receptor function derived from alternative splicing of RAGE pre-mRNA. sRAGE is lower in African-American (AA) compared with Caucasian adults and is hypothesized to contribute to elevated arterial stiffening and vascular risk in AAs. Indeed, we have previously demonstrated that sRAGE (1567±68.9 vs. 955±101.1 pg/mL, $p<0.001$) but not MMP9 is higher in Caucasian compared with AA adolescents and associated with lower carotid-femoral pulse wave velocity (CFPWV) (5.3±0.2 vs. 5.9±0.2 m/sec, $p<0.05$).

**Objectives**: We hypothesized that increased sRAGE in Caucasian versus AA adolescents is from increased circulating esRAGE through alternative splicing of RAGE pre-mRNA.

**Methods and results**: Circulating esRAGE (ELISA) was significantly higher (369±24.8 vs. 242±26.5 pg/mL, $P<0.01$) in Caucasian (n=24, age 16.5±0.3 yrs; BMI 22.9±0.8 kg/m$^2$) vs. AA (n=15, age 16.8±0.3 yrs; BMI 24.5±1.0 kg/m$^2$) adolescents ($P>0.05$). esRAGE was correlated with sRAGE (r=0.708, $P<0.001$), but esRAGE:sRAGE ratio did not differ between race (0.24±0.01