

**NORTH AMERICAN ARTERY**  
**Eighth Annual**  
**Meeting**

**POSTER**  
**ABSTRACTS**

**SERUM ALDOSTERONE, INACTIVE MATRIX GLA-PROTEIN, AND LARGE ARTERY STIFFNESS IN HYPERTENSION**

**Authors:** Jonathan Lee<sup>2</sup>; Swetha Gaddam MD<sup>1,2</sup>; Mayank Sardana MBBS<sup>3</sup>; Amer Ahmed Syed, MD<sup>2</sup>; Maheshwara R. Koppula, MD<sup>2</sup>; Swapna Varakantam, MD<sup>1,2</sup>; Izzah Vasim, MD<sup>1,2</sup>; Garrett H. Oldland, MD<sup>2</sup>; Bilal Ansari MD<sup>2</sup>; Timothy S. Phan, BSBME, BSECE<sup>2</sup>; Nadja EA Drummen, BSc<sup>4</sup>; Cees Vermeer, PhD<sup>4</sup>; Raymond R Townsend, MD<sup>2</sup>; Scott R. Akers, MD, PhD<sup>1</sup>; Wen Wei, PhD<sup>5</sup>; Edward G. Lakatta, MD<sup>5</sup>; Olga V. Fedorova, PhD<sup>5</sup>; Julio A. Chirinos MD, PhD<sup>1,2</sup>.

<sup>1</sup> Corporal Michael J. Crescenz VAMC, Philadelphia, PA, USA.

<sup>2</sup> Cardiology Division, Department of Medicine, University of Pennsylvania Perelman School of Medicine and Hospital of the University of Pennsylvania, Philadelphia PA, USA

<sup>3</sup> Cardiology Division, Department of Medicine, University of Massachusetts Medical School, Worcester, MA, USA

<sup>4</sup> R&D Group, VitaK, Maastricht University, Maastricht, The Netherlands

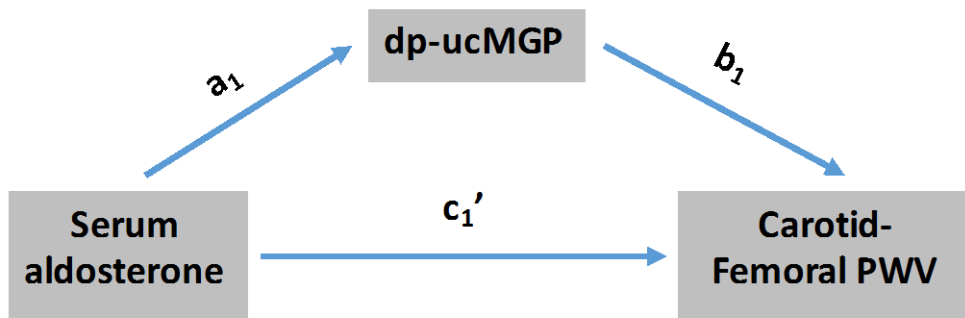
<sup>5</sup> National Institute of Aging, Baltimore, MD. USA.

**Objectives:** Vascular calcification leads to increased large artery stiffness. Matrix Gla-protein (MGP) is a vitamin K-dependent protein that inhibits arterial calcification. Aldosterone promotes vascular calcification and stiffness, but the relationships between aldosterone, MGP and arterial stiffness are unknown.

**Methods:** We studied 199 adults with hypertension. We assessed the relationship between levels of dp-ucMGP (inactive MGP), aldosterone and carotid-femoral pulse wave velocity (CF-PWV) using standard regression and mediation analyses. Serum aldosterone was measured in a subgroup of subjects (n=106).

**Results:** Aldosterone was strongly associated with dp-ucMGP (Standardized  $\beta=0.50$ ,  $P<0.001$ ), which was independent of potential confounders ( $\beta=0.37$ ,  $P<0.001$ ). Levels of dp-ucMGP were significantly associated with CF-PWV ( $\beta=0.30$ ;  $P<0.001$ ), which persisted after adjustment for potential confounders ( $\beta=0.25$ ;  $P=0.004$ ). Serum aldosterone was also significantly associated with CF-PWV (Standardized  $\beta=0.21$ ;  $P=0.035$ ). However, in a model that included aldosterone and dp-ucMGP, only the latter was associated with CF-PWV. Mediation analyses demonstrated a significant dp-ucMGP-mediated effect of aldosterone on CF-PWV, without a significant direct (dp-ucMGP-independent) effect.

**Conclusions:** Our study demonstrates a novel independent association between high aldosterone levels and dp-ucMGP, suggesting that aldosterone may influence the MGP pathway. This relationship appears to underlie the previously documented relationship between aldosterone and increased arterial stiffness.



Path	Standardized Estimate	95% Confidence Interval
Direct (dp-ucMGP-independent) aldosterone effect on CF-PWV ( $c_1'$ )	0.090	-0.095 to 0.274
Indirect aldosterone effect on CF-PWV, through dp-ucMGP ( $a_1 \cdot b_1$ )	0.102	0.012 to 0.218
Total effect of aldosterone on CF-PWV	0.179	0.013 to 0.346
Effect of aldosterone on dp-ucMGP ( $a_1$ )	0.438	0.273 to 0.603
Effect of dp-ucMGP on CF-PWV ( $b_1$ )	0.20	0.012 to 0.398

**INCREASED DP-UC-MGP, A MARKER OF VITAMIN K DEFICIENCY, IS STRONGLY ASSOCIATED WITH SARCOPENIA**

**Authors:** Bilal Ansari MD<sup>1</sup>; Swetha Gaddam MD<sup>1,3</sup>; Jessica Kim BSc<sup>1,2</sup>; Sowmya Gaddam MD<sup>2</sup>; Arpita Suri MD<sup>2</sup>; Dheera Tamvada MD<sup>2</sup>; Jagan Vanjarapu MD<sup>2</sup>; Garrett H. Oldland MD<sup>1,2,3</sup>; Rachana Miller MD<sup>2,3</sup>; Haideliza Soto-Calderon BSc<sup>2,3</sup>; Jonathan Lee<sup>1,2</sup>; Scott Akers MD, PhD<sup>3</sup>; Julio A. Chirinos MD, PhD<sup>1,2,3</sup>.

<sup>1</sup>Hospital of the University of Pennsylvania, Philadelphia, PA, USA

<sup>2</sup>University of Pennsylvania Perelman School of Medicine, Philadelphia, PA, USA

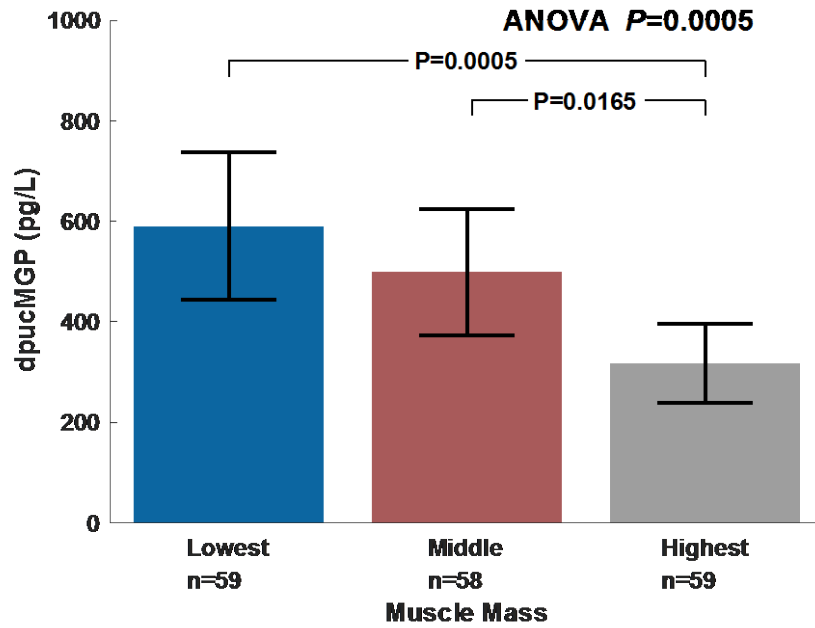
<sup>3</sup>Corporal Michael J. Crescenz VAMC, Philadelphia, PA, USA

**Objective:** In addition to regulating vascular calcification by control of the matrix-Gla protein (MGP) pathway, vitamin K2 has recently been shown to be an essential factor for mitochondrial function. Mitochondrial dysfunction and sarcopenia related to aging and other processes has been implicated as a key mediator of metabolic abnormalities, frailty, and cardiovascular disease. However, whether subclinical vitamin K2 deficiency is associated with sarcopenia in adults is unknown. We aimed to determine the relationship between dp-uc-MGP (inactive MGP) levels and sarcopenia.

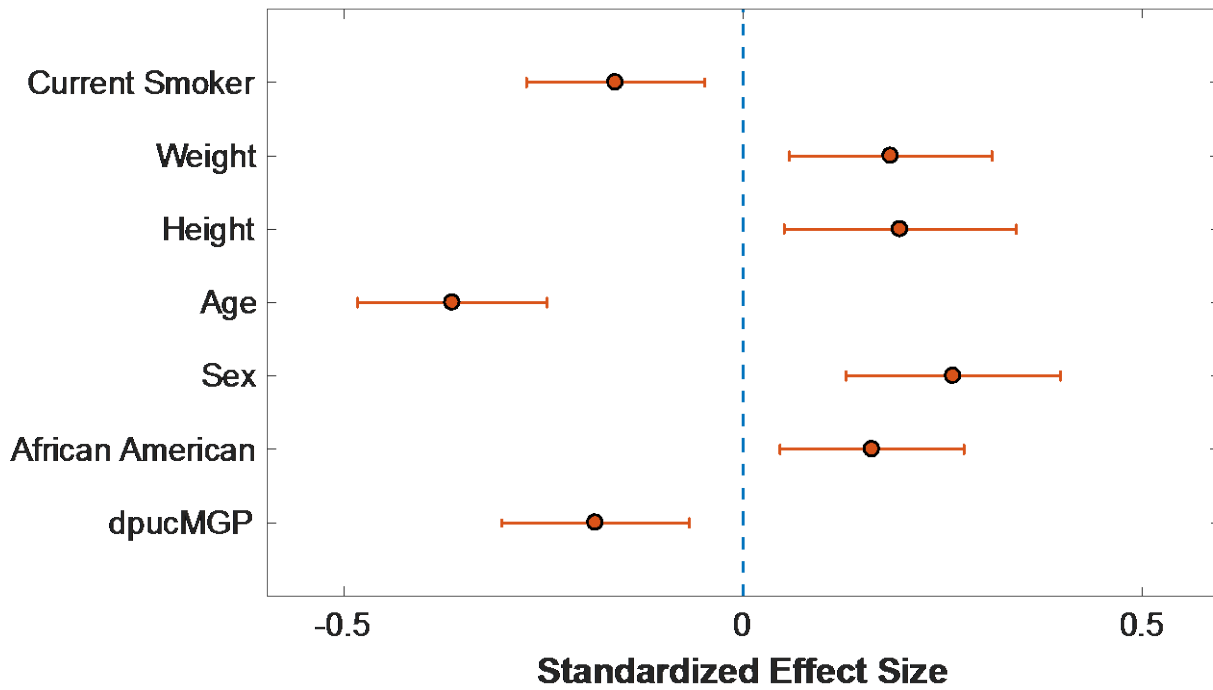
**Methods:** We studied 176 adults without heart failure. Cardiac MRI was used to estimate axial muscle mass from a stack of MRI SSFP thoracic axial images. Upper abdominal subcutaneous and visceral fat thickness was measured with SSFP MRI. Dp-uc-MGP levels were measured with ELISA (VitaK; The Netherlands).

**Results:** Dp-uc MGP levels were progressively greater as muscle mass decreased (395, 624 and 737 pmol/L in the lowest, middle and highest tertile of muscle mass, respectively; ANOVA  $P=0.0005$ ; Figure 1). Dp-uc MGP was an independent predictor of muscle mass in a model adjusting for multiple potential confounders. Stepwise regression identified age ( $\beta=-0.36$ ;  $P<0.0001$ ), dp-ucMGP ( $\beta=-0.18$ ;  $P=0.002$ ) and current smoking ( $\beta=-0.16$ ;  $P=0.009$ ) as independent predictors of lower axial muscle mass, whereas male sex ( $\beta=0.26$ ;  $P=0.0002$ ), body height ( $\beta=0.20$ ;  $P=0.008$ ) and weight ( $\beta=0.18$ ;  $P=0.004$ ) and African American ethnicity as independent predictors of greater axial muscle mass. These factors accounted for 48% of the variability in axial muscle mass (Figure 2). Dp-uc-MGP was not associated with subcutaneous or visceral fat thickness.

**Conclusions:** Sarcopenia is associated with increased dp-uc MGP (inactive MGP), a vitamin-K dependent inhibitor of vascular calcification and factor in mitochondrial function. Vitamin K2 supplementation may represent a potential therapeutic strategy to reduce sarcopenia.



Predictors of Axial Muscle Mass;  $R^2=0.48$ ;  $P<0.00001$



**ARGININE-VASOPRESSIN, ATRIAL NATRIURETIC PEPTIDE AND CARDIAC REMODELING IN HEART FAILURE WITH PRESERVED AND REDUCED EJECTION FRACTION**

**Authors:** Bilal Ansari, MD<sup>3</sup>; Mayank Sardana MD<sup>4</sup>; Garrett H. Oldland, MD<sup>1,2</sup>; Jonathan Lee<sup>2,3</sup>; Anila Hussain, MD<sup>3</sup>; Anique Mustafa, MD<sup>3</sup>; Scott R. Akers, MD, PhD<sup>1</sup>; Wen Wei, PhD<sup>4</sup>; Edward G. Lakatta, MD<sup>4</sup>; Olga V. Fedorova, PhD<sup>4</sup>; Julio A. Chirinos, MD, PhD<sup>1,2,3</sup>;

<sup>1</sup> Corporal Michael J. Crescenz VAMC, Philadelphia, PA, USA.

<sup>2</sup> University of Pennsylvania Perelman School of Medicine

<sup>3</sup> Hospital of the University of Pennsylvania, Philadelphia PA, USA

<sup>4</sup> Cardiology Division, Department of Medicine, University of Massachusetts Medical School, Worcester, MA, USA

<sup>5</sup> National Institute of Aging, Baltimore, MD, USA.

**Objectives:** Arginine-vasopressin (AVP) exerts multiple biologic effects via myocardial, vascular and renal receptors. The AVP pathway has been extensively studied in heart failure with reduced ejection fraction (HFrEF) but less is known about AVP in HF with preserved EF (HFpEF). Furthermore, atrial natriuretic peptide (ANP), is a well-known inhibitor of AVP secretion, but the relationship between AVP and ANP in heart failure is unknown.

**Methods:** We studied 28 subjects with HFpEF, 25 subjects with HFrEF, and 71 control subjects without heart failure. LV mass and LA volume were measured with steady-state free precession cardiac magnetic resonance imaging. AVP and ANP levels were measured with enzyme-linked immunosorbent assays (ELISA).

**Results:** AVP levels were significantly greater in HFpEF (0.96 pg/ml; 95%CI=0.83-1.1 pg/ml) compared to subjects without HF (0.69 pg/ml; 95%CI=0.6-0.77 pg/ml;  $P=0.0002$ ). HFpEF (but not HFrEF) was a significant predictor of higher AVP after adjustment for potential confounders. AVP levels were independently associated with a greater LA volume but also paradoxically, with lower ANP levels. Key independent correlates of higher AVP were the presence of HFpEF ( $\beta=0.30$ ; 95%CI=0.06 to 0.54;  $P=0.016$ ) and the ANP/LA volume ratio ( $\beta=-0.25$ ; 95%CI=-0.44 to -0.06;  $P=0.012$ ). AVP levels were independently associated with LV mass ( $\beta=0.26$ ; 95%CI=0.09-0.43;  $P=0.003$ ) and with an increased risk of death or HF admissions during follow-up (HR=1.61; 95%CI=1.13-2.29;  $P=0.008$ ).

**Conclusions:** AVP is increased in HFpEF and is associated with LV hypertrophy, consistent with the known biologic effects of AVP, and poor outcomes. Higher AVP is associated with the combination of greater LA enlargement with paradoxically low levels of ANP (a known suppressor of AVP release), suggesting that a relative ANP deficiency in the setting of increased LV filling pressures may contribute to AVP excess. The effects of therapeutic interventions that increase natriuretic peptide levels on AVP release and its downstream effects require further study.

Figure 2

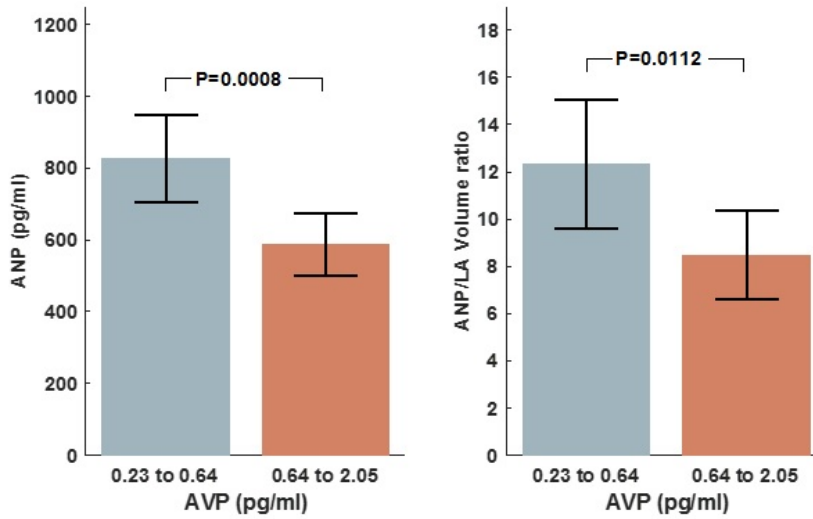
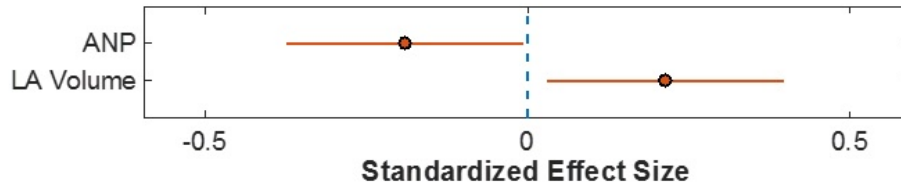
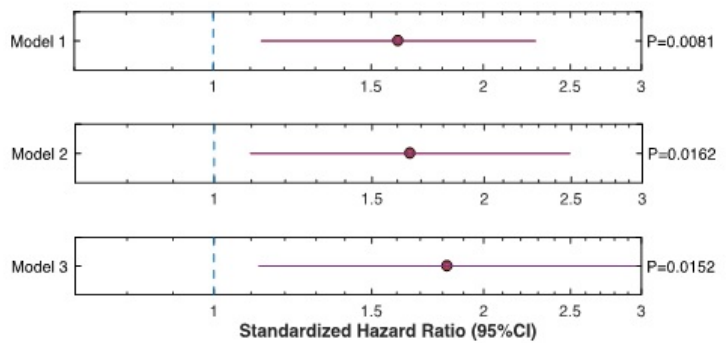
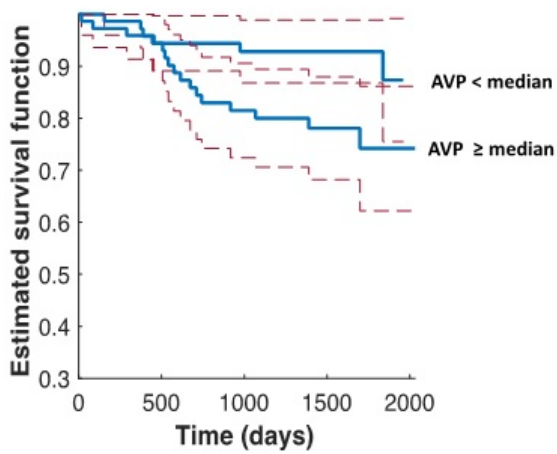


Figure 4



**POTENTIAL MICROVASCULAR COMPENSATION FOR THE REDUCTION IN ENDOTHELIAL FUNCTION DURING ACUTE INFLAMMATION: PRELIMINARY RESULTS**

ELIZABETH C. SCHROEDER, Thessa I.M. Hilgenkamp, Tracy Baynard, Bo Fernhall

UNIVERSITY OF ILLINOIS AT CHICAGO, CHICAGO, IL

Acute inflammation is associated with increased risk for cardiovascular events and leads to reductions in conduit artery (flow-mediated dilation, FMD) and resistance vessel endothelial function. Whether this dysfunction during acute inflammation is further transmitted down the arterial tree to the microvasculature, inhibiting its ability to dilate or be recruited in response to a hypoxic stimulus, has yet to be investigated. Microvascular function and reactivity can be non-invasively measured using near-infrared spectroscopy (NIRS) during and following an occlusive stimulus. **Purpose:** To investigate whether acute inflammation also impairs microvascular function in young, healthy adults. **Methods:** The typhoid vaccine was used to induce acute systemic inflammation in 10 young, healthy adults (6 male,  $27.6 \pm 3.0$  yrs;  $22.3 \pm 2.6$  kg/m<sup>2</sup>). Blood pressure, FMD of the brachial artery, and NIRS of the forearm flexor muscles were measured both at baseline and 24-h following the vaccination. NIRS was analyzed during a 5-min arterial occlusion to obtain markers of microvascular function and reactivity from the tissue saturation index (TSI): occlusion slope (muscle oxidative capacity); and reperfusion slope, reperfusion magnitude, and peak hyperemic response (microvascular reactivity). **Results:** Mean arterial pressure did not change during the inflammatory episode ( $90 \pm 7$  mmHg to  $90 \pm 7$  mmHg,  $p=0.91$ ) and FMD was significantly reduced at 24 h ( $5.8 \pm 2.7\%$  to  $4.0 \pm 1.4\%$ ,  $p=0.04$ ). The TSI peak hyperemic response was greater at 24-hours compared to baseline ( $16 \pm 6\%$  to  $19 \pm 4\%$ ,  $p=0.03$ ). There was no change noted in the TSI occlusion slope, reperfusion slope, time to peak, or reperfusion magnitude ( $p>0.05$ ). **Conclusion:** Vaccination-induced acute inflammation increased microvascular reactivity, possibly compensating for the vaccination induced reductions in endothelial function in the conduit arteries. Further investigation with a larger sample size is necessary to confirm these results.



**SLEEP QUALITY IS ASSOCIATED WITH CEREBROVASCULAR FUNCTION IN INDIVIDUALS WITH MULTIPLE SCLEROSIS**

GEORGIOS GRIGORIADIS<sup>1</sup>, Alexander J. Rosenberg<sup>1</sup>, Sang Ouk Wee<sup>1, 2</sup>, Elizabeth C. Schroeder<sup>1</sup>, Garrett Griffith<sup>1</sup>, Tracy Baynard<sup>1</sup>. <sup>1</sup>University of Illinois at Chicago, Chicago, IL. <sup>2</sup>California State University, San Bernardino, San Bernardino, CA.

**INTRODUCTION:** Individuals with multiple sclerosis (MS) exhibit impaired cerebrovascular function and have poor sleep quality. In the general population, poor sleep quality is an important contributor to cerebrovascular dysfunction and related to cardiovascular disease (CVD). Improving sleep quality might have a beneficial effect in prevention of CVD; however, the relationship between sleep quality and cerebrovascular function in MS has not been examined.

**PURPOSE:** To examine the effect of sleep quality on cerebrovascular function in individuals with MS.

**METHODS:** Ten MS individuals with good and six with poor sleep quality participated in this study. Sleep quality was measured with the Pittsburgh Sleep Quality Index. Individuals were categorized as having poor sleep quality (> 5) or good sleep quality (≤ 5). Cerebrovascular function was assessed via transcranial Doppler ultrasound with the following hemodynamic outcomes: mean middle cerebral artery velocity (mMCAv), pulsatility index (PI), and resistance index (RI). An automated blood pressure cuff was used to measure baseline blood pressure (systolic, diastolic, mean (SBP, DBP, MAP)) and heart rate in a seated position. End-tidal CO<sub>2</sub> (EtCO<sub>2</sub>) was measured by gas collection.

**RESULTS:** Those with poor sleep quality had greater PI and RI, and lower mMCAv compared to those with good sleep quality (p<0.05, see table). No group differences were seen for weight, height, BMI, CO<sub>2</sub>, or hemodynamic variables.

**CONCLUSION:** Our results suggest that individuals with MS with poor sleep quality have worsened indicators of cerebrovascular function. Therefore, sleep quality may be related to the elevated CVD risk in individual with MS, and it should be assessed in future studies evaluating cerebrovascular function in MS, including intervention studies.

	<b>Good Sleep Quality (n=10)</b>	<b>Poor Sleep Quality (n=6)</b>
<b>Sex (male/female)</b>	(2/8)	(2/4)
<b>Age (yrs)</b>	41 ± 8	44 ± 8
<b>BMI (kg/m<sup>2</sup>)</b>	29.6 ± 7.6	24.0 ± 3.6
<b>Height (cm)</b>	170 ± 11	168 ± 4
<b>Weight (kg)</b>	86 ± 25	68 ± 12
<b>SBP (mmHg)</b>	117 ± 13	113 ± 16
<b>DBP (mmHg)</b>	72 ± 11	64 ± 9
<b>MAP (mmHg)</b>	87 ± 11	80 ± 10
<b>Heart Rate (bpm)</b>	65 ± 7	59 ± 9
<b>mMCAv (cm/s)*</b>	<b>64 ± 10</b>	<b>51 ± 10</b>
<b>PI*</b>	<b>0.86 ± 0.10</b>	<b>1.03 ± 0.07</b>
<b>RI*</b>	<b>0.56 ± 0.04</b>	<b>0.63 ± 0.03</b>
<b>EtCO<sub>2</sub> (mmHg)</b>	4.6 ± 0.3	5.0 ± 0.5
All data are mean ± SD, *p<0.05		

**PERIPHERAL BLOOD FLOW REGULATION IN RESPONSE TO SYMPATHETIC STIMULATION IN INDIVIDUALS WITH DOWN SYNDROME**

THESSA I.M. HILGENKAMP<sup>1</sup>, Sangouk Wee<sup>2</sup>, Tracy Baynard<sup>1</sup>, Bo Fernhall<sup>1</sup>

<sup>1</sup> UNIVERSITY OF ILLINOIS AT CHICAGO, CHICAGO, IL

<sup>2</sup> California State University San Bernardino, San Bernardino, CA

Individuals with Down syndrome (DS) experience autonomic dysfunction, manifested as reduced sympathetic and parasympathetic control. This results in alterations in resting heart rate (HR) and blood pressure (BP) and attenuated responses to different sympathoexcitatory stimuli. It is unknown to what extent this impacts the regulation of peripheral blood flow in response to sympathetic stimuli, which is an important prerequisite to be able to exercise and perform work.

**Purpose:** To investigate differences in peripheral blood flow regulation in response to lower body negative pressure (LBNP) between individuals with and without DS.

**Methods:** Participants (n=10 males with DS and n=11 male controls, mean age 23.7 years ± 3.2) underwent 5 min of LBNP stimulations (-20 mmHg), after resting supine for 10 min. One minute steady state blood pressure and blood flow at baseline, LBNP, and 5 min recovery were obtained for analysis. Mean flow velocity and arterial diameters were recorded with ultrasonography; forerarm blood flow (FBF), shear rate and forearm vascular conductance (FVC) were calculated using brachial blood pressure measured right before ultrasound recordings.

**Results:** Participants with DS responded differently (consistent with reduced vasoconstrictive control) to the LBNP stimulus (significant ConditionxGroup interaction effect) for mean velocity (p=0.003), FBF (p=0.008), shear rate (p=0.004) and FVC (p=0.017), compared to participants without DS (see table). **Conclusion:** Young males with DS exhibit reduced peripheral blood flow regulation of blood flow in response to LBNP compared to controls, indicating a blunted sympathetic control of blood flow. Further research is necessary to explore the impact of these findings on exercise and work capacity.

	Baseline		LBNP		Recovery	
	DS	Control	DS	Control	DS	Control
MAP	90±13	88±10	85±12	87±10	92±11	85±10
Diameter (cm)	0.36±0.05	0.43±0.05	0.37±0.05	0.43±0.07	0.37±0.04	0.42±0.06
Mean velocity (cm/sec)	13±7	21±10	15±7	17±5	16±9	16±8
FBF (ml/min)	91±64	176±80	104±67	146±47	114±91	138±74
Shear Rate (sec <sup>-1</sup> )	20±12	35±16	23±12	29±8	25±17	28±14
FVC (ml/min/100 mmHg)	101±67	203±95	124±84	171±63	125±101	162±81

**HEMODYNAMIC RESPONSE FOLLOWING ACUTE MODERATE INTENSITY AEROBIC EXERCISE IN INDIVIDUALS WITH DOWN SYNDROME**

**Author Block** SANG OUK WEE<sup>1</sup>, Elizabeth C. Schroeder<sup>2</sup>, Georgios Grigoriadis<sup>2</sup>, Thessa Hilgenkamp<sup>2</sup>, Tracy Baynard<sup>2</sup>, Bo Fernhall<sup>2</sup>, 1 CALIFORNIA STATE UNIVERSITY SAN BERNARDINO, 2 University of Illinois at Chicago, Chicago, IL.

**ABSTRACT:** Individuals with Down syndrome (DS) exhibit altered autonomic function with suppressed sympathetic modulation to sympathoexcitatory tasks. However, it is unknown if individuals with DS have a normal central hemodynamic response following an acute bout of moderate intensity exercise. **OBJECTIVES:** To investigate the hemodynamic response, using wave separation analysis, following an acute bout of moderate intensity treadmill exercise in individuals with DS.

**METHODS:** A total of 38 healthy, young (DS: 25 yr  $\pm$  0.9, Control: 25 yr  $\pm$  0.8) individuals with and without DS adults (19 DS) participated in the study. Participants performed 20 min of treadmill walking at 60% of peak aerobic capacity ( $VO_{2peak}$ ). Brachial blood pressure and pulse waves were measured using a validated ambulatory blood pressure monitor (Mobil-O-Graph) and central blood pressure was generated using a validated general transfer function. Wave separation analyses were then conducted at baseline, immediate post, and 30 min post exercise.

**RESULTS:** See table. Neither group differences nor interactions were observed in brachial hemodynamic, wave separation variables, or PWV following exercise ( $p>0.05$ ). However, the following variables showed significant post-exercise changes (time effect of exercise): bSBP, bDBP, MAP, and reflected pressure. Interestingly, both groups exhibited a lack of changes in cSBP following exercise, whereas bSBP increased.

**CONCLUSIONS:** Our results suggest that young, healthy individuals with DS exhibited similar hemodynamic responses as controls following acute submaximal exercise. Interestingly, the changes in cSBP were muted compared to bSBP following exercise for both groups, suggesting that central BP was less affected by exercise. This disparate central vs brachial BP response needs further investigation.

	DS (N = 19)			Control (N = 19)		
	Baseline	Immediate post exercise	30 minute post exercise	Baseline	Immediate post exercise	30 minute post exercise
<b>bSBP (mmHg)*</b>	122 ± 14	126 ± 11	122 ± 13	120 ± 10	130 ± 12	121 ± 11
<b>bDBP (mmHg)*</b>	69 ± 9	75 ± 8	71 ± 9	72 ± 6	81 ± 8	74 ± 9
<b>MAP (mmHg)*</b>	93 ± 10	98 ± 8	94 ± 9	94 ± 7	103 ± 7	96 ± 8
<b>cSBP (mmHg)</b>	109 ± 12	110 ± 12	111 ± 14	111 ± 10	113 ± 9	110 ± 10
<b>Forward wave pressure(mmHg)</b>	24.4 ± 4.0	23.6 ± 5.6	24.3 ± 5.5	24.2 ± 4.3	22.7 ± 6.1	22.6 ± 5.8
<b>Reflected wave pressure(mmHg)*</b>	15.1 ± 3.1	13.4 ± 4.5	14.7 ± 5.8	15.8 ± 3.7	13.9 ± 5.0	13.26 ± 4.3
<b>PWV (m/s)</b>	5.2 ± 0.4	5.3 ± 0.5	5.2 ± 0.5	5.1 ± 0.4	5.4 ± 0.4	5.1 ± 0.4

\*Significant difference at immediate post time point as a group.

† Significant difference from other time points.

**THE IMPACT OF ACUTE CENTRAL HYPOVOLEMIA ON CEREBRAL HEMODYNAMICS: DOES SEX MATTER?**

ROSENBERG, A.J., Kay, V.L., and Rickards, C.A.

DEPARTMENT OF PHYSIOLOGY AND ANATOMY, UNIVERSITY OF NORTH TEXAS HEALTH SCIENCE CENTER

Trauma-induced hemorrhage is a leading cause of disability and death due, in part, to impaired perfusion and oxygenation of the brain. While cerebrovascular function differs between males and females, it is not clear if cerebrovascular responses to blood loss are differentiated based on sex. **Objective:** We examined the effect of sex on cerebral blood velocity and oxygenation responses following simulated hemorrhage induced via application of lower body negative pressure (LBNP) to presyncope. **Methods:** Healthy males (n=11, 25±1 y) and females (early follicular phase of menstrual cycle) (n=7, 27±1 y) participated in a LBNP ramp protocol (-3 mmHg/min) until presyncope. Middle cerebral artery velocity (MCAv), cerebral oxygen saturation (ScO<sub>2</sub>), end-tidal CO<sub>2</sub> (etCO<sub>2</sub>), heart rate (HR), arterial pressure (MAP) and stroke volume (SV) were measured continuously. **Results:** Baseline MCAv was higher in females vs. males (70.3±5.8 vs. 57.8 ± 2.1 cm/s, p=0.03), despite a lower etCO<sub>2</sub> (37.9±0.9 vs. 44.4±1.5 mmHg, p=0.02). While LBNP tolerance was higher for males compared with females (1675.5±123.1 vs. 1315.9±140.0 s; p=0.08), the absolute and relative (% change) increases in HR and decreases in MCAv, MAP, SV, and etCO<sub>2</sub> were similar between males and females at presyncope (p≥0.11). Males exhibited a lower rate of change in MCAv over LBNP time (-0.56±0.10 vs. -0.92±0.09 cm/s/min, p=0.03) and a greater maximum decrease in ScO<sub>2</sub> (-7.6±1.3 vs -5.3±0.9 %, p=0.08) when compared with females, most likely due to the higher tolerance in this group. **Conclusion:** Simulated hemorrhage reduced cerebral blood velocity at pre-syncope in both sexes. However, males exhibited a reduced rate of change in cerebral blood velocity to simulated hemorrhage to pre-syncope. These findings suggest that sex may influence the cerebral hemodynamic responses to simulated blood loss in young healthy adults.

**PREVALENCE AND TRENDS IN SUBTYPES OF STROKE AMONG PREGNANCY-RELATED HOSPITALIZATIONS IN THE UNITED STATES: 2002-2014****MULUBRHAN F. MOGOS, PHD, MSC**  
**ASSISTANT PROFESSOR**

1. Jason L. Salemi, PhD, MPH

Department of Family and Community Medicine, Baylor College of Medicine, Houston, TX

2. Shane Phillips, PhD

Department of Physical Therapy, University of Illinois at Chicago, Chicago, IL

3. Mariann R. Piano, PhD, FAAN, FAHA

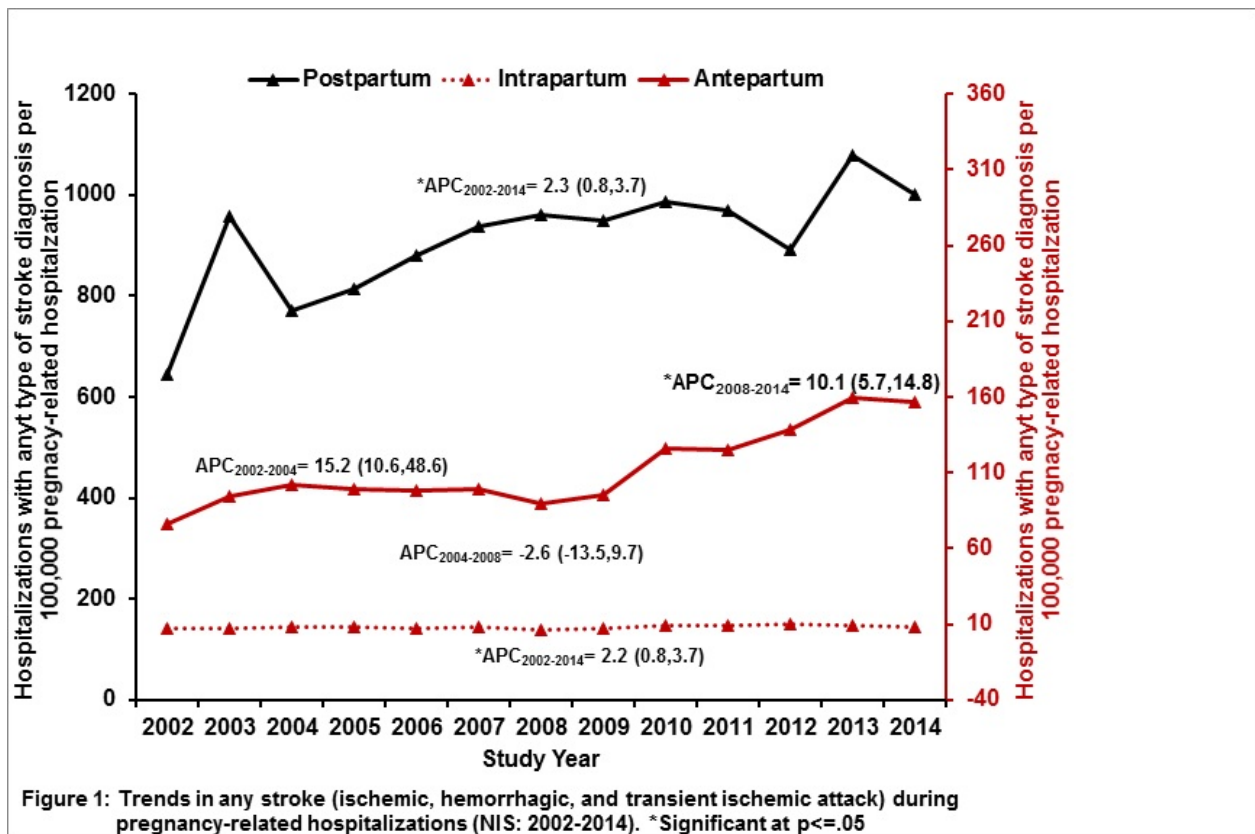
School of Nursing, Vanderbilt University; Nashville, TN

**Background:** Although the risk of stroke among young women is relatively rare, pregnant women experience an increased risk of stroke when compared to their non-pregnant counterparts. However, current data on the timing of occurrence (Antepartum, Intrapartum, and Postpartum) by sub-types of stroke is lacking in the United States.

**Method:** We used the Nationwide Inpatient Sample (NIS) dataset (2002-2014) to examine the prevalence and trends of sub-types of stroke following pregnancy-related hospitalization among adults 15-49 years of age. International Classification of Diseases, Ninth Revision, Clinical Modifications (ICD-9-CM) codes were used to define cases and behavioral characteristics. Rates of sub-types of stroke by timing of hospitalization were calculated. Joinpoint regression was used to describe temporal trends in sub-types of stroke, describing each trend using the estimated annual percent change (APC).

**Results:** Among over 58 million estimated pregnancy-related inpatient hospitalizations from 2002-2014, a total of 18,341 (0.03%) had stroke. When stratified by timing of occurrence, 0.01%, 0.91%, and 0.11% were impacted during intrapartum, postpartum, and antepartum. During postpartum and intrapartum hospitalizations, overall rates of stroke increased significantly by 2.3% and 2.2% respectively (2002-2014). During antepartum hospitalization the rate of overall stroke increased by 10.1% annually from 2008-2014 (Figure 1). Trend analyses shows a 3.46% increase in transient ischemic stroke during antepartum; 3.70%, 3.82%, and 7.98% increase in ischemic stroke during intrapartum, postpartum and antepartum hospitalizations respectively. We also found a 2.26% and 4.02% annual increase in hemorrhagic stroke during the postpartum and antepartum periods respectively.

**Conclusion:** The overall rate of stroke during pregnancy-related hospitalization is 0.03% and mostly occur among women hospitalized during the postpartum period. However, the overall rate of stroke has shown much higher (10.1%) increment among antepartum hospitalizations. Data from this study may guide future mechanistic studies examining the relationship between arterial stiffness and sub-types of stroke during pregnancy continuum.



**BLOOD PRESSURE AND BODY MASS INDEX IN WOMEN AND MEN POST-BARIATRIC SURGERY**

Awani Gohkale, Chandra Hassan, Mario Masrur, Richard Severin, Gabriela Aguiluz-Cornejo, Antonio Gangemi, Lisa Sanchez-Johnsen, Shane Philips

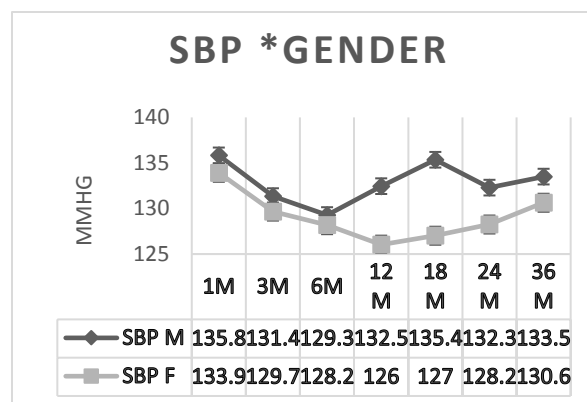
**Introduction** Approximately 40.4% of women and 37.7% of men are obese.<sup>(2)</sup> Bariatric surgery is effective in promoting weight loss and improving or resolving medical comorbidities associated with obesity such as hypertension and diabetes<sup>(4,5,6)</sup> Moreover, women have a greater weight loss than men post-bariatric surgery. However, few studies have examined differences in blood pressure between women and men post-bariatric surgery. As such, the aim of this study was to examine differences in blood pressure among women and men who underwent bariatric surgery

**Methods:** This is a retrospective review of a prospectively maintained database from 988 patients(84.42%women(n=844) who underwent bariatric surgery from January 2008 to June 2015. The following variables were extracted from the electronic medical records: sex, age, and BMI and systolic (SBP) and diastolic (DBP) blood pressure prior to surgery and at 3, 6, 12, 18, 24, 36 months following surgery at the University of Illinois Hospital and Health Sciences System.

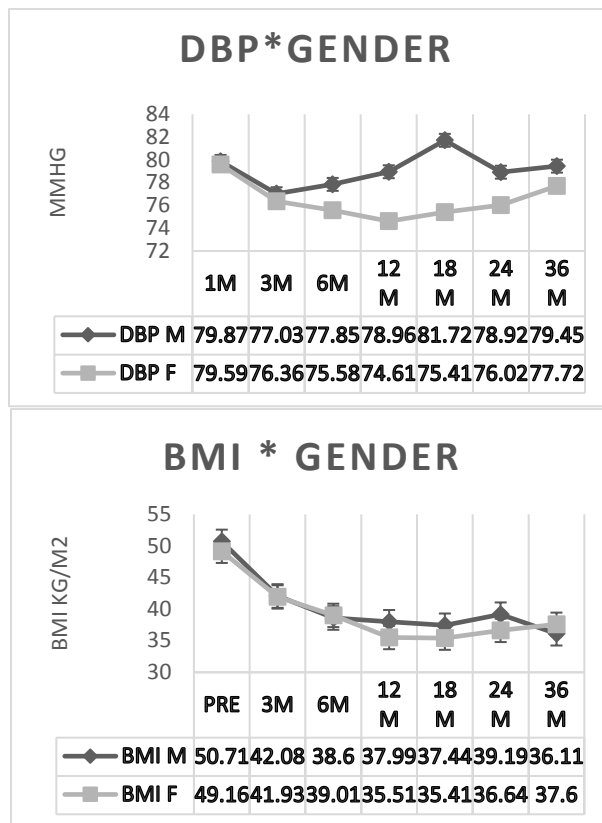
**Results:** Overall systolic and diastolic blood pressure decreased significantly at 12 months post-surgery. Systolic blood pressure was reduced significantly at 3,6,18 and 24 months post-surgery (p<.05). There was a significant reduction in SBP and DBP in women at 12 and 18 months compared to men. There was a significant decrease in BMI from pre-surgery to post-surgery at all time points (p<.05). However, the reduction in BMI when compared to 6 months post-surgery was significant only at 12 months (p<.05).

**Conclusions:** There is significant reduction in blood pressure up to 12 months following bariatric surgery. The blood pressure lowering effect following bariatric surgery plateaus after 18 months. There appears to be gender differences in blood pressure reduction following bariatric surgery whereby the magnitude of blood pressure lowering is attenuated in men compared to women. Further research is needed to examine predictors of blood pressure post-bariatric surgery in women and men.

Figures







References:

1. Adult Obesity Facts | Overweight & Obesity | CDC. (2017). Cdc.gov. Retrieved 21 November 2017, from <https://www.cdc.gov/obesity/data/adult.html>
2. Ogden CL, Carroll MD, Fryar CD, Flegal KM. Prevalence of obesity among adults and youth: United States, 2011–2014. NCHS data brief, no 219. Hyattsville, MD: National Center for Health Statistics. 2015.
3. Information, H., Statistics, H., Statistics, O., Statistics, O., Center, T., & Health, N. (2017). Overweight & Obesity Statistics | NIDDK. National Institute of Diabetes and Digestive and Kidney Diseases. Retrieved 21 November 2017
4. Re, R. (2009). Obesity-Related Hypertension. The Ochsner Journal, 9(3), 133-136. <http://dx.doi.org/10.1043 / 1524-5012-9.3.133>
5. Sweeting, A., & Caterson, I. (2017). Approaches to obesity management. Internal Medicine Journal, 47(7), 734-739. <http://dx.doi.org/10.1111/imj.13474>
6. Blum, A., Tamir, S., Hazzan, D., Podvitzky, O., Sirchan, R., & Keinan-Boker, L. et al. (2013). Gender effect on vascular responsiveness after bariatric surgery. Open Medicine, 8(5). <http://dx.doi.org/10.2478/s11536-012-0138-1>
7. Batsis, J., Miranda, W., Prasad, C., Collazo-Clavell, M., Sarr, M., Somers, V., & Lopez-Jimenez, F. (2015). Effect of bariatric surgery on cardiometabolic risk in elderly patients: A population-based study. Geriatrics & Gerontology International, 16(5), 618-624. <http://dx.doi.org/10.1111/ggi.12527>

**RELATION BETWEEN FUNCTIONAL CAPACITY AND LEVEL OF PHYSICAL ACTIVITY IN COPD AND OSA OVERLAP SYNDROME: A PILOT STUDY.**

ANDRÉA LÚCIA GONÇALVES DA SILVA, Kamila Mohammad Kamal Mansour<sup>1</sup>, Elisabete Antunes San Martin<sup>1</sup>, Cássia da Luz Goulart<sup>2</sup>, Paloma de Borba Schneiders<sup>1</sup>, Renata Trimer<sup>3</sup>, Tânia Cristina Malezan Fleig<sup>3</sup>, <sup>1</sup>Fellowship in Scientific Initiation by University of Santa Cruz do Sul (UNISC), Course of Physiotherapy, Santa Cruz do Sul/RS, Brazil.

**Objective:** To identify the relation between functional capacity and physical activity level in patients with COPD-OSA overlap syndrome.

**Methods:** Cross-section study that evaluated 12 COPD-OSA patients on a convenience non-probability sample, conducted in Santa Cruz Hospital's Pulmonary Rehabilitation Program. Patients underwent a clinical evaluating to record age, sex, and anthropometric measurements; pulmonary function test; home sleep testing with ApneaLink™ Plus for detecting apnea-hypopnea index (AHI); 6-Minute Walk Test (6MWT) and Duke Activity Status Index for functional capacity, afterwards we obtained the BODE index and applied Saint George's Respiratory Questionnaire (SGRQ). The International Physical Activity Questionnaire was used to measure individual's physical activity level. Data was tested for normality where  $p < 0.05$  was considered significant.

**Results:** Our sample was stratified in Minimally active group (MAG,  $n=7$ ) and Active group (AG,  $n=5$ ), AG had a higher AHI [42 (22-56)] vs MAG [24 (12-43)], but we did not find significant differences between the groups. We noticed a homogeneity between groups regarding the clinical characteristics. AG presented a greater estimated  $\text{VO}_2$  ( $p=0.04$ ), greater distance walked in the 6MWT ( $p=0.03$ ) and higher predicted (%)6MWT ( $p=0.001$ ) when compared to MAG. Overall SGRQ was significantly better in individuals in AG ( $p=0.03$ ). BODE index AG [1 (1-3,5)] vs MAG [4 (1-4)]. Significant Associations were found in the MAG between AHI and clinical characteristics [BMI ( $r: 0.818$   $p=0.025$ ), abdominal circumference ( $r: 0.767$   $p=0.044$ ) and waist circumference ( $r: 0.846$   $p=0.016$ )]. In AG, estimated  $\text{VO}_2$  correlated significantly with sleeping parameters: baseline peripheral oxygen saturation ( $\text{SpO}_2$ ) ( $r: 0.949$   $p=0.014$ ) and mean  $\text{SpO}_2$  ( $r: 0.949$   $p=0.014$ ).

**Conclusion:** The preliminary results suggest that a physically actives individuals with COPD-OSA overlap syndrome have better functional capacity, quality of life and survival prediction than minimally active individuals with COPD-OSA overlap syndrome.

**RELATIONSHIP BETWEEN CARDIAC AUTONOMIC MODULATION AND CARDIORESPIRATORY RESPONSE DURING THE SIX-MINUTE WALK TEST IN HEART FAILURE PATIENTS.**

ANDRÉA LÚCIA GONÇALVES DA SILVA, CÁSSIA DA LUZ GOULART<sup>1</sup>, RENAN SHIDA MARINHO<sup>1</sup>, POLLIANA BATISTA DOS SANTOS<sup>1</sup>, PATRÍCIA FARIA CAMARGO<sup>1</sup>, FLÁVIA ROSSI CARUSO<sup>1</sup>, RAMONA CABIDDU<sup>1</sup>, CLAUDIO R. OLIVEIRA<sup>1</sup>, LARISSA DELGADO ANDRÉ<sup>1</sup>, MELIZA GOI ROSCANI<sup>1</sup>, AUDREY BORGHINI-SILVA<sup>1</sup>, 1CARDIOPULMONARY PHYSIOTHERAPY LABORATORY - LACAP, FEDERAL UNIVERSITY OF SÃO CARLOS (UFSCAR), SÃO CARLOS, SÃO PAULO, BRAZIL.

**Objective:** to evaluate the association between non-linear heart rate variability (HRV) indices and cardiorespiratory response during the six-minute walk test (6MWT) in heart failure (HF) patients.

**Methods:** 13 mild to moderate HF patients were submitted to the 6MWT; subjects were requested to walk at their own maximal pace, without running, along a 30-meter perimeter during 6 minutes. Minute ventilation ( $V_E$ ), carbon dioxide production ( $V_{CO_2}$ ) and oxygen consumption ( $V_{O_2}$ ) were measured breath by breath using a portable ergospirometer (Oxycon Mobile, Jaeger) during the 6MWT. The tachogram was recorded and analyzed and the most stable 300-sample portion was selected during the final minutes of the 6MWT. Non-linear HRV variables were calculated, including Approximate entropy (ApEn) and Sample entropy (SampEn). Pearson's correlation test was applied.

**Results:** Most patients were male (n=10, 77.0%); mean age=59.3±6.4 years; Left ventricular ejection fraction=41.5±4.4%; 6MWT distance: 465.4±58.6 m, peak  $V_E$ =37.2±17.0; peak  $V_{O_2}$ =10.9±4.2 mL.kg/min; peak  $V_{CO_2}$ =919.6±489.9 mL/min; ApEn: 0.9±0.2; SampEn: 1.3±0.5. Strong and moderate correlations were found between ApEn and:  $V_E$  (r= -0.789; p=0.001);  $V_{O_2}$  (r= -0.721; p=0.005);  $V_{CO_2}$  (r= -0.721; p=0.005); SampEn and:  $V_E$  (r= -0.609; p=0.027);  $V_{O_2}$  (r= -0.593 p=0.033)

**Conclusion:** HRV complexity influences the cardiorespiratory response during submaximal activities in HF patients. Funding: FAPESP: 2015/26501-1 and 2018/03233-0.

## INTEGRATIVE INSIGHT TO INTER-ARM DIFFERENCES IN PULSE WAVE VELOCITY

MICHAEL E. HOLMSTRUP<sup>1</sup>, Brock T. Jensen<sup>1</sup>

<sup>1</sup>DEPARTMENT OF EXERCISE AND REHABILITATIVE SCIENCES, SLIPPERY ROCK UNIVERSITY, SLIPPERY ROCK, PA

**Objectives:** Information from the autonomic nervous system (e.g. heart rate variability; HRV) and vasculature (e.g. arterial stiffness) can be integrated with blood pressure measurement to improve clinical decision making. Further, bilateral measurement is suggested as standard procedure for an initial blood pressure screening, as a large inter-arm difference (IAD) in systolic blood pressure at rest is linked to premature morbidity and mortality. Similarly, arterial stiffness has been shown to differ between arms, with a greater difference linked to increased cardiovascular disease (CVD) risk. To date, there have been no investigations to expand this bilateral perspective to measures of endothelial function which also reflects CVD. **Aim:** To quantify bilateral arterial stiffness and endothelial function contextualized by inter-arm difference and autonomic function. **Methods:** Demographic measures including body fat percentage, cholesterol/glucose levels, and HRV were acquired using standard procedures. Bilateral resting pulse wave velocity (PWV) and blood pressure were measured simultaneously and sequentially, respectively. Following a five-minute occlusion protocol, a second simultaneous measure was recorded and subtracted from PWV to reflect endothelial function (ENDO). Inter-arm difference for PWV and ENDO were calculated, and organized in tertiles (e.g. lowest, middle, highest). A one-way ANOVA was utilized to compare demographic, autonomic, and blood pressure variables to PWV and ENDO tertiles. **Results:** Age, lean body mass, and resting heart rate differed based on PWV. Interestingly, individuals with the smallest inter-arm difference in PWV demonstrated decreased sympathetic (i.e. low-frequency HRV) and increased parasympathetic (i.e. high-frequency HRV) drive. Age, lean body mass, and low-density lipoprotein cholesterol differed based on ENDO. Individuals with the smallest observed inter-arm difference in ENDO also demonstrated greater IAD. **Conclusion:** The present findings support the practice of bilateral measurement of arterial stiffness and endothelial function which may provide valuable information for clinical reasoning.

## Excess Weight Loss (%EWL), Body Mass Index (BMI) and Use of Hypertension Medication after Bariatric Surgery

Pedram Ahmadnezhad, Chandra Hassan, Mario Masrur, Richard Severin, Roberto Bustos, Dustin Baldwin, Antonio Gangemi, Lisa Sanchez-Johnsen, Shane Philips

**Objective:** Bariatric surgery is associated with a reduction in medical comorbidities such as hypertension and Type 2 Diabetes Mellitus.<sup>1,2,3,4</sup> However, there is a relative dearth of research examining the use of hypertension (HTN) medication post-bariatric surgery and its relationship to percent excess weight loss (%EWL) and BMI post-surgery. Therefore, the aim of this study was to examine %EWL and BMI at 6, 12 and 24 months post-bariatric surgery, and to examine whether HTN medication status was associated with %EWL and BMI post-surgery.

**Design:** This is a retrospective review of a prospectively maintained database from 987 patients who underwent bariatric surgery from January 2008 to June 2015. The following variables were extracted from the electronic medical records: sex, age, HTN medication status (decreased/ discontinued) and BMI and % EWL at pre-surgery, 3, 6, 9, 12, 18, and 24 months post-surgery at the University of Illinois Hospital and Health Sciences System. To account for missing data in certain analyses, the time points were collapsed to create new variables of 6, 12 and 24 months.

**Results:** Of the 987 patients, 85.5% (n= 844) were female. HTN medication reduction and discontinuation status were analyzed separately and compared to BMI or % EWL post-surgery. Percent EWL was significantly different at 6, 12, and 24 months, but did not have any associations or interactions with HTN medication. BMI was associated with HTN medication reduction (p< 0.001). There was a significant difference in BMI from pre-surgery to 6, 12, and 24 months post-surgery (p< 0.001).

**Conclusions:** BMI exhibited the largest reduction and %EWL exhibited the largest increase during the first 24 months post-surgery. BMI and %EWL were not associated with post-surgery medication discontinuation. However, higher BMI post-surgery was associated with a reduction in HTN medication. Further research is needed to examine predictors of HTN medication status post-surgery and its relationship to %EWL and BMI.

**Keywords:** Hypertension medication, Excess weight loss, Body Mass Index, Bariatric surgery

### References:

1. Sweeting, A., & Caterson, I. (2017). Approaches to obesity management. *Internal Medicine Journal*, 47(7), 734-739. <http://dx.doi.org/10.1111/imj.13474>
2. Panteliou, E., & Miras, A. (2017). What is the role of bariatric surgery in the management of obesity? *Climacteric*, 20(2), 97-102. doi:10.1080/13697137.2017.1262638

3. Wilhelm, S., Young, J., & Kale-Pradhan, P. (2014). Effect of Bariatric Surgery on Hypertension. *Annals of Pharmacotherapy*, 48(6), 674-682.

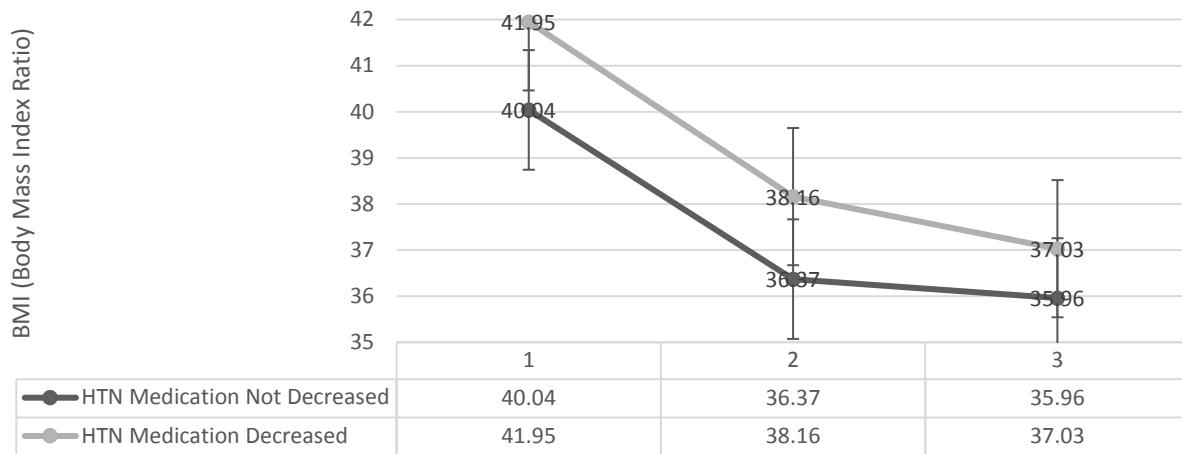
<http://dx.doi.org/10.1177/1060028014529260>

4. Buchwald, H., Avidor, Y., & Braunwald, E. (2005). Bariatric surgery. A systematic review and meta-analysis. *ACC Current Journal Review*, 14(1), 13.

<http://dx.doi.org/10.1016/j.accreview.2004.12.068>

**Graphs:**

Table 3: BMI at 6, 12, and 24 months

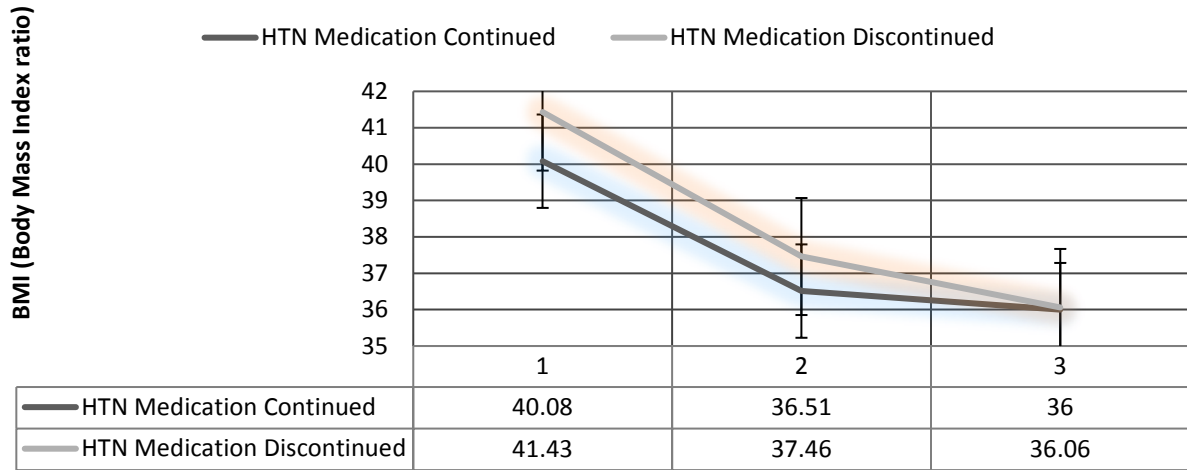


BMI at 6, 12, and 24 months in accociation with Medication Reduction Status

● HTN Medication Not Decreased      ● HTN Medication Decreased

Table 3: 1: 6 months BMI, 2: 12 months BMI, and 3: 24 months BMI

**Table 4: BMI at 6, 12, and 24 months**



BMI at; 1: 6 months post, 2: 12 months post, 3: 24 months post-surgery

Table 4: 1: 6 months BMI, 2: 12 months BMI, and 3: 24 months BMI