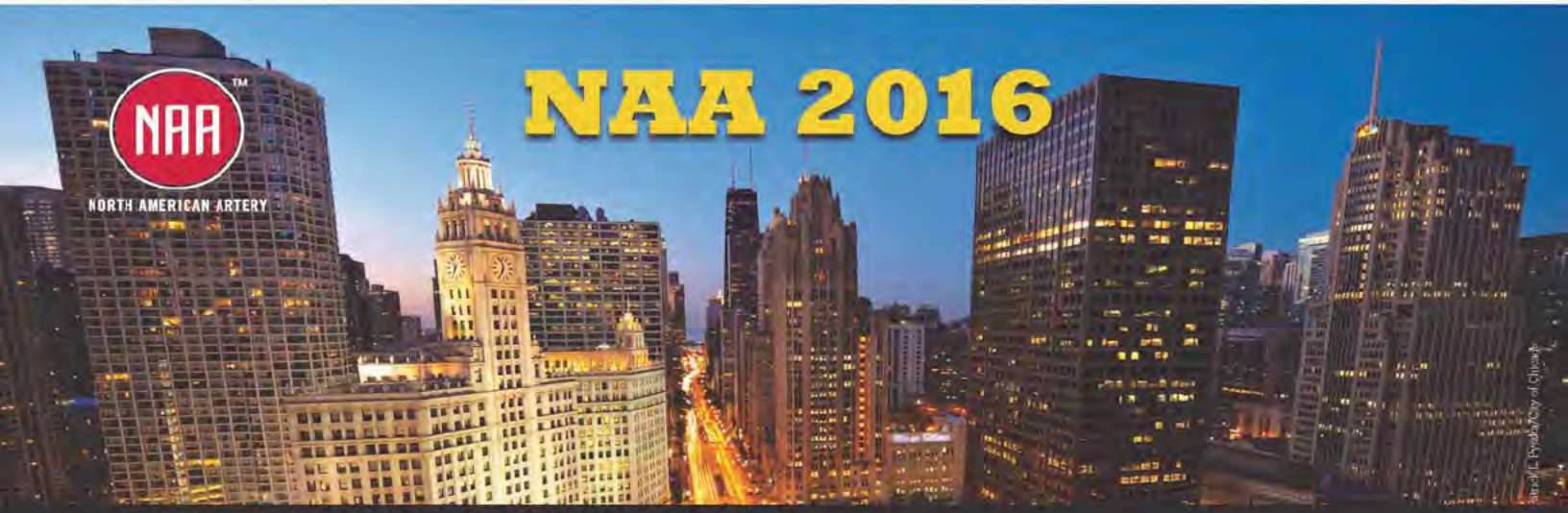

Arterial Dysfunction in Cardiometabolic Disorders: Advances in Mechanisms, Diagnosis and Treatment

North American Artery Sixth Annual Meeting

September 8-10, 2016 • University of Illinois-Chicago, Chicago, Illinois, USA

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Sharman JE et al. Hypertension. 2013. • Townsend RR et al. Journal of Clinical Hypertension. 2015.

PRESIDENT'S WELCOME

Dear Colleagues,

Please accept my personal welcome to each of you on the occasion of our Sixth Annual NAA Meeting, with this year's theme of "Arterial Dysfunction in Cardiometabolic Disorders: Advances in Mechanisms, Diagnosis and Treatment". Arterial hemodynamics remains an exciting area and the North American Artery Society continues to unite inspired speakers and energetic attendees in forums like this, leveraging our position at the cutting edge of research and the practical applications for technologies that measure arterial structure and function.

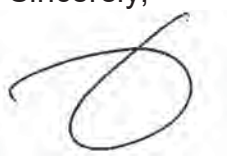
I'm indebted to the yeoman efforts of our Conference Co-Chairs, Bo Fernhall and Gary Pierce, and to the members of the Program Committee for working diligently with me to assemble this program. We are also deeply grateful for assistance from Hansen Global Event Management for such excellent logistical support.

I look forward to meeting our members, both old and new who will be here. For those of you attending who are not members, there is information about the NAA included within this book, and membership applications are available at the registration desk. Please think about becoming a part of our new and exciting organization.

This conference would not be possible without the generous support of our Conference Co-Sponsor, the University of Illinois at Chicago, our Diamond sponsors, AtCor Medical, Inc. and the National Dairy Council, and our Gold sponsors, Cardiovascular Engineering, Inc., Fukuda Denshi, Hitachi Aloka Medical America, Medical Imaging Applications, LLC and UNEX Corporation. The NAA is grateful to each of them for their support of our organization. Please visit with them in the exhibit area during our breaks.

In closing, thanks to each of you for attending the conference, and contributing your expertise to our gathering. Throughout this conference, you are encouraged to engage faculty and sponsors. My very best to you all!

Sincerely,



Raymond R. Townsend MD
University of Pennsylvania
President, North American Artery

WELCOME FROM THE CO-CHAIRS

Dear Colleagues,

On behalf of the North American Artery Society (NAA), it is our distinct pleasure to welcome you to the Sixth Annual Meeting, "Arterial Dysfunction in Cardiometabolic Disorders: Advances in Mechanisms, Diagnosis and Treatment". The NAA is a multidisciplinary society dedicated to the understanding of vascular structure and function in health and disease and its application to clinical medicine, basic/translational/population research, and pharmaceutical and medical device development. The 2016 program once again reflects these objectives with presentations focusing on the relation between arterial dysfunction and cardiometabolic diseases including a plenary lecture and symposium on macro and microvascular dysfunction in diabetes, as well as keynote lectures on mechanisms of vascular stiffness in insulin resistance and diabetes. There are also cutting edge sessions on endothelial dysfunction, vascular risk in women, debate on the origins of hypertension, tutorial lectures in forward and reflected wave hemodynamics and macro- and microvascular dysfunction in diabetes.

The Program Committee worked tirelessly to create a dynamic program that has continued to build on the success of last year's meeting. This is demonstrated by the 38 abstract presentations that are included in this year's meeting, as well as the exciting main lectures, debates, and exhibits.

We truly hope you will enjoy the 2016 NAA meeting at the University of Illinois at Chicago and that you take the opportunity to meet and network with our speakers, exhibitors, and delegates from not only the United States, but from Canada, South America, Europe and Asia as well.

We would especially like to thank our Conference Co-Sponsor, the University of Illinois at Chicago, and our supporters, AtCor Medical, Inc., Cardiovascular Engineering, Inc., Fukuda Denshi, Hitachi Aloka Medical America, Medical Imaging Applications, LLC, National Dairy Council, and UNEX Corporation for making this exciting and scientifically enriching conference possible. Thank you for participating and helping to move the NAA forward as our organization continues to grow.

Sincerely,

Bo Fernhall, Ph.D.

Gary Pierce, Ph.D.

TABLE OF CONTENTS

ADVERTISER INDEX

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GENERAL INFORMATION

Meeting Venue
University of Illinois at Chicago (UIC)
All sessions and food functions will take place in the Student Center East Tower Building located on the UIC campus at 750 S. Halsted Street, Chicago, IL.

Meeting Registration – Third Floor Foyer
All Conference materials including badges can be picked up from the registration desk during the following hours:

September 9, 2016	6:30 AM - 6:00 PM
September 10, 2016	6:30 AM - 2:00 PM

Badges are required for entry to all functions.

Conference Sessions – Meeting Room 302, Third Fl.
All sessions will take place in Meeting Room 302 except for the Dinner, which will take place in Meeting Room 605 located on the sixth floor.

Diamond Sponsored Keynote Breakfast and Dinner Lectures – Friday, September 9, 2016
The Keynote breakfast lecture, sponsored by AtCor Medical, Inc. will take place from 7:00 to 8:25 AM in Meeting Room 302 on the third floor.

The Keynote dinner lecture, sponsored by the National Dairy Council will take place from 7:30 to 9:00 PM in Meeting Room 605 on the sixth floor.

Posters on Display – Meeting Room 613, Sixth Fl.
Posters will be on display throughout the conference. Presenters will be available to discuss their posters during the lunches on Friday and Saturday.

Exhibits, Refreshment Breaks and Lunch Meeting Room 613, Sixth Fl.
All meal functions and refreshment breaks, except the dinner and breakfast, will take place in the exhibit hall as shown below.

Friday, September 9, 2016

Refreshment Breaks	9:15 to 9:45 AM
	11:00 to 11:30 AM
	3:00 to 4:00 PM
Lunch/Poster Presentations	12:10 to 1:30 PM

Saturday, September 10, 2016

Refreshment Break	10:00 to 11:00 AM
Lunch/Poster Presentations	12:45 to 2:00 PM

Participants' Reception—Friday, September 9, 2016
A reception for all participants hosted by NAA and the University of Illinois at Chicago will take place from 7:00 to 7:30 PM in Meeting Room 603 on the sixth floor.

Headquarters Hotel
Crowne Plaza Chicago Metro Downtown
733 West Madison Street
Chicago, IL 60661
Telephone: +1-800-972-2494
www.thechicagometro.com

The hotel is located less than a mile from the meeting venue.

Overnight Valet Parking – $43.00 daily
Daytime Valet Parking – $15.00 to $22.00

Shuttle Service
Shuttle service will be provided between the Crowne Plaza Chicago Metro hotel and the UIC Student Center East according to the following schedule.

Friday, September 9, 2016
From hotel to UIC: 6:00 AM, 6:40 AM & 7:20 AM
From UIC to hotel: 9:15 PM

Saturday, September 10, 2016
From hotel to UIC: 6:20 AM & 7:00 AM
From UIC to hotel: 4:30 PM

The shuttles will adhere to a strict schedule so be sure to be on time. They will depart from the front of the hotel and drop-off and pick up on Halsted Street in front of the Student Center East building.

Conflict of Interest Disclosure
North American Artery strives to ensure balance, independence, objectivity, and scientific rigor in its educational activities. Faculty members have disclosed financial relationships, in existence over the past 12 months, with commercial interests or with manufacturers with products associated with or discussed in their presentation. All Disclosure Statements are available to meeting attendees in the Program Book.

Sixth Annual Meeting Sponsors
The North American Artery Society thanks the following Sponsors for their generous support of the meeting.

We encourage all participants to visit with our sponsors' exhibit booths during the breaks.

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Associate Chief of Staff for Research and Development
Harry S Truman Memorial Veterans Hospital
Associate Professor of Medicine
Department of Internal Medicine
Division of Nephrology and Hypertension
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AGENDA—SEPTEMBER 9, 2016

7:00 - 8:25 am **Keynote Breakfast Lecture**

Moderator: Dean Winter, PhD, AtCor Medical, Inc.

7:00 am Breakfast

7:35 - 8:25 am **Demystifying CPT Coding and Payment for Arterial Pressure Waveform Analysis**

Timothy A. Pflederer, MD, Illinois Kidney Disease and Hypertension Center

Alternate CPT representative, Renal Physicians Association

CPT codes provide the mechanism for physicians to report their service and be paid for their work. Codes are developed and assigned payment through a process involving the AMA (CPT & RUC committees) and CMS. Accurately documenting the physician service is critical to payment. In the case of arterial pressure waveform analysis (CPT code 93050), this means providing a detailed analysis of the waveform in the procedure report.

The Breakfast and Keynote Lecture is sponsored by AtCor Medical, Inc. (USA)

8:25 am **Welcome Remarks**

Bo Fernhall, PhD, University of Illinois at Chicago

Gary L. Pierce, PhD, University of Iowa

President's Opening Statement

Raymond R. Townsend, MD, University of Pennsylvania

8:30 - 9:15 am **Opening Plenary Lecture**

Moderator: Raymond R. Townsend, MD

8:30 - 9:15 am **Endothelial Function in the Human Microcirculation: A Unique Window into Cardiovascular Health and Disease**

David D. Gutterman, MD, Medical College of Wisconsin

Reactive oxygen species (ROS) contribute to the onset of cardiovascular disease by promoting cell proliferation, inflammation, and thrombosis, but they can provide benefit by maintaining vasodilator control in the presence of disease when nitric oxide is lost. A novel pathway involving shear-induced release of hydrogen peroxide from endothelial mitochondria is responsible, with extranuclear telomerase is playing a critical role. Unexpectedly, this compensatory ROS-mediated dilation is also seen in athletes after brief intense exercise, blurring the line between the beneficial and detrimental role of ROS in the vasculature.

9:15 - 9:45 am **Refreshment Break—Meet the Exhibitors**

9:45-11:00 am **Endothelial Function in Health and Disease**

Moderator: Gary L. Pierce, PhD

9:45-10:05 am **Methods for Assessing Endothelial Function in Humans**

David G. Edwards, PhD, University of Delaware

Endothelial function is often assessed in both healthy and diseased individuals because of the importance of the endothelium in maintaining vascular homeostasis. There are a number of methods for assessing vascular function some of which are endothelial dependent. The choice of method depends on the research question to be asked, equipment available, operator skill, and size of the study.

10:05-10:25 am **Pharmacological Interventions for Endothelial Dysfunction in Diabetes**

Naomi M. Hamburg, MD, MS, Boston University School of Medicine

Metabolic diseases are associated with abnormal vascular function that may be reversible with novel therapeutic agents.

10:25-10:45 am **Habitual Aerobic Exercise and Vascular Endothelial Dysfunction in Aging and Prediabetes**

Gary L. Pierce, PhD, University of Iowa

Habitual aerobic exercise prevents vascular endothelial dysfunction among middle-aged/older men in part from suppression of vascular oxidative stress and inflammation.

Endothelial dysfunction in postmenopausal women appears to be resistant to improvements by aerobic exercise training, at least in part from estrogen deficiency but the cellular mechanisms remain unknown.

Prediabetes exacerbates the age-related reduction in endothelial function in sedentary middle-aged/older adults.

Habitual aerobic exercise appears to protect the endothelium from the adverse effects of impaired fasting glucose in age-related prediabetes.

10:45-11:00 am **Panel Discussion with Audience Participation**

11:00 - 11:30 am **Refreshment Break, Poster and Exhibits Viewing**

11:30 am-12:10 pm **Forward and Reflected Wave Hemodynamics**

Moderator: Raymond R. Townsend, MD

11:30-11:50 am **Hemodynamics 101: Advances in Biophysics and Hemodynamics**

Wilmer W. Nichols, PhD, University of Florida

This presentation is an overview of the hemodynamic laws that govern the flow of blood through the arterial system with special emphasis on left ventricular (LV) afterload and the separation of forward and backward (reflected) traveling pressure waves. Furthermore, the age-related changes in aortic stiffness and central aortic pressure wave morphology will be discussed. For example, when the reflected wave arrives at the left ventricle during diastole, as it does in youth, it increases coronary perfusion. When it arrives during systole, as it does in the elderly, it increases LV afterload and adversely impacts LV remodeling and systolic function.

11:50-12:10 pm **Hemodynamics 201: Physiologic and Clinical Insights**

Julio A. Chirinos, MD, PhD, University of Pennsylvania

Assessment of hemodynamics may help in various clinical situations such as:
(1) An individual's risk assessment
(2) Detailed characterization of prevalent physiology
(3) The detailed characterization of hemodynamic drug effects
(4) Deciding on which class of antihypertensive agent to use
(5) The tailored management of heart failure.

12:10 - 1:30 pm **Lunch with Exhibitors—Poster Viewing**

1:30 - 3:00 pm **Vascular Risk in Women: Preeclampsia, Postmenopause and Aging**

Moderator: Stella S. Daskalopoulou, MD, MSc, PhD, DIC

1:30-1:55 pm **Preeclampsia and Arterial Stiffness/Endothelial Function**

Stella S. Daskalopoulou, MD, MSc, PhD, DIC, McGill University

Although multi-factorial, ample evidence supports endothelial dysfunction as a key mechanism in pre-eclampsia.

Measuring arterial stiffness, even in the first trimester, may prove to be a promising clinical tool to predict pre-eclampsia in the future.

Arterial stiffness has been shown to be increased at the time of pre-eclampsia compared to uncomplicated pregnancies.

Most evidence suggests that women with a history of pre-eclampsia are at increased cardiovascular risk later in life.

(Continued on page 10)

AGENDA—SEPTEMBER 9, 2016

1:55-2:20 pm

Translational Insight Into Vascular Aging Across the Menopause Transition

Kerrie L. Moreau, PhD, University of Colorado Anschutz Medical Campus

Vascular aging, featuring endothelial dysfunction, is a major risk factor for developing cardiovascular disease (CVD). The menopause transition may be a triggering event that leads to increased vascular vulnerability and accelerated vascular aging due to changes in the hormonal environment. The acceleration in vascular aging in women during the menopause transition may be related to an increase in oxidative stress and inflammation. Intervention strategies targeting vascular oxidative stress and inflammation during the perimenopausal years may attenuate vascular aging and prevent the development of CVD in women.

2:20-2:45 pm

Systolic Blood Pressure Trajectories in Women vs. Men with Aging

Stanley S. Franklin, MD, University of California-Irvine

There is sexual dimorphism in the development of arterial hypertension. Unlike young men who are prone to diastolic hypertension, young women are largely protected from this entity during their childbearing years. On the other hand, middle-aged and older women are more prone than men to "primary" isolated systolic hypertension (ISH), related to the development of accelerated large artery stiffness. In contrast, middle-aged and older men with long-standing diastolic hypertension may transition to "secondary" ISH from what is called "burned-out" diastolic hypertension. Sexual dimorphism may explain in part why men sustain their hypertensive cardiovascular disease complications some 10 to 15 years earlier than women.

2:45-3:00 pm

Panel Discussion with Audience Participation

3:00 - 4:00 pm

Refreshment Break, Poster and Exhibits Viewing

4:00 - 4:30 pm

Featured Presentation

Moderator: Tina Ellis Brinkley, PhD, Wake Forest School of Medicine

4:00-4:30 pm

Inflammation, Vascular Function, and Exercise

Bo Fernhall, PhD, University of Illinois at Chicago

Both acute and chronic inflammation decrease endothelial function and increase arterial stiffness. Aging decreases endothelial function and increases arterial stiffness, which may be independent of inflammation. Exercise training may decrease inflammation, improve endothelial function and decrease arterial stiffness but these effects appear to be modulated by exercise mode, age, sex and type of population. Cardiovascular fitness is protective against the deleterious effects of inflammation on vascular function, especially in older individuals. Thus, increasing cardiovascular fitness should be a public health goal for most populations.

4:30 - 6:00 pm

Oral Abstract Presentations

Moderators: *Stanley S. Franklin, MD, University of California-Irvine*
Gary L. Pierce, PhD, University of Iowa

4:30 pm
OR-01

The Soluble Guanylyl Cyclase Activator Induces a Nitric Oxide Production and Decreases Reactive Oxygen Species in Endothelial Cells

Ariane Migliato Martinelli[1], Carla Nascimento dos Santos Rodrigues[1], *Gerson Jhonatan Rodrigues[1]*
[1]Departamento de Ciências Fisiológicas – Universidade Federal de São Carlos (UFSCAR), Sao Carlos, Brasil

4:45 pm
OR-02

Effect of Acute Isokinetic Resistance Exercise on Systemic Arterial Hemodynamic and Cerebral Blood Flow Dynamics: Is There a Mismatch?

Rosenberg, A., Wee, SO., Schroeder, E., Bunsawat, K., Grigoriadis, G., Fernhall, B., Baynard, T.
Integrative Physiology Laboratory, University of Illinois at Chicago, Chicago, IL

5:00 pm
OR-03

Vascular Haemodynamics In Young Adults Born Extremely Preterm

J Cockcroft[1], J Beckmann[2], C McEniery[3], K Bennett[2], N Marlow[2]
[1]University of Cardiff, [2]University College London, [3]University of Cambridge, on behalf of the EPICure Study group

5:15 pm
OR-04

Decreased Aortic Inertance Increases Susceptibility of Late-Systolic Left Ventricular Ejection to Arterial Wave Reflections

Timothy S. Phan[1,2], John K-J. Li[2], Amer Ahmed Syed[1], Harry G. Oldland[1,3], Uzma Kewan[3], Scott R. Akers[1,3], Julio A. Chirinos[1,3,4]
[1]University of Pennsylvania, Philadelphia, PA, United States, [2]Rutgers University, New Brunswick, NJ, United States, [3]Corporal Michael J. Cresenz Veterans Affairs Medical Center, Philadelphia, PA, United States, [4]Ghent University, Ghent, Belgium

5:30 pm
OR-05

Sex Differences in Vascular Structure and Function in Individuals with Multiple Sclerosis and Healthy Controls

Thessa Hilgenkamp[1], Garett Griffith[1], Robert W. Motl[2], Tracy Baynard[1], Bo Fernhall[1]
[1]Integrative Physiology Laboratory, University of Illinois at Chicago, Chicago, IL, United States, [2]Exercise Neuroscience Research Laboratory, University of Illinois at Urbana-Champaign, Champaign, IL, United States

5:45 - 5:55 pm **Break**

5:55 - 6:45 pm **Debate 2016: Origins of Hypertension**

Moderator: Bo Fernhall, PhD, University of Illinois at Chicago

Arterial Stiffness

Gary F. Mitchell, MD, Cardiovascular Engineering Inc.

After completing this activity, participants will understand that excessive aortic stiffness is the predominant contributor to the pathogenesis of wide pulse pressure hypertension in middle-aged and older individuals. Excessive aortic stiffness is associated with an increase in forward wave amplitude that accounts for the majority of the increase in pulse pressure. Patients with wide pulse pressure hypertension should be treated with destiffening agents and drugs that have been shown to lower pulse pressure, such as blockers of the renin-angiotensin system and low dose diuretics, rather than vasodilating drugs, such as calcium antagonists.

Microvascular Function/Remodeling

Ernesto L. Schiffrin, C.M., MD, PhD, McGill University

Small artery remodeling and injury as well as functional changes such as endothelial dysfunction may contribute to complications of hypertension. Whether small artery remodeling and functional changes participate in the origins of hypertension remains to be established. There is some evidence that this may be the case in some subsets of patients, particularly younger ones with systo-diastolic hypertension.

Kidney

Raymond R. Townsend, MD, University of Pennsylvania

When it comes to the origins of hypertension, and the role of the kidney, remember the FEDEX slogan!

7:00 - 7:30 pm **Participants' Reception (Room 603, 6th Floor)**

(Continued on page 12)

AGENDA—SEPTEMBER 9 & 10, 2016

7:30 - 9:00 pm **Keynote Dinner Lecture (Room 613, 6th Floor)**

Moderator: Emily Radlowski, PhD, RDN, LDN, National Dairy Council

7:30 pm Dinner

8:20 - 9:00 pm **Dairy Consumption and Vascular Function**

Lacy M. Alexander, PhD, The Pennsylvania State University

Epidemiological studies and prospective human clinical trials indicate that consuming 3-4 servings of dairy containing foods per day lowers blood pressure and improves indices of vascular function including pulse wave velocity, brachial artery flow-mediated vasodilation, and reactive hyperemia index. The beneficial vascular effects of dairy consumption are both acute and chronic, however little is known about the potential mechanisms by which dairy consumption alters vascular function. Dairy proteins themselves including lactotripepties and micronutrients have antioxidant and natural angiotensin converting enzyme-inhibitor properties. In a series of studies, the effects of acute dairy ingestion in milk (1%) and cheddar cheese on mechanisms underlying cutaneous microvascular dysfunction in older adults with pre-hypertension were investigated. Compared to nutritionally matched control meals, consuming dairy improved direct measures of nitric oxide-dependent vasodilation in the microcirculation. Furthermore, dairy cheese consumption ameliorated single-meal sodium-induced cutaneous microvascular dysfunction by reducing ascorbate-sensitive oxidants in healthy older adults.

The Dinner and Keynote Lecture is sponsored by the National Dairy Council

Saturday, September 10, 2016

7:00 - 7:55 am **Breakfast**

8:00 - 8:45 am **Keynote Lecture in Diabetes**

Moderator: Raymond R. Townsend, MD

8:00 - 8:45 am **Novel Mechanisms for Vascular Stiffness in Insulin Resistance and Diabetes**

James R. Sowers, MD, University of Missouri School of Medicine

Dietary factors such as high fructose corn syrup promote vascular stiffening.

8:45 - 10:00 am **Featured Presentations: Vascular Risk in Special Populations**

Moderator: David G. Edwards, PhD, University of Delaware

8:45 - 9:20 am **Sleep Apnea in Obesity and MetS: Vascular Risk**

Julio A. Chirinos, MD, PhD, University of Pennsylvania

1. OSA is an independent modifiable risk factor of hypertension
2. Treatment of OSA reduces BP modestly, but the effect is larger is resistant hypertension
3. The benefit of CPAP treatment most likely to be observed in HTN patients with more severe OSA who have > 4-5 hr/day PAP adherence
4. Treatment of OSA should be comprehensive and include life-style intervention targeted to weight loss along with CPAP

9:20 - 10:00 am **Gestational Diabetes and Vascular Risk**

Deborah B. Ehrenthal, MD, MPH, University of Wisconsin School of Medicine and Public Health

Women who have had gestational diabetes are at risk for diabetes, hypertension and cardiovascular disease later in life. They should be screened for diabetes with an oral glucose tolerance test 6-12 weeks after delivery, and then undergo routine testing for diabetes at least every 3 years. Strategies that focus on lifestyle modification appear to slow the development of diabetes and hypertension and should be recommended.

10:00 - 11:00 am **Refreshment Break, Poster and Exhibits Viewing**

11:00 am-12:45 pm **Special Symposium: Established Therapies for Macro- and Microvascular Dysfunction in Diabetes**

Moderator: Raymond R. Townsend, MD

11:00 - 11:30 am **Insulin Therapy & Vascular Function: Logic for New Approaches to Diabetes Care**

Stanley S. Schwartz, MD, University of Pennsylvania

The logic for not needing and minimizing use of insulin Rx in T2DM patients will be presented and is a direct result of potential adverse effects, vascular and otherwise, of insulin, i.e. hyperinsulinemia.

11:30 am-12:00 pm **SGLT-2 Inhibitors, Vascular Function, CVD Events in Diabetes**

Raymond R. Townsend, MD, University of Pennsylvania

SGLT2 medications lower glucose, lower BP, lower weight and reduce risks of heart failure and death in type 2 diabetics. The price for this is an increase in the risk of infections (urinary tract mostly) and orthostatic hypotension.

12:00 - 12:30 pm **Incretins and CVD Events in Diabetes**

Adam Whaley-Connell, DO, MSPH, MEd, University of Missouri-Columbia School of Medicine

Audience should come away with an understanding of the incretin axis in diabetes management and the potential impact it may have on cardiovascular disease.

12:30 - 12:45 pm **Panel Discussion with Audience Participation**

12:45 - 2:00 pm **Lunch, Exhibits Viewing and Poster Presentations**

1:15 - 2:00 pm **Poster Presentations**

2:00 - 3:45 pm **Lifestyle Interventions for Macro- and Microvascular Dysfunction in Human Obesity/Diabetes**

Moderator: Bo Fernhall, PhD, University of Illinois at Chicago

2:00 - 2:30 pm **Exercise and Adipose Arteriole Function In Obesity**

Shane A. Phillips, PT, PhD, University of Illinois at Chicago

These sources of acute stress to microvascular endothelium are multifactorial and linked to higher incidence of cardiovascular disease. Regular exercise protects against acute exertion induced vascular dysfunction in previously sedentary, overweight and obese individuals. The improved adipose microvascular function following acute stress such as high pressure and high intensity exercise, involves an alternative vasodilator mechanism hydrogen peroxide.

2:30 - 3:00 pm **Treatment of Accelerated Vascular Aging in Obesity**

Kevin P. Davy, PhD, Virginia Tech

There is considerable variability in large artery stiffening among middle-age and older adults. Age-related weight gain and obesity may contribute, at least in part, to this variability. As such, prevention of age-related weight gain and weight loss may be efficacious in reducing accelerated age-related large artery stiffening. Future efforts are needed to target the residual arterial stiffness that remains following interventions.

(Continued on page 14)

AGENDA—SEPTEMBER 10, 2016

3:00 - 3:30 pm **Nitrates and Vascular Function in Diabetes**

Andrew Webb, BSc, MBBS, MRCP, PhD, British Heart Foundation Centre, King's College London/Guy's & St Thomas' NHS Foundation Trust

Dietary inorganic nitrate (found in green leafy vegetables such as rocket and in beetroot) is now recognised to be an important source of nitric oxide, via the nitrate-nitrite-nitric oxide (NO) pathway.

Dietary nitrate appears to confer several cardiovascular beneficial effects, for example on blood pressure, arterial stiffness, endothelial function, platelets, mitochondrial efficiency and exercise.

Dietary nitrate appears to be an important component of 'healthy diets', such as the DASH diet to lower blood pressure and the Mediterranean diet, with its potential to lower cardiovascular risk.

Use of dietary nitrate may be a beneficial additional approach in patients with high blood pressure (Actionable item).

Patients with diabetes have a high incidence of cardiovascular disease, associated with endothelial dysfunction and decreased NO bioavailability.

Therefore, dietary nitrate has the potential to improve cardiovascular parameters and risk in patients with diabetes; however, studies to date have not demonstrated significant improvements in this population, suggesting that patients with diabetes may be partially resistant to dietary nitrate/ the nitrate-nitrite-NO pathway.

We have now completed the longest and largest placebo-controlled intervention study to date of dietary nitrate, as beetroot juice (6 months') in 126 patients with, or at risk of diabetes (Vasera study), exploring vascular effects, and will present preliminary data from this study at the meeting.

3:30 - 3:45 pm **Panel Discussion with Audience Participation**

3:45 - 3:55 pm **Awards Presentations**

Best Abstract and Young Investigator Awards

3:55 - 4:00 pm **Concluding Remarks**

PO-01 Animal Models of Local Aortic Stiffening: The Effect of Salt in SHRSP

PO-02 No Sex Differences In the Cardiovascular Response to Mental-Stress in Older Adults.

PO-05 Psoriasis is associated with Increased Arterial Stiffness: a Systematic Review and Meta-Analysis

PO-06 Increased Arterial Stiffness in Behcet's Disease: A Systematic Review and Meta-Analysis

PO-07 Association between White-Coat Hypertension and Arterial Stiffness: A Systematic Review and Meta-Analysis

PO-08 Association between Masked Hypertension and Arterial Stiffness: A Systematic Review and Meta-Analysis

PO-09 Central Hemodynamics and Arterial Stiffness in Young Obese Adults: the Preliminary Finding

PO-10 Vascular Function in individuals with Down Syndrome

PO-11 Multiple Sclerosis Patients Experience More Decrements in Carotid Artery Functional Properties with Aging than Age-Matched Peers

PO-13 Sex Differences in Vascular Function Following Antioxidant Supplementation

PO-14 Pulse Wave Velocity Is Increased With Experimental Sleep Restriction in Healthy Humans

PO-16 Blood Pressure Variability and Baroreceptor Sensitivity in Normotensive Obese In Response to Aerobic Exercise

PO-17 Role of Nitric Oxide in B2-adrenergic Mediated Vasodilation in Postmenopausal Women

PO-19 Associations of Walking with Sarcopenic Obesity and Cardiovascular Disease Risk Factors in Older Adults

PO-20 A Hydrogen Sulfide Prodrug Augments Angiogenesis in a Swine Model of Critical Limb Ischemia via a Nitric Oxide Dependent Mechanism

PO-22 Body Mass Index as an Independent Predictor of Change in Arterial Stiffness Parameters with Change in Body Position

PO-23 A Systematic Review on the Effect of Acute Aerobic Exercise on Arterial Stiffness Reveals A Differential Response in the Upper and Lower Arterial Segments

PO-24 Relationship between Step Counts and Carotid Femoral Pulse Wave Velocity in Adults Treated For Hypertension and Diabetes

PO-25 Higher Central and Brachial Systolic Blood Pressure is Selectively Associated with Weaker Cognitive Performance in Postmenopausal Women but Not Older Men

PO-26 Bilateral Symmetry of Brachial Pulse Waveform Analysis in a Clinical Population

PO-27 A New Software for Determining Changes In Arterial Diameter Over Time

PO-29 Greater Early and Late Arterial Loading with Advancing Age is Associated with Impaired Hemodynamic Efficiency in a Community Dwelling Population

PO-30 Effect of Low-Dose Acetylsalicylic Acid on Arterial Stiffness in High-Risk Pregnancies: An Observational Longitudinal Study

PO-31 Effect of Poor Glycemic Control on Arterial Stiffness in Pregnancy

PO-32 Dietary Calcium Intake And Cardiovascular Health: Is There Any Relationship?

PO-33 Statin Therapy in Rheumatoid Arthritis May Improve Arterial Stiffness in Women but Not In Men: A Preliminary Analysis

PO-35 First in Man Measurement of Arterial Stiffness Using a Connected Bathroom Scale: Calibration against SphygmoCor

PO-36 Effects of fixed versus auto-titrating continuous positive airway pressure on vascular function in patients with resistant hypertension and obstructive sleep apnea

PO-37 The Impact of Intradialytic Pedaling Exercise On Arterial Stiffness in a Hemodialysis Population

EXHIBITORS

Diamond Sponsor

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AtCor Medical develops and markets the SphygmoCor® system, the industry standard for performing central arterial pressure waveform analysis and measuring arterial stiffness. SphygmoCor is essential for hypertension management, providing information that helps physicians individualize treatment decisions. The SphygmoCor system is used by clinicians, researchers and pharmaceutical companies globally. For more information, visit atcormedical.com or email info@atcormedical.com.

Gold Sponsor

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Over the past 2 decades, Cardiovascular Engineering, Inc., (CEI) has designed and manufactured the Noninvasive Hemodynamics (NIHem) family of workstations, which are used at research centers around the world to perform comprehensive assessments of vascular function. The new NIHem-WF system is a compact research solution that provides ECG and tonometry data via a direct WiFi link to a tablet, laptop or desktop computer. The device allows for rapid and robust assessment of carotid-femoral pulse wave velocity, central blood pressure and central pressure waveform analysis using the gold standard direct tonometry approach.

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The VaSera model VS-2000 is our new generation vascular function testing system that provides fast and accurate measures of ankle-brachial index (ABI) and the cardio-ankle vascular index (CAVI; a measure of arterial stiffness), and a clinical blood pressure evaluation protocol. CAVI is calculated using measures of pulse wave velocity (PWV), heart sounds, and blood pressure. The VS-2000 system is designed with test efficiency improvements and greater flexibility for use in clinics or research studies. VaSera is very easy to use, and reduces operator error through an automated measurement protocol—making its test measurements highly reproducible.

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- Medical Imaging Applications, LLC (MIA) offers FDA-compliant and research-only software for quantification of arterial images (ultrasound, CT, MR).
- Brachial Analyzer - assessment of endothelial function via brachial-artery flow-mediated dilatation (brachial FMD).
- Carotid Analyzer - quantification of carotid and aortic intimal-medial thickness (carotid/aortic IMT), analysis of both near and far arterial walls at multiple locations, assessment of plaque characteristics, vascular distensibility/compliance.
- Aortic Analyzer - 3-D, 4-D, and longitudinal analysis of multi-phase aortic MR. MRA, CT, CTA images. Location-specific quantification of aortic morphology, distensibility/compliance, aortic plaque, and motion from aortic annulus to diaphragm.
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UNEXEF is the semi-automated device specified for FMD test presented by UNEX, Japan. NAA 2016 participants will have a great opportunity to experience the demonstration of UNEXEF and see how easy it is to operate the FMD test just like an expert sonographer.

More than eight hundred facilities have been using UNEXEF in Japan for not only their research studies but also for practical clinical usage. We hope the FMD test will be a very important part of the Society's Focus to protect people from CVD.

FACULTY BIOS

LACY M. ALEXANDER, PhD, FACSM is an Associate Professor of Kinesiology at The Pennsylvania State University, University Park, PA.

Dr. Alexander is an expert in the mechanisms underlying microvascular dysfunction in clinical populations in human skin. Dr. Alexander uses *in vivo* and *in vitro* approaches to study the effects of pharmacological and lifestyle interventions on microvascular dysfunction in humans. She has authored and co-authored >60 publication in her area of expertise and has received merit based awards from both the American Physiological Society and the American College of Sports Medicine for her work in microvascular control and integrative physiology including the Exercise and Environmental Physiology Section New Investigator Award in 2011. Dr. Alexander is on the editorial board for several journals including the *Journal of Applied Physiology* and is an elected member of the Exercise and Environmental Physiology Steering Committee and Joint Programming Committee for the American Physiological Society. Dr. Alexander has maintained a record of extramural funding with grants from the National Institutes of Health (NIH), the National Dairy Management Inc., and other industry sponsors. Her current NIH grant focuses on the role of hydrogen sulfide as a gasotransmitter in the microcirculation of humans with essential hypertension.

JULIO A. CHIRINOS, MD, PhD, FAHA is an Assistant Professor of Medicine, Director of the Cardiovascular Phenotyping Unit, Clinical Translational Research Center, and Adjunct Faculty, Center for Magnetic Resonance and Optical Imaging at the University of Pennsylvania Perelman School of Medicine. He is also a Visiting Professor at Ghent University, Belgium.

Dr. Chirinos earned his MD from the Catholic University of Santa Maria, Arequipa, Peru, and his PhD in Biomedical Sciences from the University of Ghent, Belgium.

His research interests include the non-invasive assessment of arterial function and ventricular-vascular coupling and its role in left ventricular remodeling, dysfunction and heart failure risk. Dr. Chirinos has a particular interest in the role of the left ventricular loading sequence in patients with heart failure and normal ejection fraction as well as in the role of arterial stiffness and central arterial pressures as predictors of cardiovascular risk. He is also interested in the cardiovascular consequences of obstructive sleep apnea.

STELLA DASKALOPOULOU, MD, MSc, PhD, DIC is a tenured Associate Professor of Medicine at McGill University. She established and directs the Vascular Health Unit at the McGill University Health Centre, where she conducts research projects on vascular structure and function, including the identification of novel pathways of atherosclerotic plaque instability, image analysis of atherosclerosis plaque composition, and arteriosclerosis, with special interest in arterial stiffness and hemodynamics in subjects with cardiovascular risk factors, such as hypertension, smoking, diabetes, dyslipidemia, and pre-eclampsia. Dr. Daskalopoulou's research has led to over 100 peer-reviewed publications. She is a member of the central review committee of the Canadian Hypertension Education Program (CHEP), the Executive Committee of the Société des Sciences Vasculaires du Québec, and the North America Artery Society. Dr. Daskalopoulou has received several personal awards, including the Hypertension Canada New Investigator Award, the Young Researcher Award of Excellence from the Heart and Stroke Foundation of Quebec, the Canadian Foundation for Women's Health Research, the Bourse Fonds de recherche du Québec–Santé (FRQS) ─La Société Québécoise d'Hypertension Artérielle Jacques-de-Champlain, and the Canadian Society of Internal Medicine New Investigator award. Dr. Daskalopoulou holds several research grants from the Canadian Institutes of Health Research, FRQS, Heart and Stroke Foundation of Canada and Canada Foundation for Innovation Leaders Opportunity Fund, amongst others. She is a clinical research scholar of the FRQS.

KEVIN P. DAVY, PhD received his B.S. degree at the State University of New York at Cortland; his M.A. in Exercise Physiology from Adelphi University; and his Ph.D. in Applied Physiology from Virginia Tech. He performed postdoctoral training in Integrative Physiology at the University of Colorado at Boulder. He has served on the faculties of Colorado State University and University of Mississippi Medical Center. During this period, he was a recipient of both an NIH Career Development Award and Independent Scientist Award. Dr. Davy is currently a Professor of Translational Physiology in the Department of Human Nutrition, Foods, and Exercise at

Virginia Tech. He has held a number of leadership positions including the Assistant Vice President of Research at Virginia Tech. He now serves as Director of the Human Integrative Physiology Laboratory and the Fralin Translational Obesity Research Center and Interdisciplinary Graduate Education Program at Virginia Tech. Dr. Davy has been continuously funded by NIH, industry, and non-profit agencies for >20 years. He has served on numerous American Heart Association and NIH study sections including a 4 year term on the NIH Clinical Integrative and Diabetes Study section. His research interests are related to understanding how obesity, weight gain, and therapeutic interventions influence cardiometabolic dysfunction in obesity and aging. Much of his work has been driven by the objective to determine the human clinical relevance of observations made in animal models. Dr. Davy has published over 80 peer reviewed manuscripts, reviews, and book chapters. He has trained numerous fellows who now hold positions in academia, government, and industry in the U.S. and South America. His current work is focused on testing and identifying efficacious therapeutic interventions to mitigate the cardiometabolic consequences of high fat diets and obesity.

DAVID G. EDWARDS, PhD is a Professor in the Department of Kinesiology and Applied Physiology at the University of Delaware in Newark, DE. He also directs the recently launched Center for Cardiovascular Health at the University of Delaware. His research focus is in the area of vascular physiology and is funded by the National Institutes of Health. His current work is focused on studying vascular function in patients with chronic kidney disease as well as studying the effects of dietary sodium on vascular function in normotensive adults.

(Continued on page 20)

FACULTY BIOS

DEBORAH B. EHRENTHAL, MD, MPH, FACP is Associate Professor at the University of Wisconsin-Madison School of Medicine and Public Health, where she holds the Lifecourse Initiative for Healthy Families Chair. She attended the University of Massachusetts Medical School, completed her internship and residency training in General Internal Medicine at Yale-New Haven Hospital, and received an MPH from the Johns Hopkins Bloomberg School of Public Health. She moved to the University of Wisconsin-Madison in 2014, where she has appointments in Obstetrics & Gynecology, Population Health Sciences, and Medicine. She is the Faculty Director of the Wisconsin Partnership Program's Lifecourse Initiative for Healthy Families, and Director of the Health Disparities Research Scholars T32 post-doctoral training program. Her research integrates women's health and maternal and child health, focusing on the health outcomes of women and children over the life course.

BO FERNHALL, PhD started his career with a focus on physical fitness and cardiac rehabilitation, and he spent over 20 years directing university based cardiac rehabilitation programs. This shaped his current research interests in exercise physiology with a specialization in cardiovascular function and health throughout the lifespan. He currently has an active, funded research program on the effect of exercise and physical activity on heart and arterial health. He is especially interested in the how exercise impacts the interaction of heart, arterial and autonomic function and how these factors are affected by inflammation. Dr. Fernhall's research program has a special focus on aging, racial and ethnic health disparities and cardiovascular health and function in individuals with disabilities or chronic disease conditions.

Dr. Fernhall is currently Dean of the College of Applied Health Sciences and Professor of Kinesiology and Nutrition at the University of Illinois at Chicago. Together with several other faculty, he founded the Integrative Physiology Laboratory in the College of Applied Health Sciences at UIC in 2012.

STANLEY S. FRANKLIN, MD, FACP, FACC is Clinical Professor of Medicine at the University of California, Irvine and Investigator with the Framingham Heart Study. His main research interest is the epidemiology of hypertension with more than 220 peer-reviewed original articles and chapters. The European Society for Artery Research has honored him with their 2013 "Lifetime Research Achievement Award."

DAVID D. GUTTERMAN, MD, FCCP, FAPS is the Senior Associate Director of the Cardiovascular Center and Northwestern Mutual Professor of Cardiology. He is actively involved in clinical practice and supervises an NIH funded research laboratory. Dr. Gutterman's investigative interests focus on regulation of human vascular reactivity both at the fundamental and translational research levels. His work has defined a critical role for mitochondria in generating the hydrogen peroxide responsible for flow-mediated dilation in the coronary microcirculation of patients with coronary artery disease. The cellular mechanism of this ROS-mediated dilation is under active investigation with emerging roles for extranuclear telomerase and short chain ceramides in the generation of mitochondrial ROS. Dr. Gutterman has also undertaken studies to examine the earliest changes that occur in the development of atherosclerosis (clinical endothelial dysfunction) and has used this approach to examine the beneficial and detrimental roles of various physical and dietary stresses on cardiovascular health in human subjects. Dr. Gutterman has authored more than 160 peer-reviewed publications related to cardiovascular function and pathophysiology.

NAOMI M. HAMBURG, MD, MS, FACC is an Associate Professor in the Cardiovascular Medicine Section and Chief of the Section of Vascular Biology at Boston University School of Medicine. She runs an active translational research program investigating the mechanisms of vascular dysfunction in metabolic diseases and peripheral artery disease. She is a specialist in Cardiology and Vascular Medicine, a member of the Board of Directors of the Society of Vascular Medicine and an Associate Editor of the journal Vascular Medicine and Senior Editor of the Journal of the American Society of Hypertension.

GARY F. MITCHELL, MD is a cardiologist and internationally acknowledged leader in the field of vascular stiffness and pulsatile hemodynamics. He received his medical degree from Washington University in St. Louis and completed his training in Medicine and Cardiology at

Brigham and Women's Hospital in Boston, where he served as a staff cardiologist until 1998. He left the Brigham in 1998 to become founder and president of Cardiovascular Engineering, Inc., which is an NIH-funded small business that designs and develops innovative devices that measure arterial stiffness and uses those devices to examine genetic and environmental correlates of arterial stiffness and the role that arterial stiffness plays in the pathogenesis of hypertension and target organ damage. He joined the Framingham Heart Study as a Framingham Investigator in 1999 and became a collaborator on the AGES-Reykjavik study in 2006 and the Jackson Heart Study in 2010. Using devices designed and built by Cardiovascular Engineering, Dr. Mitchell has performed detailed assessments of arterial stiffness and pulsatile hemodynamics in more than 20,000 research participants, including participants in all 3 generations of the Framingham Heart Study as well as participants in the AGES-Reykjavik study, the REFINE study and the Jackson Heart Study.

KERRIE L. MOREAU, PhD was born in Superior, WI and received her BA degree in exercise science from the College of St. Scholastica in Duluth, MN. From there she received her MS in clinical exercise physiology from Ball State University and her PhD in exercise and applied physiology from the University of Tennessee, Knoxville. She pursued postdoctoral training in the laboratory of Dr. Douglas Seals at the University of Colorado, Boulder and has been a faculty member at the University of Colorado Denver School of Medicine since 2005. Her research career has focused on the modulatory influence of sex hormones on vascular aging, with an

(Continued on page 22)

emphasis on vascular endothelial dysfunction, large elastic arterial stiffening, intimal-medial thickening, and maladaptations in cardiac structure/function, and on the role of regular exercise in the primary and secondary prevention of vascular aging. She has developed a successful clinical research program to study the mechanisms underlying vascular aging, particularly as it relates to menopause and andropause in women and men, respectively. She has authored 36 peer-reviewed publications and has been consistently funded by NIH. She currently is the PI on an NIH R56 investigating the mechanisms mediating vascular dysfunction with aging and the menopause transition in women, and an RO1 examining the cardiovascular consequences of low testosterone in aging men. Additionally, she is the director of the Cardiovascular BioImaging Core at the University of Colorado Denver Anschutz Medical Campus, a dedicated research facility for protocols utilizing cardiovascular imaging.

WILMER W. NICHOLS, PhD is an Adjunct Professor of Medicine in the Department of Medicine in the College of Medicine and in the Division of Cardiovascular Medicine at the University of Florida.

He served as the Director of Basic Cardiovascular Research, Division of Cardiology, Department of Medicine at the University of Florida for nearly 30 years. Dr. Nichols, who earned his PhD degree from the University of Alabama Medical School at Birmingham, Alabama, coauthored the book, McDonald's Blood Flow in Arteries: Theoretic, Experimental and Clinical Principles and has authored more than 200 articles that have appeared in numerous journals.

Dr. Nichols' major interests are related to pulsatile pressure and flow in arteries. His investigations over the years have dealt with sophisticated measurements of aortic impedance and left ventricular responses to acute changes in afterload in man. He is also interested in Hemodynamics, Cardiovascular Aging, Hypertension, Exercise, and Cardiovascular Disease in Women.

TIMOTHY A. PFLEDERER, MD, FASN, FASDIN completed medical school at the University of Illinois College of Medicine and both internal medicine residency and nephrology fellowship at the University of Iowa. He practices general and interventional nephrology. He is President of his practice, Illinois Kidney Disease and Hypertension Center, in Peoria IL, which is a group of 14 nephrologists and 2 surgeons serving much of central IL. He serves on the board of directors of the Renal Physicians Association (RPA), chairs the Medical Review Board for Renal Network 10, and is a past president of the American Society of Diagnostic and Interventional Nephrology (ASDIN). He also is the alternate advisor to the AMA CPT committee for the Renal Physicians Association.

SHANE A. PHILLIPS, PT, PhD, FAHA is Professor and Associate Head in the Department of Physical Therapy at the University of Illinois at Chicago. Phillips has clinical expertise in physical therapy and cardiovascular rehabilitation. His PhD degree in physiology was completed at the Medical College of Wisconsin, he completed post-doctoral training in Cardiovascular Medicine. He is Director of the Vascular Biology Laboratory in the College of Applied Health Sciences where he studies obesity and the control of blood flow and responses of the microcirculation to surgery, diet and exercise interventions. Other research interests include the impact of cardiovascular risk factors such as high blood pressure, alcohol and high cholesterol on macro and microcirculatory function. His laboratory is funded by the National Institute of Health.

GARY L. PIERCE, PhD, FAHA is an Assistant Professor of Health and Human Physiology at the University of Iowa with secondary faculty appointments in the Abboud Cardiovascular Research Center, the Center for Hypertension Research and the Fraternal Order of Eagles Diabetes Research Center. Dr. Pierce obtained his Ph.D. in Applied Physiology and Kinesiology at the University of Florida in 2005 with an emphasis in cardiovascular exercise physiology. At Florida he worked with Dr. Wilmer Nichols and Randy Braith investigating effects of chronic exercise training on vascular endothelial function, arterial stiffness and central blood pressure hemodynamics patients in patients with heart failure and heart transplantation. From 2005-2009, Dr. Pierce was a postdoctoral research fellow in the lab of Dr. Doug Seals in the Department of Integrative Physiology at the University of Colorado Boulder where he studied the mechanisms by which select pharmacological interventions or habitual aerobic exercise improved vascular endothelial function

in middle aged and older adults. From 2009-11, Dr. Pierce was an Assistant Professor at the Medical College of Georgia where he studied the influence of arterial stiffness and central pulsatile hemodynamics on target organ damage in youth with obesity. In 2011, Dr. Pierce joined the University of Iowa where he continues to examine the integrative mechanisms by which aging, obesity, hypertension, COPD, chronic anxiety and preeclampsia alters large and small artery structure and function in humans. Dr. Pierce is a member of the North American Artery Society, American Physiological Society and a Fellow of the American Heart Association.

ERNESTO L. SCHIFFRIN, C.M., MD, PhD, FRSC, FRCPC, FACP is Physician-in-Chief of the Sir Mortimer B. Davis-Jewish General Hospital. He holds a tier 1 Canada Research Chair in Hypertension and Vascular Research at the Lady Davis Institute of Medical Research, and is Professor and Vice-Chair (Research), Department of Medicine, McGill University, all since January 2006.

Dr. Schiffrin's research deals with molecular and cellular mechanisms of vascular disease and hypertension and their treatment, supported currently by a Foundation Grant from the Canadian Institutes for Health Research (2015-2022) and an Industry discovery grant. He is author of 540 peer-reviewed publications, many book chapters and is editor of four books, on molecular and clinical aspects of vascular disease and hypertension. Dr. Schiffrin was Associate Editor of Hypertension (American Heart Association [AHA] journal) from July 2003 to September 2015, and since January 2016, Dr. Schiffrin is the Editor-in-Chief of the American Journal of Hypertension.

Dr. Schiffrin is President of Hypertension Canada (2013-

(Continued on page 24)

2016). Previously, he was Chair of the High Blood Pressure Research Council (now Hypertension Council) of the American Heart Association (2002-2004), and President of the International Society of Hypertension (2012-2014), and is now its Immediate Past President.

Dr. Schiffrin was elected Fellow of the Royal Society of Canada in 2006, and received the 2007 Irvine Page-Alva Bradley Lifetime Achievement Award of the High Blood Pressure Research Council of the AHA, the 2010 Bjorn Folkow Award of the European Society of Hypertension, the 2011 Excellence Award in Hypertension Research of the AHA, the 2013 American Society of Hypertension Robert Tigerstedt Award, the Distinguished Scientist Award of the Canadian Society of Cardiology in 2013 and of the Canadian Society of Clinical Investigation in 2015. He was appointed Member of the Order of Canada (C.M.) in 2010.

STANLEY SCHWARTZ, MD, FACP, FACE is an Affiliate of the Main Line Health System, and an Emeritus Associate Professor of Medicine at the University of Pennsylvania, currently in a private practice in Ardmore, Pa. Dr. Schwartz received his MD in 1973 from the University of Chicago in Chicago, Illinois. He then completed his residency at the University of Pennsylvania, followed by a fellowship in endocrinology and metabolism at the University of Chicago.

Dr. Schwartz actively lectures nationally, as well as internationally, about diabetes and its treatment. In the past 4 years, he has been a speaker in Turks/Caicos, Istanbul, Switzerland, Belgium, Tunisia, Spain, 11 cities in China, Philippines (AACE), Israel, Singapore and AACE 2011/2014, ADA 2014- (2015). He has authored numerous articles in peer-reviewed scientific journals and has been a lead or co-investigator for many clinical trials (DCCT-EDIC, LOOK AHEAD). He has created a call for a New Classification for All Diabetes and presented it in Jerusalem, at ADA 2014 and ADA/JDRF symposium 2015 and now published in Diabetes Care.

Since leaving Penn, he has received an R-01 from the NIH for studying the Genes related to LADA (with 2 articles published), written 3 book chapters, had posters at 2012 AACE, ADA and EASD in 2014 and 2015), has had 16 peer-reviewed article's published (Incretins in Hospital, hypoglycemia, a critique of ADA DM guideline, incretins in NODAT, weight reduction in DM, and the incretin story). He is an author of the Self-Assessment Program of the American Diabetes Assoc. (2007 and 2008) , the 2009/2010 AACE Diabetes Guidelines, and slide modules for primary care for AACE, 2011 and 2013, and Diabetes in Control 2013-2016 . He is a member or fellow of international, national, and local professional societies including an Overseas Fellow of the Royal Society of Medicine (London), European Association for the Study of Diabetes (EASD), the International Diabetes Federation (IDF), the American Diabetes Association (ADA), the American Endocrine Society (ES), College of Physicians in Philadelphia, and the American College of Physicians (ACP). He's been elected by his peers for inclusion in Best Doctors in America® from 1996 to 2015. Though he does some research and teaches a great deal, most of his time is spent caring for patients.

JAMES R. SOWERS, MD, is Professor of Medicine, Pharmacology/Physiology and Director of the Endocrinology, Diabetes & Metabolism Division at the University of Missouri School of Medicine. In addition, he is the Director of the Thomas and Joan Burns Cardiovascular and Diabetes Research Center and holds the Thomas W. and Joan F. Burns Missouri Chair in Dialectology. Dr. Sowers serves as a reviewer on several study sections for the National Institutes of Health and for the Department of Veterans Affairs. He is the editor of Cardiorenal Medicine, Associate Editor of Diabetes and on editorial boards of Hypertension, Endocrinology, and Metabolism. Dr. Sowers has published more than 500 peer-reviewed papers.

Dr. Sowers achieved the Irvin Page Lifetime Achievement Award from the American Heart Association in 2012. He serves as a PI on one NIH funded grant and a VA Merit grant, and is a Co-Investigator on several NIH grants with colleagues at the University of Missouri, as well as other research colleagues at academic institutions around the country.

Dr. Sowers has been examining the cellular mechanisms of insulin action in cardiovascular, renal and skeletal muscle tissue for three decades, focusing primarily on in vitro and in vivo/ex-vivo studies of animal models. Recently his research has been directed to the role of overnutrition/angiotensin II, aldosterone, estrogen, and immune function on T regulatory cells and in site-specific

serine phosphorylation of insulin sensitivity and associated cardiovascular functional abnormalities. As part this continuing cardiovascular renal diabetes research program, his program plans to pursue the role of angiotensin II, aldosterone and sex differences in metabolic cardiovascular insulin resistance in mice subjected to a "Westernized" diet.

RAYMOND R. TOWNSEND, MD, is a Professor of Medicine and an Associate Director of the General Clinical Research Center/CTSA at the University of Pennsylvania. He is currently a Principal Investigator on a 7-center U01 grant (DK-060984) to evaluate the role of demographic, phenotypic, humoral and genetic factors in the progression of kidney disease and the development and progression of cardiovascular disease in patients with chronic kidney disease. His formal certifications are in internal medicine (ABIM), nephrology (ABIM), clinical pharmacology (ASCP) and hypertension (ASH). He is a fellow in the American Heart Association and a fellow of the Council for High Blood Pressure Research. Research interests include role of vascular dynamics in CKD progression and the incidence/development of CVD in CKD. He was an empanelled member of JNC 8, a recent co-chair of the 8th AHA Hypertension Summer School (Summer 2013), and was named the American Heart Association's Physician of the Year for 2016.

ANDREW WEBB, BSc, MBBS, FRCP, PhD is a Clinical Senior Lecturer/Honorary Consultant Physician in Cardiovascular Clinical Pharmacology/General Medicine at the British Heart Foundation Centre, King's College London/Guy's & St Thomas' NHS Foundation Trust. Andrew was awarded his PhD in 2007 at Barts & The London/Queen Mary University London on "nitrite (NO2-)-derived NO in the cardiovascular system", which demonstrated the protective effects of nitrite in ischaemia-

(Continued on page 26)

reperfusion injury and the role of xanthine oxidase. He also performed studies with beetroot juice/ dietary nitrate (NO3-) and demonstrated blood pressure-lowering, antiplatelet and vasculo-protective effects. Andrew was appointed Senior Lecturer at King's College London in 2010 where he established a lab to further investigate vascular/ haemodynamic/metabolic mechanisms involving nitrate and nitrite. He has demonstrated the paradoxical, normoxia-dependent selective effects of nitrite in conduit arteries - with selective effects on central haemodynamics (with colleague Prof Phil Chowienczyk), and recently completed a study of the vascular effects of 6 months' intervention with dietary nitrate as beetroot juice in 126 patients with diabetes (with colleague Prof Kennedy Cruickshank).

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The Soluble Guanylyl Cyclase Activator Induces a Nitric Oxide Production and Decreases Reactive Oxygen Species in Endothelial Cells

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Introduction: Previously, we have verified that the relaxation to NO donor sodium nitroprusside is potentiated in the presence of endothelium. Thus, the aim of this study was verify if the activation of soluble guanylyl cyclase (sGC) by ataciguat in endothelial cells induces a NO production, as well as identify the mechanism of this action.

Methods: Male wistar rats were used (400–500 g). To vascular reactivity study, thoracic aortas were used. The relaxation was performed to Ataciguat in aortas with (E+) and without (E-) endothelium. The potency (pD2) was measured. In Human Umbilical Vein *Endothelial Cells* (*HUVEC*) in culture, we have measured intracellular NO (by DAF-2DA fluorescence intensity FI) and reactivity oxygen species (ROS) by DHE fluorescence. HUVECs were treated for 30 minutes with Ataciguat (0.1, 1.0 and 10μM) or 100μM Tempol (SOD mimetic), without and with non-selective NOS inhibitor (L-NAME), or sGC inhibitor (ODQ), or calcium channel blocker (Verapamil). The Ethical Committee of the UFSCar (n º 012/2013) approved all protocols with rats.

Results: The presence of endothelium potentiated the relaxation induced by Ataciguat (pD2 E+: 4.22±0.23, n=4 > pD2 E-: 2.99±0.18, n=5, $p<0.05$). In the presence of L-NAME the effect of endothelium was abolished (pD2 E+ L-NAME: 3.34±0.31, n=5). In HUVECs, the Ataciguat induced the NO production. In the presence of L-NAME or ODQ the NO production induced by Ataciguat 0.1μM was abolished, with no difference in the presence of Verapamil. ROS was increased in HUVECs stimulated with angiotensin II and Ataciguat treatment decreased the ROS production induced by Angiotensin II, with similar results to Tempol.

Conclusion: Taken together our results indicate that the activation of sGC in endothelial cells can induces a NO production by a mechanism independent of calcium influx and is able to decreases the ROS.

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Effect of Acute Isokinetic Resistance Exercise On Systemic Arterial Hemodynamics And Cerebral Blood Flow Dynamics: Is There a Mismatch?

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Resistance exercise (RE) is currently recommended for most adults and is important for reducing risk factors for cardiovascular and metabolic diseases, and improving quality of life. Despite functional and musculoskeletal benefits, high-intensity RE has been shown to acutely increase arterial stiffness and blood pressure, with reduced cerebral blood flow velocity and greater flow pulsatility in the cerebral circulation, which may be detrimental to cerebral microvasculature. Objective: The purpose of this study was to investigate the effects of an acute bout of RE on hemodynamics and cerebral vascular responses in recreationally active, young adults.

Methods: Fifteen healthy adults aged 18-35 years (~26 years, male=7) performed RE, which consisted of 3 sets of 10 repetitions of isokinetic concentric/concentric unilateral knee flexion/extension. All measurements were obtained at baseline and post-exercise (1,5,30-minutes). Beat-to-beat heart rate (HR), brachial blood pressure (bSBP, bDBP, bMAP), cardiac output (CO), stroke volume (SV), total vascular resistance (TVR) and end-tidal CO2 were collected. Cerebral vascular blood flow velocity (CBFv) was measured by Transcranial Doppler technology. Central blood pressures (cSBP, cDBP, cMAP), and central pulse wave velocity (PWV) were obtained using an automated ambulatory blood pressure monitor. Carotid artery beta-stiffness index was measured by ultrasonography.

Results: Mean CBFv increased at 1-minute post ($p<0.01$), but decreased below baseline values post 5-minute ($p<0.001$). In contrast, CBFv pulsatility increased following RE and remained significantly elevated at 5-minute post ($p<0.001$). TVR decreased post-RE ($p<0.001$), and returned back to baseline at post 30-mintue (See Table). PWV increased 1-minute post RE ($p<0.001$), returning to baseline values at 5-minutes. There were no increases in beta-stiffness index.

Conclusion: RE increased aortic stiffness, mean CBFv and CBFv pulsatility. Despite an increase in CO at 5-minute, mean CBFv drops below baseline values and CBFv pulsatility continued to rise further above baseline. This temporary disruption in cerebral autoregulation may impact brain health.

Variables	Baseline	1-minute	5-minute	30-minute
Heart Rate (bpm) *	68 ± 9 abc	89 ± 11 bc	78 ± 11	76 ± 9
CO (L/min) *	5.2 ± 1.0 ab	8.1 ± 1.5 bc	6.3 ± 1.2 c	5.5 ± 1.1
SV (ml/min) *	76.0 ± 17.5 a	90.0 ± 20.0 bc	80.1 ± 16.8 c	72.2 ± 17.0
bSBP (mmHg) *	122 ± 10 a	139± 12 bc	124± 11	123± 8
bDBP (mmHg) *	73 ± 7 a	77 ± 8 b	72 ± 6	75 ± 5
bMAP (mmHg) *	93 ± 8 a	102 ± 9 bc	93 ± 7	95 ± 5
TVR (mmHg*min / L)	18 ± 3 ab	13 ± 2 bc	15 ± 2 c	18 ± 3
cSBP (mmHg) *	108± 11 a	120± 11 bc	113± 8	109± 9
cDBP (mmHg) *	79± 9 a	87± 8 bc	81± 6	81± 8
cMAP (mmHg) *	97± 9 a	107± 8 bc	100± 7	98± 7
PWV (m/s) *	5.2 ± 0.5 a	5.6 ± 0.5 bc	5.3 ± 0.5	5.2 ± 0.4
Carotid Max Diameter (mm) *	6.90 ± 0.54 abc	6.61 ± 0.54	6.62 ± 0.54	6.70 ± 0.55
Carotid Min Diameter (mm) *	6.39 ± 0.50 ab	6.04 ± 0.54	6.10 ± 0.50	6.23 ± 0.53
Beta-Stiffness Index	6.3± 1.6	5.9 ± 1.5	6.1± 1.2	6.4 ± 1.3
CBFv Mean (cm/s) *	59 ± 15 ab	70 ± 23 bc	55 ± 13 c	58 ± 13
CBFv Pulsatility Index *	0.86 ± 0.09 b	0.97 ± 0.19 c	1.02 ± 0.12 c	0.84 ± 0.11
End-Tidal CO2 *	1.95 ± 0.44 ac	2.23 ± 0.55 bc	1.82 ± 0.47	1.75 ± 0.53

All Data are mean ± SD, * Exercise effect, $p<0.05$. **a** significantly different from 1 min, **b** significantly different from 5 min. **c** significantly different from 30 min, $p<0.05$.

Vascular Haemodynamics In Young Adults Born Extremely Preterm

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Objectives: Adverse cardiovascular outcomes following preterm birth have been described in the literature, but few studies have described these in detail in children and adults born extremely preterm (EP). The EPICure study previously reported on vascular haemodynamics in 11-year-olds born <26 weeks gestation,1 and this cohort has now been reassessed to determine outcomes in young adulthood.

Methods: Young adults born EP (n=130) and controls (n=64) were evaluated for detailed haemodynamic evaluations, including direct and indirect measurements for blood pressure (BP), augmentation index (AIx), aortic pulse wave velocity (aPWV), cardiac output (CO), stroke volume (SV) and total peripheral pressure (TPR). Outcomes were compared between EP subjects with and without neonatal bronchopulmonary dysplasia (BPD) and term-born controls.

Results: At 19 years, there were no differences in seated systolic and diastolic BP between groups, but EP subjects had higher supine brachial diastolic BP and mean arterial pressure (MAP). Similar to findings at 11 years, AIx remained significantly higher in EP subjects, and likewise there was no difference in aPWV between groups. Cardiac index was similar between groups, but with faster heart rate and lower stroke volume index, TPR was significantly higher in the EP group. There were no differences between EP subjects with and without BPD, and differences between EP and control groups persisted on adjustment for confounders, including socioeconomic status. (Table 1)

Table 1: Baseline demographic and haemodynamic characteristics of EP and Control groups

	EP n=130	Controls n=64	EP-C Difference in means (95% CI)	P value
Gestational age (weeks)	24.9	≥ 37		
Male, n (%)	60 (45)	25 (38)		
Age (years)	19.3	19.2		
Height (m)	1.64	1.68	-0.04 (-0.07, -0.01)	0.004
Weight (kg)	62.6	69.7	-7.0 (-11.6, -2.5)	0.003
Body Mass Index	23.3	24.7	-1.4 (-2.8, 0.1)	0.063
Waist : Hip ratio	0.85	0.81	0.04 (0.02, 0.06)	<0.001
Seated brachial SBP (mmHg)	119	118	1.4 (-1.7, 4.6)	0.360
Seated brachial DBP (mmHg)	73	72	1.6 (-0.8, 3.7)	0.172
Supine central SBP (mmHg)	101	97	4.2 (1.8, 6.6)	0.001
Non-augmented central SBP (mmHg)	28	30	-1-1 (-2.9, 0.7)	0.225
Supine central DBP (mmHg)	70	67	3.4 (1.2, 5.5)	0.002
Central pulse pressure (mmHg)	31	30	0.8 (-1.1, 1.9)	0.379
MAP (mmHg)	85	81	3.7 (1.5, 5.9)	0.001
AIx, %	6.5	0.4	6.1 (3.4, 8.7)	<0.001
Adjusted AIx[b]	6.6	0.3	6.4 (3.8, 8.9)	<0.001
aPWV (m/s)	5.1	4.9	0.1 (-0.1, 0.3)	0.181
Adjusted aPWV[c]	5.0	5.0	-0.04 (-0.2, 0.1)	0.664
Heart Rate (b.p.m.)	71	67	4.3 (1.0, 7.7)	0.011
Adjusted Heart Rate[a]	71	67	4.7 (1.5, 7.9)	0.004
Cardiac Index (L/min/m^2)	4.3	4.3	0.02 (-0.2, 0.3)	0.849
Adjusted Cardiac Index[a]	4.3	4.3	0.02 (-0.2. 0.3)	0.854
TPR (dyne cm^{-5} s)	972	875	97 (27, 166)	0.007
Adjusted TPR[a]	972	875	100 (31, 169)	0.005

Data are means. AIx, augmentation index; aPWV, aortic pulse wave velocity; b.p.m., beats per minute; BMI, body mass index; DBP, diastolic blood pressure; MAP, mean arterial pressure; SBP, systolic blood pressure; SD, standard deviation; TPR, total peripheral resistance. [a]Data adjusted for sex, height and heart rate. [b]Data adjusted for sex and MAP. [c]Data adjusted for sex.

Conclusion: Vascular haemodynamics remain persistently altered in young adulthood following extremely preterm birth, as shown by increase in AIx but not aPWV. Our findings suggest abnormalities in the resistance vasculature, although it is unclear whether this is structural or functional in origin. Long-term monitoring of cardiovascular risk would be highly recommended in this population.

References: McEniery, C. M. *et al.* Cardiovascular consequences of extreme prematurity: the EPICure study. *J Hypertens* **29,** 1367–1373 (2011).

Decreased Aortic Inertance Increases Susceptibility of Late-Systolic Left Ventricular Ejection to Arterial Wave Reflections

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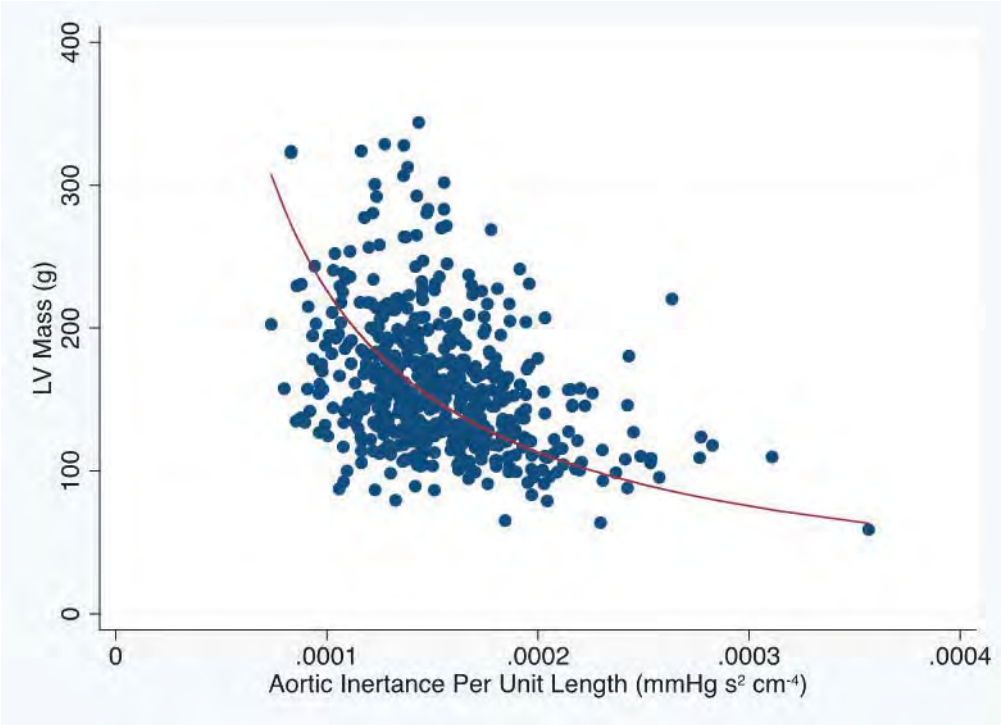
Background: Left ventricular (LV) afterload patterns consisting of late-systolic loading has been linked to LV remodeling and fibrosis in a number of studies. The contributions from arterial wave reflections (WR) has therefore garnered much interest. Aortic dilation may facilitate the adverse effects of WRs through its effect on aortic inertance. Decreased aortic inertance from aortic dilation is particularly important in late-systole, when the LV-aortic pressure gradient generally reverses and ejection decelerates until time of aortic valve closure.

Hypothesis: Decreased aortic inertance from aortic dilation is associated with LV hypertrophy.

Methods: We measured carotid-femoral pulse wave velocity (PWV; a measure of arterial stiffness) and LV mass (LVM) with SSFP-MRI in 409 subjects (mean age = 61 years). Aortic geometry was measured using SSFP-MRI, with a novel 3D aortic analyzer (Medical Imaging Applications, Coralville, Iowa). We computed compliance and inertance from PWV and geometric measurements. Reflection magnitude (RM) was calculated from pressure-flow analysis of calibrated carotid tonometry and aortic flow (PC-MRI).

Results: A non-linear relationship between inertance and LVM was found, with a more pronounced slope at lower inertance values (Figure). After log-transformation of LVM and adjusting for age, height, weight, sex, and area compliance of the thoracic aorta, decreased aortic inertance was independently associated with increased LVM (standardized b=-0.382; P<0.001). Aortic inertance was the strongest predictor of LVM in this model, whereas area compliance was not predictive. There was significant interaction between inertance and RM (P=0.029) such that the negative relationship between inertance and LVM was stronger for greater RM.

Conclusions: Reduced inertance from aortic dilation is independently associated with LV hypertrophy. This is consistent with the principle that reduced inertance diminishes the buffer between pressure gradient transients and aortic flow. In late-systole, augmentation of the negative LV-aortic pressure gradient by WRs imposes a greater deceleration force on LV ejection.



Sex Differences In Vascular Structure And Function In Individuals With Multiple Sclerosis And Healthy Controls

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Objectives: Cardiovascular disease is a leading cause of death in multiple sclerosis (MS), and recent data showed that subclinical markers of atherosclerosis are higher in MS as well. Prevalence of MS in men is much lower than in women, but their prognosis is much worse. Men with MS also have higher rates of hypertension and diabetes than women with MS. Whether vascular function and structure differs in men than in women with MS, and whether potential sex differences are similar to those in healthy controls, is unknown. **Aim:** To compare vascular function and structure between men and women in a group with MS and in healthy controls. **Methods:** After a 10 minute rest in the supine position, resting heart rate (HR) and brachial blood pressure (BP) were collected. Augmentation index (AIX), HR normalized AIX (AIX@HR75) and pulse wave velocity were measured with applanation tonometry. Carotid intima-media thickness (IMT) and beta-stiffness were measured with carotid ultrasound, and Forearm Blood Flow (Baseline, Peak and Area Under the Curve) was measured with strain gauge plethysmography. Data were analyzed with a two-way independent ANOVA for factors group, sex and group*sex. **Results:** In both groups, men were taller and heavier than the women, had higher SBP, lower AIX and AIX@HR75, larger IMT and higher baseline and peak FBF. Different patterns were observed in the sex differences for AIX and AIX@HR75 (in women similar in MS and controls, in men higher in MS than in controls). **Conclusions**: People with MS demonstrate a vascular profile consistent with a higher cardiovascular risk compared to controls. Sex differences were similar in subjects with and without MS, except for the significantly higher AIX and AIX@HR75 in men with MS vs male controls, suggesting males with MS may be particularly at risk for cardiovascular disease.

	Control		MS		p-values factors [#]		
	Female (n=21)	Male (n=18)	Female (n=52)	Male (n=18)	Group	Sex	Interaction
Age	49 ± 10	41 ± 9	48 ± 12	48 ± 13	0.228	0.126	0.109
Height (cm)	164 ± 6	177 ± 5	163 ± 7	179 ± 6	0.867	**<0.001****	0.264
Weight (kg)	69 ± 10	89 ± 13	73 ± 14	88 ± 17	0.679	**<0.001****	0.431
BMI	26 ± 4	28 ± 5	28 ± 6	28 ± 6	0.579	0.218	0.243
HR rest	59 ± 9	60 ± 12	65 ± 8	66 ± 12	**0.004****	0.582	0.739
SBP rest	120 ± 12	128 ± 8	119 ± 16	125 ± 12	0.440	**0.015***	0.672
DBP rest	76 ± 9	76 ± 11	72 ± 10	77 ± 8	0.429	0.269	0.341
MAP rest	91 ± 10	94 ± 10	88 ± 11	93 ± 9	0.410	0.081	0.688
AIX	31 ± 10	10 ± 15	27 ± 12	17 ± 12	0.510	**<0.001****	**0.038***
AIX@HR75	23 ± 8	3 ± 16	22 ± 11	13 ± 9	0.074	**<0.001****	**0.018***
PWVc	6 ± 1	7 ± 1	7 ± 2	7 ± 2	0.058	0.695	0.675
PWVc/MAP	0.07 ± 0.01	0.07 ± 0.01	0.08 ± 0.02	0.08 ± 0.02	**0.013***	0.525	0.445
IMT	0.45 ± 0.08	0.51 ± 0.11	0.53 ± 0.12	0.6 ± 0.13	**0.001****	**0.010***	0.985
Beta	7.04 ± 2.21	6.64 ± 2.04	7.25 ± 2.03	8.07 ± 3.57	0.104	0.675	0.227
FBF Baseline	3.1 ± 1.3	3.7 ± 1	1.9 ± 0.9	2 ± 0.9	**<0.001****	0.099	0.203
FBF Peak	20.6 ± 7.1	27.2 ± 7	15.6 ± 5.8	20.5 ± 6.6	**<0.001****	**<0.001****	0.533
FBF AUC	70 ± 23.3	94 ± 27.7	58 ± 22.2	68 ± 26.6	**<0.001****	**0.001****	0.160

[#] two-way independent ANOVA with Group, Sex and Group*Sex as factors

* $p<0.05$

** $p<0.01$

Animal Models of Local Aortic Stiffening: The Effect of Salt in SHRSP

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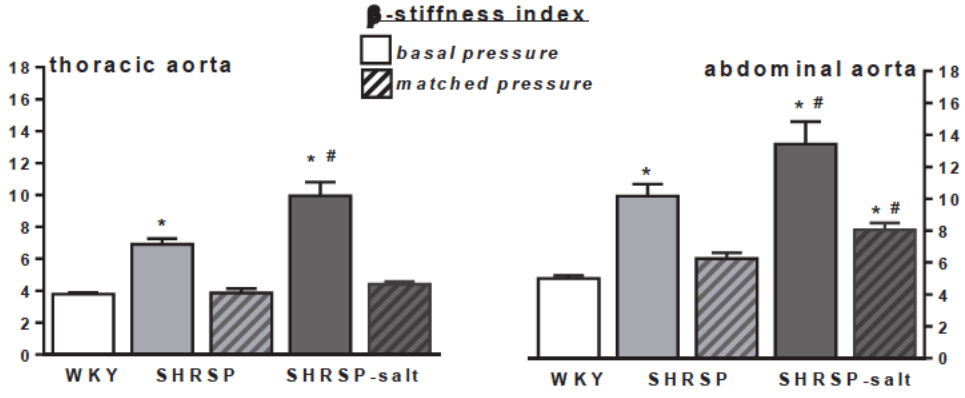
Objectives: the cardiovascular risk associated with central artery stiffening is recognized but better understanding of the mechanisms and therapeutic strategies are needed. Therefore animal models for evaluating stiffness are crucial. We and others use different approaches to calculate aortic stiffness. Pulse wave velocity (PWV) is relatively easily measured and is indispensable, despite being highly influenced by blood pressure. Local determination using echotracking allows us to determine stiffness at various levels of the arterial tree and to measure the pulsatile diameter wave (distension). Previously, using this technique, we have shown the presence of an aortic wall stiffening in spontaneously hypertensive rats (SHR) when the pressure effect is complicated by a reduction of nitric oxide or by aging (1-2). Age, endothelial dysfunction and salt are main contributors in human cardiometabolic pathologies.

Methods: we have evaluated the effect of salt in stroke prone SHR (SHRSP). SHRSP-salt (4.5 % NaCl diet 5 weeks, n=7), compared to normotensive rats Wistar Kyoto (WKY) and SHRSP with normal diet (n=6-6). After pentobarbital anesthesia, parameters were measured at basal and again at reduced blood pressures (following acute clonidine administration), in the thoracic (TA) and abdominal aorta (AA).

Results: at basal pressure both the TA and AA presented decreased distensibility, distension and distension-pressure loop, increased β-stiffness index (figure) and local PWV in the SHRSP and SHRSP-salt. Following clonidine administration to match the WKY basal blood pressure (130 mmHg), only parameters acquired from the AA of SHRSP-salt remained altered.

Conclusions: this study confirms the potency of ultrasonic derived stiffness measurements and that aortic remodeling is non-uniform along the aortic trunk. It shows that salt in addition to hypertension develops central artery stiffening; after 5 weeks the TA presents a pressure-dependent and AA both pressure dependent and independent stiffening.

(1: Vayssettes-Courchay et al., 2011, 2: Lindesay et al., 2016)



No Sex Differences in the Cardiovascular Response to Mental-Stress in Older Adults

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Mental stress elicits increases in blood pressure (BP) and arterial stiffness, the magnitude of which, is subject to sex differences. Women tend to have blunted increases in BP compared to men that are driven by cardiac excitation, rather than changes in peripheral resistance. These sex differences have primarily been documented in younger, pre-menopausal women, and through the measurement of peripheral (i.e. brachial) BP, which may differ from responses in the central vasculature (i.e. carotid artery).

PURPOSE: Investigate sex differences in the cardiovascular response to mental stress among older adults.

METHODS: 91 older adults (n=46 men, 68±6 yrs, BMI 27.1±3.7 $kg \cdot m^{-2}$; n=45 women, 67±7 yrs, BMI 25.3±3.6 $kg \cdot m^{-2}$) underwent cardiovascular measures at rest and during a mental stress protocol. Mental stress was induced using a 4-minute computerized incongruent Stroop task. Brachial and carotid systolic (SP), diastolic (DP) and pulse pressure (PP) were measured via a brachial oscillometric cuff and applanation tonometry, respectively. Carotid waveforms were calibrated to brachial mean pressure and DP. Carotid-femoral pulse wave velocity and common carotid artery (CCA) elastic modulus (Ep, calibrated to carotid SP and DP) were assessed as measures of aortic and carotid stiffness, respectively.

RESULTS: Significant group effects were detected for brachial SP, PP, HR, and PWV, with men having greater PWV, but lower BP than women ($p<0.05$). Significant time effects were observed for brachial and carotid pressures, HR, PWV, and CCA Ep, which increased during mental stress. No significant sex-by-time interactions were detected, indicating similar responses to mental stress between sexes.

CONCLUSIONS: Mental stress resulted in acute increases in peripheral and central blood pressure and large artery stiffness. Although men had consistently higher PWV and lower brachial BP than women at rest and during mental stress, the magnitude of the cardiovascular responses to mental stress were similar between sexes.

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Table 1: Cardiovascular responses to mental-stress among older men and women. (Mean ± SD)

	Men (n=46)		*Women (n=45)*		*Effects*		
Variable	Rest	Mental-stress	Rest	Mental-stress	Sex	Time	SxT
Brachial SP (mmHg)	124 ± 12	139 ± 16	127 ± 14	145 ± 20	**0.035**	**0.001**	0.545
Brachial DP (mmHg)	79 ± 7	86 ± 7	79 ± 7	86 ± 9	0.769	**0.001**	0.677
Brachial PP (mmHg)	45 ± 8	53 ± 12	49 ± 9	59 ± 12	**0.004**	**0.001**	0.558
Carotid SP (mmHg)	116 ± 12	129 ± 17	118 ± 13	131 ± 16	0.306	**0.001**	0.769
Carotid PP (mmHg)	37 ± 9	43 ± 14	39 ± 9	45 ± 10	0.218	**0.001**	0.903
HR ($b \cdot min^{-1}$)	60 ± 10	66 ± 10	63 ± 9	70 ± 13	**0.023**	**0.001**	0.735
PWV ($m \cdot s^{-1}$)	10.2 ± 2.6	11.2 ± 2.6	9.3 ± 2.7	10.1 ± 3.1	**0.020**	**0.023**	0.763
CCA Ep (kPa)	100.58 ± 35.40	125.01 ± 50.23	107.89 ± 46.99	123.64 ± 55.57	0.674	**0.005**	0.540

SP, systolic pressure; DP, diastolic pressure; PP, pulse pressure; HR, heart rate; PWV, pulse wave velocity; CCA, common carotid artery; Ep, elastic modulus; SxT, sex-by-time interaction.

Improvement in Post-Transplant Hypertension in Living Donor Renal Transplantation

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Objectives: Since genetic factor determines part of hypertensive phenotype, we aim to demonstrate the role of transplanted kidney from normotensive living donors in post-transplant hypertension (HTN).

Methods: From 1.5-year-medical record review yielded 103 kidney transplant recipients in whom living-donor renal transplantation (LDRT) was performed in 32 (15 living-related renal transplantation (LRRT) and 17 living-unrelated renal transplantation (LURT)).

Results: Of all 32 recipients, mean age was 51.30 years old (21.42-79.53) and 50% were male. Mean duration of follow-up was 8.4 months (0.63-16.33). Up to 93.75% of recipients had pre-transplant hypertension, and 56.25% became non-hypertensive after transplantation, which was defined as SBP≤140, DBP≤90, or being on ≤2 BP agents regardless SBP or DBP (Figure 1). Mean post-transplant systolic blood pressure (SBP) was lower than pre-transplant SBP but not statistically significant (132.88+/-2.54 vs.134.75+/-3.01, p= 0.6366) as same as mean DBP (77.84+/-1.88 vs. 82.25+/-2.39, p= 0.1520). The number of pre- and post-transplant blood pressure medications was 1.94 and 1.28, respectively. in LRRT group, 5 of 13 (38.46%) pre-transplant hypertensive patients became normotensive while 11 of 17 (64.71%) patients in LURT group were non-hypertensive (Figure 2). Mean post-transplant SBP was higher than mean pre-transplant SBP in LRRT group (1.33.73+/-3.33 vs.129.67+/-4.40, p=0.4680); however, mean post-transplant DBP in LRRT group (77.93+/-2.68 vs.79.40+/-3.20, p=0.7273) as well as mean SBP (132.12+/-3.85 vs. 139.24+/-3.93, p=0.2049) and mean DBP (77.76+/-2.71 vs. 84.76+/-3.48, p=0.1223) in LURT were lower than those during pre-transplant periods. The mean number of antihypertensive medications was decreased in post-transplant compared to pre-transplant in both LRRT (1+/-0.24 vs.1.73+/-0.33, p=0.0844) and LURT (1.53+/-0.12 vs.2.12+/-0.28, p=0.0616) groups.

Conclusion: Hypertension was resolved in more than half of the pre-transplant hypertensive patients after kidney transplantation. Since higher number of LURT recipients becomes normotensive, the possibility of hypertensive genotype in living-related donor kidneys may contribute to post-transplant HTN in some LRRT recipients.

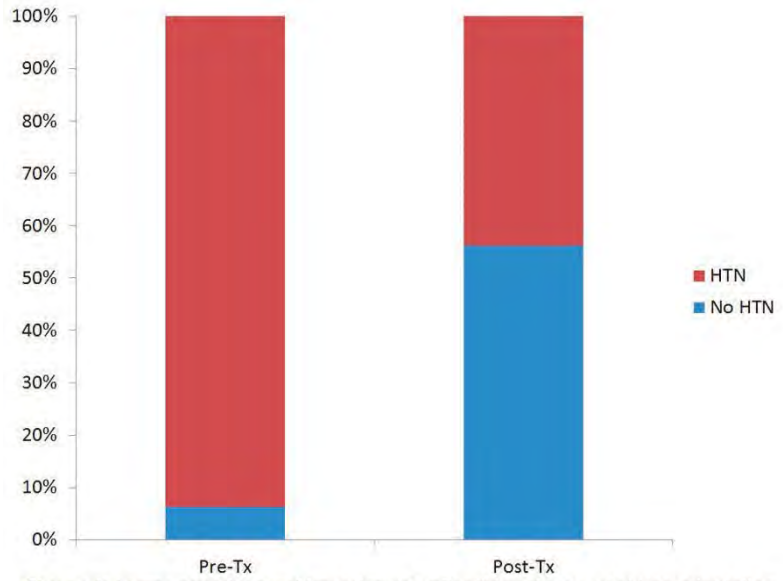

Figure 1: Lower prevalence of hypertension after living donor renal transplantation

Rebound Weight Gain and Blood Pressure Control after Living Kidney Donation and Kidney Transplantation

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Objective: Overweight and obesity are known risk factors of hypertension in both donors and recipients after kidney donation and transplantation, respectively. We aim to study the correlation between blood pressure (BP) and body mass index (BMI) in donor post-donation and in recipients post-transplantation.

Methods: A consecutive 24 paired living kidney donors and recipients were reviewed. Demographic data, systolic blood pressure (SBP), diastolic blood pressure (DBP), and BMI were collected.

Results: Of all 24 donors and recipients, donors trends to be younger than their recipients (mean age 46.54±2.81 vs. 50.32±3.16 years old). Half of the donors and 54.17% (13/24) of the recipients were male. In donor group, mean SBP, but not DBP decreased overtime after donation (SBP 125.58±2.9 vs. 123.69±1.97; p=0.5924 vs. 121.33±3.02; p=0.3181. DBP 74.92±1.7 vs. 75.73±1.12; p=0.6926 vs. 76.85±1.82; p=0.4437). However, BMI decreased at 2-week post-donation, but rebounded above pre-donation BMI at 6 months (BMI 28.19±0.87 vs. 28±0.82; p=0.8750 vs. 28.92±1.03; p=0.5884) (Figure 1A). For recipient group, mean SBP, DBP, and BMI trended down after transplantation. However, these values increased to almost the same levels of pre-transplantation at 3-month post-transplant, and only DBP and BMI trended up beyond pre-transplant values at 6-month post-transplant (Figure 1B). Among 24 donors, 13 and 11 patients were living-related (LRD) and living unrelated donors (LUD), respectively. SBP, but not DBP continuously decreased in both LRD and LUD. Conversely, BMI was up trending in LRD, but decreased at 2-week post-donation, and then rebounded at 6-month (Figure 2A and 2B). Of all 24 recipients, 13 and 11 patients were living-related (LRR) and living unrelated renal transplant recipients (LUR), respectively. SBP, DBP, and BMI in LRR decreased until 1-month post-transplant and increased to above pre-transplant levels at 6-month post-transplant without statistical significance (Figure 2C). LUR group had the same patterns of SBP, DBP, and BMI, but SBP and DBP at 1-week and 1-month post-transplantation almost significantly decreased from the pre-transplant levels (Figure 2D).

Conclusion: BP and BMI in both donors and recipients seem to be positively correlated, and BMI rebounded beyond the pre-donation and pre-transplant levels. Early post-transplant SBP and DBP appear to be better improved in LUR than LRR group.

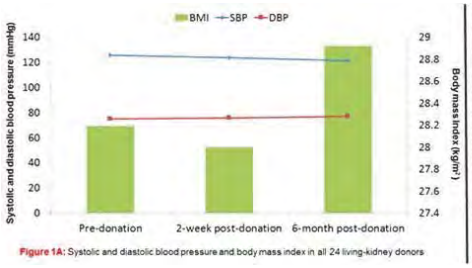

Figure 1A: Systolic and diastolic blood pressure and body mass index in all 24 living-kidney donors

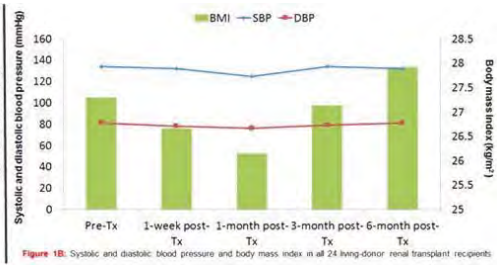

Figure 1B: Systolic and diastolic blood pressure and body mass index in all 24 living-donor renal transplant recipients

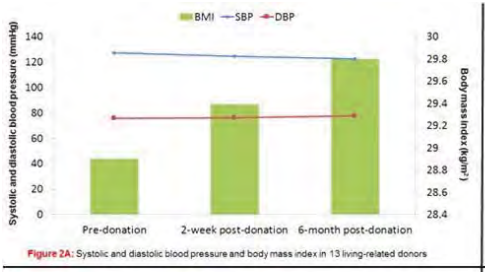

Figure 2A: Systolic and diastolic blood pressure and body mass index in 13 living-related donors

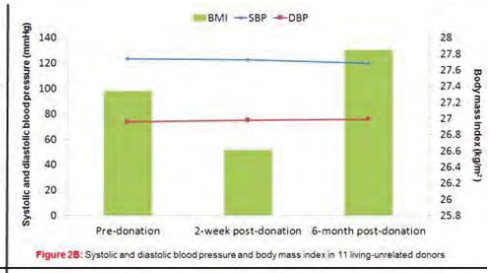

Figure 2B: Systolic and diastolic blood pressure and body mass index in 11 living-unrelated donors

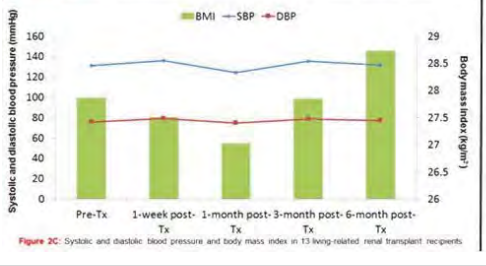

Figure 2C: Systolic and diastolic blood pressure and body mass index in 13 living-related renal transplant recipients

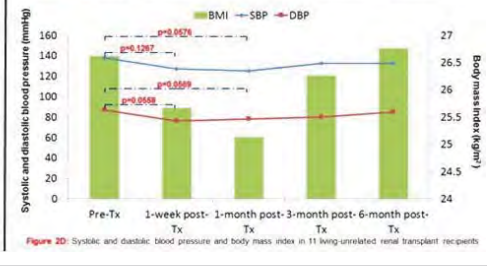

Figure 2D: Systolic and diastolic blood pressure and body mass index in 11 living-unrelated renal transplant recipients

Psoriasis Is Associated with Increased Arterial Stiffness: A Systematic Review and Meta-Analysis

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Background

Studies have shown that patients with psoriasis have higher risk of cardiovascular disease (CVD), independent of traditional CVD risk factors. However, pathophysiology of the development of CVD from psoriasis is not well known. arterial stiffness has been recognized as an independent predictor of cardiovascular risk. It is controversial whether psoriasis and arterial stiffness is associated. In this systematic review and meta-analysis, we sought to assess the hypothesis that patients with psoriasis have increased arterial stiffness compared with controls.

Methods

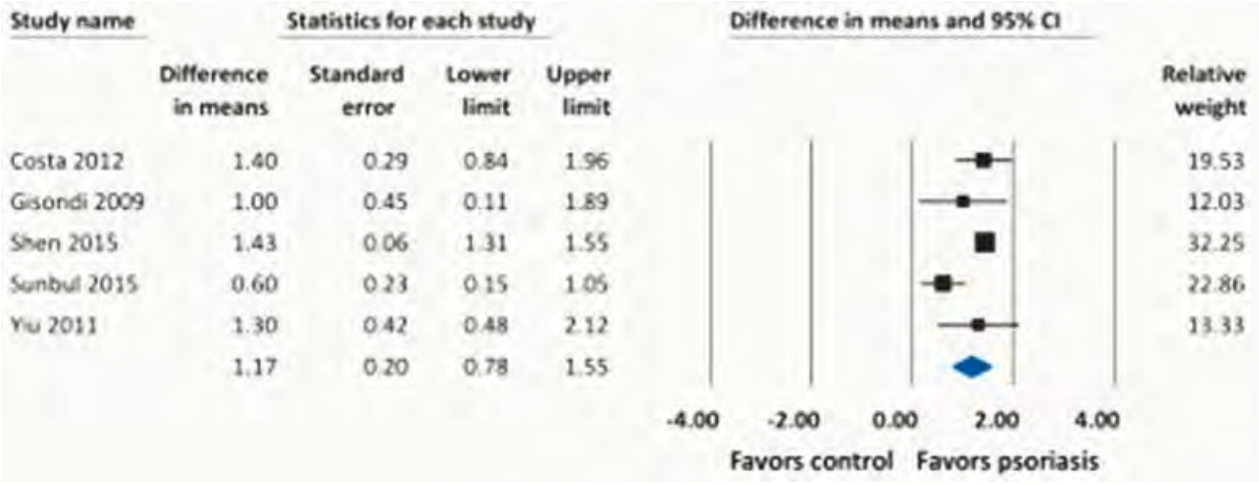
Systematic literature search was performed using MEDLINE and EMBASE databases from inception to May 2016. We included original research publications that contained data on arterial stiffness and psoriasis. Aortic pulse wave velocity (aPWV) is the non-invasive marker for assessment of arterial stiffness. Aortic PWV was calculated from two different recording sites (carotid and femoral) and the heart. We compared aPWV between patients with psoriasis controls and estimated the pooled mean difference (MD) of aPWV using a random-effects model meta-analysis. Heterogeneity across the included studies was quantified using Q statistic and I^2.

Results

Data from five observational studies involving 438 participants (233 with psoriasis) were extracted and included in the meta-analysis. Pooled MD of aPWV was 1.17 m/sec higher in patients with psoriasis compared with controls (95 % CI: 0.78-1.55, P-value<0.01, I^2 = 69%). There is no change in the direction or statistical significance of MD of aPWV after removing each study at a time in sensitivity analysis.

Conclusion

Psoriasis is associated with increased arterial stiffness. Assessment of arterial stiffness parameters may be important for early detection of cardiovascular deterioration in psoriasis patients.



Increased Arterial Stiffness in Behcet's Disease: A Systematic Review and Meta-Analysis

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Background:

Behçet's disease (BD) a systemic vasculitis characterized by genital, oral or skin lesions, uveitis, and vascular complications. Studies have shown increased arterial stiffness in systemic immune and inflammatory diseases, such as rheumatoid arthritis and systemic lupus erythematosus. However, whether patients with BD have increased arterial stiffness is still debatable. This meta-analysis aimed to compare arterial stiffness parameter in subjects with diagnosis of BD to normal subjects.

Methods:

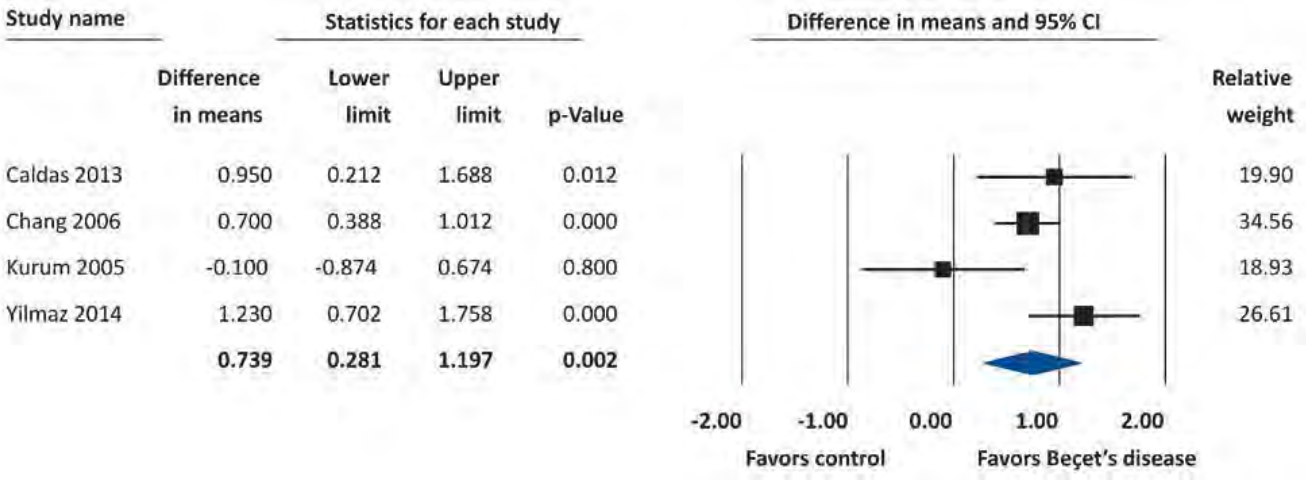
A comprehensive search of the databases of the MEDLINE and EMBASE was performed from inception through May 2016. The inclusion criterion was the observational studies' assessment of the association between BD and arterial stiffness in adult subjects. BD patients met the International Study Group criteria for diagnosis of Behçet's disease. Aortic stiffness was assessed using carotid-femoral pulse wave velocity (PWV) measurements as an indicator. Pooled mean difference (MD) of PWV and 95% confidence interval (CI) were calculated using a random -effect, generic inverse variance meta-analysis. The between-study heterogeneity of effect-size was quantified using the *Q* statistic and I^2.

Results:

Data were extracted from 4 observational studies involving 303 subjects. PWV is significantly higher in patients with Behçet's disease compared with controls (MD=0.74;95% CI: 0.28-1.20, P-value=0.002, I^2=63%).

Conclusion:

In this meta-analysis, we observe that PWV, an ideal indicator of arterial stiffness, is increased in patients with Behçet's disease compared with controls. Prospective studies in a large population should be done to determine the pathophysiological and prognostic implications of increased arterial stiffness in BD.



Association between White-Coat Hypertension and Arterial Stiffness: A Systematic Review and Meta-Analysis

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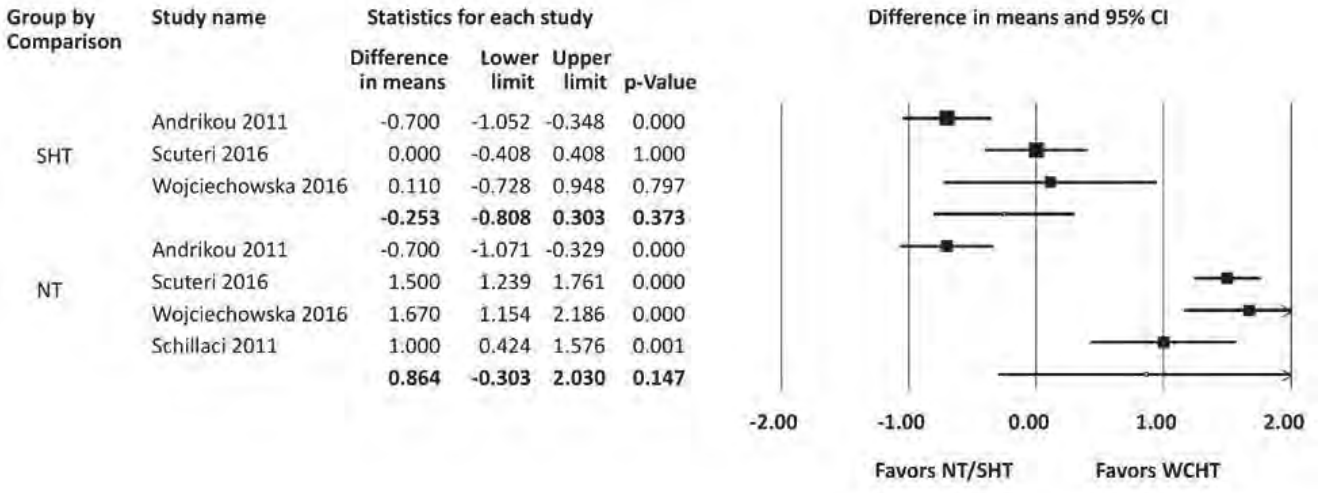
Background: Previous studies have shown inconclusive effects of target organ damage from white-coat hypertension (WCHT). Arterial stiffness is involved in the atherosclerotic processes in the setting of sustained hypertension. This meta-analysis aimed to compare arterial stiffness in subjects with diagnosis of WCHT to subjects with normotension (NT) and SHT.

Methods: A comprehensive search of the databases of the MEDLINE and EMBASE was performed from inception through May 2016. The inclusion criterion was the observational studies' assessment of the association between WCHT and NT or SHT in adult subjects. European Society of Hypertension practice guidelines for ambulatory blood pressure (BP) monitoring was used to define WCHT (office BP≥140/90mmHg and daytime BP <135/85mmHg), and SHT (office BP≥140/90mmHg and daytime BP≥135/85mmHg). Aortic stiffness was assessed using Carotid-femoral pulse wave velocity (PWV) measurements. Pooled mean difference (MD) of PWV and 95% confidence interval (CI) were calculated using a random-effect, generic inverse variance method.

Results: Data were extracted from 4 observational studies (1-4) involving 2,413 subjects. PWV is not different in patients with WCHT compared with SHT (pooled MD= -0.25 m/sec; 95% CI, -0.81 to 0.30; P-value=0.37, I^2=74%). PWV in WCHT is also not different when compared with PWV in NT (MD= 0.86 m/sec; 95% CI, -0.30 to 2.03; P-value=0.15, I^2=97%).

Conclusion: In a meta-analysis, we observed that arterial stiffness measured by pulse wave velocity is not different in patients with white-coat hypertension when compared with sustained hypertension or normotension.



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Association between Masked Hypertension and Arterial Stiffness: A Systematic Review and Meta-Analysis

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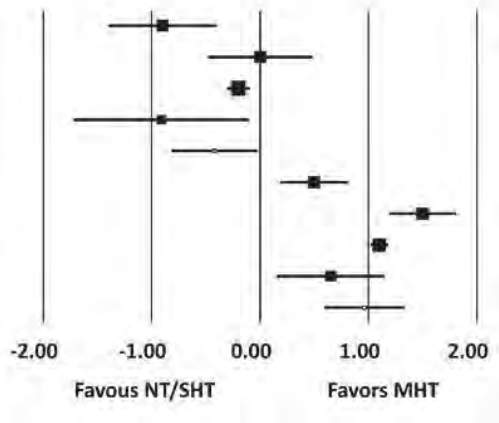
Background: Previous studies have shown inconclusive effects of target organ damage from masked hypertension (MHT). Arterial stiffness is involved in the atherosclerotic processes in the setting of sustained hypertension. Few studies assessed the role of MH on arterial stiffness compared with sustained hypertension (SHT), but results are still debatable. This meta-analysis aimed to compare arterial stiffness parameter in subjects with diagnosis of MHT to subjects with normotension (NT) or SHT.

Methods: A comprehensive search of the databases of the MEDLINE and EMBASE was performed from inception through May 2016. The inclusion criterion was the observational studies' assessment of the association between MHT and normotension or sustained hypertension in adult subjects. European Society of Hypertension practice guidelines for ambulatory blood pressure (BP) monitoring was used to define MHT (office <140/90mmHg and daytime BP≥135/85mmHh) and SHT (office BP≥140/90mmHg and daytime BP≥135/85mmHg). Aortic stiffness was assessed using Carotid-femoral pulse wave velocity (PWV) measurements. Pooled mean difference (MD) of PWV and 95% confidence interval (CI) were calculated using a random-effect, generic inverse variance method. The between-study heterogeneity of effect-size was quantified using the *Q* statistic and I^2.

Results: Data were extracted from 4 observational studies (1-4) involving 3,288 subjects. PWV is significantly lower in patients with MHT compared with SHT (MD=-0.42; 95% CI -0.82 to -0.02, P-value=0.04, I^2=72%). Compared with patients with NT, patients with MHT had significantly higher PWV (MD=0.96; 95% CI 0.59 to 1.34, P-value<0.001, I^2=87%).

Conclusion: In this meta-analysis, we observe that arterial stiffness in masked hypertension is higher than in normotension. However, arterial stiffness in masked hypertension is lower than in sustained hypertension. Physician should raise awareness of arterial stiffness detection in patients with masked hypertension.

Group by Comparison	Study name	Difference in means	Lower limit	Upper limit	p-Value
SHT	Andrikou 2011	-0.900	-1.403	-0.397	0.000
	Scuteri 2016	0.000	-0.484	0.484	1.000
	Ware 2016	-0.200	-0.311	-0.089	0.000
	Wojciechowska 2016	-0.910	-1.725	-0.095	0.029
		-0.423	**-0.824**	**-0.021**	**0.039**
NT	Andrikou 2011	0.500	0.183	0.817	0.002
	Scuteri 2016	1.500	1.186	1.814	0.000
	Ware 2016	1.100	1.014	1.186	0.000
	Wojciechowska 2016	0.650	0.151	1.149	0.011
		0.961	**0.588**	**1.335**	**0.000**



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Central Hemodynamics and Arterial Stiffness in Young Obese Adults: the Preliminary Finding

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Changes in central hemodynamics and arterial stiffness are associated with augmented cardiovascular risks and have been reported in obese adults with metabolic syndrome. It is unclear whether this observation may also be present in young healthy obese adults with normal metabolic profile. **Objectives:** To compare measures of central hemodynamics and arterial stiffness in young normal-weight vs. obese adults. **Methods:** There were 11 normal-weight (female=6; age 25±2 yrs; BMI 22.4±0.6 kg/m^2) and 13 obese adults (female=6; age 27±1 yrs; BMI 32.7±0.6 kg/m^2). Central hemodynamics were measured using SphygmoCor and wave separation analysis. Ultrasonography was used to measure carotid intima-media thickness (cIMT) and arterial stiffness (beta stiffness (β), elastic modulus (Ep), arterial compliance (AC)). Cardio-ankle vascular index (CAVI) was measured using VaSera and is another index reflecting the stiffness of the artery from the heart to ankles. Percent fat was determined using DEXA. **Results:** Obese adults exhibited higher percent body fat and cIMT than normal-weight adults (P<0.05), with no group differences in metabolic profile. No group differences were observed for brachial and aortic blood pressures, heart rate, arterial stiffness, and wave separation variables. **Conclusion:** The larger carotid intima-media thickness in young obese adults suggest early remodeling of the vasculature as a result of obesity. However, young obese adults with normal metabolic profile still exhibited comparable central hemodynamics and arterial stiffness as normal-weight adults, suggesting preserved vascular health despite initial carotid vascular remodeling.

Table 1. Comparisons of central hemodynamics and arterial stiffness in normal-weight and obese adults.

	Normal-Weight (n=11)	Obese (n=13)
Percent body fat (%) *	31.1±1.7	41.9±1.7
Total cholesterol	180±14	176±11
High density lipoprotein (mg/dL)	62±3	51±5
Low density lipoprotein (mg/dL)	103±14	110±12
Triglycerides (mg/dL)	100±19	85±11
Glucose (mg/dL)	96±4	98±5
Brachial SBP (mmHg)	109±1	109±3
Brachial DBP (mmHg)	70±2	73±2
Aortic SBP (mmHg)	93 ±3	96±3
Aortic DBP (mmHg)	65±2	69±2
HR (bpm)	62±2	58±3
cIMT (mm) *	0.37±0.01	0.44±0.02
CAVI	6.0±0.2	6.0±0.2
β-Stiffness	5.5±0.4	5.2±0.4
Ep (kPA)	66.3±5.3	62.8±5.3
AC (%)	1.02±0.07	1.20±0.09
AIx (%)	7±4	6±3
AIx@75 (%)	0±3	-2±3
AP (mmHg)	2±1	2±1
FPH (mmHg)	25±1	25±1
RPH (mmHg)	38±6	34±6
RI (%)	19±4	24±9

Data are mean±SE. BMI, body mass index; cIMT, carotid intima-media thickness; CAVI, cardio-ankle vascular index; β-stiffness, beta stiffness; Ep, elastic modulas; AC, arterial compliance; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; HR, heart rate; AIx, augmentation index; AIx@75, augmentation index normalized to heart rate of 75 bpm; FPH, forward pulse height; RPH, reflected pulse height; RI, reflection index. *significant group difference based on an independent *t*-test (P<0.05).

Vascular Function in individuals with Down Syndrome

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Individuals with Down syndrome (DS) experience premature aging. Arterial stiffness increases with advancing biological age and predicts cardiovascular disease. . However, only limited studies investigated arterial function in individuals with DS. Thus, the impact of DS on vascular function still remains poorly understood.

Purpose: To compare vascular function between individuals with and without DS (control).

Methods: Twenty-seven volunteers (DS=13, Control=14) participated in this study. Central arterial stiffness indices (β-stiffness, Ep and circumferential strain) were measured by carotid ultrasonography and analyzed with B-mode, echo tracking and strain analysis. Cardio-ankle Vascular Index (CAVi) and carotid blood pressure (carBP) were measured using a limb cuff system and applanation tonometry (SphygmoCor), respectively. In addition, heart rate (HR) was recorded by finger photoplethymography.

Results: There were significant differences in CAVi (lower) and circumferential strain (higher) in individuals with DS compared to individuals without DS (p<0.05). No group differences were observed for β-stiffness and Ep.

Conclusions: Our results suggest that individuals with DS have lower arterial stiffness than that of individuals without DS. Interestingly, circumferential carotid strain was greater in persons with DS, with no differences in B-stiffness, suggesting the greater strain may have been a function of greater pulse pressure in individuals with DS.

	DS (N = 13)	Control (N = 14)
carSBP (mmHg)	133 ± 22	126 ± 14
carDBP (mmHg)	74 ± 8	75 ± 9
HR (bmp)	62.5 ± 11.9	64.3 ± 13.1
Circumferential Strain ‡	9.94 ± 3.37	7.48 ± 2.56
R-CAVI ‡	4.86 ± 0.83	5.84 ± 0.68
L-CAVI ‡	4.97 ± 0.95	5.81 ± 0.68
B-Stiffness	5.24 ± 1.40	5.65 ± 1.99
Ep	72.38 ± 20.84	76.29 ± 20.48

‡ Significant group difference. Mean ± SD, Significance level, p < 0.05

Multiple Sclerosis Patients Experience More Decrements in Carotid Artery Functional Properties with Aging than Age-Matched Peers

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INTRODUCTION: Peak prevalence of multiple sclerosis (MS) is approaching 60 years of age, suggesting an aging patient population compared to past reports. Aging is independently associated with increased cardiovascular disease risk. Additionally, arterial function is compromised with aging. Carotid artery stiffness serves as a non-invasive method to quantify aspects of arterial function. As MS patients increase their average lifespan, it is unclear if they may experience differential changes in aspects of carotid artery function compared to their healthy age-matched peers.

OBJECTIVE: To compare carotid artery structure and function between young and older subjects with and without MS.

METHODS: After 10 minutes of supine rest, 120 subjects (MS=89, Control=31) underwent applanation tonometry and ultrasonography of the carotid artery. Subjects were classified as young or older (<50 and ≥50 years, respectively).

RESULTS: See table below. In those with MS, carotid artery pulse pressure (PP), carotid intima media thickness (IMT), beta stiffness, and elastic modulus were higher, and arterial compliance was lower, in the older group compared to young subjects, whereas no differences were detected between young and older subjects in the control group.

CONCLUSION: These data show that older subjects with MS exhibit more structural and functional alterations in carotid artery indices than older controls compared to their young counterparts. This highlights the importance of increased efforts to explore early interventions to preserve arterial function in those with MS.

	Control (n=31)		MS (n=89)	
	Young (n=15)	Older (n=16)	Young (n=44)	Older (n=45)
Carotid SBP (mmHg)	113.5 ± 3.7	116 ± 3.0	103.2 ±1.6	112.0 ± 3.6
Carotid DBP (mmHg)	74.8 ± 2.2	76.8 ± 2.2	70.3 ± 1.2	74.1 ± 1.7
Carotid MAP (mmHg)	90.1 ± 2.5	91.9 ± 2.4	84.0 ± 1.3	90.3 ± 1.9
Carotid PP (mmHg)	38.7 ± 2.2	39.2 ± 2.4	32.9 ± 0.9	40.1 ± 1.4*
Carotid IMT (mm)	0.41 ± 0.02	0.48 ± 0.02	0.48 ± 0.01	0.61 ± 0.02*
Beta Stiffness (AU)	6.30 ± 0.46	7.38 ± 0.56	6.02 ± 0.30	8.68 ± 0.40*
Elastic Modulus (kPa)	77.31 ± 6.68	93.67 ± 7.32	68.76 ± 3.57	109.27 ± 5.56*
Arterial Compliance (mm^2/kPa)	1.11 ± 0.10	0.87 ± 0.05	1.17 ± 0.67	0.81 ± 0.05*

Mean ± SEM. *Significant difference between Young and Older groups.

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Relations Between Aortic Stiffness And Left Ventricular Mechanical Function

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Objectives: Left ventricular contraction produces longitudinal strain in the proximal aorta. As a result, aortic stiffening may impair optimal mechanical ventricular-vascular coupling and left ventricular (LV) systolic function, particularly in the long axis. LV global longitudinal strain (GLS) has recently emerged as a sensitive measure of early cardiac dysfunction. In this study, we investigated the relation between aortic stiffness and GLS in a large community-based sample.

Methods: In 2516 participants (age 39-90 years, 57% women) of the Framingham Offspring and Omni cohorts, free of cardiovascular disease, we performed tonometry to measure aortic stiffness and echocardiography to assess cardiac function. Aortic stiffness was evaluated as carotid-femoral pulse wave velocity (CFPWV) and as characteristic impedance (Zc), and GLS was calculated using speckle tracking-based measurements.

Results: In multivariable analyses adjusting for age, sex, height, systolic blood pressure, augmentation index, LV structure, and additional cardiovascular disease risk factors, increased CFPWV (β±SE: 0.122±0.030 SD strain per SD CFPWV, P<0.0001) and Zc (0.091±0.029 SD/SD, P=0.002) were both associated with worse (less negative) GLS. We observed effect modification by sex of the relation between Zc and GLS (P=0.004); in sex-stratified multivariable analyses, the relation between greater Zc and worse GLS persisted in women (0.145±0.040, P=0.0003) but not in men (P=0.73).

Conclusion: Higher aortic stiffness was associated with worse GLS after adjusting for hemodynamic variables. Parallel reductions in LV long axis shortening and proximal aortic longitudinal strain in individuals with a stiffened proximal aorta may represent a manifestation of abnormal direct mechanical ventricular-vascular coupling.

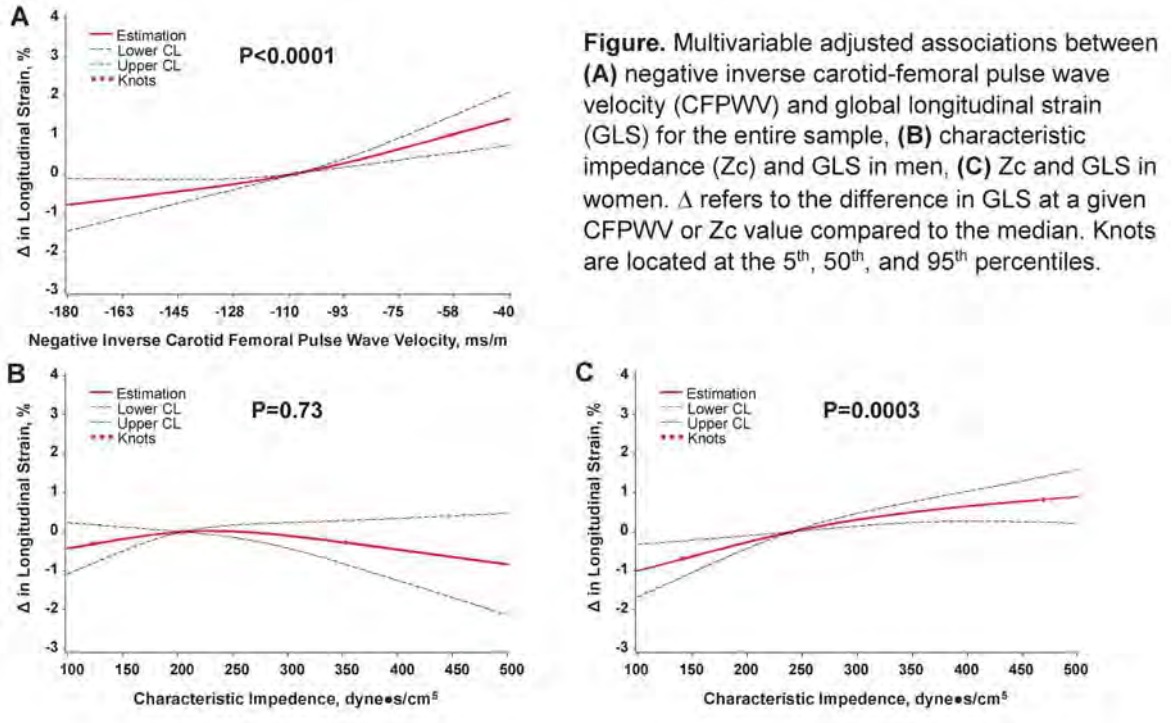

Figure. Multivariable adjusted associations between **(A)** negative inverse carotid-femoral pulse wave velocity (CFPWV) and global longitudinal strain (GLS) for the entire sample, **(B)** characteristic impedance (Zc) and GLS in men, **(C)** Zc and GLS in women. Δ refers to the difference in GLS at a given CFPWV or Zc value compared to the median. Knots are located at the 5th, 50th, and 95th percentiles.

Sex Differences in Vascular Function Following Antioxidant Supplementation

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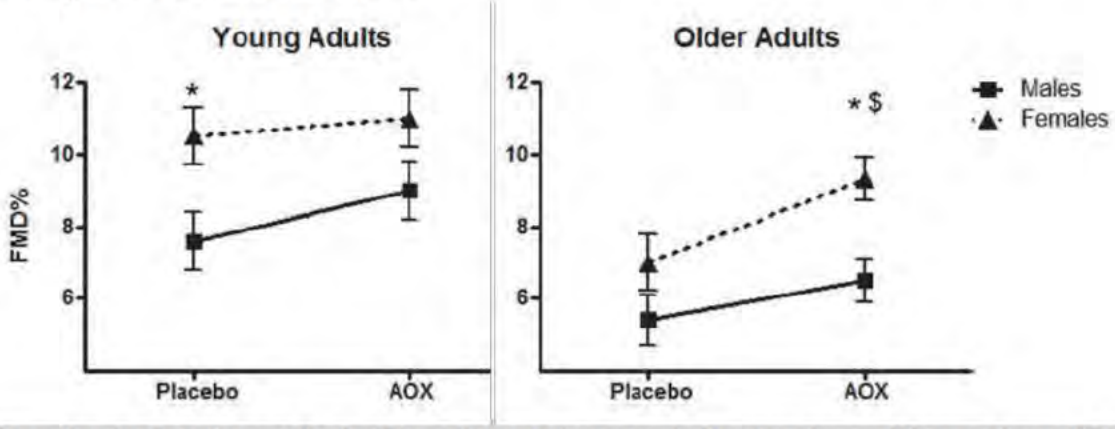
Objectives: Sex differences in cardiovascular disease risk and progression are well established. Estrogen loss following menopause leads to vascular dysfunction, potentially due to elevations in oxidative stress and subsequent decrements in nitric oxide. It is possible a reduction in oxidative stress utilizing an antioxidant supplement could improve vascular function in older females. **Methods:** Forty-seven young (27 ± 0.5 years, 23 M and 24 F) and 46 older (59 ± 0.7 years, 23 M and 23 F) subjects underwent measures of vascular function following both placebo and antioxidant supplementation in a randomized, double-blind, crossover study. **Results:** Young males displayed higher central and peripheral pressures, stiffer arteries and decreased macrovascular endothelial function when compared to young females, and this was reversed with aging, with females developing stiffer arteries, higher pressures and endothelial dysfunction to match the older male group. Young males were more responsive to AOX and showed improvements in macrovascular function following AOX. In the older group, although both males and females improved FMD% with AOX, females were more responsive and improved significantly more. **Conclusions:** These results demonstrate the potential role of oxidative stress in estrogen loss and subsequent arterial dysfunction, possibly due to reductions in nitric oxide bioavailability.

Table: Pressure and vascular response following placebo and AOX supplementation in Young and Older Adults.

	Young (n=47)				Older (n=46)			
	Males (n=23)		Females (n=24)		Males (n=23)		Females (n=23)	
	Placebo	AOX	Placebo	AOX	Placebo	AOX	Placebo	AOX
bSBP (mmHg) #	126±2*	125±2*	106±2	105±2	128±4	127±3	127±4	125±3
bDBP (mmHg) #	71±1*	69±1*$	64±1	65±1	76±2	75±2	77±2	77±2
aSBP (mmHg) #	106 ± 1	105 ± 2	93 ± 1	91 ± 2	119 ± 4	118 ± 3	120 ± 4	119 ± 3
cPWV (m/s) #	6.4±0.2	6.1±0.6	5.9±0.2	6.4±0.6	8.1±0.5	8.5±0.4	8.4±0.5	7.4±0.5
Carotid Arterial Compliance (mm^2/kPa) #	1.1±0.8*	1.1±0.6 *	1.5±0.8	1.4±0.6	0.95± 0.59*	0.91± 0.51	0.77± 0.59	0.80± 0.51

Significance p<0.05, Mean ± SEM. AOX, antioxidant supplementation; bSBP, brachial systolic blood pressure; bDBP, brachial diastolic blood pressure; aSBP, aortic systolic blood pressure; cPWV, central pulse wave velocity.
*significant sex difference
$ significantly different from placebo
# significant age group differences

Figure: Flow Mediated Dilation Following Placebo and AOX supplementation in Young and Older Adults. There were significant differences between age groups at both placebo and AOX condition. *denotes a significant difference between sexes, $ denotes a significant difference from placebo.



Pulse Wave Velocity Is Increased With Experimental Sleep Restriction in Healthy Humans

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Objectives: Increased carotid-femoral pulse wave velocity is indicative of vascular stiffening of the central arterial tree. Aortic stiffness is a key risk factor for the development of hypertension and cardiovascular disease. Following acute (24-hour) sleep deprivation, healthy adults exhibit an increase in carotid-femoral pulse wave velocity; however, acute sleep deprivation poorly represents sleep patterns observed in everyday life. With this information in mind, we hypothesized a prolonged (9 day) exposure to restricted sleep (4 hours of sleep per night) would result in increases in carotid-femoral pulse wave velocity in healthy humans.

Methods: Seven (3M, 5F) young (23±1 yrs), healthy adults underwent a 4-day period of acclimation followed by 9 days of experimental sleep restriction (4 hours of sleep per night – from 12:30 AM to 4:30 AM). High-fidelity radial arterial pressure waveforms and carotid-femoral pulse wave velocity were assessed using applanation tonometry (SphygmoCor, AtCor Medical). Subjects were studied on Day 2 (Acclimation) and Day 13 (Restriction).

Results: Sleep restriction resulted in an increase in carotid-femoral pulse wave velocity (5.6±0.2 to 5.9±0.2 m/s, $p=0.05$) and a decrease in round trip time (179±8 to 150±11 ms, $p<0.01$) when compared to the acclimation period. A reduction in the Buckberg subendocardial viability ratio (SEVR, indicative of myocardial oxygen supply/demand, $p=0.02$) and an increase in the Pressure-Time Integral Systole (PTI, an index of cardiac load, $p=0.01$) were also observed following sleep restriction.

Conclusions: prolonged (9-day) exposure to experimental sleep restriction in young healthy humans results in unfavorable changes in central macrovascular function, including an increase in central arterial stiffness and cardiac load. These results may have important implications for the increase in cardiovascular disease risk in individuals experiencing limited sleep.

Blood Pressure Variability and Baroreceptor Sensitivity in Normotensive Obese in Response to Aerobic Exercise

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BACKGROUND: Autonomic dysfunction, with increased sympathetic activity at rest has been reported in obese individuals. Indices of blood pressure variability (BPV) and baroreceptor sensitivity (BRS) can provide insight into aspects of autonomic function, particularly following an aerobic exercise bout.

PURPOSE: To examine BPV and BRS in normotensive obese individuals in response to aerobic exercise.

METHODS: Normal-weight (n=8; 25 yr; 23.0 kg/m^2) and obese individuals (n=9; 27 yrs; 32.2 kg/m^2) performed a 60-min leg cycling exercise at 60% of VO_{2peak}. Beat-by-beat blood pressure was recorded at baseline, immediately post-exercise and 30 min into passive recovery using finger plethysmography. R-R intervals were obtained at 1,000 Hz with a digital acquisition system. Power spectral analysis was conducted using WinCPRS software for estimates of BPV (very low and low frequency (VLF, LF), and systolic and diastolic deviation (SDev, DDev)). BRS was estimated using the sequence technique. Natural log-transformed was performed on LF BPV (LnLF) to account for non-normal distribution.

RESULTS: HR increased from baseline similarly in both groups ($p<0.05$). The control group decreased SBP at immediately post-exercise compared to baseline measurements ($p<0.05$), but not the obese group. A main effect of time and group ($p<0.05$) existed for BRS. No group differences were found on DBP, LF, LnLF, VLF, SDev and DDev.

CONCLUSION: The results showed no difference in the BPV indices between the obese and control groups. The different response in SBP suggests that control group may have better BRS; however, this is not supported by the lower values in BRS. A limitation of this study may be the small number of participants.

	Control			Obese		
	Baseline	**Immediate**	**30min**	**Baseline**	**Immediate**	**30min**
HR (bpm)*	66 ± 11	88 ± 12	80 ± 12	60 ± 6	79 ± 11	74 ± 11
SBP (mmHg)*#&	116 ± 11	104 ± 8^{abc}	115 ± 9	122 ± 5	119 ± 6	122 ± 4
DBP (mmHg)	64 ± 10	64 ± 4	68 ± 7	69 ± 5	71 ± 5	72 ± 5
Raw LF (mmHg^2)	9.00 ± 5.37	15.91 ± 15.03	15.24 ± 12.29	5.23 ± 4.65	6.89 ± 4.93	9.64 ± 8.13
LnLF (mmHg^2)	2.07 ± 0.53	2.40 ± 0.89	2.41 ± 0.89	1.37 ± 0.75	1.73 ± 0.66	1.89 ± 0.98
VLF (mmHg^2)	20.83 ± 14.39	29.63 ± 19.77	22.69 ± 13.67	11.91 ± 7.96	18.68 ± 14.70	15.29 ±10.87
BRS (ms/mmHg)*#	15.95 ± 7.92	5.20 ± 3.48	8.05 ± 4.52	19.38 ± 6.79	12.74 ± 8.70	14.49 ± 7.79
SDev (mmHg)	5.61 ± 1.75	7.14 ± 2.71	6.36 ± 2.25	4.77 ± 1.48	5.83 ± 2.42	5.50 ± 2.02
DDev (mmHg)	3.70 ± 1.08	4.06 ± 1.56	3.84 ±.31	3.67 ± 1.33	4.18 ± 1.67	3.90 ± 1.30

All data are mean ± SEM. *Time effect, # Group effect, & time x group effect, a Within-Subjects effect vs Baseline, b Within-Subjects effect vs 30min, c Between-Subject effect vs obese group.

Role of Nitric Oxide in B_2-Adrenergic Mediated Vasodilation in Postmenopausal Women

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Objectives: Postmenopausal (PM) women have a blunted β_2-adrenergic receptor-mediated responsiveness when compared to young premenopausal women in part due to a reduction in the relative contribution of nitric oxide (NO) to β_2-adrenergic mediated vasodilation. Hence, we tested the contribution of NO to β_2-adrenergic receptor-mediated vasodilation during terbutaline infusion.

Hypothesis: We hypothesized that the contribution of NO to β_2-adrenergic mediated vasodilation would be attenuated in PM women as compared to young women.

Methods: Venous occlusion plethysmography was used to measure forearm blood flow (FBF) in 7 healthy young premenopausal women and 9 healthy PM women (mean age = 27 ± 1 and 60 ± 1 years, respectively). FBF was measured at baseline and during terbutaline infusion at 0.1, 0.5, 1.0, 2.0 µg/100ml tissue/min before (with saline co-infusion) and during NO synthase inhibition with L-NMMA. Forearm vascular conductance was calculated from FBF and mean arterial pressure.

Results: In young women, there was a significant L-NMMA effect on forearm vascular conductance during terbutaline infusion with and without L-NMMA (1.7 ± 0.14, 3.56 ± 0.41, 7.13 ± 1.11, 7.87 ± 0.74, 10.54 ± 1.81 versus 2.08 ± 0.28, 5.54 ± 0.50, 9.32 ± 1.10, 10.77 ± 1.49, 13.29 ± 1.94 ml/100ml tissue/min/mmHg, respectively). However, there was no effect of L-NMMA in PM women during terbutaline infusion with and without L-NMMA (1.34 ± 0.26, 2.37 ± 0.32, 5.21 ± 0.99, 4.71 ± 0.99, 6.43 ± 1.37 versus 1.62 ± 0.31, 3.11 ± 0.55, 5.41 ± 1.12, 6.26 ± 1.38, 7.26 ± 1.44 ml/100ml tissue/min/mmHg, respectively).

Conclusions: These data suggest that NO contributes to β_2-adrenergic mediated vasodilation in young premenopausal women. In contrast, no contribution of NO to β_2 mediated vasodilation was observed in PM women. These data suggest a lower β_2-adrenergic responsiveness in PM women may be due to a reduced contribution of NO.

Associations of Walking with Sarcopenic Obesity and Cardiovascular Disease Risk Factors in Older Adults

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Objectives: To investigate the associations of walking (steps/day) with sarcopenic obesity (SO) and cardiovascular disease (CVD) risk factors in older adults.

Methods: This cross-sectional study included 297 older adults aged ≥65 years (mean age 72, ranged 65-95). Walking was assessed using an accelerometer (Omron HJ-321) and categorized into thirds (tertile) based on the average daily steps. SO was defined based on physical function (gait speed), muscle strength (handgrip strength), and muscle mass (appendicular lean mass [ALM] index) according to the Foundation for the National Institutes of Health Sarcopenia Project diagnostic criteria, and % body fat (obesity as ≥25% in men and ≥30% in women) using Dual Energy X-Ray absorptiometry.

Results: Each 10,000 steps/day increase was associated with improved SO variables and CVD risk factors, specifically with 0.008 faster gait speed (m/s), 0.006 higher muscle mass index (ALM/BMI), 0.59 lower % body fat (%), and 0.68 lower fasting glucose (mg/dl)(all $p < 0.05$) in the linear regression after adjusting for age, sex, smoking status, and alcohol intake. Compared to low walking group, odds ratios (ORs)(95% confidence intervals [95% CIs]) in moderate and high walking groups were 0.18 (0.02-1.54) and 0.22 (0.03-2.01) for slow walking, 0.42 (0.14-1.30) and 0.34 (0.09-1.29) for weak handgrip strength, 0.45 (0.23-0.87) and 0.44 (0.22-0.88) for low muscle mass, 0.58 (0.13-2.57) and 0.46 (0.11-2.06) for high % body fat, and 0.62 (0.17-2.28) and 0.21 (0.02-1.78) for SO, respectively, in the multivariable logistic regressions. Compared to individuals without SO, ORs (95% CIs) in individuals with SO were 2.04 (0.58-7.18) for hypertension, 1.27 (0.39-4.22) for hypercholesterolemia, and 1.87 (0.37-9.45) for type 2 diabetes in the multivariable logistic regression. However, these associations appeared to be weaker after further adjustment for walking (steps/day).

Conclusion: This study suggests that walking in older adults is associated with lower risks of SO and CVD risk factors.

A Hydrogen Sulfide Prodrug Augments Angiogenesis in a Swine Model of Critical Limb Ischemia via a Nitric Oxide Dependent Mechanism

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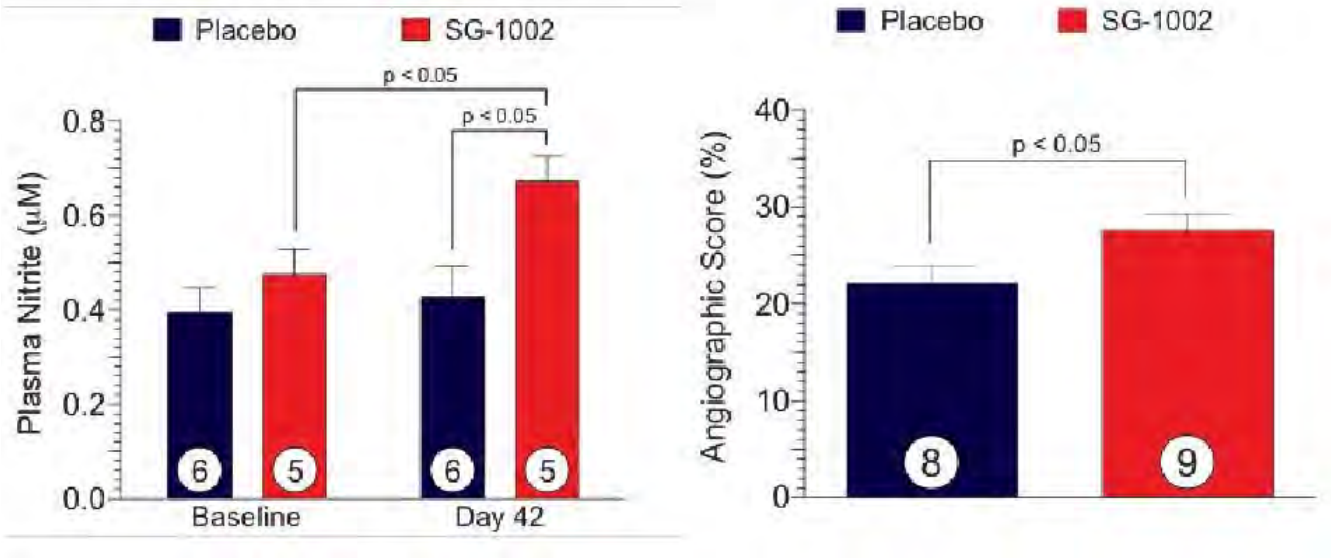
Introduction: Despite advances in revascularization, treatments for critical limb ischemia (CLI) have been largely unsuccessful. Hydrogen sulfide (H_2S) and nitric oxide (NO), are endogenous gasotransmitters which exert potent vasodilatory and proangiogenic effects. Resent experimental evidence suggest that the proangiogenic effects of H_2S are medicated in part through the NO pathway. We sought to determine whether a novel H_2S prodrug, SG-1002 (Sulfagenix, Inc. Cleveland OH), increases NO production and promotes peripheral revascularization.

Methods: CLI was generated in Yucatan miniswine (n=17) via carotid cutdown and placement of an Amplatzer vascular plug deployed within a Viabahn stent positioned proximally in the external iliac artery. At day 7 post-CLI pigs, received daily placebo or SG-1002 (1600 mg PO). Cuff-pressures were measured weekly by ankle/brachial index (ABI). Plasma H_2S, H_2S metabolite sulfane sulfur (SS), and NO metabolite, nitrite (NO_2) were measured. At day 42 post-CLI, digital subtraction angiography (DSA) was performed and opacified vessels quantitated.

Results: ABI was reduced to 0 following CLI induction. ABI improved in both groups but continued to demonstrate persistent ischemia with values below 0.25 at day 42 and showed no difference between groups. Circulating H_2S levels were similar between groups. SS levels were increased from baseline to day 42 in SG-1002 treated pigs ($p < 0.001$) but remained unchanged in placebo treated animals. At day 42, SG-1002 treatment increase circulating NO_2 levels ($p < 0.05$) compared to placebo. There was an increase in NO_2 levels from baseline to day 42 in SG-1002 treated pigs ($p < 0.05$). DSA revealed an increase of CLI limb vessel number in SG-1002 treated pigs compared to placebo ($p < 0.05$).

Conclusions: Treatment with the H_2S prodrug, SG-1002, results in increased metabolites of H_2S and NO signaling. H_2S treatment increased vascular density in the setting of severe CLI in a clinical relevant swine model.



Body Mass Index as an Independent Predictor of Change in Arterial Stiffness Parameters with Change in Body Position

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Changing from supine to a seated position creates an orthostatic challenge due to the effects of gravity on the distribution of blood. This redistribution of volume unloads baroreceptors and may evoke sympathetic activation. The sympathetic activation may lead to increases in arterial stiffness, but it is unknown as to how different demographic variables may impact these changes.

Objective: To investigate whether the change in arterial stiffness parameters between two positions is influenced by factors such as age, sex, or body mass index (BMI).

Methods: Thirty healthy, young adults (24±4 years) were randomly positioned supine or semi-supine, at two different angles (0°,72°) on an adjustable table. After 5 minutes rest, arterial stiffness parameters of the common carotid artery were obtained via ultrasound: beta stiffness index, elastic modulus (Ep), arterial compliance (AC), and distensibility, as well as cardio-ankle vascular index (CAVI) from the VaSera (Fukuda Denshi, Tokyo, Japan). Linear regression was used on the change value for each arterial stiffness parameter adjusting for age, sex, BMI, and baseline values of each outcome measure.

Results: BMI was a significant independent predictor of changes in each measured arterial stiffness parameter after controlling for age and sex. Increasing BMI is related to greater changes in beta stiffness (β=0.55,p=0.001) and Ep (β=0.58,p=0.001) with change in position. Concomitantly, increasing BMI is associated with smaller changes in AC (β=-0.31,p=0.03), distensibility (β=-0.54,p=0.001), and CAVI (β=-0.48,p=0.001). Sex was only a significant independent predictor when assessing change in CAVI (β**=**-0.44,p=0.001).

Conclusion: When measuring arterial stiffness parameters in different positions, it is important to account for the effect of BMI in the analyses. Although obesity is associated with increased baseline sympathetic activity and reduced baroreceptor sensitivity, the change in position creates a larger change in arterial stiffness which may relate to the greater displacement of blood volume with a larger body size.

A Systematic Review on the Effect of Acute Aerobic Exercise on Arterial Stiffness Reveals A Differential Response in the Upper and Lower Arterial Segments

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Objectives: The overall impact of resistance-type exercises and chronic physical activity on the modulation of arterial stiffness has been well characterized; however, the impact of acute aerobic exercise remains unclear. Therefore, we aimed to synthesize evidence pertaining to acute changes in arterial stiffness shortly following aerobic exercise in healthy individuals.

Methods: Electronic databases (MEDLine, EMBASE, Cochrane Library, Sport Discus, and Web of Science) were searched to identify articles assessing the effects of acute aerobic exercise on parameters of arterial stiffness. Eligible studies included arterial stiffness measurements before and after acute exercise in healthy adults, who were free of any cardiovascular risk factors, and were not taking cardioprotective medications.

Results: A total of 43 studies were included. The effect of acute aerobic exercise on arterial stiffness was found to be dependent on the anatomical segment assessed, and on the time at which the measurement was performed post-exercise. Arterial stiffness of the *central* and *upper body peripheral arterial segments* was found to be increased relative to resting values immediately post-exercise (0-5 minutes), while thereafter (>5 minutes) was decreased to a level at, or below resting values. In the *lower limbs*, proximal to the primary working muscles, arterial stiffness decreased immediately post-exercise (0-5 minutes), which persisted into the recovery period post-exercise (>5 minutes).

Conclusions: This systematic review reveals a differential response to acute exercise in the lower and upper/central arterial segments in healthy adult subjects. We further showed that the effect of acute exercise on arterial stiffness is dependent on the time at which the measurement is performed following acute aerobic exercise. Therefore, when assessing the overall impact of exercise on arterial stiffness it is important to consider the arterial segment being analysed and the measurement time point, as failure to contextualize the measurement can lead to conflicting results and misleading clinical inferences.

Relationship between Step Counts and Carotid Femoral Pulse Wave Velocity in Adults Treated For Hypertension and Diabetes

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Objectives: 'Step counts' captured by wearable physical activity tracking devices are associated with reductions in cardiovascular disease. However, in individuals on cardioprotective medications the impact of step counts may not be captured by the measurement of traditional cardiometabolic risk factors. To address this gap, we aimed to assess the relationship between pedometer-derived step counts and carotid-femoral pulse wave velocity (cfPWV), a summative measure of arterial health.

Methods: 369 adults (46% men, 60% White, mean age 59.6±11.2 years, BMI 31.3±4.5 kg/m^2) with hypertension and/or type 2 diabetes were recruited in Montreal, Canada (2011-2015). Step counts (Yamax SW-701 pedometer), moderate-to-vigorous physical activity (MVPA) (ActiGraph GT3x+), arterial stiffness (applanation tonometry; SphygmoCor), and cardiometabolic risk factors including blood pressure, haemoglobin AIc, and lipids were assessed.

Results: Blood pressure was well controlled (mean 125/77±15/9 mmHg), low-density lipoprotein cholesterol (LDL-C) was close to target (mean 2.5±1.0 mmol/L), and A1c in diabetes was acceptable (mean 7.7±1.3%). Participants averaged 5,125±2,722 steps/day (low active) and mean cfPWV was 9.8±2.2 m/s. Step counts correlated with cfPWV, but not with any other cardiometabolic risk factors. A 1,000 step/day increment was associated with a 0.1m/s (95% CI -0.19, -0.02) decrement in cfPWV in a model adjusted for age, sex, BMI, ethnicity, immigration status, employment, education, diabetes, hypertension, medication classes, and MVPA.

Conclusion: In patients with hypertension and/or diabetes who were well-controlled on cardioprotective medications, cfPWV is responsive to step counts and may emerge as a useful health indicator to track the arterial health impact of physical activity strategies in clinical practice.

Higher Central And Brachial Systolic Blood Pressure Is Selectively Associated With Weaker Cognitive Performance In Postmenopausal Women But Not Older Men

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Introduction: Higher aortic stiffness and central blood pressure (BP) are associated with reduced cognitive performance in older adults. Cognitive performance tends to be higher in older women compared with older men, unexplained by differences in years of formal education and/or presence of atherosclerotic vascular disease (AVD). However, whether gender-related differences in cognitive function are explained by alterations in aortic stiffness or central blood pressure (BP) is unclear. We hypothesized that higher aortic stiffness and central systolic BP would be associated with weaker cognitive performance in middle-aged/older (MA/O) men but not postmenopausal women.

Methods/Results: A total of 135 MA/O men and postmenopausal women (age 55-85 yrs) were recruited. Brachial systolic BP was higher in men, however, there were no differences in aortic stiffness (carotid-femoral pulse wave velocity, cfPWV), central systolic BP or pulse pressure (PP) (Table 1). Women scored higher than men on the RBANS Total Scale Score and Delayed Memory Index (both P<0.05) (Table 1). In the entire cohort, higher central and brachial systolic BP were associated with weaker Stroop Color Naming (r=-0.24, P<0.05, r=-0.25, P<0.05) and Stroop Interference (r=-0.30, P<0.01, r=-0.32, P<0.01) performance. Interestingly, years of education was associated with RBANS Total Scale Score (r= 0.64, P<0.001) and WRAT-3 Reading (r=0.63, P<0.001) scores in men but not women (P>0.05). Adjusting for age, AVD status, BMI, insulin, estrogen therapy and medications, higher Stroop Interference scores were associated with lower central systolic (r= -0.52, P=0.001), brachial systolic (r= -0.50, P=0.001) BPs and central PP (r=-0.31, P=0.05) in women but not men. Lower WRAT-3 Reading scores were associated with higher central (r= -0.44, P<0.01) and brachial PP (r= -0.50, P<0.01) in women only.

Conclusion: Higher central and brachial systolic BP and PP is selectively associated with weaker cognitive performance in postmenopausal women but not MA/O men independent of aortic stiffness and AVD.

Mean ± SE	**Men (n=68)**	**Women (n=67)**	*p-value*
Demographics:			
Age (yrs)	66.3 ± 1.0	68.3 ± 1.0	0.14
Atherosclerosis Vascular Disease, no. (%)	46 (67.6)	27 (40.3)	**0.001**
Education (yrs)	15.1 ± 0.3	14.3 ± 0.3	0.08
Body Mass Index (kg/m^2)	29.9 ± 0.7	28.7 ± 0.8	0.25
Total cholesterol (mg/dL)	145 ± 3.8	177 ± 4.4	**<0.001**
HDL cholesterol (mg/dL)	47.5 ± 1.6	57.3 ± 2.2	**<0.001**
Triglycerides (mg/dL)	100 ± 5.7	117 ± 8.0	0.09
Glucose (mg/dL)	109 ± 3.3	95 ± 2.8	**0.002**
Total insulin uU/mL	10.9 ± 1.2	8.9 ± 1.0	0.22
Statins, no. (%)	38 (55 9)	29 (43.3)	0.15
Anti-hypertensives, no. (%)	48 (70.6)	38 (56.7)	0.10
Aspirin, no. (%)	46 (67.6)	39 (58.2)	0.12
Estrogen therapy at baseline, no (%)	-	7 (10.4)	**<0.001**
Vascular:			
Brachial systolic blood pressure (mmHg)	140 ± 2.1	133 ± 2.4	**0.032**
Brachial diastolic blood pressure (mmHg)	78 ± 1.2	68 ±1.4	**<0.001**
Brachial pulse pressure (mmHg)	62 ± 2.4	65 ± 2.1	0.37
Mean arterial pressure (mmHg)	98 ± 1.1	89 ± 1.5	**<0.001**
Aortic systolic blood pressure (mmHg)	129 ± 2.2	125 ± 2.3	0.16
Aortic pulse pressure (mmHg)	51 ± 2	56 ± 2	0.13
cfPWV (m/sec)	10.5 ± 0.3	10.3 ± 0.24	0.54
Cognitive:			
Global Cognitive Function			
RBANS Total Scale Score	98.6 ± 1.6	104.7 ± 1.5	**0.007**
WRAT-3 Reading Standard Score	103.6 ± 9.5	110.8 ± 4.1	0.09
Memory			
RBANS Immediate Memory	96.6 ± 1.7	101.6 ± 1.9	0.06
RBANS Delayed Memory	99.3 ± 1.6	106.0 ± 1.4	**0.002**
Processing speed			
Stroop Color Naming	67.0 ± 1.3	69.5 ± 1.8	
Stroop Word Reading	87.4 ± 1.7	91.1 ± 2.1	0.26
Executive function/working memory			
Stroop Interference	33.1 ± 0.9	35.2 ± 0.9	0.11

Table 1; displays demographic, vascular rand cognitive performance data. All data are presented as mean ± SE. HDL, High density lipoprotein, cfPWV, carotid femoral pulse wave velocity; RBANS, Repeatable Battery for the Assessment of Neuropsychological Status; WRAT, Wide Range Achievement Test.

Bilateral Symmetry of Brachial Pulse Waveform Analysis in a Clinical Population

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Background: Pulse waveforms are modified as they propagate along the arterial tree. Small differences in the arterial pathways from the heart to the left and right brachial artery may impact pulse waveform analysis (PWA) for the purpose of hemodynamic assessment. The VaSera VS-1500AU (Fukuda Denshi) is a cuff-based device that permits simultaneous acquisition of bilateral brachial pulse volume recordings. To determine if interchangeability between left and right brachial pulse waveforms is possible, we assessed whether there are significant differences in pulse waveform analysis variables between each arm.

Methods: In 20 subjects (mean age=67±11 years) from a clinical population, simultaneous pulse waveforms were acquired at both the left and right brachial arteries. Following an initial recording, the cuffs were switched and a second series of continuous waveforms were acquired. Phonocardiograms were continuously acquired to determine timing of aortic valve closure. All PWA variables were averaged across the standard- and switched-cuff configurations to minimize the impact that slight variations in cuffs may impart on recordings. Extracted PWA variables include (1) brachial form factors (bFF), (2) heart-brachial transit time (hb-TT), and (3) brachial augmentation index (bAIx).

Results: Paired t-test revealed no statistically significant differences in left and right pulse waveform features (P>0.05 for BFF, hb-TT, bAIx). Bland-Altman analysis revealed no significant bias in extracted waveform features between each arm (mean bias [limits of agreement]=0.3 [-3.2, 2.7]%, -2.65[-1571.1, 1041.1] msec, 0.3[-1.15, 1.21]% for bFF, hb-TT, and bAIx, respectively).

Conclusion: No significant systematic differences exist between left and right pulse waveforms. Despite minor differences in arterial pathways between left and right brachial arteries, we found agreement in PWA variables between both arms. The side of measurement did not influence pulse waveform analysis results in this clinical sample.

A New Software for Determining Changes in Arterial Diameter over Time

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Objectives: The purpose was to investigate the ability of a new software, developed by our group, to provide continuous measures of arterial diameter from recorded ultrasound video.

Methods: Software (MAUI) was developed to assess arterial diameter using active contours to accurately detect the vessel walls in recorded ultrasound video. Ultrasound imaging was used to acquire longitudinal, B-Mode images of the common carotid artery (CCA) with videos recorded for later analysis. A single recorded 10s video was used to gain an indication of the reproducibility and repeatability of MAUI. For this assessment, two investigators (E1 and E2) each performed 10 measurements of the test video using the MAUI software. MAUI was then used to process several longer videos (~5min) to assess the ability of the software to continuously process data over longer periods of time.

Results: MAUI provided a measurement of vessel diameter (media to media border) for each frame of the recorded video. The ten assessments of the test video resulted in average standard deviation of 0.002±0.003cm for E1 and 0.003±0.003cm for E2 for each frame measurement. Overall analysis of the test video resulted in an average diameter, measured across eight cardiac cycles, of 0.781±0.0005cm and 0.780±0.0007cm for E1 and E2 respectively. Measures by E1 and E2 ranged from 0.781 to 0.782cm and 0.779 to 0.781cm respectively. When processing the 5min videos, MAUI was able to continuously track the vessel walls throughout the entire video.

Conclusions: Preliminary assessments suggest that MAUI software represents a viable method for the continuous assessment of arterial diameter over time with high repeatability and low interrater variability. Use of this software may be especially applicable for studies investigating acute changes in vessel dimensions as well as the study of vascular properties in health and disease.

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Lower Ankle-Brachial Index within the Normal Range is Associated with Reduced Mitochondrial Energy Production, Explaining the accompanying Poor Walking Endurance

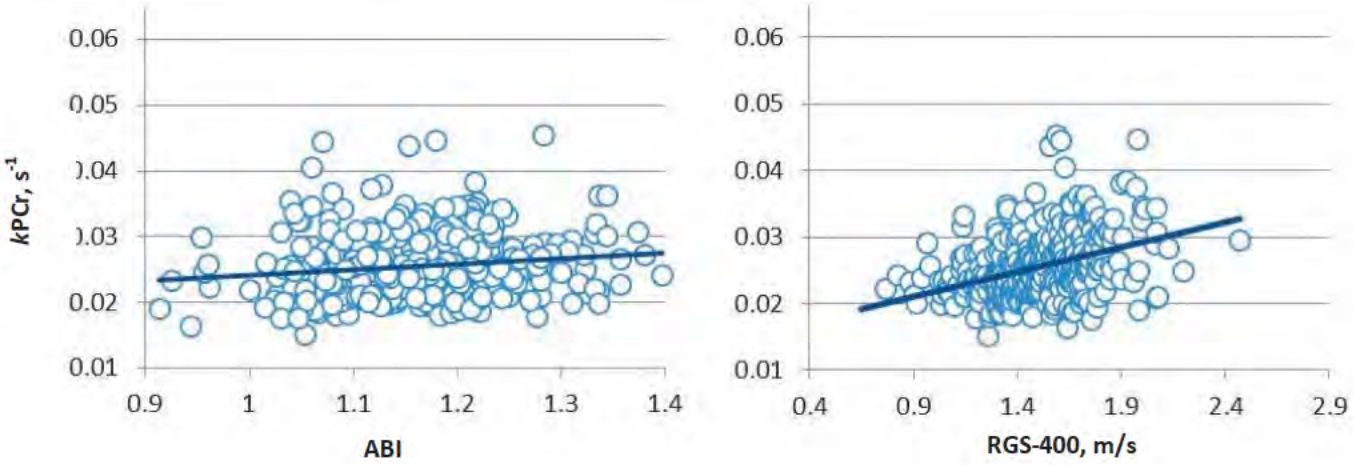
Majd AlGhatrif, Ariel Zane, Matt Oberdier, Elizabeth Ratchford, Maya Salameh, Rajiv Thakkar, Stephanie Studenski, Eleanor Simonsick, Richard Spencer, Kenneth Fishbein, David Reiter, and Luigi Ferrucci

Objective:
Reduced walking endurance in peripheral arterial disease, defined as ABI <0.9, has been attributed to impaired muscle mitochondrial energetics. Individuals with borderline ABI between 0.9 and 1.0, or low normal ABI between 1.0 and 1.1 have been also found more likely to develop reduced walking endurance than those with higher ABI. Therefore, we hypothesized that poor walking endurance in individuals with lower ABI in borderline and normal ranges is mediated by impaired mitochondrial energy production.

Methods:
We examined data of 382 men and women participating in the Baltimore Longitudinal Study of Aging, who were free of PAD. Walking endurance was assessed by 400 meter rapid gate speed (RGS-400). Muscle mitochondrial energy production was assessed by post-exercise phosphocreatine recovery rate constant (KPCr) measured by phosphorus magnetic resonance spectroscopy (31P-MRS) of the left thigh; reduced KPCr reflects decreased mitochondria energy production. Ipsilateral ABI was measured by the Colin system. Multivariate models were adjusted for age, gender, glucose, and smoking status.

Results:
The sample mean age was 71 ± 12 years; about 18% of the participants had diabetes; 4% were current and 40% were former smokers. There were significant associations between KPCr and each of ABI and RGS-400 (Figure 1); these remained significant after adjustment. Lower ABI was independently associated with slower RGS-400 (β=-0.60, P=0.0339). Adding KPCr to the model weekend the association between RGS-400 and ABI by 15%, rendering it statistically insignificant (β=-0.51, P=0.09.)

Conclusion:
The association between lower ABI in the normal range and slower walking speed is mediated by decreased mitochondrial energy production. Hence, even within the normal range, lower ABI could represent a preclinical reduction in lower extremity perfusion negatively affecting energy production. Prospective studies are needed to confirm these association and their long terms effects.



Greater Early and Late Arterial Loading with Advancing Age is Associated with Impaired Hemodynamic Efficiency in a Community Dwelling Population

Matt Oberdier, Stephanie Studenski, Edward Lakatta, Majd AlGhatrif

Objective:
Aortic elastic properties are pivotal for proper arterial-ventricular coupling and optimal hemodynamic efficiency, minimizing wasted, potentially damaging, energy. Major alterations in arterial properties ensue with aging, potentially reducing hemodynamic efficiency. Therefore, we hypothesized that hemodynamic efficiency is reduced with advancing age in a community dwelling population free of cardiovascular disease; this decline is explained in part by alterations in arterial loading parameters.

Methods:
We studied 382 participants (185 men, age range 26-95) from the Baltimore Longitudinal Study of Aging who had carotid tonometry and left-ventricular outflow Doppler testing performed. Pressure and flow waveforms were analyzed using custom-designed Matlab software to calculate total and steady state power. Efficiency was defined as the quotient of mean and total power. Early loading was assessed by characteristic impedance (Zc), while late loading was assessed by reflected wave transit time (RWTT), and reflection coefficient (RC); nonpulsatile loading was assessed by total vascular resistance (TVR).

Results:
Efficiency declined with advancing age (β=-0.1, P<0.0001), adjusting for gender, race, weight hypertension, and diabetes. Hemodynamically, efficiency was directly associated with heart rate, RWTT, and RC while it was inversely associated with Zc and TVR. In standardized multivariate analysis, RWTT had the highest standardized coefficient (β=34.2, P=0.0002) followed by RC (β=21.5, P<0.0001), Zc (β=-19.9, P=0.0024), TVR (β=-5.4, P<0.0001), and HR (β=0.2, P<0.0001). The hemodynamic variables explained 27% of the reduced efficiency with advancing age.

Conclusion:
Hemodynamic efficiency is reduced with advancing age. This decline is associated with greater nonpulsatile, early arterial loading, and shorter reflection time. Such a decline in efficiency implies greater wasted energy with aging that is potentially dissipated in the central arteries and high-flow organs, contributing to arterial remodeling and chronic diseases of aging. Further prospective analyses of larger samples is needed to examine whether hemodynamic efficiency and wasted energy parameters better predict the longitudinal decline in end-organ function.

Effect of Low-Dose Acetylsalicylic Acid on Arterial Stiffness in High-Risk Pregnancies: An Observational Longitudinal Study

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Objectives: Low-dose acetylsalicylic acid (ASA) has been shown to reduce the risk for pre-eclampsia in high-risk pregnancies when prescribed before 16 weeks of gestation. It remains unknown whether this anti-inflammatory agent has effects on arterial stiffness. Our objective was to characterize arterial stiffness indices throughout pregnancy in women with high-risk pregnancies who were and were not prescribed low-dose ASA.

Methods: In this prospective longitudinal study, women with high-risk singleton pregnancies were recruited from obstetrical clinics in Montreal, Canada. Arterial stiffness was measured using applanation tonometry (SphygmoCor; AtCor) in the 1st trimester, every 4 weeks thereafter until delivery, and at 6 weeks' post-partum. Arterial stiffness was compared between women who were prescribed low-dose ASA (81 mg) before 16 weeks' gestation and women who were not prescribed any prophylactic medication for pre-eclampsia.

Results: Of the 152 participants who delivered in this ongoing study, 26 women were prescribed ASA. Longitudinal analyses adjusted for family history of pre-eclampsia, past history of pre-eclampsia, and development of an outcome showed no significant differences in carotid-femoral pulse wave velocity (cfPWV), carotid-radial PWV, augmentation index adjusted for a heart rate of 75 beats per minute, or start time of wave reflection (T1R) throughout pregnancy in women who were taking low-dose ASA (all $p > 0.05$). Additionally, 13 women developed pre-eclampsia and ASA did not confer any significant change in adjusted odds for the complication (OR: 4.85 95% CI: 0.5 – 41; p = 0.15).

Conclusion: In this high-risk pregnant population, ASA before 16 weeks' gestation was not associated with differences in arterial stiffness or wave reflection throughout pregnancy and did not have an effect on the odds for developing pre-eclampsia. Our ongoing study will provide definite evidence on the association between ASA use and arterial stiffness.

Effect of Poor Glycemic Control on Arterial Stiffness in Pregnancy

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Objectives: Poor glycemic control during pregnancy is associated with increased adverse perinatal outcomes. Our objective was to characterize the association between glycemic control and arterial stiffness in pregnancy.

Methods: In this prospective longitudinal study, women with high-risk singleton pregnancies were recruited from obstetrical clinics in Montreal, Canada. Arterial stiffness was measured in women with gestational diabetes (GDM) or pre-existing diabetes mellitus (DM) using applanation tonometry (SphygmoCor; AtCor) starting at 24 weeks' gestation (the period at which GDM screening is performed for all women according to standard clinical practice) and every 4 weeks thereafter until delivery. Arterial stiffness indices were compared between women with poor glycemic control and women with adequate glycemic control. Poor glycemic control was defined as average HbA1C > 7%, average fasting glucose > 5.3 mmol/L, average 1h post-prandial glucose > 7.8 mmol/L, insulin dosage > 30 units, large for gestational age fetus, or maximal vertical pocket > 8 cm.

Results: Of the 35 women who delivered in this ongoing study and had GDM (n=18) or DM (n=17), 12 had poor glycemic control throughout their pregnancy. Longitudinal analyses adjusted for maternal age, body mass index, and medical history, showed women with poor glycemic control had significantly increased carotid-radial pulse wave velocity (PWV) at each timepoint: 26-30 weeks: 8.4 vs. 8.0 m/s, p = 0.04; 30-34 weeks: 8.4 vs. 8.1 m/s, p <0.01; 34-38 weeks: 8.5 vs. 8.1 m/s, p = 0.02. No differences were found in carotid-femoral PWV, augmentation index adjusted for a heart rate of 75 beats per minute, or start time of wave reflection between these 2 cohorts.

Conclusion: Women who had poor glycemic control throughout pregnancy showed increased peripheral arterial stiffness from the late 2nd trimester until delivery. Our ongoing study will provide more definite conclusions with increased population size.

Dietary Calcium Intake and Cardiovascular Health: Is There Any Relationship?

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Introduction: Calcium intake, recommended for osteoporosis prevention, has been associated with cardiovascular (CV) outcomes. We examined the association of dietary calcium intake (dCa) with surrogate CV markers, including carotid intima-media thickness (cIMT), arterial stiffness and hemodynamics in healthy postmenopausal women.

Methods: Healthy postmenopausal women without any CV risk factors, from a randomized controlled trial studying the effect of calcium supplementation vs. dietary calcium on vascular heath, were recruited. Cross-sectional analyses of baseline data of the participants are presented. Peripheral systolic and diastolic blood pressures (pSBP, pDBP) were measured by BpTRU. cIMT of both common-carotid arteries was measured by B-mode ultrasonography (Philips-iU22). Arterial stiffness (carotid-to-femoral pulse wave velocity [cfPWV] and carotid-to-radial PWV), central SBP and DBP (cSBP, cDBP), mean arterial pressure (MAP), and hemodynamic parameters (pulse pressure, augmentation pressure, augmentation index corrected for 75 bpm) were obtained non-invasively (SphygmoCor). Usual dCa intake was estimated using a validated food frequency questionnaire. Measurements were compared across groups (<600, 600-1000 and >1000 mg/day of dCa) by one-way analysis of variance and covariance.

Results: We evaluated 83 postmenopausal women (mean age 60.4±6.3 years; BMI 25.6±3.8 kg/m^2). Mean dCa was 857±333 mg/day. Although within normal range, vascular parameters had a non-significant, U-shaped relationship with dCa. In unadjusted analyses, women with dCa >1000 mg/day had significantly higher cfPWV, pSBP, cSBP, and MAP compared to those with 600-1000 mg/day; however, significance was lost for all other parameters except for MAP after adjustment for pertinent covariates (Table).

Conclusion: In healthy postmenopausal women, a non-significant, U-shaped relationship of vascular parameters across the 3 dCa groups was noted; dietary calcium may have favourable effect on MAP for those consuming 600-1000 mg/day compared to >1000 mg/day intake. Of note, our population had optimal/normal BP. Our ongoing study including a larger sample-size will determine the relationship between dCa and surrogate CV markers.

Statin Therapy in Rheumatoid Arthritis May Improve Arterial Stiffness in Women but Not In Men: A Preliminary Analysis

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Objectives: Patients with rheumatoid arthritis are at increased risk for cardiovascular disease. Statins have anti-inflammatory and immunomodulatory effects, thereby reducing cardiovascular risk. Arterial stiffness is a composite indicator of cardiovascular health and a predictor of cardiovascular risk. We assessed the effect of statin therapy on arterial stiffness and hemodynamics in subjects with rheumatoid arthritis.

Methods: A prospective cohort study including adults with rheumatoid arthritis and an indication for statin therapy (cases) or not (controls) is being conducted. Peripheral systolic and diastolic blood pressures were measured by BpTRU. Arterial stiffness (carotid-to-femoral pulse wave velocity [cfPWV] and carotid-to-radial PWV), central systolic and diastolic blood pressures, mean arterial pressure, and augmentation index corrected for 75 bpm were obtained non-invasively (SphygmoCor, AtCor, Australia). All measurements were performed prior to statin initiation and at 6-month post-treatment. Independent *t*-tests evaluated differences in changes between groups. Carotid intima-media thickness (cIMT) measurements were also performed.

Results: To date, 14 subjects (mean age 61.4±9.5 years, 9 females), have completed the study. All cases achieved recommended lipid level targets by 6 months. There were no statistical differences in patient characteristics (beyond lipid levels) at baseline or 6-months between cases and controls among the whole cohort. In sex-specific analyses, statin therapy was associated with a significant decrease in cfPWV in women taking statins compared to women in the control group (-0.71±0.18 m/s vs +0.96±1.13 m/s, respectively; $p<0.05$), which was not observed in men. No other associations were observed. cIMT analyses are underway.

Conclusion: Our preliminary results suggest that in women with rheumatoid arthritis, statin therapy may reduce cfPWV, a predictive marker of cardiovascular disease and events, which was not observed in men. Whether sex differences in the effect of statin on arterial stiffness are sustained with a larger sample size of rheumatoid arthritis patients will be addressed in our ongoing study.

First in Man Measurement of Arterial Stiffness Using a Connected Bathroom Scale: Calibration against SphygmoCor

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Background: Measurement of arterial stiffness (AS) is still considered difficult. We developed a non-invasive technique to assess AS from a connected bathroom scale, based on ballistocardiography (BCG) and impedance plethysmography (IPG).

Methods: We included 192 subjects and patients, 106 for calibration study (cal), 86 for validation study (val), 33% hypertensives, mean age 48±17 years, 48% women. The scale pulse transit time (WS- PTT) was calculated as the difference between BCG systolic signals and IPG blood flow in the foot. Distance was estimated from body height and PWV was calculated. Carotid to femoral transit time (CF-PTT) was measured using SphygmoCor. Spearman and robust multivariate regressions were used.

Results: The WS-PTT correlated well with CF-PTT with R=0.73 in pooled population (cal 0.79, val 0.66). WS-PWV correlated with CF-PWV with R=0.76 (cal 0.80, val 0.70). The standard deviation of difference was 1.39 m/s with a bias of 0.25 m/s compared with CF-PWV. Correlations of WS-PWV with age and blood pressure were similar (R= 0.72 and 0.59, resp.) to those of CF-PWV (R=0.67 and 0.61, resp.). These good correlations were non-trivial given the differences in wave paths, the fact that measurements are made in orthostatic position and totally investigator-free.

Conclusion: We show in two distinct populations that a simple user-oriented instrument such as a connected bathroom scale can estimate arterial stiffness with accuracy close to healthcare-oriented systems. Because these devices will be used by the general population, the availability of arterial stiffness data on very large, non-medicalized populations will change our management of well-being and health.

Effects of Fixed Versus Auto-Titrating Continuous Positive Airway Pressure on Vascular Function in Patients with Resistant Hypertension and Obstructive Sleep Apnea

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Introduction: Obstructive sleep apnea (OSA) is a common cause of resistant hypertension. We investigated the effects of 2 modalities of positive airway pressure; fixed continuous airway pressure (fCPAP) versus auto-titrating positive airway pressure (APAP) on arterial function in subjects with resistant hypertension and severe OSA.

Objective: To assess in participants with resistant hypertension and OSA the effects of fCPAP vs. APAP on 24h ambulatory blood pressure monitoring (ABPM), as well as sleep indices, heart rate variability (HRV), and arterial stiffness.

Methods: We randomized 14 subjects (56±11 years, baseline SBP and DBP 137±10 and 77±12 mm Hg, respectively, apnea-hyponea index [AHI] 58±31 events/h, Epworth sleepiness scale 7±5) to fCPAP or APAP for 6 weeks, followed by crossover to the other modality for another 6 weeks. Overnight polysomnography, 24h ABPM, HRV, and carotid-femoral pulse wave velocity (cfPWV, arterial stiffness 'gold-standard' measure) were measured at baseline and after each intervention period.

Results: fCPAP and APAP were associated with similar improvements in sleep quality, AHI and oxygen desaturation indices, while the nadir SpO_2 was significantly higher with fCPAP than APAP (z=-2.251, p=0.03). There were no significant effects of either modality on central BP or 24h ABPM, likely due to controlled BP at baseline. Both fCPAP and APAP improved cfPWV compared to baseline, (fCPAP, p=0.017; APAP, p=0.056), suggesting that their effects are BP independent. CPAP significantly decreased HR and HRV, whereas APAP had no effect.

Conclusions: No differences in vascular function was observed with treatment with fCPAP or APAP, but there is some suggestion that fCPAP is associated with improved measures of arterial health, i.e.: cfPWV and HR. The effects of fCPAP on arterial stiffness may be independent of BP and potentially mediated by changes in sympathovagal activity. Our results of mild favorable effects of fCPAP need to be confirmed in larger studies.

The Impact of Intradialytic Pedaling Exercise on Arterial Stiffness in a Hemodialysis Population

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Objectives: Hemodialysis patients are at greater risk of increased arterial stiffness. Regular aerobic exercise has been shown to reduce arterial stiffness in hemodialysis patients. However, the impact of a more realistic intradialytic form of exercise, such as pedaling, is unclear. Therefore, we aimed to examine 1) the effect of intradialytic pedaling exercise on arterial stiffness over 4 months, and 2) the durability of the pedaling effect 4 months after finishing the exercise intervention.

Methods: We performed a 4-month randomized control trial in patients on a stable in-center hemodialysis regimen (3 days/week). Subjects were block randomized to either pedaling exercise (EX) or to a control group receiving usual dialysis (nonEX) for 4 months. At baseline and 4 months, augmentation index heart rate corrected (AIx75), and carotid-femoral pulse wave velocity (cfPWV) were assessed (applanation tonometry; SphygmoCor XCEL). Measurements were repeated in the EX group 4 months after the exercise intervention.

Results: 11 exercisers (58±16 years, BMI 26±5kg/m^2, 3 female) and 10 controls (53±15 years, BMI 27±6kg/m^2, 3 female) were included. Overall exercise compliance was 60±25%, and subjects exercised on average 47±25 mins per session. AIx75 was unchanged in the EX group, however an increase of 4.4±4.5% was noted in the nonEX group (*P=0.020*). We observed a greater absolute decrease in cfPWV in the EX group compared to the nonEX group: -1.44±2.06 vs. 0.27±0.55 m/s (*P=0.037*) (Figure 1). This difference in cfPWV was maintained after adjustments for age, Charlson comorbidity score, and the baseline cfPWV value (*P=0.041*). Interestingly, the decrease in cfPWV observed in the EX group was partially preserved 4 months after exercise cessation (Figure 2).

Conclusions: The relationship between intradialytic pedaling exercise and improved arterial stiffness is promising, and warrants further investigation. Moreover, we have demonstrated that pedaling exercise is a realistic form of aerobic training in hemodialysis patients.

Figure 1. Post-Exercise Absolute Change in cfPWV

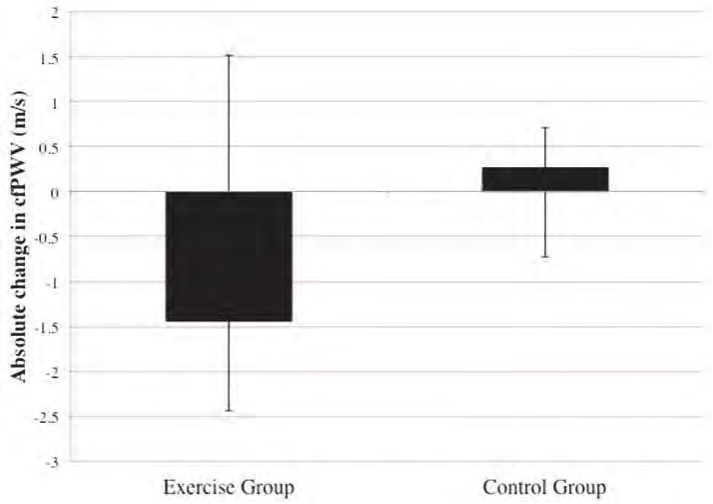
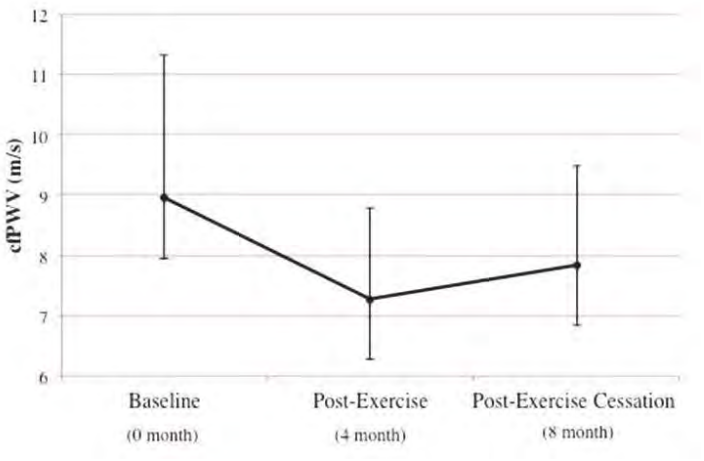


Figure 2. cfPWV at baseline, post-exercise and 4 months after exercise cessation



ABOUT NORTH AMERICAN ARTERY

Mission Statement

The Mission of North American Artery Society is to:

- Support education on arterial structure and function appropriate to the various medical communities, such as scientific researchers, clinical specialists, primary care specialists, medical students, and pharmaceutical researchers, as well as the patient community;
- Develop mechanisms and venues for disseminating information on the understanding and application of arterial structure and function and its measurement among the various medical communities;
- Participate in and encourage the study of improved application of technologies in the measurement of arterial structure and function;
- Participate in and encourage clinical trials that develop the understanding of how arterial structure and function and its measurement can guide and inform patient selection and treatment;
- Guide and support efforts to standardize arterial structural and functional measurements for clinical practice and clinical/scientific studies;
- Direct efforts to include arterial structure and function measurements in appropriate national guidelines;
- Formulate a consensus statement regarding what is known in regards to arterial structure and function.

Society Objectives

North American Artery is a non-profit, non-partisan professional society dedicated to the encouragement, support, and understanding of vascular structure and function and its application to clinical medicine, research and pharmaceutical and medical device development. The Society Objectives are to:

- Support education on arterial mechanics appropriate to the various medical communities, such as scientific researchers, clinical specialists, primary care specialists, and pharmaceutical researchers, as well as the patient community;
- Develop mechanisms and venues for disseminating information on the understanding and application of arterial mechanics and its measurement among the various medical communities;
- Participate in and encourage the study of arterial mechanics in basic and applied research to further especially the clinical applications derived from an improved understanding of arterial mechanics;
- Participate in and encourage clinical trials that develop the understanding of how arterial mechanics and its measurement can guide and inform patient treatment;
- Guide and support efforts to standardize arterial mechanics measurements for clinical practice and clinical/scientific studies;
- Direct efforts to include arterial mechanics measurements in appropriate national guidelines;
- Provide the knowledge for the critical understanding and application of technologies to measure arterial mechanics.

JOIN OUR EXCITING ORGANIZATION TODAY!

An active membership to this growing and influential research community is extremely beneficial to anyone associated with or interested in arterial research. As a member of North American Artery, you can view our member database, participate in our forum, as well as enjoy a host of other benefits.

Membership is open to all individuals and organizations that have a research, clinical, or scientific interest in arterial mechanics and hemodynamics. There are three (3) classes of membership:

- **Individual Voting Members - $60.00**
  All dues-paying individuals, are voting members.
- **Sponsor Member Organizations - $500.00**
  This membership permits an organization to identify up to five (5) individuals from its organization to be Individual Voting Members. Additional members may be added according to guidelines developed by the Executive Committee. An organization may have an unlimited number of members.
- **Student Members – Free**
  This membership is open to all individuals who are currently still in training (residents, fellows, post-doctoral candidates). Student Members are non-voting members. A letter from the training director is required to be submitted with the application for membership.

Membership in NAA is based on a calendar year (July 1 – June 30). Payments of dues at any time during the year are considered dues for that calendar year. Membership renewal invoices are sent on June 1 and due by July 1.

MEMBERSHIP BENEFITS

Here are seven specific reasons why you should join North American Artery Society (NAA) today.

1. **On-line subscription to ARTERY RESEARCH.** ARTERY, the Association for Research into Arterial Structure and Physiology, is a European society with similar goals and objectives to NAA; ARTERY RESEARCH is its peer-reviewed journal featuring articles, case studies, meeting abstracts and other relevant publications on arterial structure and function. The on-line subscription comes with NAA membership. Without a membership, the purchase price of the journal on-line is $31.50 per article.

2. **Be an active participant.** NAA is active in developing a multidisciplinary approach to research in and applications of arterial structure and function. We recognize the value of many voices, opinions, and disciplines, and invite you to get involved.

3. **Enjoy reduced registration fees.** Membership in NAA provides you with significant savings on registration fees for all NAA sponsored events.

4. **Join the Forum.** Membership in NAA makes you part of the conversation on artery research and applications. You can contribute to and learn from presentations in workshops, seminars, on-line videos, and other avenues of sharing information.

5. **Make key connections.** Participation in NAA provides a focal point for developing working relationships with others active in the field.

6. **Lead the pack.** NAA will be leading the development of consensus positions on a number of related issues, and participating in the design of upcoming studies in the field of artery research.

7. **Become a decision maker.** NAA is an organized voice in the development of clinical applications of arterial research, including setting validation standards and application guidelines. As a member, you can be part of our voice to both the pharmaceutical as well as the device manufacturing industries.

Membership Application

Name & Principal Mailing Address **(please type or print legibly and complete ALL information requested)**

Last Name:_____First Name:_____MI:____ Degree:_____

Affiliation:_____

Address:_____

City:_____State/Province:_____Postal Code:_____

Country (if not U.S.)_____ Telephone:_____Fax:_____

E-mail:_____

Please indicate Specialty

Physician Specialty: ☐ Cardiology ☐ Family Practice ☐ Endocrinology ☐ Nephrology

☐ Internal Medicine ☐ Other_____

☐ Physiologist (Exercise) ☐ Allied Health Professional ☐ Scientist ☐ Physician Assistant

☐ Other _____

Membership Category

☐ Individual Voting Member - $60.00

☐ Sponsor Member Organization - $500.00 (Includes up to five (5) Voting Members. List names below.)

If applying for Sponsor Member Organization membership, please include up to four (4) additional individuals who will also be Voting Members.

1) Name: _____ Email Address: _____

2) Name: _____ Email Address: _____

3) Name: _____ Email Address: _____

4) Name: _____ Email Address: _____

Payment Information: No CASH or BANK TRANSFERS. Membership dues may be paid with VISA, MasterCard, American Express, Check or Money Order. Checks and money orders must be drawn on American Banks and must be in US Dollars.

Payment Method: ☐ VISA ☐ MasterCard ☐ American Express ☐ Check payable to North American Artery

Cardholder's Name:_____ Exp. Date:_____

Credit Card Number Card:_____ Verification Code:_____

Cardholder's Billing Address:_____

Cardholder's Signature:_____

Return this form to: North American Artery Society, c/o Hansen Global Event Management, LLC, 68 Carlton Terrace, Stewart Manor, NY 11530 or fax the form to 866-383-6027.

Contact Matthew Hansen with any questions at 516-361-4415 or via email to naa@hansenglobalevents.com.

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