



NORTH AMERICAN ARTERY

NAA 2015



Photo © Choose Chicago / Patrick J. Pyserka / City of Chicago

North American Artery Fifth Annual Meeting
**Hemodynamics and Target Organ Damage:
Mechanisms, Measurements, and Management**

September 11-12, 2015, Chicago Marriott O'Hare, Chicago, IL

FINAL PROGRAM

September 11-12, 2015
Chicago Marriott O'Hare
Chicago, IL

SphygmoCor[®]
XCEL



SIMPLY THE GOLD STANDARD

**At the heart of
blood pressure management.**

info@atcormedical.com • atcormedical.com

ABOUT THE NORTH AMERICAN ARTERY SOCIETY

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.

2014 PROGRAM COMMITTEE

Co-Chair
Bo Fernhall, PhD
Chicago, IL

Co-Chair
Gary L. Pierce, PhD
Iowa City, IA

Industry Liaison
Peter U. Feig, MD
Guilford, CT

Tina E. Brinkley, PhD
Winston-Salem, NC

Michael D. Brown, PhD
Chicago, IL

Julio A. Chirinos, MD, PhD
Philadelphia, PA

Stella Daskalopoulou, MD,
MSc, DIC, PhD
Montreal, Quebec, Canada

Daniel Duprez, MD, PhD
Minneapolis, MN

David G. Edwards, PhD
Newark, DE

Keith C. Ferdinand, MD
New Orleans, LA

Stanley S. Franklin, MD
Irvine, CA

Raymond R. Townsend, MD
Philadelphia, PA

Elaine M. Urbina, MD, MS
Cincinnati, OH

Dean Winter, PhD
Portland, OR

ACKNOWLEDGEMENT

North American Artery sincerely thanks the following firms for their support of the Fifth Annual Meeting.

DIAMOND SPONSORS



GOLD SPONSORS



TABLE OF CONTENTS

2015 Program Committee Members.....	2
President’s Welcome.....	4
Program Committee Chairmen Welcome.....	5
General Information.....	6
Faculty List.....	7
Takeaway Messages.....	8
Meeting Agenda-Friday, September 11, 2015.....	10
Meeting Agenda-Friday & Saturday, September 11 & 12, 2015.....	12
Meeting Agenda-Saturday, September 12, 2015.....	13
Conflict of Interest Disclosures.....	14
Posters.....	15
Faculty Bios.....	16
Exhibitors.....	22
Abstracts (Accepted for Oral Presentation).....	24
Abstracts (Accepted for Poster Presentation).....	30
NAA Membership Information.....	59
Membership Application.....	60

PRESIDENT'S WELCOME



NORTH AMERICAN ARTERY

Executive Committee

President

*Raymond R. Townsend, MD
University of Pennsylvania*

Vice President

*Samer Najjar, MD
Medstar Washington Hospital Center*

Secretary-Treasurer

*Dean C. Winter, PhD
AtCor Medical, Inc.*

Chairman of the Board

*Stanley Franklin, MD
University of California Irvine*

Industry Liaison

*Peter U. Feig, MD
PF Pharmaceutical Development, LLC*

Board of Directors

*David M. Herrington, MD, MHS,
Wake Forest Baptist Medical Center
Winston-Salem, NC*

*Edward G. Lakatta, MD
National Institute on Aging
Baltimore, MD*

*Harold Smulyan, MD
University Health Care Center
Syracuse, NY*

*Ernesto L. Schiffrin, MD, PhD
Lady Davis Institute for
Medical Research at the
Jewish General Hospital
Montreal, Quebec, Canada*

*Elaine M Urbina MD,
Cincinnati Children's Hospital
Medical Center
Cincinnati, OH*

North American Artery
68 Carlton Terrace
Stewart Manor, NY 11530

Dear Colleagues,

I'd like to personally welcome each of you to our Fifth Annual Meeting, "Hemodynamics and Target Organ Damage: Mechanisms, Measurements, Management". The field of arterial hemodynamics is an exciting area and the North American Artery Society will continue to meet and bring inspired people together in forums like this, to ensure the NAA remains at the cutting edge of research and the practical applications for technologies that measure arterial structure and function.

I appreciate the efforts of our Conference Co-Chairs, Bo Fernhall and Gary Pierce, as well as the members of the Program Committee for working tirelessly with me to put together this program. We are also indebted to 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. I encourage you to think about being a part of our new and exciting organization.

This conference would not be possible without the generous support of our Diamond sponsors, AtCor Medical, Inc. and Fukuda Denshi, and our Gold sponsors, Cardiovascular Engineering, Inc., Hitachi Aloka Medical America, and Welch Allyn Inc./I.E.M. GmbH. 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, I would like to thank each of you for attending the conference and bringing your expertise to our gathering. Your vision, knowledge, and experience will help us pave the way for future developments in arterial studies. Throughout this conference, you are encouraged to engage faculty and sponsors. My personal respect and thanks to all!

Sincerely,

Raymond R. Townsend, M.D.
NAA President

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 Fifth Annual Meeting, “Hemodynamics and Target Organ Damage: Mechanisms, Measurements, Management”. The NAA is a multidisciplinary society dedicated to understanding vascular structure and function and its application to clinical medicine, basic/translational research, and pharmaceutical and medical device development. The 2015 once again reflects these objectives with presentations focusing on the relationship between vascular function and diseases of target organs such as the brain and kidney, as well as clinical applications of arterial mechanisms, measurement, and management.

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 35 abstract presentations that are included in this year’s meeting, as well as the exciting main lectures, debates, and exhibits. We have also listened to you and have added a 45-minute informal roundtable discussion period that will allow participants, particularly students, trainees and junior faculty, the opportunity for individualized, informal discussion of the presentations and research with our expert faculty.

We truly hope you will enjoy the 2015 NAA meeting at the Chicago Marriott O’Hare and that you take the opportunity to meet our speakers, exhibitors, and delegates from not only the United States, but from Canada, Europe, and Australia as well.

We would especially like to thank our sponsors AtCor Medical, Cardiovascular Engineering Inc., Fukuda Denshi, Hitachi Aloka Medical America, and Welch Allyn/ I.E.M. 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,

A handwritten signature in black ink, appearing to read 'Bo Fernhall'.

Bo Fernhall, Ph.D.

A handwritten signature in black ink, appearing to read 'Gary Pierce'.

Gary Pierce, Ph.D.

GENERAL INFORMATION

Meeting Venue/Headquarters Hotel Marriott Chicago O'Hare

Located 15 miles from downtown, this renowned hotel combines the distinguished atmosphere of a Marriott Hotel with modern accommodations and innovative amenities. Visitors to downtown Chicago have 'L'-train access located just one block from this Chicago hotel near the airport. A departure from ordinary Chicago Airport hotels, the recently renovated Chicago Marriott O'Hare Airport hotel represents a first-class landing offering convenience, an established reputation and state-of-the-art technology. Each of the hotel's 470 guest rooms and 10 suites feature high-speed Internet and plush bedding for a restful night's sleep.

Attendees staying at the hotel receive complimentary internet access in their rooms.

Airport Shuttle

The Chicago Marriott O'Hare offers complimentary shuttle service, which runs every 20 minutes between 6:00am and 11:00pm. After hours please contact the hotel (773) 693-4444. Proceed to Baggage Claim, follow signs "Bus/Shuttle Center" to nearest underground walkway, wait by Door 2. Vans arrive every 20 minutes.

Valet and Self-Parking

On-site Parking - \$28 daily

Daytime Parking - \$18.00 Monday-Friday and \$11.00 Saturday-Sunday

Hotel Amenities

- Fitness Center & Indoor Pool
- Room Service available from 6:30 am to midnight
- Staffed, Full Service Business Center with internet printing
- Complimentary wireless access in all public spaces

Restaurants

- Brickton Restaurant & Lobby Bar - American Cuisine
- Starbucks Kiosk

Conflict of Interest Disclosure

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

Meeting Registration – Chicago Ballroom Foyer

All Conference materials including badges can be picked up from the registration desk during the following hours:
September 11, 2015 11:00 AM - 7:00 PM
September 6, 2014 5:30 AM - 3:00 PM

Badges are required for entry to all functions.

Sessions – Chicago Ballroom A

All sessions will take place in Grand Ballroom H except for the Dinner, which will take place in the United Room (lower level).

Posters on Display—Chicago Ballroom D

Posters will be on display throughout the conference. Presenters will be available to discuss their posters during the Lunch on Saturday.

Exhibits—Chicago Ballroom D

All meal functions and refreshment breaks, except the dinner and breakfast, will be held in the exhibit hall as shown below.

Friday, September 5, 2014

Opening Reception	1:00 to 1:30 PM
Refreshment Break	4:10 to 5:10 PM
Reception (Grand Ballroom 1-2)	7:15 to 7:45 PM
Dinner (Grand Ballroom 1-2)	7:35 to 9:00 PM

Saturday, September 6, 2014

Breakfast (Grand Ballroom 1-2)	6:30 to 8:00 AM
Refreshment Break	9:00 to 9:15 AM
Refreshment Break	10:15 to 11:15 AM
Lunch/Poster Presentations	1:00 to 2:30 PM

Fifth Annual Meeting Sponsors

The North American Artery Society wishes to acknowledge the following Corporate Sponsors for their generous support of the Fifth Annual Meeting.

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

Diamond Dinner Sponsor

AtCor Medical, Inc. (USA)

Diamond Breakfast Sponsor

Fukuda Denshi

Gold Sponsors

Cardiovascular Engineering Inc.
Hitachi Aloka Medical
Welch Allyn Inc./I.E.M. GmbH

Alberto Avolio, BE, PhD, FIAMBE

Professor of Biomedical Engineering
The Australian School of Advanced Medicine, Macquarie University
North Ryde, NSW, Australia

Julio A. Chirinos, MD, PhD, FAHA

Assistant Professor of Medicine
Director, Cardiovascular Phenotyping Unit, Clinical Translational Research Center
Adjunct Faculty, Center for Magnetic Resonance and Optical Imaging
University of Pennsylvania
Perelman School of Medicine
Philadelphia, PA
Visiting Professor
Ghent University
Ghent, Belgium

John Cockcroft, MD

Professor of Cardiology
Wales Heart Research Institute
Cardiff, Wales, United Kingdom

Daniel Duprez, MD, PhD

Donald and Patricia Garofalo Chair in Preventive Cardiology
Professor of Medicine/Cardiology
Director of Research of the Rasmussen Center for Cardiovascular Disease Prevention
Professor, Epidemiology and Community Health (School of Public Health)
University of Minnesota,
Minneapolis MN

David G. Edwards, PhD

Associate Professor
Kinesiology & Applied Physiology
University of Delaware
Newark, DE

Stanley S. Franklin, MD

Clinical Professor
Department of Medicine
University of California, Irvine
Irvine, CA

David R. Jacobs, Jr., PhD

Professor, Epidemiology & Community Health
University of Minnesota
Minneapolis, MN

Donald M. Lloyd-Jones, MD, ScM

Senior Associate Dean for Clinical and Translational Research
Chair, Department of Preventive Medicine
Director, Northwestern University Clinical and Translational Sciences (NUCATS) Institute
Eileen M. Foell Professor of Heart Research
Northwestern University
Feinberg School of Medicine
Chicago, IL

Carmel McEniery, PhD

Senior Research Associate
Clinical Pharmacology Unit
University of Cambridge
Cambridge, United Kingdom

Gary F. Mitchell, MD

President
Cardiovascular Engineering Inc.
Norwood, MA

Wilmer W. Nichols, PhD

Adjunct Professor of Medicine
University of Florida
Gainesville, FL

Vasan S. Ramachandran, MD, DM

Professor of Medicine
Chief, Section of Preventive Medicine & Epidemiology
Boston University School of Medicine
Boston, MA

Douglas R. Seals, PhD

Professor, Department of Integrative Physiology
University of Colorado Boulder
Boulder, CO

James Sharman, PhD

Associate Professor and Senior Research Fellow
Menzies Institute for Medical Research, University of Tasmania
Hobart, Tasmania, Australia

Raymond R. Townsend, MD

Professor of Medicine
Director, Hypertension Program
University of Pennsylvania
Health System
Philadelphia, PA

Elaine M. Urbina, MD, MS

Director, Preventive Cardiology
Associate Professor, UC
Department of Pediatrics
Cincinnati Children's Hospital
Medical Center
Cincinnati, OH

TAKEAWAY MESSAGES

Friday, September 11, 2015

2:20 - 2:40 pm Forward Pressure Wave Amplitude and CVD Risk—Gary F. Mitchell, MD

At the completion of this activity, attendees will understand relations of variables derived from central pressure-flow analysis with pulsatile hemodynamic load and cardiovascular disease risk. The distinction between variables derived from measurement of pressure only as compared to measurement of pressure and flow will be examined and relations of each set of variables with clinical events will be presented. The information presented will allow researchers to make an informed decision about best practices for assessing components of pulsatile hemodynamic load.

2:40 - 3:00 pm Indices of Wave Reflection and Late Systolic Hypertension as Predictors of CVD Risk
Julio A. Chirinos, MD, PhD

- 1) *There is more than central pressure profiles than just the level of peak pressure (central SBP).*
- 2) *Augmentation index is a poor index of wave reflections and a limited predictor of CV risk*
- 3) *Assessment of reflection magnitude via wave separation analysis using pressure and flow signals is optimal. Pressure-only approaches using physiologic flow waveforms has proven to be highly predictive of heart failure in the general population. This is consistent with animal and experimental studies indicating that late systolic load is deleterious for the heart.*
- 4) *The cardiac effects of interventions that reduce wave reflections should be tested in clinical trials.*

3:20 - 3:40 pm Is There a Difference in CVD Prediction from Old versus New Derived Arterial Waveform Parameters?—Daniel Duprez, MD, PhD

There is evidence that in asymptomatic populations free of overt cardiovascular disease carotid-femoral PWV, Augmentation Index and C1 and C2 predict cardiovascular disease events.

New derived arterial waveform parameters are predictive for heart failure events. Ongoing analysis for predictive CVD outcome by these new derived arterial waveform parameters are underway.

Saturday, September 12, 2015

8:30 - 9:00 am The Brain as a Target Organ of the Pulse—Alberto Avolio, BE, PhD

Pulsatility of blood pressure and flow can contribute to cerebral dysfunction.

9:15 - 9:45 am Hemo Math: $[\uparrow\text{CPP}] + [\uparrow\text{PWV}] = \text{Proteinuria}$ —Raymond R. Townsend, MD

Arterial stiffness adds an additional hemodynamic stress to the micro-circulation of the kidney. This is partly due to the high blood flow, low vascular resistance state of the kidney circulation

A consequence of the baro-trauma associated with higher stiffness is proteinuria.

Proteinuria, in turn, is a significant predictor of CKD progression, as well as CV outcomes in CKD.

9:45 - 10:15 am Vascular Function and Change in Kidney Function: Are They Related?
David G. Edwards, PhD

Alterations in endothelial function and arterial stiffness have the potential to accelerate declines in kidney function. Epidemiological evidence is emerging to support a role for arterial stiffness in the progression of kidney disease however research is needed to understand the role of impaired endothelial function may play in the progression of kidney disease.

11:15 - 11:45 am Accelerated Vascular Aging in Youth with Cardiovascular Risk Factors
Elaine M. Urbina, MD, MS

Arterial stiffness can be reliably measured in youth with proper training.

CV Risk Factors measured in adolescents are associated with increased arterial stiffness indicating accelerated vascular aging.

Medications and lifestyle modifications can slow this accelerated atherosclerosis.

Pediatricians should promote healthy lifestyles to prevent future heart attack and stroke.

11:45 - 12:10 pm Aldosterone as an Important Mediator of EVA and Arterial Stiffness
Vasan Ramachandran, MD, DM

Aldosterone concentrations are inappropriately high in many patients with high blood pressure, in some individuals with the metabolic syndrome and/or obesity. Substantial evidence suggests that aldosterone contributes to the development of high blood pressure in individuals (even in the absence of primary hyperaldosteronism). Aldosterone also is a key mediator of cardiovascular remodeling, cardiac and vascular fibrosis, early vascular aging and vascular stiffness. Such cardiovascular remodeling can antedate the development of high blood pressure, and characterized by ventricular and vascular hypertrophy and fibrosis, inflammation and impaired vascular reactivity, and increased conduit artery stiffness manifesting as elevated pulse wave velocity. Aldosterone blockade by pharmacological means has been shown to reverse some of these effects, especially in the setting of chronic heart failure, and in post-myocardial infarction patients with heart failure. The mineralocorticoid receptor is a key player in mediating the vascular effects of aldosterone possibly via increased vascular expression of integrin alpha 5. Recognizing the contribution of aldosterone to early vascular aging and its potential reversibility with mineralocorticoid receptor blockade is critical for all clinicians managing patients with arterial disease including elevated vascular stiffness.

12:10 - 12:35 pm Arterial Stiffness and Low Perfusion Pressure as Consequences of EVA
Stanley S. Franklin, MD

This community-based Framingham Heart Study is the first to show that early vascular aging may be an effect modifier that increases pulse pressure and decreases diastolic blood pressure as central elastic arteries stiffen, resulting in premature isolated systolic hypertension and low diastolic perfusion pressure that is independent of antihypertensive therapy. Furthermore, it is hypothesized that large artery stiffness increases pulsatility to the microcirculation, and low perfusion pressure impairs intrinsic autoregulation of blood flow with acceleration of ischemic damage to heart, brain, and kidneys.

12:35 - 1:00 pm Effective Primordial and Primary Prevention Health Measures in Prevention of EVA and Arterial Stiffness—Donald M. Lloyd-Jones, MD, ScM

Early interventions that promote primordial prevention and cardiovascular health preservation can have significant impact in delaying vascular damage and preventing cardiovascular disease.

2:45 - 3:15 pm Lifestyle and Pharmacological Strategies for Age-Associated Arterial Stiffening
Douglas R. Seals, PhD

Regular aerobic exercise, reduced energy intake-associated weight loss (for overweight-obese individuals) and maintaining a dietary sodium intake within current medical guidelines should be considered for minimizing stiffening of the large elastic arteries with aging. In general, the efficacy of nutraceutical and other supplements remains to be established in middle-aged and older adults.

AGENDA—SEPTEMBER 11, 2015

- 1:00 - 1:30 pm** **Welcome Reception in Exhibit Hall**
- 1:30 - 1:35 pm** **Welcome Remarks**
Bo Fernhall, PhD, University of Illinois at Chicago
Gary L. Pierce, PhD, University of Iowa
- 1:35 - 1:45 pm** **President's Opening Statement**
Raymond R. Townsend, MD, University of Pennsylvania
- 1:45 - 2:15 pm** **Opening Plenary Lecture**
Moderator: Bo Fernhall, PhD, University of Illinois at Chicago
- 1:45 - 2:10 pm **Measuring Central Blood Pressure: Testing New Methods for Truth**
2:10 - 2:15 pm Q&A
James Sharman, PhD, University of Tasmania
- 2:20 - 4:10 pm** **NAA Special Symposium – Measuring the Central Arterial Wave Form: New Methods versus Tried and True**
*Moderators: Alberto Avolio, BE, PhD, Macquarie University
Wilmer W. Nichols, PhD, University of Florida*
- 2:20 - 2:35 pm **Forward Pressure Wave Amplitude and CVD Risk**
2:35 - 2:40 pm Q&A
Gary F. Mitchell, MD, Cardiovascular Engineering, Inc.
- 2:40 - 2:55 pm **Indices of Wave Reflection and Late Systolic Hypertension as Predictors of CVD Risk**
2:55 - 3:00 pm Q&A
Julio A. Chirinos, MD, PhD, University of Pennsylvania School of Medicine
- 3:00 - 3:15 pm **Morphologic and Physiologic Connections of the Radial Artery Waveform with Incident CVD**
3:15 - 3:20 pm Q&A
David R. Jacobs, Jr., PhD, University of Minnesota School of Public Health
- 3:20 - 3:35 pm **Is There a Difference in CVD Prediction from Old versus New Derived Arterial Waveform Parameters?**
3:35 - 3:40 pm Q&A
Daniel Duprez, MD, PhD, University of Minnesota
- 3:40 - 4:00 pm **Panel Discussion with Audience Participation**
Faculty and Moderators
- 4:10 - 5:10 pm** **Refreshment Break, Meet the Vendors, and Poster Viewing**

5:10 - 6:40 pm **Oral Abstract Presentations**

Moderators: Stella Daskalopoulou, MD, MSc, DIC, PhD, McGill University

Elaine M. Urbina, MD, MS, Cincinnati Children's Hospital Medical Center

5:10 pm
OR-01

Resting Systolic Blood Pressure and Body Mass Index but Not Aortic Stiffness Independently Predict Systolic Blood Pressure Response to Maximal Exercise in Healthy Adults

Abbi D Lane-Cordova¹, Lyndsey E Dubose¹, Kaitlyn Dubishar¹, Michelle W. Voss², Maggie Swift², Gardar Sigurdsson³, Philip G Schmid^{3,6}, Gary L Pierce^{1,4,5,6}

¹Departments of Health and Human Physiology, ²Psychology, ³Internal Medicine, and the

⁴Fraternal Order of Eagles Diabetes Research Center, ⁵Center for Hypertension Research,

⁶Aboud Cardiovascular Research Center, University of Iowa, Iowa City, IA

5:25 pm
OR-02

Aortic Stiffness, Obesity and Left Atrial Volume in Older Participants in the Age, Gene/Environment Susceptibility—Reykjavik Study

Benjamin A. Mitchell, BA^{}, Alec Trub, BA^{*}, Alyssa A Torjesen, BS^{*}, Sigurdur Sigurdsson, MS[†], Jos JM Westenberg, PhD[‡], Vanessa C Bell, BS^{*}, Thor Aspelund, PhD^{†,§}, Lenore J Launer, MS, PhD^{||}, Tamara B Harris, MD, MS^{||}, Albert de Roos, MD[‡], Vilmundur Gudnason, MD, PhD^{†,§} and Gary F Mitchell, MD^{*}*

^{*}Cardiovascular Engineering, Inc., Norwood, MA; [†]Icelandic Heart Association, Kopavogur,

Iceland; [‡]Department of Radiology, Leiden University Medical Center, Leiden, The Netherlands;

[§]Faculty of Medicine, University of Iceland, Reykjavik, Iceland; and ^{||}Laboratory of

Epidemiology, Demography, and Biometry, Intramural Research Program, National Institute on Aging, National Institutes of Health, Bethesda, MD

5:40 pm
OR-03

The Effects of High-Intensity Aerobic Interval versus Moderate-Intensity Continuous Aerobic Exercise on Post-Exercise Cardiovascular Responses

D.S. Kimmerly, D. Ramsay, and A.M. Irwin

Dalhousie University, School of Health And Human Performance, Division of Kinesiology

5:55 pm
OR-04

Carotid Arterial Circumferential Strain Has a Greater Association to Vascular Aging than Conventional Carotid Arterial Stiffness

Rosenberg, A., Lane-Cordova, AD.², Kappus, RM.¹, Bunsawat, K¹, Wee, SO¹, Baynard, T.¹, Fernhall B¹

¹Integrative Physiology Laboratory, University of Illinois at Chicago, Chicago, IL; ²University of Iowa, Iowa City, IA

6:10 pm
OR-05

Forward Wave Amplitude Is Not Solely Dependent on Proximal Aortic Properties: Importance of Wave Reflections

Timothy S. Phan^a, Julio A. Chirinos^b, John K-J. Li^a

^aRutgers University, Piscataway, NJ, USA, ^bUniversity of Pennsylvania, Philadelphia, PA, USA

6:25 pm
OR-06

Effect of Nitrate Ingestion on Central Hemodynamics in Hypoxia

Wesley K. Lefferts, William E. Hughes, Kevin S. Heffernan

Department of Exercise Science, Syracuse University, Syracuse, NY

5:40 - 7:10 pm **NAA Business Meeting**

7:15 - 7:45 pm **Pre-Dinner Reception**

AGENDA—SEPTEMBER 11 & 12, 2015

7:45 - 9:15 pm Diamond Dinner & Lecture

7:45 - 8:45 pm Dinner

8:45 - 9:15 pm Validation of Non-Invasive Central BP Measurement Systems
Alberto Avolio, BE, PhD, Macquarie University

The Diamond Dinner and Lecture is sponsored by AtCor Medical, Inc. (USA)

Saturday, September 12, 2015

6:30 - 8:00 am Diamond Breakfast & Lecture

6:30 - 7:30 am Breakfast

7:30 - 8:00 am Comprehensive Phenotyping of Arterial Stiffness and Hemodynamics in Multicenter Studies: In Search of a Practical Approach
Julio A. Chirinos, MD, PhD, University of Pennsylvania Perelman School of Medicine

The Diamond Breakfast and Lecture is sponsored by Fukuda Denshi USA

8:00 - 8:30 am Keynote Presentation

Moderator: Raymond R. Townsend, MD, University of Pennsylvania Health System

8:00 - 8:25 am Isolated Systolic Hypertension in Youth: Clinical Importance and Underlying Mechanisms

8:25 - 8:30 am Q&A
Carmel McEniery, PhD, University of Cambridge

8:30 - 9:00 am Target Organ Lecture – The Brain

Moderator: Gary F. Mitchell, MD, Cardiovascular Engineering, Inc.

8:30 - 8:55 am The Brain as a Target Organ of the Pulse

8:55 - 9:00 am Q&A
Alberto Avolio, BE, PhD, Macquarie University

9:00 - 9:15 am Refreshment Break

9:15 - 10:15 am Target Organ Lecture – The Kidney

Moderator: Michael D. Brown, PhD, UIC College of Applied Health Sciences

9:15 - 9:35 am Hemo Math: [\uparrow CPP] + [\uparrow PWV] = Proteinuria

9:35 - 9:45 am Q&A
Raymond R. Townsend, MD, University of Pennsylvania

9:45 - 10:05 am Vascular Function and Change in Kidney Function: Are They related?

10:05 - 10:15 am Q&A
David G. Edwards, PhD, University of Delaware

10:15 - 11:15 am Refreshment Break, Meet the Vendors & Poster Viewing

AGENDA—SEPTEMBER 12, 2015

11:15 - 1:00 pm **Early Vascular Aging (EVA) Symposium**

Moderator: Stanley S. Franklin, MD, University of California, Irvine

11:15 - 11:40 am **Accelerated Vascular Aging in Youth with Cardiovascular Risk Factors**

11:40 - 11:45 am Q&A

Elaine M. Urbina, MD, MS, Cincinnati Children's Hospital Medical Center

11:45 - 12:05 pm **Aldosterone as an Important Mediator of EVA and Arterial Stiffness**

12:05 - 12:10 pm Q&A

Vasan Ramachandran, MD, DM, Boston University School of Medicine

12:10 - 12:30 pm **Arterial Stiffness and Low Perfusion Pressure as Consequences of EVA**

12:30 - 12:35 pm Q&A

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

12:35 - 12:55 pm **Effective Primordial and Primary Prevention Health Measures in Prevention of EVA and Arterial Stiffness**

12:55 - 1:00 pm Q&A

Donald M. Lloyd-Jones, MD, ScM, Northwestern University Feinberg School of Medicine

1:00 - 2:30 pm **Lunch and Poster Presentations**

2:00 - 2:45 pm **Roundtable Discussions with our Expert Faculty**

This informal 45-minute, open discussion session will allow participants, particularly students, trainees and junior faculty, the opportunity for individualized, informal discussion of the presentations and research with our expert faculty and committee members. There will be six numbered tables with each table featuring three faculty. No topics will assigned as it is an open discussion.

2:45 - 3:15 pm **Keynote Presentation**

Moderator: Stanley S. Franklin, MD, University of California, Irvine

2:45 - 3:10 pm **Lifestyle and Pharmacological Strategies for Age-Associated Arterial Stiffening**

3:10 - 3:15 pm Q&A

Douglas R. Seals, PhD, University of Colorado-Boulder

3:15 - 3:50 pm **Debate 2015**

Moderator: Raymond R. Townsend, MD, University of Pennsylvania Health System

Cardiovascular Risk Assessment: Coronary Calcium Scans versus Arterial Stiffness

David R. Jacobs, Jr., PhD, University of Minnesota School of Public Health

John Cockcroft, MD, University of Wales College of Medicine

3:50 - 3:55 pm **Awards Presentations**

Best Abstract and Young Investigator Awards

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

FACULTY DISCLOSURES

Alberto Avolio, BE, PhD

Grant/Research Support: AtCor Medical, Inc.

Julio A. Chirinos, MD, PhD

Consultant: Bristol-Myers Squibb, Merck, OPKO Health Inc., Fukuda Denshi

Grant/Research Support: Bristol-Myers Squibb, Fukuda Denshi, NIH, American College of Radiology, VA

Other: Named as inventor in patent application for the use of inorganic nitrates in HFPEF

John Cockcroft, MD

Consultant: GlaxoSmithKline, Nivalis Therapeutics, Inc., Novartis, I.E.M. GmbH

Speaker's Bureau: I.E.M. GmbH

Grant/Research Support: GlaxoSmithKline

Daniel Duprez, MD, PhD

Consultant: Amgen, Astra-Zeneca, Merck, Lundbeck

Speaker's Bureau: Amgen, Astra-Zeneca

Grant/Research Support: NIH, Sanofi, Regeneron, Pfizer

Honoraria: Amgen, Astra-Zeneca, Merck, Lundbeck

David G. Edwards, PhD

Grant/Research Support: NIH

Stanley S. Franklin, MD, has no conflict of interests to disclose.

Donald M. Lloyd-Jones, MD, ScM, has no conflict of interests to disclose.

Gary F. Mitchell, MD

Consultant: Novartis, Servier, Merck

Grant/Research Support: NIH

Stock Shareholder (self-managed): Cardiovascular Engineering Inc.

Full-time/part-time Employee: Cardiovascular Engineering Inc.

Vasan Ramachandran, MD, DM, has no conflict of interests to disclose.

Douglas R. Seals, PhD, has no conflict of interests to disclose.

James Sharman, PhD, has no conflict of interests to disclose.

Raymond R. Townsend, MD

Consultant: Medtronic, Janssen Pharmaceuticals, GlaxoSmithKline

Grant/Research Support: NIH, Fukuda Denshi

Elaine M. Urbina, MD, MS

Other: AtCor Medical

- PO-01 Estimations of Total Arterial Compliance from Carotid vs. Generalized Transfer Function-Derived Central Pressure Waveforms
- PO-02 Endogenous Hydrogen Sulfide Mediated Cutaneous Vasodilation is Attenuated in Essential Hypertensive Humans
- PO-03 Independent and Combined Effects of Aerobic and Resistance Training on Sarcopenic Indices and Its Associations with Peripheral and Central Blood Pressure
- PO-04 The Influence of Body Composition on Arterial Stiffness and Cardiorespiratory Fitness in Young Adults
- PO-05 Relationship between Arterial Stiffness and Functional Capacity in Kidney Transplant Patients
- PO-06 Effects of Hyperphosphatemia on Cerebral Small Vessel Diseases in Chronic Kidney Disease
- PO-07 CA VI Measurements in a North American Normal Population
- PO-08 Differences in Carotid Arterial Characteristics Based on Years since Diagnosis in Relapsing Remitting Multiple Sclerosis Patients
- PO-09 Influence of Body Position and Venous Pooling on Aortic Blood Pressure and Wave Reflection in Young Adults
- PO-10 Comparison of the Flow-Mediated Dilation Response between the Popliteal and Fibular Arteries
- PO-11 Independent Modifications to Backward and Forward Pressure Waves Lead to Non-Physiological Aortic Flow
- PO-12 Reconciling the Increased Pulse Wave Velocity and Reflected Wave Transit Time Paradox
- PO-13 Evaluating the Logical Relationships of Reflected Wave Transit Time with the Complex Global Reflection Coefficient, Height, and Pulse Wave Velocity
- PO-14 "Impedance Matching" Between the Aorta and Large Muscular Arteries? Misinterpretation of Pulse Wave Velocity Gradients
- PO-15 Transplant Renal Artery Stenosis: A Treatable Cause of Resistant Hypertension and Renal Allograft Dysfunction
- PO-16 Pre-Operative Pulse Wave Velocity (PWV) Values and Refractory Systemic Hypotension after Induction of General Anesthesia in Patients Treated with Angiotensin Converting Enzyme Inhibitor: A Pilot Study
- PO-17 Blood Pressure Is the Strongest Component Associated with Arterial Stiffness in Mexican Patients with Metabolic Syndrome
- PO-18 Central Blood Pressure, Wave Reflection and Subendocardial Viability Ratio in Women with a History of Hypertensive Pregnancy
- PO-19 Reproducibility of Central Systolic Blood Pressure, Augmentation Index, Measurements Calculated With the Omron HEM-9000AI Device in a Mexican Population of Young Individuals
- PO-21 Altered Vessel Hemodynamics at Rest and After Acute Physical Stress in Young Smokers
- PO-22 Arterial Stiffness and Central Systolic Blood Pressure Response to Dietary Sodium in Young and Middle-Aged Adults
- PO-23 Hemodynamic Responses Following 12 Weeks of Home-Based Exercise in Individuals with Multiple Sclerosis: Wave Separation Analysis
- PO-24 Arterial Stiffness Throughout Pregnancy in Women Who Conceive via In Vitro Fertilization
- PO-25 The Influence of Resting Heart Rate on Low- and Very Low-Frequency Blood Pressure Variability
- PO-26 A New Pathway to Increase Arterial Flexibility: Investigating Oils with Respect to Arterial Flexibility Using Photoplethysmography (The IOWA Study)
- PO-27 Longitudinal Strain Is Not Associated with Clinic Central Systolic Blood Pressure in a Young Normotensive Population
- PO-28 Resting Heart Rate Is a Factor in Acute Blood Pressure Variability Responses to Cycling
- PO-29 The Blood Pressure Variability Responses to Light Cycling in Individuals with Different Resting Heart Rates
- PO-30 Independent and Combined Effects of Aerobic and Resistance Training on Blood Pressure (ART-B)

FACULTY BIOS

ALBERTO AVOLIO, BE, PhD is Professor of Biomedical Engineering in the Faculty of Medicine and Health Sciences, Macquarie University, Sydney, Australia. His research interests include pulsatile relationships between blood pressure and flow, cardiovascular modeling and biological signal processing. He has made significant contributions to hemodynamic applications of arterial stiffness and pulse wave analysis and has recently expanded his research to include cellular and molecular mechanisms of vascular function. He serves on a number of journal editorial boards, is a reviewer for over 45 international scientific journals and has over 200 publications.

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.

JOHN R. COCKCROFT, MD is Professor of Cardiology at the Wales Heart Research Institute in Cardiff. He is also visiting Professor in the Department of Cardiology at Columbia Presbyterian Hospital New York and adjunct Professor in the Australian School of Advanced Medicine at Macquarie University, Sydney, Australia.

His major research interests focus on endothelial function and arterial stiffness, and vascular ageing in health and disease, and recently, Professor Cockcroft has become interested in the relationship between COPD and cardiovascular disease and the mechanisms involved in the increased CV risk associated with COPD. He is a founding member and past president of the Association for Research into Arterial Structure and Physiology (ARTERY) and is past Secretary of the European Association of Clinical Pharmacology and Therapeutics (EACPT). He is principal

investigator of the Assessment of Risk in Chronic Airways Disease Evaluation (ARCADE) Study. He is Editor-in-Chief of Artery Research and on the editorial board of Hypertension, the American Journal of Hypertension and the American Journal of Nephrology, and has published over 350 peer-reviewed articles and has co-authored books on hypertension and coronary heart disease.

Professor Cockcroft's clinical interests focus on hypertension and cardiovascular disease prevention, and he was a member of the committee, which produced the Welsh National Service Framework for cardiovascular disease. He is especially interested in patient empowerment and the promotion of more informed involvement with their care and treatment. He has lectured widely to patient groups on hypertension and cardiovascular disease; established the first patient self-referral clinic in the UK; and has run mobile cardiac vascular risk factor screening clinics throughout Wales and England.

Professor Cockcroft is a member of the British, European, American, and International societies of Hypertension, the British Pharmacology Society, the British Cardiac Society, and the European Association for the Study of Diabetes.

DANIEL DUPREZ, MD, PhD is the Donald and Patricia Garofalo Chair in Preventive Cardiology at the University of Minnesota where he also serves as Professor of Medicine/Cardiology, Director of Research of the Rasmussen Center for Cardiovascular Disease Prevention. He is also a Professor of, Epidemiology and Community Health at the University of Minnesota School of Public Health.

Dr. Duprez completed medical school, a residency, a cardiology fellowship, a doctorate in biomedical sciences, and a Ph.D. in cardiology. He also completed a physiology fellowship at the Mayo Clinic in Rochester, Minnesota. From research studies to clinical practice, Dr. Daniel Duprez's cardiology interests span the prevention of cardiovascular disease in the general population to its occurrence in patients with organ transplants, patients with HIV. Dr. Duprez is the author of more than 250 articles and 55 book chapters, and more than 1000 lectures nationally and internationally on a wide range of cardiology topics.

DAVID G. EDWARDS, PhD is an Associate 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.

STANLEY S. FRANKLIN, MD, FACP, FACC, FAHA, FASN, FASH 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 and arterial stiffness in the elderly with more than 200 peer-reviewed original articles and chapters.

Dr. Franklin received a degree in biology and chemistry Summa Cum Laude from the University of California at Los Angeles, his M.D. degree from Harvard Medical School, and did his post-graduate training in nephrology at Peter Bent Brigham in Boston and in clinical pharmacology at Royal Postgraduate Medical School, London.

Dr. Franklin is a Fellow of the American College of Physicians, the American College of Cardiology, the American Society of Nephrology, the American Society of

Hypertension, the High Blood Pressure Council, the Council on Geriatric Cardiology, and the American Heart Association.

The European Society for Artery Research has honored him with their 2013 "Lifetime Research Achievement Award" for his pioneering work on the importance of pulse pressure as a marker of arterial stiffness in the prediction of cardiovascular disease risk and as a metric for pathologic ageing, based on his many publications from the Framingham Heart Study.

DAVID R. JACOBS, JR., PhD, is a Professor of Public Health, division of Epidemiology and Community Health at the University of Minnesota School of Public Health. He is also a Visiting Professor at the University of Oslo, Norway.

David Jacobs's research is in nutrition and cardiovascular disease and other diseases such as diabetes. He is co-investigator of CARDIA, a national study of the evolution of

(Continued on page 18)

HITACHI ALOKA MEDICAL Vascular Ultrasound

HITACHI
Inspire the Next

Exceptional performance and an advanced ergonomic design balance the challenges of today's dynamic healthcare environment at a cost you can afford



Call Today!
844.HIALOKA



Ultrasound Solutions Clearly Defined

 **Hitachi Aloka Medical America, Inc.**

www.hitachi-alka.com/cardiology

FACULTY BIOS

cardiovascular risk in young adults and principal investigator of CARDIA's YALTA ancillary study of oxidative stress. He is co-investigator of MESA, a national longitudinal study of middle-aged and older adults and principal investigator of its MESA Air ancillary study of air pollution. He directs studies of whole grain intake and chronic disease. He has been a statistical analyst or co-investigator in experiments involving the interrelationship of drugs, diet, and exercise. He has also worked with studies of cancer and infectious disease outcomes and with drug and behavioral intervention studies.

DONALD M. LLOYD-JONES, MD, SCM, FACC, FAHA, is the Chair of the Department of Preventive Medicine and Senior Associate Dean for Clinical and Translation Research. He is the Director of the Northwestern University Clinical and Translational Sciences (NUCATS) Institute and is the Eileen M. Foell Professor of Preventive Medicine and Medicine.

Dr. Lloyd-Jones' research interests include cardiovascular disease epidemiology, risk estimation, and prevention. A main focus of his research has been investigation of the lifetime risks for cardiovascular diseases and factors which modify those risks.

He chaired the committee and authored the monograph that defined and set the American Heart Association's Strategic Impact Goals for 2010-2020, including a bold new focus on cardiovascular health promotion. He was co-chair of the Risk Assessment Work Group and a member of the Cholesterol Treatment Guidelines Panel for the 2013 ACC/AHA Guidelines for Cardiovascular Disease Risk Reduction.

Dr. Lloyd-Jones earned his MD degree from Columbia University College of Physicians and Surgeons and a Master of Science degree in Epidemiology from the Harvard School of Public Health. He is a Fellow of the American College of Cardiology and the American Heart Association, and is an inducted member of the American Society for Clinical Investigation. He is a recipient of the Patterson Award for Teacher of the Year from the Department of Medicine and the Teacher of the Year award from the Division of Cardiology at Northwestern. In 2013, Dr. Lloyd-Jones was awarded Northwestern's Tripartite Legacy Award in recognition of his achievements as a mentor, leader, and translational physician-scientist.

CARMEL MCENIERY, PhD is a Senior Research Associate in the Clinical Pharmacology Unit, University of Cambridge and is a Fellow and College Lecturer in Physiology, Churchill College, Cambridge, United Kingdom. Her research interest lies in the haemodynamic consequences of

ageing, with a particular focus on arterial stiffening and central blood pressure. She is also interested in the factors underlying the development of hypertension in young individuals, including the influence of ethnicity. She is centrally involved in the Anglo-Cardiff Collaborative Trial, a large, community-based investigation into the influence of ageing on blood pressure and arterial haemodynamics and she leads the ENIGMA and ENIGMA-Ethnicity Studies, both large, longitudinal follow-up studies investigating the natural history of blood pressure in young adults.

GARY F. MITCHELL, MD, founder and President of Cardiovascular Engineering, Inc., 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 for measuring 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.

VASAN S. RAMACHANDRAN, MD, DM is currently a Professor of Medicine at Boston University School of Public Health and the Principal Investigator of the Framingham Heart Study. He is also the Director of the Vascular and Echocardiography Laboratory at the Framingham Heart Study and the Co-Director of the Framingham Heart Study fellowship in cardiovascular epidemiology. Dr. Ramachandran's research interests include heart failure, echocardiography, high blood pressure, vascular function, biomarkers, and risk prediction. He has published on a wide range of cardiology-related topics, authoring or co-authoring over 610 referenced articles, book chapters, and abstracts. His research has appeared in such journals as *The New England Journal of Medicine*, *Lancet*, *Journal of the*

American Medical Association, British Medical Journal, Circulation, Stroke, and Journal of the American College of Cardiology.

DOUGLAS R. SEALS, PhD, College Professor of Distinction, Department of Integrative Physiology at the University of Colorado Boulder is a native of St. Louis, Missouri. He received undergraduate degrees in Education and Business from William Jewell College in Liberty, Missouri, and his M.S. and Ph.D. degrees from the University of Wisconsin-Madison in Applied Physiology. His postdoctoral research training was performed in the area of aging and applied human physiology at Washington University School of Medicine in St. Louis.

Dr. Seals subsequently held faculty appointments at the University of Iowa and University of Arizona before moving to the University of Colorado Boulder in 1992. Presently he is a College of Arts and Sciences Professor of Distinction in Integrative Physiology on the CU Boulder campus, and a Professor of Medicine (Divisions of Cardiology and

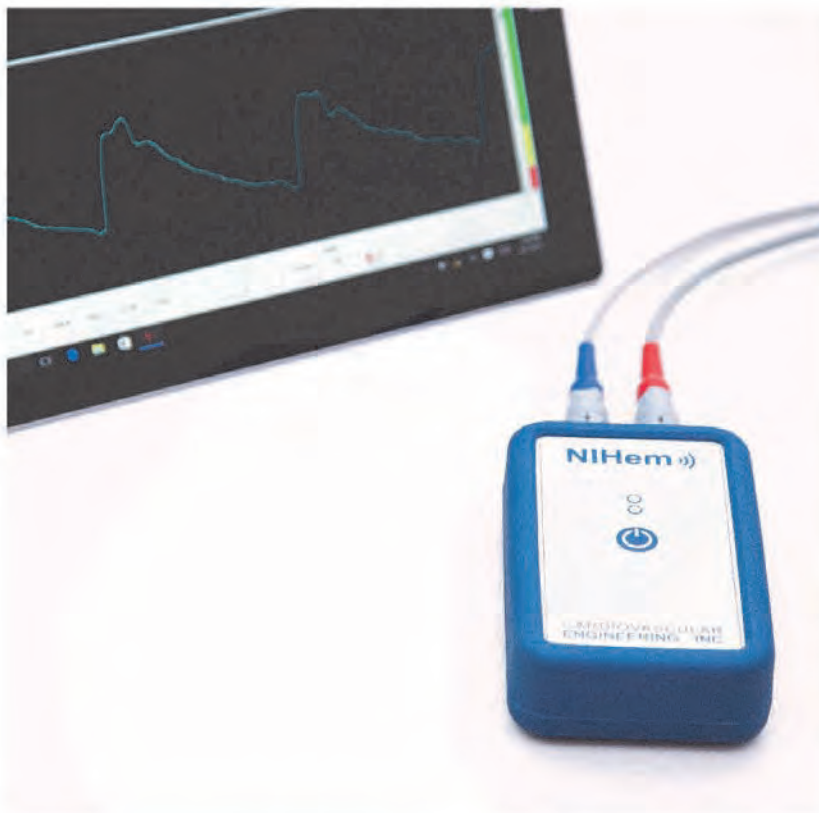
Geriatric Medicine) at the University of Colorado Denver Anschutz Medical Campus.

Dr. Seals' primary research interest is in establishing lifestyle and pharmacological strategies that delay physiological dysfunction with aging and prevent age-associated chronic degenerative diseases and disability. Much of his recent work has focused on prevention of adverse vascular aging and age-related cardiovascular diseases. He has published over 285 papers in top scientific journals.

Dr. Seals' research laboratory provides scientific training at the undergraduate, M.S., Ph.D. and postdoctoral levels. His research primarily is supported by research grants from the National Institutes of Health (NIH), particularly the National Institute on Aging (NIA) and the National Heart, Lung and Blood Institute.

Dr. Seals founded the University of Colorado Boulder NIH

(Continued on page 20)



NIHem-WF

The ultimate compact research solution for wireless tonometry data acquisition and analysis.

CARDIOVASCULAR ENGINEERING INC
cardiovascularengineering.com



FACULTY BIOS

Clinical and Translational Research Center in 1999, which provides a core facility for conducting biologically-based biomedical research on human subjects on our campus. He also established the first formal Responsible Conduct of Research program for the campus and served as its director in 2011. Professor Seals has taught undergraduate courses in physiology, and graduate courses in the physiology of aging, as well as professional skills for the research scientist. In 2003, Professor Seals received the Citation Award from the American College of Sports Medicine for career achievement in original research, scientific mentoring, and service related to his contributions in cardiovascular health, exercise and aging. In 2004, he received a MERIT Award from NIA to support his research focusing on lifestyle and pharmacological interventions for the prevention and treatment of vascular dysfunction with aging. In 2005, Professor Seals received the Herbert H. deVries Award for Distinguished Research in the Field of Aging. In 2006, Dr. Seals received the University of Colorado Boulder Faculty Assembly Award for Research, Scholarly and Creative Work. In 2008, Dr. Seals was named Professor of Distinction in the College of Arts and Sciences at the University of Colorado. Most recently, Professor Seals was named by the American Physiological Society as its 2013 Edward F. Adolph Distinguished Lecturer for his work in exercise and vascular aging.

JAMES SHARMAN, PhD, Associate Professor & Senior Research Fellow, Menzies Institute for Medical Research, University of Tasmania heads the Cardiometabolic Research Theme and the Blood Pressure Research Group at the Menzies Institute for Medical Research, Hobart, Tasmania. He undertakes research in human cardiovascular physiology, encompassing basic blood pressure physiology, central hemodynamics, exercise physiology, and clinical trials of new blood pressure methods. James holds an NHMRC R.D. Wright Biomedical Career Development Fellowship and is an Honorary Associate Professor at The University of Queensland, School of Medicine. He completed his undergraduate and Honours degree at the University of Tasmania in 2000. His PhD was at The University of Queensland and the Wales Heart Research Institute, Cardiff, United Kingdom. Prior to moving to Tasmania in 2009, he was a Postdoctoral Fellow with the Cardiac Imaging Group and Centre for Clinical Research Excellence in Cardiovascular Disease and Metabolic Disorders at the Princess Alexandra Hospital, Brisbane, Australia.

RAYMOND R. TOWNSEND, MD

is a Professor of Medicine at the Hospital of the University of Pennsylvania and the Director of the Hypertension Section in the Department of Internal Medicine/Renal at the University of Pennsylvania School of Medicine.

Dr. Townsend received his MD degree from Hahnemann University Hospital. His formal certifications are in internal medicine (ABIM), nephrology (ABIM), clinical pharmacology (ASCP) and hypertension (ASH). He is a Fellow of the American Heart Association and a Fellow of the Council for High Blood Pressure Research. An empanelled member of the NHLBI Joint National Committee (JNC8), Dr. Townsend is the Principal Investigator of the Pulse Wave Velocity in CKD ancillary project in the Chronic Renal Insufficiency Cohort Study (CRIC), and the Principal Investigator of the Penn Clinical Center in the CRIC Study.

Dr. Townsend's principal research interests focus on the role of hypertension and in particular mechanisms of kidney damage that are related to pulse wave travel and pulse wave reflection in the circulation; and in the role of metabolism, specifically the linkages between insulin resistance and kidney disease progression in people with chronic kidney disease. These two areas (arterial stiffness and metabolism) link the role of hemodynamics (as reflected in both blood pressure and vascular stiffness) to a variety of outcomes in CKD including heart failure, cognitive function changes, retinopathy, CKD progression, and vascular calcification.

ELAINE M. URBINA, MD, MS, is the Director of Preventive Cardiology and Professor of Pediatrics (Cardiology) at Cincinnati Children's Hospital Medical Center. As Director of Preventive Cardiology, her clinical activities and industry sponsored grants focus on prevention (obesity, hypertension and dyslipidemias) while her research grants (AHA, NIH) and masters in epidemiology training concentrate on new non-invasive methods of assessing atherosclerotic CV target organ damage in youth related to CV risk factors especially those that cluster with obesity.

Dr. Urbina has over 20 years of experience in non-invasive imaging of CV structure and function in large epidemiologic studies such as the Bogalusa Heart Study. She is PI of a National Institutes of Health (NHLBI R01) following the cardiac and vascular effects of obesity and type 2 diabetes on adolescents and a member of the International Childhood CV Cohorts Consortium that will

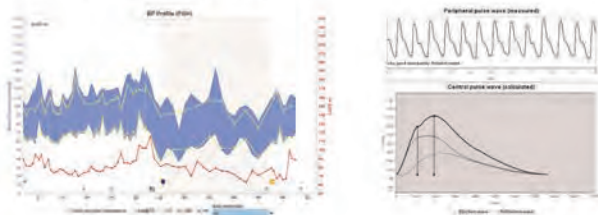
be following Bogalusa, Muscatine, Young Finns and other cohorts that collect CV risk factor data in children over 40 years ago as the participants are now entering middle age. She is also PI of an AHA Strategically Focused Network in HTN grant that will be exploring population, clinical and epigenetic determinants of target organ damage in hypertensive youth.

Dr. Urbina's team also supplies training for many multi-center pediatric studies including CKiDs (chronic kidney disease) and we are the Vascular Imaging Core for TODAY2 (type 2 diabetes), SEARCH 3 (type 1 diabetes), Udenafil (single ventricle), DoIT (dyslipidemia) grants funded by NIH's Pediatric Heart Network.



Pulse Wave Monitoring for Improvement of Therapy Decisions and Risk Stratification of Arterial Hypertension

- Peripheral blood pressure
- Central aortic blood pressure (cBP)
- Augmentation index (AIx): (\geq Age 40)
- Augmentation pressure



Simplified Clinical Routine

- Provides information about vital hemodynamic and vascular parameters
- Procedure is identical to conventional office BP and/or 24h-ABPM.
- The PWA algorithm has been validated against invasive and non-invasive reference methods.



I.E.M. GmbH | Cockerillstrasse 69 | 52222 Stolberg | Germany | T + 49 (0)2402 9500-0 | F + 49 (0)2402 950011 | info@iem.de | www.iem.de

EXHIBITORS

Diamond Sponsor

AtCor Medical, Inc. (USA)
One Pierce Place, Suite 225W
Itasca, IL 60143
Contact: Doug Kurschinski
Phone: 630-228-8874
Fax: 815-366-5953
Website: www.atcormedical.com



AtCor Medical developed and markets SphygmoCor® systems, the gold standard in non-invasive central blood pressure and pulse wave velocity assessment, used globally in research, clinical practice and as part of a turnkey service in pharmaceutical clinical trials. SphygmoCor XCEL is a brachial-cuff based system for central blood pressure assessment, providing both brachial and central pressures as well as the central blood pressure waveform and clinical indices. See more at www.atcormedical.com.

Diamond Sponsor

Fukuda Denshi USA
17725-C NE 65th Street
Redmond, WA 98052
Phone: 425-241-7396
Fax: 425-869-2018
Website: www.fukuda.com



The VaSera System

VaSera™ brings reliable measurement of arterial stiffness (heart-to-ankle PWV) to the busy clinician's practice. Our cuff-based system requires no special user training, and includes automated ABI- and TBI-measurement functionality. Chose VaSera for research-grade accuracy and quality, and fully automated ease-of-use.

Gold Sponsor

Cardiovascular Engineering, Inc.
1 Edgewater Drive, Suite 201
Norwood, MA 02062
Phone: 781-255-6930
Email: Info@CardiovascularEngineering.com
Website: www.CardiovascularEngineering.com



Over the past 17 years, 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.

Gold Sponsor

Hitachi Aloka Medical America
10 Fairfield Blvd
Wallingford, CT 06492
Phone: 844-HIALOKA
Website: www.hitachi-aloka.com/cardiology

 **Hitachi Aloka Medical America, Inc.**

Hitachi Aloka Medical is proud of the reputation we've built as an industry leader in diagnostic ultrasound. Known for our unparalleled Doppler performance, superior system reliability and intuitive use of cutting edge technology, Hitachi Aloka Medical remains the ideal choice for exceptional diagnostic ultrasound imaging in the field of Cardiac and Vascular medicine. Our focused dedication to diagnostic ultrasound imaging allows us to offer a full range of products to meet the needs for all your Cardiac and Vascular applications.

Gold Sponsor

Welch Allyn Inc. / I.E.M. GmbH
4341 State Street Road
Skaneateles Falls, NY 13153
Contact: Mr. Frits Kroon
Phone: 315-554-4032
E-mail: frits.kroon@welchallyn.com
Website: www.welchallyn.com

 **Welch Allyn®**

Celebrating its 100th anniversary this year, Welch Allyn offers a wide range of devices for blood pressure screening and management, including manual aneroid sphygmomanometers, standalone digital blood pressure devices and multi-parameter electronic vital signs monitors.

I.E.M. GmbH is the researcher, developer and manufacturer of validated medical products and services, having its business started in 1993.

The Mobil-O-Graph ABP monitor is commercialized in the US as the Welch Allyn ABPM-7100. The device has successfully been validated both invasively and non-invasively and is considered the gold standard technology for the ambulatory 24h monitoring of central blood pressure.

Resting Systolic Blood Pressure and Body Mass Index but Not Aortic Stiffness Independently Predict Systolic Blood Pressure Response to Maximal Exercise in Healthy Adults

Abbi D Lane-Cordova¹, Lyndsey E Dubose¹, Kaitlyn Dubishar¹, Michelle W Voss², Maggie Swift², Gardar Sigurdsson³, Philip G Schmid^{3,6}, Gary L Pierce^{1,4,5,6}

¹Departments of Health And Human Physiology, ²Psychology, ³Internal Medicine, and the ⁴Fraternal Order of Eagles Diabetes Research Center, ⁵Center for Hypertension Research, ⁶Abboud Cardiovascular Research Center, University of Iowa, Iowa City, IA

Objectives: Exaggerated systolic blood pressure (BP) response to maximal exercise is associated with incident hypertension and cardiovascular disease (CVD) death. Aortic stiffness, measured by carotid-femoral pulse wave velocity (cfPWV), is a robust predictor of CVD and is related to systolic BP at the 2nd stage of a graded exercise treadmill test. However, because stage 2 of a graded exercise test represents a different relative workload (% of maximal exercise oxygen uptake, VO₂max) for each participant, differences in submaximal exercise systolic BP response may be related to variation in VO₂max rather than cfPWV. Therefore, we hypothesized that systolic BP at maximal exercise would be associated with cfPWV independent of VO₂max in healthy adults. **Methods:** Heart rate (HR, 12-lead ECG) and systolic BP (measured by an experienced exercise physiologist) were assessed during a maximal graded exercise test (cycle ergometer), and cfPWV was measured via applanation tonometry at rest in 37 healthy adults (11M/26F; age=46 ± 3 yrs; range: 19-71 yrs). **Results:** Peak exercise systolic BP was associated with age (r=0.30, P=0.046), body mass index (BMI, r=0.49, P=0.001), cfPWV (r=0.44, P=0.005), and resting systolic BP (r=0.73, P<0.001), but not maximal exercise HR (r=-0.15, P=0.33) or VO₂max (r=-0.24, P=0.16). In a hierarchical multiple regression model including age, sex, BMI, VO₂max, peak HR and cfPWV (model 1), only BMI predicted maximal exercise systolic BP (β=0.370, Model R²=0.38, P=0.02). Adding resting systolic BP to the model (model 2), significantly improved the overall R² by 31% and resting systolic BP (β=0.700, P<0.01) and BMI (β=0.278, P=0.03) both predicted maximal exercise systolic BP (Model R²=0.69, P<0.01, Table 1). **Conclusions:** resting systolic BP and BMI, not cfPWV, predict the systolic BP response to maximal exercise. Reducing resting BP and adiposity may decrease the risk of hypertension and CVD in part by attenuating repeated exaggerated BP responses to physical exertion.

Supported by NIH AG043722-01, AHA 13SDG143400012 and NIH 5T320078638-28.

Table 1. Hierarchical Regression Analysis: Determinants of Peak Exercise SBP.

Model	Predictor	Coefficients for Individual Predictors				Model Coefficients (p-value)	
		B (SE)	β	Partial r	p-value	Model R ²	ΔR ² from Previous
1	Age (yr)	0.715 (0.444)	0.412	0.282	0.118	0.38 (0.020)	0.38 (0.020)
	Sex	-0.035 (14.046)	-0.001	-0.001	0.997		
	BMI (kg/m ²)	3.649 (1.623)	0.370	0.380	0.032		
	VO ₂ max(ml/kg/min)	-0.49 (0.742)	-0.012	-0.012	0.948		
	Peak HR (bpm)	0.611 (0.409)	0.311	0.263	0.146		
	cfPWV(cm/s)	0.36 (0.32)	0.237	0.200	0.273		
2	Resting SBP(mmHg)	1.762 (0.330)	0.700	0.704	0.000	0.69 (0.012)	0.31 (<0.001)
	Age (yr)	0.193 (0.335)	0.111	0.106	0.570		
	Sex	12.692 (10.425)	0.167	0.220	0.233		
	BMI (kg/m ²)	2.739 (1.185)	0.278	0.394	0.028		
	VO ₂ max(ml/kg/min)	0.312 (540)	0.079	0.107	0.568		
	Peak HR (bpm)	0.410 (298)	0.209	0.248	0.179		
	cfPWV(cm/s)	0.025 (0.023)	0.167	0.197	0.288		

Table Legend. “B” = Unstandardized Coefficient, “SE” = Standard Error, “β” = Standardized Coefficient.

Aortic Stiffness, Obesity and Left Atrial Volume in Older Participants in the Age, Gene/Environment Susceptibility—Reykjavik Study

Benjamin A. Mitchell, BA^{}, Alec Trub, BA^{*}, Alyssa A Torjesen, BS^{*}, Sigurdur Sigurdsson, MS[†], Jos JM Westenberg, PhD[‡], Vanessa C Bell, BS^{*}, Thor Aspelund, PhD^{†,§}, Lenore J Launer, MS, PhD^{||}, Tamara B Harris, MD, MS^{||}, Albert de Roos, MD[‡], Vilmundur Gudnason, MD, PhD^{†,§} and Gary F Mitchell, MD^{*}.*

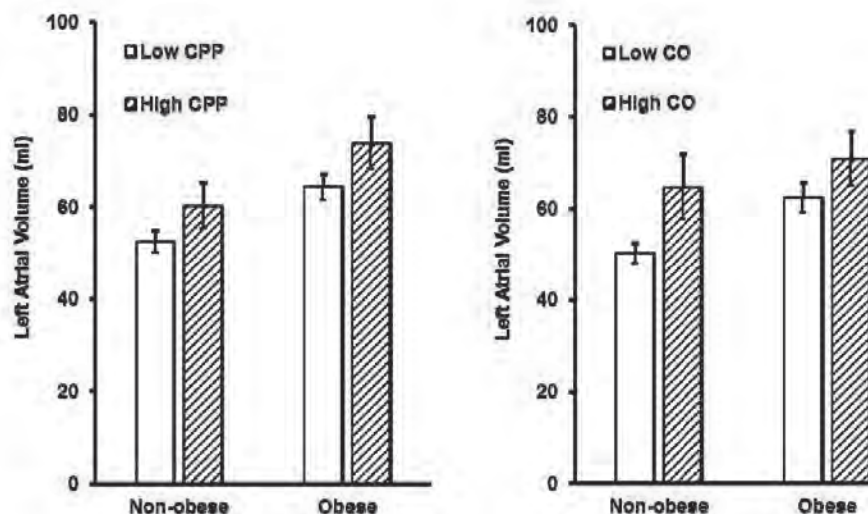
^{*}Cardiovascular Engineering, Inc., Norwood, MA; [†]Icelandic Heart Association, Kopavogur, Iceland; [‡]Department of Radiology, Leiden University Medical Center, Leiden, The Netherlands; [§]Faculty of Medicine, University of Iceland, Reykjavik, Iceland; and ^{||}Laboratory of Epidemiology, Demography, and Biometry, Intramural Research Program, National Institute on Aging, National Institutes of Health, Bethesda, MD.

Objectives: Larger left atrial volume (LAV) is associated with increased risk for atrial fibrillation, stroke, and other cardiovascular diseases. Relations of LAV with obesity and various hemodynamic measures have been investigated separately. We evaluated the combined relations of LAV with obesity, volume load on the heart, and aortic stiffness in order to determine whether relations between obesity and LAV are attributable to hemodynamic abnormalities.

Methods: We used biplane magnetic resonance imaging to measure LAV in 423 participants (72 to 89 years of age, 245 women) in the community-based Age, Gene/Environment Susceptibility-Reykjavik Study. Aortic stiffness was assessed as carotid-femoral pulse wave velocity and central aortic pressure-flow relations. Adiposity was assessed as body-mass index (BMI).

Results: In a single multivariable model that included age, sex, left ventricular diastolic volume, and standard cardiovascular disease risk factors, larger LAV was associated with higher BMI (B=1.08 fold higher LAV per SD higher BMI; 95% CL=1.04,1.11; P<0.001), cardiac output (CO) (B=1.08; 95% CL=1.04,1.11; P<0.001), central pulse pressure (CPP) (B=1.06; 95% CL=1.03,1.09; P<0.001) and carotid-femoral pulse wave velocity (B=1.04; 95% CL=1.01,1.07; P=0.007). In a risk factor-adjusted multivariable model that examined components of CPP, larger LAV was associated with higher forward wave amplitude (B=1.09; 95% CL=1.05, 1.14; P<0.001) but not backward wave amplitude (P=0.7). In a dual impedance-flow model, larger LAV was associated with higher characteristic impedance (B=1.09; 95% CL=1.05,1.12; P<0.001) and peak flow (B=1.12; 95% CL=1.08,1.16; P<0.001). Obesity (BMI≥30 kg/m², P<0.001) and CPP above the sex-specific median (P<0.001) were associated with larger LAV (interaction, P=0.7, **Figure**). Similarly, obesity (P<0.001) and CO above the sex-specific median (P<0.001) were associated with larger LAV (interaction, P=0.12, **Figure**).

Conclusions: Higher pulsatile load (attributable to increased characteristic impedance, forward wave amplitude, and a stiffer aorta), higher steady-flow volume load and obesity are jointly associated with larger LAV.



The Effects of High-Intensity Aerobic Interval versus Moderate-Intensity Continuous Aerobic Exercise on Post-Exercise Cardiovascular Responses

D.S. Kimmerly, D. Ramsay, and A.M. Irwin

Dalhousie University, School of Health And Human Performance, Division of Kinesiology

Objectives: After an acute bout of moderate-intensity continuous aerobic exercise (CAE), leg blood flow (LBF) and vascular conductance (LVC) remain elevated contributing to a corresponding decrease in arterial blood pressure (ABP). However, little is known about the vascular and ABP response following high-intensity interval aerobic exercise (HIAE). The current study tested the hypothesis that HIAE, matched for total work to CAE, would elicit greater post-exercise reductions in ABP and larger increases in LBF and LVC.

Methods: Heart rate (HR), ABP, blood lactate and LBF (popliteal artery, Doppler) were measured in 10 healthy participants (7M/3F, 20.2 ± 1.6 years) in a side-lying position before and every 5 minutes for 1 hour following CAE and HIAE treadmill protocols. LVC was calculated as LBF divided by mean ABP. The HIAE consisted of four, 4-minute intervals at $85\%VO_{2peak}$ separated by 3-minute active recovery intervals at $55\% VO_{2peak}$. The CAE protocol consisted of treadmill running at $\sim 55\% VO_{2peak}$ for a duration that produced the same total oxygen consumption as HIAE.

Results: Compared to pre-exercise values, peak decreases in mean ABP (-6 ± 1 mmHg vs. 0 ± 1 mmHg) and diastolic ABP (-5 ± 2 mmHg vs. -1 ± 1 mmHg), as well as, increases in blood lactate (10.5 ± 3.2 mmol/L vs. 3.4 ± 1.4 mmol/L), HR (37 ± 10 beats/minute vs. 21 ± 6 beats/minute) and leg vascular conductance (8 ± 5 mL/min/mmHg vs. 5 ± 4 mL/min/mmHg) were greater during the passive post-exercise recovery period following HIAE than CAE (all, $p < 0.05$).

Conclusions: These results highlight that a matched-work HIAE treadmill protocol resulted in greater skeletal muscle vasodilation during a 1 hour passive post-exercise recovery period than CAE. These data suggest that HIAE may prove to be more beneficial as a therapeutic strategy for patient populations with peripheral blood flow limitations (e.g., peripheral arterial disease).

*Supported by a Canadian Foundation for Innovation: Leader's Opportunity Fund grant to D.S. Kimmerly.

Carotid Arterial Circumferential Strain Has a Greater Association to Vascular Aging than Conventional Carotid Arterial Stiffness.

Rosenberg, A.¹, Lane-Cordova, Ad.², Kappus, Rm.¹, Bunsawat, K.¹, Wee, So.¹, Baynard, T.¹, Fernhall B.¹

¹Integrative Physiology Laboratory, University of Illinois at Chicago, Chicago, IL; ²UNiversity of Iowa, Iowa City, IA

Objective: Arterial stiffness is closely related to the risks of CVD and increases with aging. Functional impairment of the arterial wall can occur before structural changes and can be detectable before CVD symptoms. The elastic properties of the carotid arterial wall during the cardiac cycle can be evaluated by either standard 2-dimensional (2D) ultrasound longitudinal or 2D ultrasound circumferential imaging of vascular deformation (Strain) using speckle tracking. The purpose of this study was to compare standard longitudinal scans with 2D circumferential ultrasound imaging of vascular tissue motion and Strain using speckle tracking with in young and old people.

Methods: Young and older adults had 2D ultrasound circumferential and longitudinal axis images of the common carotid artery recorded. Circumferential carotid strain (CS) and CS rate were obtained and analyzed via speckle tracking software. Following the strain analysis, the circumferential strain beta-stiffness (C- β) was calculated. Longitudinal beta-stiffness (L- β) was calculated and non-invasive blood pressure measurements obtained from carotid artery pressure measurements (carSBP, carDBP, carPP, carMAP) in a resting supine position using applanation tonometry.

Results: C- β was significantly higher than L- β , and the association with age was greater ($r = .824$ vs $r = .547$). Resting CS and CS rate were significantly higher in the young compared to the older group. See table below for descriptive characteristics.

Conclusion: Conventional longitudinal beta-stiffness does not explain as much of the age-dependent differences in the carotid artery compared with circumferential strain beta-stiffness by two-dimensional speckle tracking imaging. This is possible due to inclusion of the whole arterial wall motion and deformation that is observed in the circumferential strain image. Circumferential strain Beta-stiffness appears to provide additional information when comparing age-dependent changes in vascular stiffness. The ability of circumferential strain beta-stiffness to accurately predict the future risk of CVD independent of age still needs to be investigated.

Variables	Young (n=13)	Older (n=16)
Age (yrs) *	26 ± 1	62 ± 2
BMI (kg/m ²) *	24.1 ± 0.9	28.0 ± 1.3
Heart Rate (bpm)	57 ± 3	63 ± 2
carSBP (mmHg)	104 ± 4	116 ± 5
carDBP (mmHg) *	66 ± 2	73 ± 2
carMAP (mmHg) *	83 ± 2	91 ± 3
carPP (mmHg)	45 ± 2	52 ± 3
AC (mm ² /kPa) *	1.48 ± 0.20	0.96 ± 0.14
L-Systolic Diameter (mm) *	6.82 ± 0.22	7.79 ± 0.29
L-Diastolic Diameter (mm) *	6.15 ± 0.22	7.34 ± 0.29
L- β *	4.5 ± 0.4	8.8 ± 0.8
C-Systolic Diameter (mm) *	6.86 ± 0.10	7.66 ± 0.23
C-Diastolic Diameter (mm) *	6.24 ± 0.11	7.24 ± 0.22
C- β *	6.0 ± 0.6	16.7 ± 1.4
CS (PK%) *	8.3 ± 0.8	3.1 ± 0.3
CS Rate (PK 1/s) *	0.65 ± 0.06	0.25 ± 0.02
Radial Displacement (PK mm) *	0.27 ± 0.02	0.12 ± 0.01
Radial Velocity (cm/s) *	0.23 ± 0.02	0.10 ± 0.01

All Data are mean ± SEM, * Age Difference, p<0.05

Forward Wave Amplitude Is Not Solely Dependent on Proximal Aortic Properties: Importance of Wave Reflections

Timothy S. Phan^a, Julio A. Chirinos^b, John K-J. Li^a

^aRutgers University, Piscataway, NJ, USA, ^bUniversity of Pennsylvania, Philadelphia, PA, USA

Background: The contribution of proximal aortic properties and wave reflections to increased pulse pressure with aging remains controversial. The forward wave amplitude (FWA) paradigm proposes an important role for mismatch between flow and proximal aortic properties. This proposition assumes that the morphology and amplitude of the forward wave depend exclusively on the aortic root. This is unlikely, since FWA depends on the LV ejection pattern, which itself is sensitive to wave reflections.

Methods: Simultaneous aortic pressure and flow were measured in anesthetized, open-chest dogs (n=5). Wave reflections were modified through i.v. infusion of methoxamine and nitroprusside to increase and decrease reflections, respectively (Figure 1). Pressure waves were decomposed into forward and backward waves using standard methods.

Results: The time of peak flow and FWA were only approximately equal in the case of minimal reflections (Figure 2). In the presence of reflections, FWA occurred later than the time of peak flow. Increased reflections impart an inflection point on the forward wave, analogous to the inflection point on measured pressure waveforms. In the presence of normal/increased reflections, FWA was systematically greater than peak flow multiplied by aortic characteristic impedance (23.3 vs. 18.5 mmHg; P=0.006)

Conclusion: Only in cases of minimal reflections does FWA primarily reveal the interaction between peak flow and proximal aortic diameter/stiffness. Forward and backward waves are derived under the assumption of steady-state oscillations, in which both the forward and backward waves are determined by reflections. FWA is therefore influenced by wave reflections. When interpreted out of context with the hemodynamic principles of its derivation, the FWA amplitude paradigm erroneously amplifies the role of the proximal aorta on pulse pressure. We conclude that the FWA paradigm reinforces rather than precludes the role of increased reflections on the increased PP with age and disease.

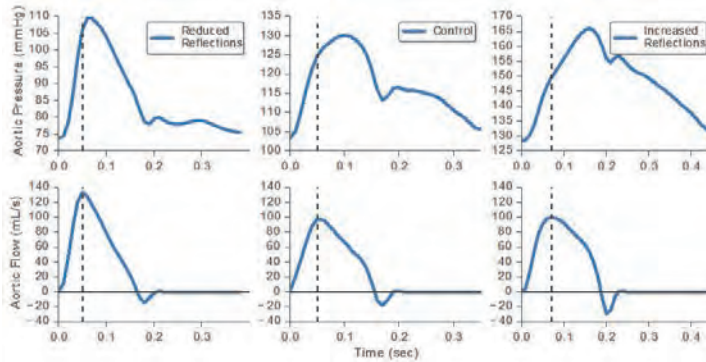


Figure 1. Simultaneously measured aortic pressure (top) and flow (bottom) waveforms. Dashed lines indicate time of peak flow.

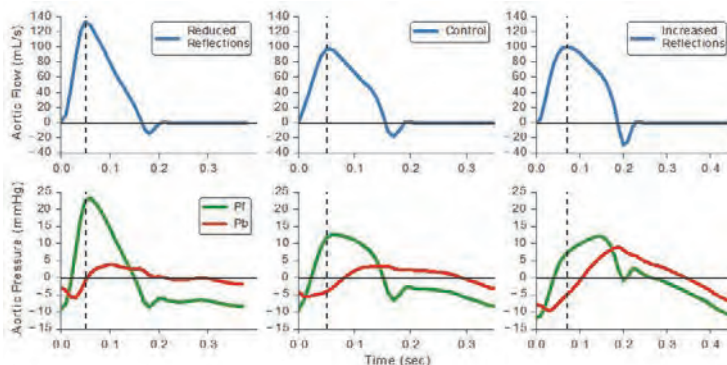


Figure 2. Measured aortic flow (top) compared with backward and forward pressure waves (bottom). The dashed lines indicate time of peak flow.

Effect of Nitrate Ingestion on Central Hemodynamics in Hypoxia

Wesley K. Lefferts, William E. Hughes, Kevin S. Heffernan

Department of Exercise Science, Syracuse University, Syracuse, NY

Acute hypoxia results in local vasodilation that may temporarily unload the left ventricle (LV) through nitric-oxide (NO)-mediated mechanisms. Whether increasing NO levels augments LV unloading and improves ventricular-vascular coupling in hypoxia remains unknown. **PURPOSE:** Investigate the effect of nitrate ingestion on central hemodynamics in hypoxia. **METHODS:** 20 Healthy men (23±3 yrs, BMI 24.6±2.8 kg·m⁻²) consumed 70 mL of either a) a 0.45 g nitrate (NIT) or b) an inert placebo (PLA) prior to 105 min of normobaric hypoxia (11.6±0.1%) in this randomized, double-blind, crossover-design study. Central hemodynamic variables were derived from the aortic blood pressure (BP) waveform at normoxic baseline and in hypoxia. Wave reflection index (RIx; ratio of forward to reflected wave pressure), augmentation index (AIx75) and pulse wave velocity were assessed as measures of wave reflection and aortic stiffness, respectively. LV wasted pressure effort (WPE) was calculated as an index of LV work due to wave reflections, and subendocardial viability ratio (SEVR) as a measure of myocardial O₂ supply/demand ratio. Arterial O₂ saturation was measured to quantify the hypoxic stimulus. **RESULTS:** Hypoxia significantly reduced arterial oxygen saturation compared to normoxia (p<0.05). Aortic diastolic BP, RIx, AIx75, and LV WPE significantly decreased in hypoxia compared to normoxia (p<0.05). SEVR and PWV were unaffected by hypoxia (p>0.05). Nitrate ingestion did not significantly alter central hemodynamics in hypoxia (p<0.05). **CONCLUSIONS:** Acute hypoxic exposure unloads the LV and reduces myocardial energetics without disturbing myocardial O₂ supply-demand ratio via reductions in pressure from wave reflections and not from changes in aortic stiffness per se. Nitrate ingestion did not improve LV unloading or ventricular-vascular coupling in acute hypoxia.

This research was supported by an American College of Sports Medicine Foundation Research Grant

Table 1: Central hemodynamics, wave reflections, and indices of left ventricular work across treatments and conditions (mean ± SD)

	Placebo (PLA)		Nitrate (NIT)		n
	Normoxia	Hypoxia	Normoxia	Hypoxia	
Aortic SBP (mmHg)	97 ± 8	97 ± 7	97 ± 7	96 ± 10	20
Aortic DBP (mmHg)	69 ± 6	74 ± 7*	70 ± 6	72 ± 8*	20
Heart rate (b·min ⁻¹)	59 ± 9	64 ± 7*	57 ± 8	64 ± 8*	20
AIx75 (%)	-10 ± 11	-19 ± 7*	-13 ± 10	-22 ± 12*	20
LV WPE	-542 ± 856.0	-1089.7 ± 691.1*	-534.0 ± 902.1	-1312.0 ± 932.6*	14
Aortic SEVR (%)	176 ± 37	178 ± 29	183 ± 31	177 ± 31	20
Aortic PWV (m·s ⁻¹)	6.6 ± 0.8	6.2 ± 0.6	6.4 ± 0.7	6.2 ± 0.6	15
Aortic RIx	40 ± 8	32 ± 7*	36 ± 7	32 ± 5*	16

SBP, systolic blood pressure; DBP, diastolic blood pressure; AIx75, augmentation index at 75 bpm; WPE, wasted pressure effort; SEVR, subendocardial viability ratio; PWV, pulse wave velocity; RIx, wave reflection index.

* p<0.05 time effect, significantly different vs normoxia

Estimations of Total Arterial Compliance from Carotid vs. Generalized Transfer Function-Derived Central Pressure Waveforms

Timothy Phan¹, Izzah Vasim¹, Maheshwara R. Koppula¹, Swapna Varakantam¹, Amer A. Syed¹, Ejaz M. Shah¹, Zoubair Ahmed¹, Julio A Chirinos¹, Scott R. Akers²

¹University of Pennsylvania, Cardiovascular Medicine, ²VA Medical Center, Philadelphia, Department of Radiology

Background: Generalized transfer functions from radial pressure waveforms are often used to estimate the peak central aortic pressure. However, time-resolved information in the waveform can also be used to assess the total arterial compliance, using central pressure and flow waveforms. Although carotid waveforms are preferred over peripheral arterial waveforms, high quality carotid waveform recordings are difficult to obtain in multicenter studies.

Objectives: Total arterial compliance estimated using radial artery tonometry (using a generalized transfer function, GTF) strongly correlates with the results obtained with carotid tonometry.

Methods: We studied 212 adults (mean age = 62.8, % male = 84.5). Radial and carotid arterial waveforms were obtained with arterial tonometry (Sphygmocor device). A GTF was used to obtain a central pressure waveform from the radial pressure waveform. Aortic flow was assessed with phase-contrast MRI. Total arterial compliance was estimated using 3 methods: - (1) pulse pressure method (PPM) (2) area method (AM) (3) diastolic decay method (DDM, fitting an exponential decay to the diastolic waveform).

Results: Correlation coefficients between carotid and GTF-derived arterial compliance estimates were 0.92 for the PPM, R = 0.91 for the AM, and R=0.77 for the DDM. When comparing absolute values, estimates obtained with the PPM were significantly different when using carotid vs. radial/GTF-derived pressure waveform, with a systematic small positive bias when using radial waveforms. ($\Delta=0.152$; $P<0.001$). In contrast, values obtained with carotid vs. radial waveforms did not show any significant differences with the DDM ($\Delta=0.035$; $P=NS$) and AM ($\Delta=-0.0158$; $P=NS$).

Conclusions: Total arterial compliance assessed with radial GTF-derived central pressure waveforms provide acceptable estimates, compared to those obtained with carotid pressure waveforms. The area method provides consistent results between the 2 sites, without significant systematic bias.

Endogenous Hydrogen Sulfide Mediated Cutaneous Vasodilation is Attenuated in Essential Hypertensive Humans

Lacy M. Alexander¹, Jessica L. Kutz¹, Lakshmi Santhanam², W. Larry Kenney¹, and Jody L. Greaney¹

¹The Pennsylvania State University, Department of Kinesiology, University Park, PA, ²Johns Hopkins University School of Medicine, Departments of Anesthesiology and Critical Care Medicine and Biomedical Engineering, Baltimore, MD

Hydrogen sulfide (H₂S) is an endothelium-dependent hyperpolarizing factor (EDHF) implicated in the pathogenesis of hypertension-induced vascular dysfunction. H₂S is synthesized enzymatically through cystathione-γ-lyase (CSE) and 3-mercaptopyruvate transferase (MPST) in the cutaneous vasculature and induces vasodilation directly and through nitric oxide synthase (NOS)-dependent mechanisms.

Objective: Our aim was to determine the role of endogenously produced H₂S in the cutaneous microcirculation of essential hypertensive humans. We hypothesized that *in vivo* H₂S-mediated vasodilation would be attenuated and *in vitro* H₂S enzymatic activity would be reduced in Stage I hypertensive adults.

Methods: Seven Stage I unmedicated hypertensive (HTN: 24-hour ambulatory systolic 144±5mmHg, diastolic 86±3mmHg) and 7 normotensive (NTN: 110±5mmHg, 72±2mmHg) men and women were instrumented with intradermal microdialysis fibers serving as (1) control (Ringers), and (2) NOS-inhibited (L-NAME), (3) enzymatic H₂S-inhibited (aminooxyacetic acid; AOAA), and (4) dual enzymatic H₂S+NOS inhibition during dose-response perfusion of acetylcholine (Ach: 0.001, 0.01, 0.1, and 1.0mM). Red blood cell flux (laser-Doppler flowmetry) was measured and cutaneous vascular conductance was calculated (%CVC_{max}). Skin biopsy samples were obtained and H₂S producing enzymatic activity was measured (amperometric assay).

Results: Ach-induced vasodilation was attenuated in HTN compared to NTN adults (p<0.001). Enzymatic H₂S-inhibition blunted the cutaneous vasodilatory response to Ach in NTN (80±6 vs. control: 90±5 %CVC_{max}; p=0.002) adults but had no effect in HTN adults (70±6 vs. control: 70±8). Similarly, NOS-inhibition reduced the response to Ach in NTN adults (50±8 %CVC_{max}, p<0.001 vs. control), but had no effect in HTN adults (59±4%CVC_{max}). Dual inhibition did not further reduce Ach-induced vasodilation compared to NOS-inhibition alone in either group. CSE and MPST were expressed in all skin samples and enzymatic activity was reduced in HTN samples (45±1 vs 35±4 H₂S[nM]/h; p<0.05).

Conclusions: Endogenous H₂S-mediated vasodilation is functionally absent and H₂S producing enzymatic activity is reduced in the cutaneous microcirculation of essential hypertensive humans.

Independent and Combined Effects of Aerobic and Resistance Training on Sarcopenic Indices and Its Associations with Peripheral and Central Blood Pressure

Duck-chul Lee, Elizabeth C. Schroeder

Iowa State University, Ames, IA

Objectives: To investigate the effects of aerobic and/or resistance training on sarcopenic indices and associations between changes in appendicular lean mass (ALM) and blood pressure (BP) and heart rate (HR).

Methods: This 8-week randomized controlled exercise intervention includes 69 adults aged ≥45 years (mean age 58) with pre/stage1 hypertension, overweight/obese, and sedentary individuals. Participants were randomly assigned in aerobic exercise, resistance exercise, a combination of both, and control group. Exercise participants exercised 3 days/week for 60 minutes/session. Sarcopenic indices include ALM, appendicular lean mass index (ALMI), total lean mass (TLM), and total lean mass index (TLMI) using multifrequency bioelectrical impedance (InBody 720). ALM was derived as the sum of the lean mass of the four limbs, and TLM as total lean mass. ALM and TLM were then normalized by dividing by body mass index (kg/m²) to yield ALMI and TLMI. BP and HR were measured using the SphygmoCor device.

Results: ALM and TLM appeared to increase in all exercise groups, but decrease in control group. ALMI significantly increased in aerobic exercise group (p=0.03), and this increase was significantly different from the control group (p=0.03) after adjusting for age, sex, and baseline ALMI using linear mixed-effects model. We found a similar results in TLMI. In the analyses on the associations between changes in ALM and BP or HR, participants who increased ALM were more likely to decrease peripheral systolic, central systolic and diastolic BP, and HR. In multivariable linear regression, although not significant (p>0.05), participants who increased ALM appeared to decrease 1.5, 1.2, and 1.2 mmHg and 1.9 bpm in peripheral systolic, central systolic and diastolic BP, and HR, respectively, after adjusting for age, sex, baseline value of BP or HR, and weight change.

Conclusion: Aerobic exercise prevents sarcopenia, and increasing ALM appeared to reduce BP and HR.

Table 1. Baseline, Follow-up, and Changes in Sarcopenic Indices by Exercise Training Type

	No. of Participants	Mean (SE)		Mean (95% Confidence Interval)		Pair-Wise P Value
		Baseline Values	Follow-up Values	Within-Group Changes	Between-Group Comparison vs Control Group Changes	
Appendicular Lean Mass (kg)						
Aerobic	17	23.90 (0.19)	24.11 (0.19)	0.20 (-0.32, 0.72)	0.39 (-0.35, 1.12)	0.30
Resistance	17	23.91 (0.19)	23.90 (0.19)	0 (-0.52, 0.52)	0.18 (-0.55, 0.92)	0.62
Combination	18	23.90 (0.18)	24.18 (0.18)	0.28 (-0.23, 0.78)	0.46 (-0.26, 1.18)	0.21
Control	17	23.90 (0.18)	23.71 (0.18)	-0.18 (-0.70, 0.33)		
Appendicular Lean Mass/BMI*						
Aerobic	17	0.742 (0.005)	0.760 (0.005)	0.017 (0.002, 0.032)	0.024 (0.002, 0.045)	0.03
Resistance	17	0.742 (0.005)	0.742 (0.005)	0 (-0.016, 0.015)	0.006 (-0.016, 0.028)	0.58
Combination	18	0.742 (0.005)	0.745 (0.005)	0.003 (-0.012, 0.018)	0.009 (-0.012, 0.031)	0.39
Control	17	0.742 (0.005)	0.735 (0.005)	-0.006 (-0.022, 0.009)		
Total Lean Mass (kg)						
Aerobic	17	50.60 (0.33)	50.94 (0.33)	0.34 (-0.64, 1.33)	0.49 (-0.90, 1.88)	0.49
Resistance	17	50.29 (0.32)	50.51 (0.32)	0.22 (-0.76, 1.20)	0.37 (-1.02, 1.75)	0.60
Combination	18	49.95 (0.31)	50.53 (0.31)	0.58 (-0.38, 1.53)	0.72 (-0.65, 2.09)	0.30
Control	17	50.20 (0.32)	50.06 (0.32)	-0.14 (-1.13, 0.84)		
Total Lean Mass Index/BMI*						
Aerobic	17	1.560 (0.010)	1.593 (0.010)	0.033 (0.005, 0.061)	0.039 (-0.001, 0.079)	0.06
Resistance	17	1.560 (0.010)	1.566 (0.010)	0.006 (-0.022, 0.034)	0.012 (-0.028, 0.052)	0.55
Combination	18	1.559 (0.010)	1.565 (0.010)	0.005 (-0.022, 0.033)	0.011 (-0.028, 0.050)	0.57
Control	17	1.558 (0.010)	1.552 (0.010)	-0.006 (-0.034, 0.022)		

Values are expressed as fitted mean and all are adjusted for age, sex, and baseline value

*Appendicular/Total Lean Mass Index based on the US Foundation for the National Institutes of health (FNIH) Sarcopenia Project definition

Table 2. Changes in Blood Pressure and Heart Rate by Appendicular Lean Mass Increase

	Peripheral Blood Pressure (mmHg)		Central Blood Pressure (mmHg)		Heart Rate (bpm)
	SBP	DBP	SBP	DBP	
Linear Regression*					
β coefficient (SE)	-1.46 (2.31)	0.58 (1.99)	-1.16 (2.04)	-1.16 (1.59)	-1.92 (1.83)
p-value	0.53	0.77	0.57	0.47	0.30

Values are means (SE).

*Adjusted for age, sex, baseline value, and weight change

The Influence of Body Composition on Arterial Stiffness And Cardiorespiratory Fitness in Young Adults

Adam J. Berrones, Stephanie M. Moore, Bradley S. Fleenor

University of Kentucky, Department of Kinesiology & Health Promotion, Lexington, KY

Objectives: Carotid-femoral pulse wave velocity (cfPWV) is associated with increased cardiovascular disease risk, which is due, in part, to reduced cardiorespiratory fitness (CRF). The influence of body composition on the relationship between cfPWV and CRF has been equivocal. Thus, identifying the influence of body composition on CRF in young adults for future prevention is clinically relevant. We hypothesized the inverse relation between cfPWV and CRF is due to greater fat mass (FM) and fat free mass (FFM) in young adult males.

Methods: Forty-three apparently healthy, young adult males (22.19 ± 0.49 yrs) were recruited. A resting measure of aortic stiffness (carotid-femoral pulse wave velocity, cfPWV) was acquired, and body composition was assessed with bioelectrical impedance analysis using a two-compartment model (FM and FFM). Subjects completed a maximal treadmill test to assess peak oxygen uptake (VO_{2peak}) as a measure of CRF.

Results: Relative VO_{2peak} (ml/kg/min), which accounts for overall body mass, was inversely associated with cfPWV ($P < 0.001$). However, absolute VO_{2peak} (L/min) independent of body mass was not related to cfPWV ($P = 0.34$). Subjects divided into tertiles based on absolute VO_{2peak} demonstrated no differences in cfPWV ($P = 0.47$). However, an analysis of covariance (ANCOVA) revealed a significant main effect for total body fatness ($P < 0.001$). Bivariate analyses with total body mass, total fat mass, total fat free mass, and percent fat mass were all positively associated with cfPWV ($P < 0.05$, all). Percent fat free mass, however, was inversely related to cfPWV ($P < 0.01$).

Conclusions: CRF is not associated with aortic stiffness in apparently healthy, young adult males after controlling for total body mass. An increase in either FM or FFM was associated with greater aortic stiffness, suggesting that total body mass – independent of its composition – modulates the aortic stiffness and CRF relationship. Thus, reducing body mass early in life may prevent aortic stiffness and early vascular aging.

Figure 1.

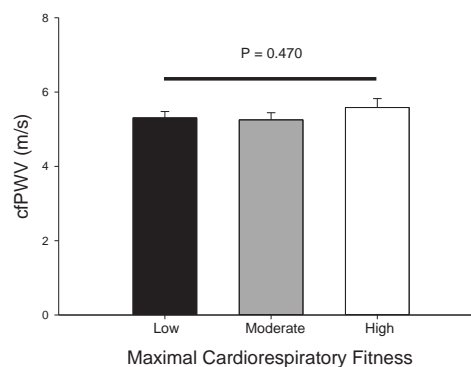
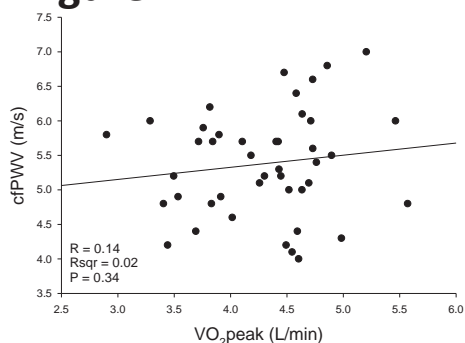
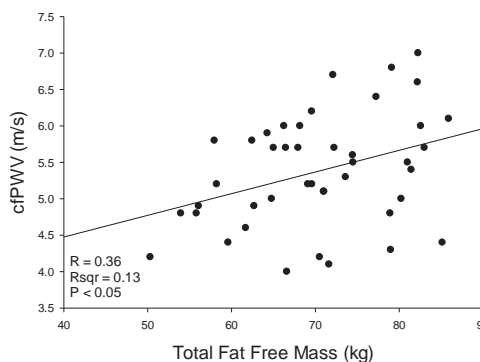
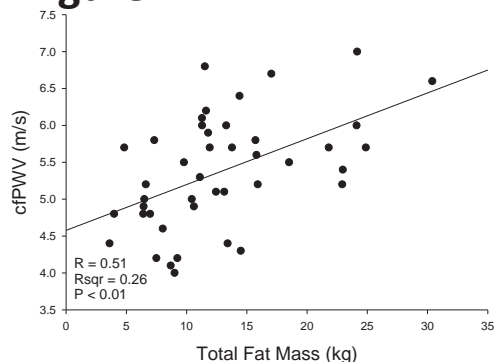


Figure 2.



Relationship between Arterial Stiffness and Functional Capacity in Kidney Transplant Patients

Alexandra Kastelz^{ab}, Ivo G. Tzvetanov^a, Rebecca Kappus^b, Garrett Griffith^b, Alexander Rosenberg^b, Bo Fernhall^b, Lorenzo Gallon^c, Aneesha Shetty^c, Enrico Benedetti^a

^a Department of Surgery, College of Medicine, University of Illinois University at Chicago, Chicago, IL, USA. ^b Integrative Physiology Laboratory, College of Applied Health Sciences, University of Illinois at Chicago, IL, USA. ^c Department of Nephrology, Northwestern Medicine, Chicago, IL, USA.

Introduction: While survival rates are much greater in kidney transplant (KT) than in dialysis patients, significant issues remain. Elevated pulse pressure (PP), carotid beta stiffness, and central pulse wave velocity (cPWV) have all been shown to be associated with cardiovascular disease, which is the main cause of death in kidney transplant patients. **Purpose:** To evaluate the relationship between physical activity levels, functional-capacity and arterial health in KT patients. **Methods:** Thirty-six persons with KT (means ± SE: 44 ± 2.23 yrs , 97.51 ± 5.096 kg, 18 males) that were 2-18 months post-transplant had measures of arterial stiffness performed (PP, carotid beta stiffness, and cPWV) in addition to functional-capacity via 6 minute walk test (6MWT) and a 1-week free living physical activity assessment via accelerometry. Results were analyzed using bivariate correlations. Statistical significance was set at p< 0.05. **Results:** Both aortic and brachial PP as well as carotid beta stiffness were negatively correlated with 6MWT distance (r= -0.353, P=0.037; r= -0.341, P= 0.042; r= -0.336, P=0.045) however central cPWV was not (r= 0.10, P=0.962). 6MWT distance was negatively correlated with percent time spent in sedentary behavior (r = -0.398, P=0.044) and positively correlated with percent time spent in light physical-activity (r= 0.424, P=0.031). Percent time in sedentary, light, and moderate activity were not correlated with PP, carotid beta stiffness, or cPWV. **Conclusions:** These findings suggest lower functional-capacity is associated with higher aortic and brachial PP and carotid beta stiffness in persons with KT. The lack of significance between cPWV and 6MWT distance may suggest a lack in correlation between functional capacity and central aortic stiffness or the possibility KT somehow alters cPWV. Further research is required to investigate this. Additionally, these findings suggest greater daily physical-activity may improve functional-capacity but may not affect arterial stiffness in these patients.

Supported by Gift of Hope and the University of Illinois at Chicago Surgery Department

	Aortic PP	Beta Stiffness	Central PWV
Percent Sedentary	r= 0.082, P= 0.691	r= -0.005, P= 0.980	r= 0.140, P= 0.555
Percent Light	r=-0.083, P=0.687	r= -0.184, P=0.369	r= 0.074, P=0.757
Percent Moderate	r= -0.223, P= 0.274	r=-0.135, P=0.511	r= -0.017; P=0.944

r=Pearson Correlation

Effects of Hyperphosphatemia on Cerebral Small Vessel Diseases in Chronic Kidney Disease

Chih-Ping Chung, MD, PhD^{1, 2}; Chin-Sern Yong, MA^{1, 2}; Kenneth Lim, MD, PhD⁴; Po-Tsang Lee, MD, PhD^{2, 3}; Tzongshi Lu, PhD⁵

¹Department of Neurology, Taipei Veterans General Hospital, Taipei, Taiwan; ²School of Medicine, National Yang Ming University, Taipei, Taiwan; ³Renal Division, Department of Internal Medicine, Kaohsiung Veterans General Hospital, Kaohsiung, Taiwan; ⁴Division of Nephrology, Massachusetts General Hospital, Harvard Medical School, Boston, MA; ⁵Renal Division, Brigham and Women's Hospital, Harvard Medical School, Boston, MA

Objectives: Chronic kidney disease (CKD) has been recently identified as a significant risk factor for stroke as well as for subclinical vascular diseases such as cerebral small-vessel diseases (CSVD). Endothelial dysfunction is a major contributor to CSVD in CKD. Interestingly, both kidney and cerebral microvasculature share similar anatomical and physiologic characteristics. The goal of this study was to elucidate the effects of hyperphosphatemia in cerebral microvasculature using human brain microvascular endothelial cells (HBMECs) and CKD mice as our models.

Methods:

CKD mice model was generated by 5/6 nephrectomy on 8-week-old C57BL/6 mice. Mice were sacrificed at four months after 5/6 nephrectomy. β -glycerolphosphate disodium (2mM, 5mM) was used in HBMECs for 72 hours, in vitro.

Results:

Our results showed that CKD mice had decreased myelinated nerve fibers in the corpus callosum and cerebral cortex and loosening myelinated fibers in the corpus callosum. We found collagen IV accumulation in brain microvasculature, a pattern associated with aging. Serum phosphate levels were significant increased in CKD mice compared to control mice. In HBMECs, β -glycerolphosphate disodium decreased cell viability and increased caspase-3 mediated apoptosis. In addition, Collagen IV expression increased in a dose-dependent manner at 72 hours after β -glycerolphosphate disodium treated HBMECs.

Conclusion:

Our data show for the first time that cerebral white matter and microvascular dysfunction occurs in uremic environments in CKD, in vitro and in vivo. Furthermore, CSVD can be driven in part, by CKD induced hyperphosphatemia. Our data provides a potential mechanism for the development of CSVD in CKD patients.

CAVI measurements in a North American Normal Population

George Maliha, Raymond R. Townsend MD

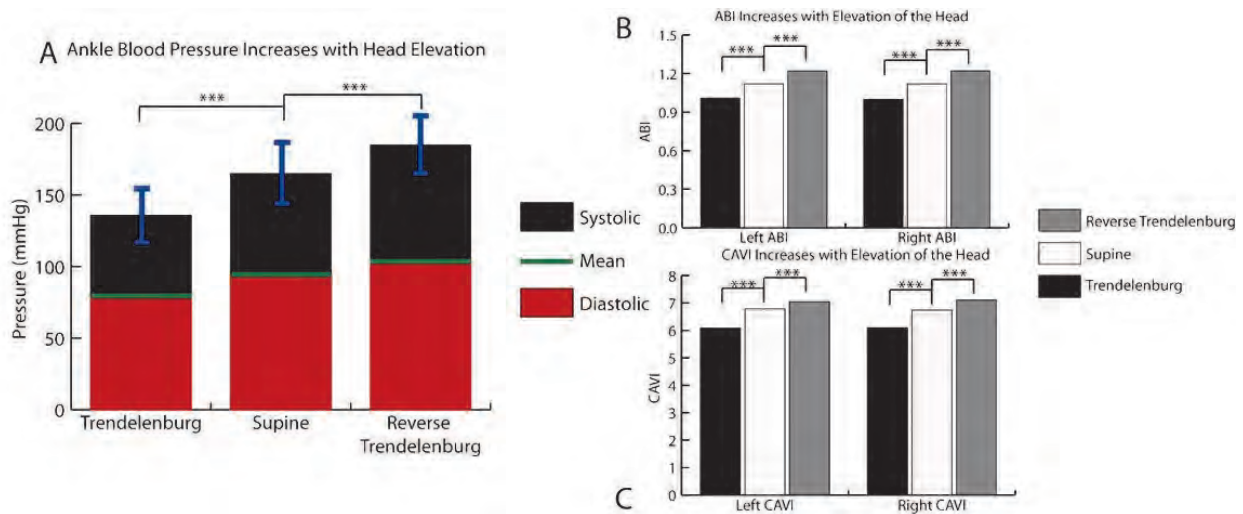
Perelman School of Medicine, University of Pennsylvania, Philadelphia PA 19104 USA

Introduction: The Cardio-Ankle Vascular Index (CAVI) represents a promising index of arterial stiffness, however, the measure or its device, the VaSera, have not been extensively tested in North American clinical settings, nor is much known about the pressure independency of the measure and the accuracy of the ankle-brachial index measure when validated by Doppler in North Americans.

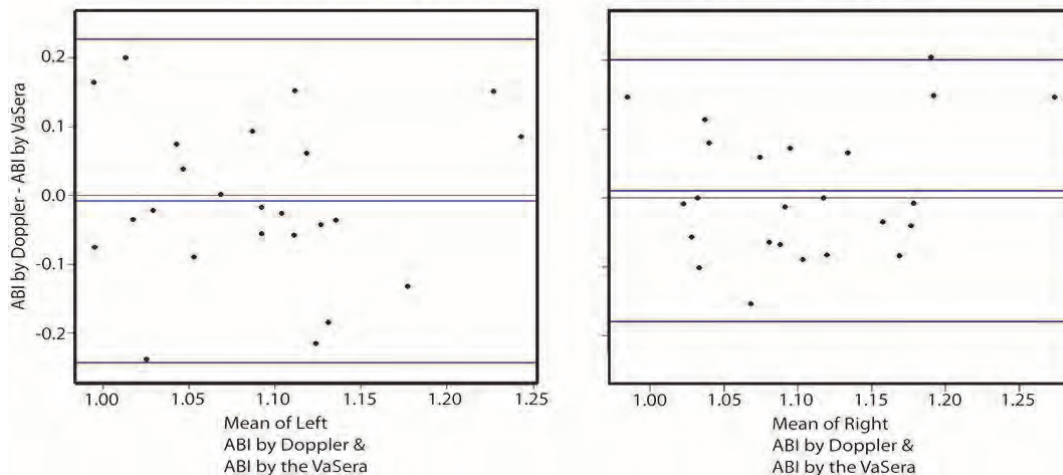
Methods: To provide normal baselines, we recruited 20 male and 28 female volunteers free of known cardiovascular or renal disease and no history of smoking. Their CAVIs, Ankle-Brachial Indices (ABIs), and 4-limb blood pressures were measured in 3 positions: supine, 7° Trendelenburg, and 7° Reverse Trendelenburg. In addition, the VaSera ABI function was validated using a standard Doppler ABI measurement technique in both legs in a subset of our cohort (n=24).

Results: Subjects were 33±13 years old, with a BMI of 24±3 kg/m². Position was found to significantly affect CAVI, indicating that CAVI is sensitive to positional variations (Upper Figure right side) which are pressure dependent (Upper Figure left side). ABI performed well by Bland-Altman analysis (Lower Figure).

Summary: This study represents a first step in bring the VaSera device and its CAVI measurement into clinical practice in a North American (US) normal cohort.



Bland-Altman Plot Comparing ABI by Doppler vs. VaSera



Differences in Carotid Arterial Characteristics Based On Years since Diagnosis in Relapsing Remitting Multiple Sclerosis Patients

Garett Griffith¹, Rachel E. Klaren², Sang Ouk Wee¹, Rebecca M. Kappus¹, Robert W. Motl², Tracy Baynard¹, Bo Fernhall¹

¹University of Illinois at Chicago, Chicago, IL; ²University of Illinois at Urbana-Champaign, Champaign, IL

INTRODUCTION: Persons with multiple sclerosis (MS) experience a decreased life expectancy and greater burden of cardiovascular disease (CVD) compared to age-matched healthy peers. Carotid artery dysfunction in the form of increased stiffness and reduced compliance are often precursors to the clinical manifestation of CVD. Carotid artery dysfunction is a common effect of aging, and may occur earlier in those with MS as the progression of CVD may be accelerated in this population. **OBJECTIVE:** To study the differences in carotid arterial characteristics in both young and older persons with MS based on length of clinical diagnosis. **METHODS:** Twenty seven persons with MS (Expanded Disability Status Scale of 0 – 4.0) were divided into young (i.e. ≤ 50 years) or older (i.e. > 50 years) cohorts. Additionally, length of diagnosis (Dx) was determined to be short (i.e. ≤ 10 years) or long (i.e. > 10 years). Subjects completed echo tracking ultrasound assessments of the carotid artery (Aloka Hitachi) and results were analyzed using a 2 X 2 analysis of variance (ANOVA). Statistical significance was set at p<0.05. **RESULTS:** Carotid arterial function parameters were significantly better in the young subjects compared to older subjects. In the young group, Beta Stiffness Index and Elastic Modulus were significantly lower, and Arterial Compliance was significantly higher, in the short Dx group compared to the long Dx group. In the older group, no significant differences were observed in the short Dx group compared to the long Dx group. **CONCLUSION:** These data show an expected decline in arterial function as individuals with MS age. The differential stiffness and compliance properties seen in the young group based on length of diagnosis suggests that MS influences arterial health independent of age. This may be a result of the decrease in physical activity seen in chronic disease populations such as MS.

Supported by the National Multiple Sclerosis Society RG 4702A1/2

	Young (n=16)		Older (n=11)	
	Short Dx (n=8)	Long Dx (n=8)	Short Dx (n=3)	Long Dx (n=8)
Age (years) [#]	35 ± 2	40 ± 2	54 ± 2	59 ± 2
Beta Stiffness Index (AU) [#]	4.89 ± .44*	6.14 ± .31	8.23 ± 1.37	8.05 ± .66
Elastic Modulus (kPa) [#]	55.01 ± 5.50*	69.19 ± 3.62	123.17 ± 23.28	99.50 ± 10.30
Arterial Compliance (mm ² /kPa) [#]	1.47 ± .12*	1.16 ± .08	1.04 ± .35	.96 ± .13

All data are presented as Mean ± SEM. *Significant difference between short and long Dx groups.

[#]Significant difference between young and older groups.

Influence of Body Position and Venous Pooling On Aortic Blood Pressure and Wave Reflection in Young Adults

William E. Hughes, Darren P. Casey

University of Iowa, Iowa City, IA, USA

Objective: Indices of wave reflection (Augmentation Index, Alx; Augmentation Index at 75 bpm, Alx@75) decrease during a passive head up tilt (HUT). We aimed to examine whether decreases in wave reflection during HUT were a result of venous pooling in the lower limbs, or change in body position (supine vs. 60° tilt).

Methods: 23 healthy, normotensive, young adults (12M/11F; 23±1yr) participated in 3 randomized orthostatic challenge conditions; 60° head up tilt (HUT), 60° HUT with rhythmic blood pressure cuff inflation on calves (75 mmHg) to minimize venous pooling, and lower body negative pressure (LBNP; -30 mmHg) to mimic HUT condition without change in body position. High fidelity radial artery pressure waveforms using applanation tonometry were taken at baseline (rest) and at 2:30 and 5:00 min during each challenge. Aortic blood pressure and wave reflection were analyzed from a synthesized aortic blood pressure waveform (SphygmoCor, AtCor Medical).

Results: Table 1 shows peripheral and central hemodynamics and pressures during each condition. Alx decreased during LBNP compared to baseline (P<0.01) and HUT conditions (P<0.05; Figure 1). Likewise, Alx@75 decreased across time points during LBNP (P<0.01); whereas, it increased slightly during HUT conditions (with and without cuff inflations) (P<0.05). Aside from diastolic pressures, there was no significant condition x time interactions for any of the other peripheral or central pressures.

Conclusion: In contrast to previous research, Alx did not decrease during HUT conditions; however, when standardized for heart rate, HUT (with and without cuff inflations) elicited slight increases in aortic wave reflection. Conversely, LBNP elicited large reductions in indices of aortic wave reflection. These data suggest that the change in aortic wave reflection in response to an orthostatic challenge is dependent on the specific test and possibly body position.

Table 1: Peripheral and Central Pressure and Hemodynamics

	Tilt			Tilt with Cuffs			LBNP			Condition	Time	Interaction
	BL	2:30	5:00	BL	2:30	5:00	BL	2:30	5:00			
HR (bpm)	58±2	74±2	74±3	58±2	71±2	68±3	59±2	71±3	72±3	0.47	<0.01	0.47
Alx@75 (%)	-8±2	-4±3*	-1±2*	-9±2	-7±2	-3±2*	-8±2	-18±2**†	-14±2**†	<0.01	<0.01	<0.01
BSP (mmHg)	116±2	107±2	106±2	115±2	108±2	106±2	115±2	111±2	109±2	0.28	<0.01	0.17
BDP (mmHg)	72±1	66±2*	66±2*	71±1	66±1*	66±2*	70±2	69±2	68±2	0.38	<0.01	0.01
MAP (mmHg)	85±1	79±2	79±2	84±1	79±1	78±1	83±2	80±1	79±2	0.59	<0.01	0.10
BPP (mmHg)	44±2	41±2	40±2	44±1	41±2	40±2	45±2	41±2	40±2	0.80	<0.01	0.80
ASP (mmHg)	99±2	92±2	91±2	99±2	92±1	91±2	98±2	94±2	92±2	0.59	<0.01	0.10
ADP (mmHg)	72±1	67±1*	67±2*	72±1	67±1*	67±2*	70±2	71±2†	70±2	0.27	<0.01	0.01
APP (mmHg)	27±1	24±1	24±1	27±1	24±1	24±1	28±1	23±1	23±1	0.73	<0.01	0.19
AP (mmHg)	0±1	-1±1	0±1	0±1	-1±1	0±1	0±1	-4±1**†	-3±1**†	<0.01	<0.01	0.01

BL, Baseline; HR, Heart Rate; Alx@75, Heart Rate Standardized Augmentation Index; BSP, Brachial Systolic Blood Pressure; BDP, Brachial Diastolic Blood Pressure; MAP, Mean Arterial Pressure; BPP, Brachial Pulse Pressure; ASP, Aortic Systolic Blood Pressure; ADP, Aortic Diastolic Blood Pressure; APP, Aortic Pulse Pressure; AP, Augmented Pressure; * P < 0.01 vs. Baseline; † P < 0.01 vs. HUT conditions

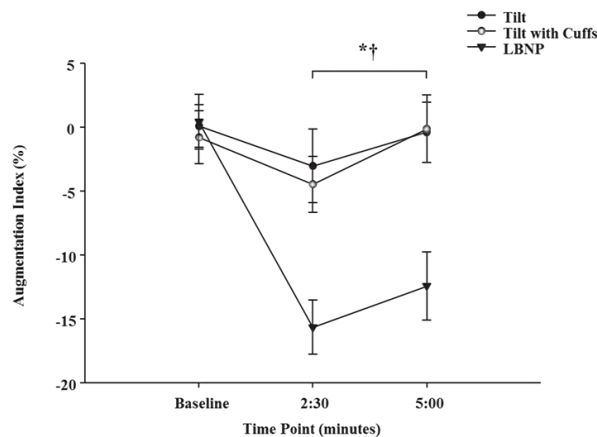


Figure 1: Augmentation Index during orthostatic challenges * P < 0.05 LBNP vs. Baseline; † P < 0.05 LBNP vs. HUT conditions

Comparison of the Flow-Mediated Dilation Response between the Popliteal and Fibular Arteries

D. Ramsay¹, J. Gilby², and D.S. Kimmerly¹

¹ *Dalhousie University, School of Health And Human Performance, Division of Kinesiology*

² *Dalhousie University, School of Health Sciences, Division of Diagnostic Medical Ultrasound*

Objectives: Vascular health is controlled by competing neural sympathetic vasoconstrictor and endothelial-derived vasodilator mechanisms. However, the relationship between these mechanisms is poorly understood. In humans, muscle sympathetic nerve activity (MSNA) is commonly measured in the fibular nerve to assess vasoconstrictor activity, while flow-mediated dilation (FMD) measures of lower-limb endothelial dilatory function are routinely measured in the popliteal artery (PA). Ideally, FMD measures would come from the fibular artery (FA). However, fibular FMD responses are rarely reported and whether it responds similarly to the PA is unknown. Past literature has shown upper limb arteries with a larger baseline diameter exhibit smaller FMD responses, although this relationship isn't as strong in lower limb arteries (1). The aim of this study was to compare PA and FA FMD and corresponding changes in shear rate.

Methods: Ultrasound-derived diameters and Doppler flow velocities of the PA and FA were measured in 13 healthy participants (5F/8M, 22±2 years) at rest and after 5-minutes of distal cuff occlusion. Data were analyzed using automated edge-detection software. FMD was expressed as percent increase from rest (%FMD) and normalized to shear rate (%FMDnorm).

Results: Resting and peak post-occlusion diameters were higher in the PA than FA (4.95mm±0.59mm vs. 2.54mm±0.56mm, P<0.001 and 5.38mm±0.71mm vs. 2.82mm±0.63mm, P<0.001, respectively). However, %FMD and %FMDnorm were higher in the FA than PA (8.56±2.19 vs. 11.22±2.13, P=0.001 and 8.71±2.42 vs. 11.09±2.80, P=0.01, respectively). Total shear rate area under the curve was not significantly different between the PA and FA (P=0.48).

Conclusions: These results correspond with previous literature, indicating that arteries with smaller baseline diameters have greater FMD responses (1). There were no differences in shear rates despite different FMD responses. Our findings have implications for future research examining the effect of age and aerobic fitness on concurrent measures of FMD and resting MSNA.

*Supported by a Canadian Foundation for Innovation: Leader's Opportunity Fund and a Faculty of Health Professions Research Development grant.

References:

1. **Thijssen DH, et al.** Heterogeneity in conduit artery function in humans: impact of arterial size. *Am J Physiol Heart Circ Physiol.* 295: 5: H1927-34, 2008.

Independent Modifications to Backward and Forward Pressure Waves Lead to Non-Physiological Aortic Flow

Timothy S. Phan^a, Julio A. Chirinos^b, John K-J. Li^a

^a Rutgers University, Piscataway, NJ, USA

^b University of Pennsylvania, Philadelphia, PA, USA

Background: Arterial wave reflections are important contributors to LV afterload. So as to demonstrate the role of increased forward wave amplitude and morphology in late systolic pressure augmentation, a recent study [1] modified backward waves independently of forward waves to form simulated aortic pressure waveforms, without regard for the underlying flow necessary to meet those altered conditions.

Methods: Simultaneous aortic pressure and flow waveforms were measured in an anesthetized open-chest dog under i.v. infusion of methoxamine. Pressure and flow waves were decomposed into backward and forward waves using standard methods. As done in the recent study, the backward waves were shifted in time to simulate earlier arrival of reflected waves. The forward and backward waves were then recombined to generate simulated aortic pressure waveforms. Unlike the recent study, we also recombined forward and backward flow waves to assess the flow that is necessary for the proposed backward wave modification.

Results: Arbitrarily modifying backward waves independently of forward waves resulted in markedly non-physiologic flow waveforms with positive flow in diastole (Figure, panel D, dashed line), despite simulated pressure waveforms that appear physiological (Figure, panel B, dashed line).

Conclusions: Modification of the backward pressure wave requires the same modification of the backward flow wave in order to satisfy the hemodynamic definition of aortic characteristic impedance. When backward waves are modified independently of the forward wave, basic hemodynamic principles are violated and non-physiological aortic flows are produced. Previous findings using this method cannot be used to support the importance of the forward wave. Our findings illustrate the key influence of wave reflections on any given combination of forward wave and flow waveforms. Changes in reflections for any given forward wave will necessarily change flow. Similarly, changes in reflections in the presence of any given flow wave will necessarily change the forward wave.

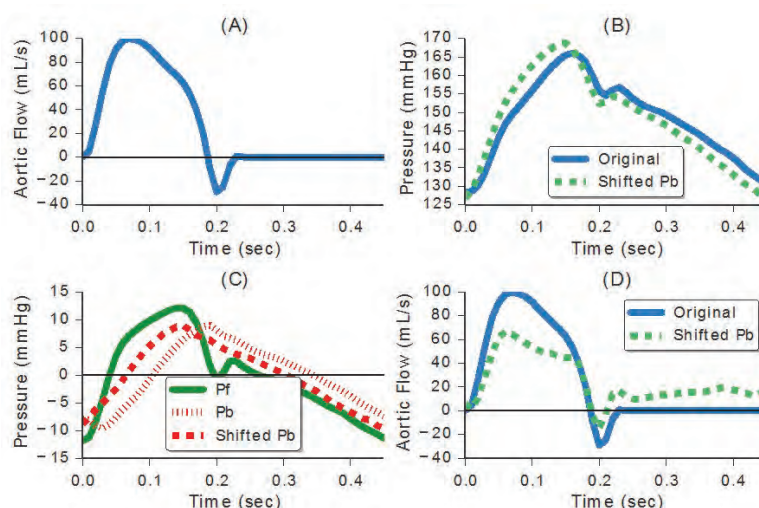


Figure. Simultaneously measured aortic flow (panel A) and pressure (panel B). Pressure waveform decomposed into forward (Pf) and backward (Pb) waves (panel C). Pb is shifted in time to simulate earlier arrival of reflections (panel C, dashed line). Despite a simulated pressure waveform that appears physiological (panel B, dashed line), the corresponding flow waveform is non-physiological (panel D, dashed line).

References

[1] Torjesen, Alyssa A., Na Wang, Martin G. Larson, Naomi M. Hamburg, Joseph A. Vita, Daniel Levy, Emelia J. Benjamin, Ramachandran S. Vasan, and Gary F. Mitchell. "Forward and backward wave morphology and central pressure augmentation in men and women in the Framingham Heart Study." *Hypertension* 64, no. 2 (2014): 259-265.

Reconciling the Increased Pulse Wave Velocity and Reflected Wave Transit Time Paradox

Timothy S. Phan^a, John K-J. Li^a, Swapna Varakantam^b, Vandan Panchal^c, Amer A. Syed^c, Ejaz M. Shah^c, Scott R. Akers^b, Julio A Chirinos^c

^a Rutgers University, Piscataway, NJ, USA

^b Philadelphia VA Medical Center, Philadelphia, PA, USA

^c University of Pennsylvania, Philadelphia, PA, USA

Background: Previous studies suggest that, despite the clear increase in PWV with aging, reflected wave transit time (RWTT) does not change significantly. This has led to the proposition of a “distal shift” of reflecting sites with aging. This conclusion is critically dependent on the methods to assess RWTT. We aimed to assess how different RWTT measures vary with age and show that increased PWV with age is indeed associated with decreased RWTT when a proper RWTT method is used.

Methods: We studied 200 subjects (mean age 62 years; range=26 to 93). Carotid tonometry and phase-contrast MRI were used to assess central pressure and flow, respectively. We assessed RWTT based on: (1) wave separation analysis (RWTT_{WSA}); (2) inflection point of the pressure waveform (RWTT_{INF}); (3) partially distributed tube-load arterial system modeling (RWTT_{TUBE}). Tube-load based RWTT takes into account information contained in entire pressure and flow waveforms, rather than arbitrarily chosen regions of interest and empirical methods to impose a ‘foot’ to waves.

Results: Consistent with previous reports, RWTT_{INF} (B=-0.0488 msec/year; P=0.65) and RWTT_{WSA} (B=-0.217 msec/year; P=0.075) demonstrated relatively “flat” relationships with age. RWTT_{TUBE} demonstrated a much greater reduction in RWTT (B=-0.968 msec/year; P<0.001), corresponding to changes with aging ~20 times greater than by the timing of the inflection point (and ~4.5 times greater than those assessed via RWTT_{WSA}).

Conclusion: Tube-load modeling suggests that RWTT declines much more rapidly with age, consistent with earlier arrival of reflections to the aorta as age and PWV increases in tandem. The apparent asymptotic RWTT from standard methods misleads to the identification of a reflection-free period in early systole, at odds with the assumption of steady-state oscillation. Tube-load modeling resolves the paradox and leads to logical trends of RWTT that is consistent with increased PWV and reflections with age.

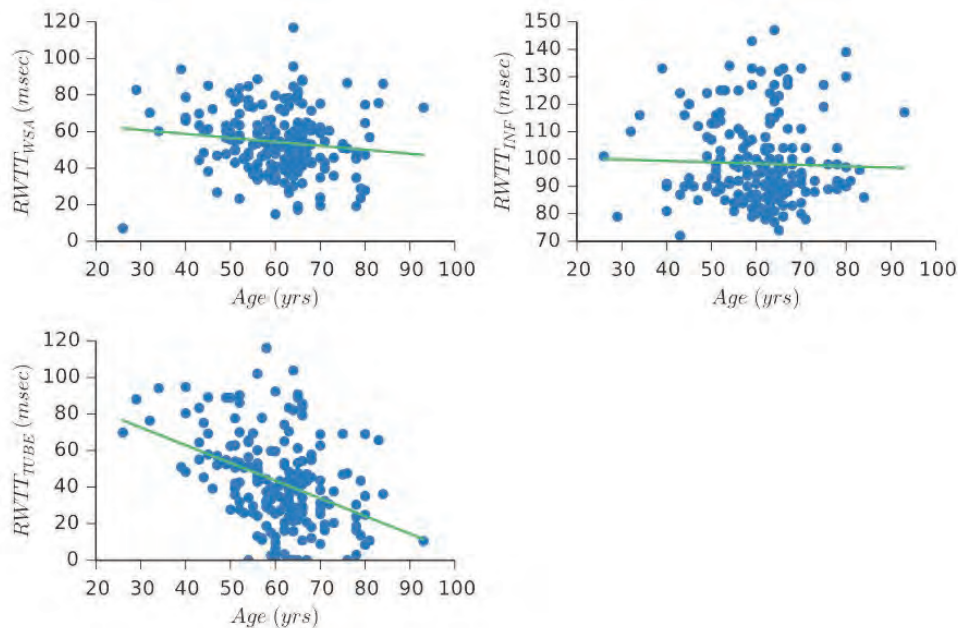


Figure. RWTT relationship with age via wave separation analysis method (top left), pressure waveform inflection point method (top right), and tube-load modeling method (bottom left).

Evaluating the Logical Relationships of Reflected Wave Transit Time with the Complex Global Reflection Coefficient, Height, and Pulse Wave Velocity

Timothy S. Phan^a, *John K-J. Li*^a, *Izzah Vasim*^b, *Maheshwara R. Koppula*^c, *Swapna Varakantam*^b, *Vandan Panchal*^c, *Amer A. Syed*^c, *Ejaz M. Shah*^c, *Scott R. Akers*^b, *Julio A. Chirinos*^c

^a Rutgers University, Piscataway, NJ, USA

^b Philadelphia VA Medical Center, Philadelphia, PA, USA

^c University of Pennsylvania, Philadelphia, PA, USA

Background: Reflected wave transit time (RWTT) represents the time at which arterial wave reflections begin to exert significant effects on aortic pressure and flow. Different methods exist to compute RWTT, each yielding conflicting interpretations regarding underlying cardiovascular changes. RWTT should sensibly correlate with body height and pulse wave velocity (PWV). Furthermore, RWTT should correlate strongly with the phase of the complex global reflection coefficient ($\Gamma_{\phi,1}$), as this variable describes the time lag between backward and forward waves in the frequency-domain. We evaluated the relationship between RWTT measured with 3 methods and the following 3 variables: height, PWV and $\Gamma_{\phi,1}$, to determine the method that is most logically consistent.

Methods: Central pressure and flow waveforms were measured noninvasively using carotid tonometry and PC-MRI, respectively, in 201 subjects (mean age = 61 years; range 26 to 93), along with carotid-femoral PWV (cfPWV). RWTT was computed using (1) wave separation analysis (RWTT_{WSA}); (2) inflection point on pressure waveform (RWTT_{INF}); (3) tube-load modeling (RWTT_{TUBE}).

Results: RWTT_{WSA} did not show significant relationships with either height (standardized $\beta=0.14$; $P=0.08$) or CF-PWV (standardized $\beta=-0.006$; $P=0.94$). RWTT_{INF} was significantly related only to cfPWV ($\beta=-0.15$; $P=0.01$). In contrast, RWTT_{TUBE} was strongly related to $\Gamma_{\phi,1}$ ($\beta=-0.724$; $P<0.001$) and was significantly related to body height ($\beta=0.29$; $P<0.0001$), and cfPWV ($\beta=-0.28$; $P<0.001$).

Conclusions: RWTT_{WSA} and RWTT_{INF} demonstrated inconsistent relationships with $\Gamma_{\phi,1}$, despite their presumed relation to backward wave timing. Only RWTT_{TUBE} demonstrated the logically expected relationships with $\Gamma_{\phi,1}$, height, and PWV. Closer inspection of the putative methods (RWTT_{WSA}, RWTT_{INF}) reveals that both equate the time around peak flow to reflection timing. This imposes an artificial restriction on RWTT. RWTT_{TUBE} does not have such artificial constraints, uses information contained in entire pressure and flow waveforms, and produce values that have consistent and logical trends with $\Gamma_{\phi,1}$, height, and PWV.

“Impedance Matching” Between The Aorta And Large Muscular Arteries? Misinterpretation of Pulse Wave Velocity Gradients

Timothy S. Phan^a, John K-J. Li^a, Izzah Vasim^b, Zoubair Ahmed^b, Maheshwara R. Koppula^c, Julio A. Chirinos^c

^a Rutgers University, Piscataway, NJ, USA

^b Philadelphia VA Medical Center, Philadelphia, PA, USA

^c University of Pennsylvania, Philadelphia, PA, USA

Background: Given the increase in carotid-femoral Pulse Wave Velocity (cfPWV) with aging, without an increase in carotid-radial PWV (CR-PWV), some investigators have suggested an age-related “impedance matching” between the aorta and large muscular arteries, leading to a reduction of reflections arriving at the proximal aorta and a distal shift in reflection sites. However, characteristic impedance is much more sensitive to diameter than wall stiffness and PWV “matching” should not be equated to “impedance matching”. If PWV “matching” indeed produces a distal shift of reflection sites, it should be related to a greater reflected wave transit time (RWTT) for any given PWV.

Methods: Central pressure and flow waveforms were measured noninvasively using carotid tonometry and phase-contrast MRI, respectively, along with cfPWV and CR-PWV (n=175; mean age 62 years). Reflected wave transit time was calculated using (1) wave separation analysis (RWTT_{WSA}); (2) partially distributed modeling of the arterial system (RWTT_{TUBE}). The “matching” of large artery and muscular artery PWV was calculated as the difference (“gradient”) between CR-PWV and cfPWV.

Results: The PWV gradient was not related to RWTT computed with either WSA or partially distributed model of the arterial system (table). Similarly, this gradient was not related to the reflection coefficient measured at the proximal aorta. Distributed modeling demonstrated that RWTT occurs earlier with aging, in tandem with increases in PWV, therefore not supporting a “distal shift” in reflection sites, but rather supporting the logical presence of earlier wave reflections.

Conclusion: As dictated by well-established theoretical principles, impedance matching that determines reflection sites is defined by gradients of impedances, and not of PWVs. The controversial proposition of “impedance matching” and distal shift of reflection sites with aging is therefore not supported by either theoretical or experimental observations. Equating “PWV matching” to “impedance matching” can result in important misinterpretations of reflection phenomena.

	Predictors of RWTT _{WSA}	Predictors of RWTT _{TUBE}
Age	-0.110 (P=ns)	-0.280 (P=0.008)
Sex	0.018 (P=ns)	0.000 (P=ns)
cfPWV	0.232 (P=ns)	-0.169 (P=ns)
ΔPWV	0.282 (P=ns)	0.146 (P=ns)
Height	0.157 (P=ns)	0.210 (P=0.046)

Table. All values are standardized coefficients. Significance level is set at 0.05.

Transplant Renal Artery Stenosis: A Treatable Cause of Resistant Hypertension and Renal Allograft Dysfunction

¹Ekamol Tantisattamo, MD, FASN; ²Praveen Ratanasrimetha, MD; ³Siwadon Pitukweerakul, MD; ¹Aneesha A. Shetty, MD, MPH; ¹Lorenzo Gallon, MD

¹Division of Nephrology and Hypertension, Department of Medicine, Northwestern University Feinberg School of Medicine, Chicago, Illinois

²Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand 10700

³Department of Internal Medicine, Presence St. Francis Hospital, Evanston, Illinois

Objectives: Atherosclerosis is the most common cause of transplant renal artery stenosis (TRAS). It is unknown whether therapeutic angioplasty and/or renal-artery stenting is superior to medical management for TRAS. With donor renal vascular manipulation or reconstruction and exposure to immunosuppression, the pathogenesis of RAS in renal transplant recipients may differ from non-transplant patients. We aim to identify potential risk factors of TRAS and outcomes after renal artery angioplasty and/or renal-artery stenting.

Methods: The new cases of TRAS from July 2014 to June 2015 in our institutes were reviewed. The renal transplant donors' and recipients' information and outcomes including renal allograft function, blood pressure (BP), and change in the number of blood pressure medications were reviewed. All patients underwent balloon angioplasty with/without stent placement.

Results: A total of 4 newly diagnosed TRAS were identified. Age at the time of diagnosis was 46.5 +/- 5.0 (SEM) years. All patients were African American and three were male. The most common cause leading to work up for TRAS was resistant hypertension. Mean serum creatinine at the time of diagnosis and after intervention were 2.2 +/- 0.3 and 1.9 +/- 0.2 mg/dL (p = 0.4372), respectively. All patients had the stenosis at the ostium of the transplant renal artery. Three donor kidneys were required arterial or venous reconstruction before anastomosed to the external iliac vessels and two of these kidneys were the right kidneys. Only 1 patient had normalized BP and decreased the number of BP medications (Table 1).

Conclusions: As the same to non-transplant patients, angioplasty +/- renal-artery stenting may not significantly improve renal allograft function or BP control in kidney transplant recipients. African American may be the risk factor of TRAS. Larger studies and interventional trials are required to determine the prevalence, risk factors, and therapeutic strategies for TRAS.

Table 1: The patients' and donor kidneys' data and outcomes

AA, African American; BP, blood pressure; DDRT, deceased donor renal transplantation; DGF, delayed graft function; ESRD, end-stage renal disease; F, female; HTN, hypertension; LRRT, living-related renal transplantation; M, male; No, number; PKD, polycystic kidney disease; Post-, post-angioplasty; Pre-, pre-angioplasty; PSV, peak systolic velocity; RA, renal artery; RAS, renal artery stenosis; RI, resistive index; SCR, serum creatinine; SGF, slow graft function; T2DM, type 2 diabetes mellitus; U/S, ultrasound

Pt	Cause of ESRD / Kidney transplant	Recipient co-morbidity / Smoking	Posttransplant complications	Time to diagnose RAS (months posttransplant)	Donor kidney side / RA reconstruction	Doppler renal U/S: PSV (cm/sec) / RI	Angiography: Site of RAS / Intervention	Outcomes					
								SCR		BP		No. of BP medication	
								Pre-	Post-	Pre-	Post-	Pre-	Post-
(1) 51 y/o AA M	HTN / DDRT	Prostate cancer / No	SGF	12	Left / 2 RA with Carrel patch	362-547 / 0.57	Ostium / Balloon + Stent	2.4	1.95	155 / 85	128 / 74	4	3
(2) 55 y/o AA F	T2DM / LRRT	HTN / No	• DGF requiring dialysis • RA thrombosis requiring thrombectomy on POD1	26	Left / None	250 / 0.66	Ostium / Balloon + Stent	1.7	1.75	160 / 65	209 / 92	4	4
(3) 48 y/o AA M	PKD / DDRT	HTN / No	None	3.5	Right / Anastomosis of the lower pole RA end-to-side onto the main RA	341 / 0.69	Ostium / Balloon	1.7	1.5	160 / 90	151 / 83	3	4
(4) 32 y/o AA M	HTN / DDRT	None / Yes	SGF	3	Left / 2 RA (cranial and caudal) in 1 aortic patch	395 / 0.63	The smaller caudal RA / Balloon	2.8	2.45	155 / 90	155 / 85	1	2

Pre-operative Pulse Wave Velocity (PWV) Values and Refractory Systemic Hypotension after Induction of General Anesthesia in Patients Treated with Angiotensin Converting Enzyme Inhibitor: A Pilot Study

Janiczek, D., Casey, DP., Ueda, K.

The University of Iowa Roy J. and Lucille A. Carver College of Medicine, Iowa City, Iowa, USA

Objective: During the induction of anesthesia moderate hemodynamic disturbances are common due to concurrent autonomic nervous system suppression and the stress response from endotracheal intubation. Previous studies have shown that patients chronically taking angiotensin converting enzyme (ACE) inhibitors have a higher incidence of developing hypotension under general anesthesia and are refractory to adrenergic vasoconstrictor medications given to help restore blood pressure. We aimed to investigate whether differences in baseline levels of arterial stiffness, as measured by PWV, contribute to this phenomenon.

Methods: Twenty three patients (69±7 years), chronically taking an ACE-inhibitor and were scheduled for morning surgery under general anesthesia with endotracheal intubation were enrolled. Applanation tonometry (SphygmoCor®) was utilized to measure carotid-femoral PWV (cfPWV) preoperatively. Blood pressure was recorded at 1 minute intervals for 10 minutes after intubation. Three groups were defined by systolic blood pressure (SBP) response; 1) non-hypotension: SBP never dropped greater than 20% from baseline, 2) hypotension: SBP dropped greater than 20% from baseline, however, it was restored with less than 200 mcg phenylephrine, 3) refractory hypotension: SBP dropped greater than 20% from baseline that required more than 200 mcg of phenylephrine to return SBP to baseline.

Results: Of the 23 patients enrolled, 11 (47%) patients developed hypotension within 10 minutes of endotracheal intubation. Among those who developed hypotension, 4 (17%) patients were refractory to phenylephrine (200 mcg bolus). cfPWV values in the non-hypotension, hypotension, and refractory hypotension group were 11.0±1.1, 10.0±0.9 and 8.2±0.9 m/sec, respectively (P=0.2).

Conclusion: Our preliminary results suggest there was a trend for lower pre-operative cfPWV values being associated with the development of hypotension or refractory hypotension during the induction of anesthesia in patients on an ACE inhibitor. These results may help anesthesiologists predict and manage hypotension after induction of general anesthesia and consequently reduce the adverse events from persistent hypotension.

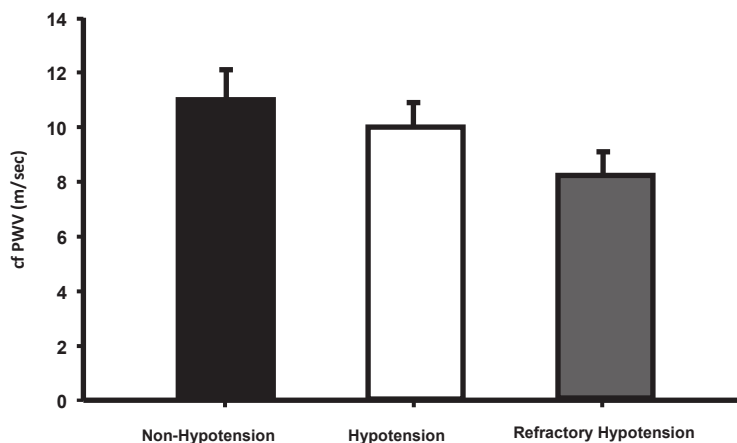


Figure 1: Pre-operative mean cfPWV based on the patient's systolic blood pressure response after induction of general anesthesia.

Blood Pressure is the Strongest Component Associated with Arterial Stiffness in Mexican Patients with Metabolic Syndrome

*Grover-Paez Fernando*¹, *Ramos-Becerra Carlos G*², *Alanis-Sánchez Guillermo A*³, *Ognianov-Iantchoulev Assen*⁴, *Cardona-Müller David*⁵, *Totsuka-Sutto Sylvia E*⁶, *Pascoe-González Sara*⁷, *Cardona-Muñoz Ernesto G*⁸.

^{1,2,3,4,5,6,7,8}, *Institute of Experimental and Clinical Therapeutics, Department of Physiology, University Center for Health Sciences, University of Guadalajara, México.*

The prevalence of Metabolic Syndrome (MetS) in Mexico increased from 26.6% to 45% in 2012. Cardiovascular disease is the main cause of death in Mexico and arterial stiffness (AS) is an early subclinical vascular disease marker.

Objective: The aim of this cross-sectional study was to evaluate the correlation between the components of MetS and AS.

Methods: Sixty-three non-smoking male patients, 40 to 77 years old, were included (22 without MetS and 41 with MetS). Blood pressure and baPWV were measured with the “Vascular Profiler 1000” device (VP-1000) (Omron, Kyoto, Japan). The methodological details have been described previously (1). Mean right and left baPWV values were used for the analysis. Fasting plasma glucose (FPG) was measured using a glucose oxidase technique. Total cholesterol, high-density lipoprotein cholesterol (HDL-c), and triglycerides were assessed by enzymatic methods. All of these measurements were performed using commercially available kits (Beckman Instruments Inc, Brea, Calif.) All values are expressed as an average ± the standard deviation (SD); the association between MetS and AS measurements was analyzed using Pearson’s coefficient. P < 0.05 was accepted as statistically significant.

Results: Patient characteristics are listed in Table 1. Concerning AS parameters, we observed statistical differences in baPWV and peripheral pulse pressure (PP).

Table 1. Anthropometric, metabolic, hemodynamic and arterial stiffness characteristics of patients without MS (n=22) and with (n=41) and

Data	Metabolic Syndrome		p ^a
	No (n=22)	Yes (n= 41)	
Age, years	55.2 ± 7.8	58.1 ± 7.48	.083
BMI, (Kg/m ²)	28.0 ± 3.2	28.7 ± 2.6	.292
Waist, (cm)	101.5 ± 9.1	101.81 ± 7.1	.988
FPG, (mg/dL)	90. ± 12.1	101.4 ± 19.9	.018
Triglycerides, (mg/dL)	118.6 ± 62.9	194.1 ± 31.46	.002
HDL-c, (mg/dL)	57.6 ± 14.9	48.5 ± 14.4	.019
LDL-c, (mg/dL)	98.5 ± 34.7	103.3 ± 42.1	.653
Total cholesterol, (mg/dL)	182.5 ± 26.3	175.1 ± 47.6	.524
Systolic blood pressure , (mmHg)	123.3 ± 7.8	146. 4 ± 18.0	<0.01
Diastolic blood pressure (mmHg)	76.1 ± 7.2	86.4 ± 9.4	<0.01
Peripheral pulse pressure (mmHg)	47.3± 9.4	59.7±15.4	<0.01
Mean baPWV (cm/s)	1347.0± 173.5	1578.7± 295.0	<0.05

^a = Student t test was used to analyze difference of continuous variables between groups. Regarding the association of AS and MetS components (Table 2),

Table 2. Correlation between the different components of Metabolic syndrome and arterial stiffness (n=41).

Data	baPWV ^b	p	PP ^b	p
BMI	-.145	.443	-.283	.073
Waist	-.087	.052	-.314	.052
SBP	.753	<0.01	.853	<0.01
DBP	.603	<0.01	-.005	.976
Triglycerides	.231	.256	-.154	.363
HDL-c	-.236	.246	-.245	.144
FPG	-.065	.758	-.091	.598

^b= Pearson’s r, was used to analyze the correlation between AS variables and MS

among patients with MetS, we observed strong and significant correlations between SBP and baPWV (r=.753, p=<0.001), between DBP and baPWV (r=.603, p=<0.001), and between SBP and PP(r=.853, p=<0.001).

Conclusions: This is the first study conducted in a Mexican patient population that demonstrates that blood pressure is the component of MetS that is most strongly associated with AS.

References: 1.- Yamashina A 2002

Central Blood Pressure, Wave Reflection and Subendocardial Viability Ratio in Women with a History of Hypertensive Pregnancy

Sushant M. Ranadive¹, Ronee E. Harvey¹, Michael J. Joyner¹, Virginia M. Miller^{2,3}, Jill N. Barnes^{1,4}

¹. Department of Anesthesiology, Mayo Clinic, Rochester, MN; ². Department of Surgery, Mayo Clinic, Rochester, MN; ³. Department of Physiology and Biomedical Engineering, Mayo Clinic, Rochester, MN. ⁴. Department of Kinesiology, University of Wisconsin, Madison

Objective: History of hypertensive pregnancy (HTNP) is considered a risk factor for future cardiovascular disease. However, only some women with a history of HTNP become hypertensive later in life. Higher augmentation index (AIx) is associated with increased cardiovascular risk; additionally, lower (below 50%) subendocardial viability ratio (SEVR) represents subendocardial ischemia and serves as an index of myocardial oxygen supply and demand. Hence, the aim of the study was to determine underlying phenotypes that might differentiate these women.

Methods: Pulse wave analysis using applanation tonometry was performed at rest in postmenopausal women (58±1 years) with a history of HTNP. Generalized transfer function was used to determine central aortic pressures and central arterial pressure waveform characteristics.

Results: Aortic systolic pressure, aortic mean arterial pressure and augmentation index did not differ between women with a history of HTNP currently using anti-hypertensive medications (n=18) and non-medicated (n=15) HTNP women groups (p>0.05). However, medicated women compared to non-medicated women had a significantly higher aortic diastolic blood pressure (81±2 versus 74±2 mmHg, respectively; p=0.04) and significantly higher systolic pressure-time integral and lower subendocardial viability ratio (2551 ± 106 and 129 ± 6 versus 2179 ± 84 mmHg*s and 154 ± 6 %, respectively; p=0.01 and p=0.03).

Conclusions: These results identify differences in central blood pressure, systolic pressure-time integral and subendocardial viability ratio, an index of myocardial oxygen supply/demand in women with a history of HTNP that are currently hypertensive versus normotensive. These data suggest the presence of distinct phenotypes in women with a history of HTNP, which may be identified non-invasively using arterial pressure waveforms. Further investigation is needed to evaluate if these changes can be primarily attributed to a history of HTN pregnancy and how this affects overall cardiovascular risk.

Funding: NIA 1P50AG044170-01, CTSA UL1 TR000135, HL 118154, HL83947

Reproducibility of Central Systolic Blood Pressure, Augmentation Index, Measurements Calculated with the Omron HEM-9000AI Device in a Mexican Population of Young Individuals

Ramos Carlos G¹, Grover Fernando², Alanis Guillermo A³, Ognianov Assen⁴, García Catherine A⁵, Cardona David⁶, Totsuka Sylvia E⁷, Cardona Ernesto G.⁸

Objective: To assess the agreement and reproducibility of the non-invasive measurement of the central systolic blood pressure (cSBP), second systolic shoulder (SYS2), and augmentation index (AIx) with the HEM-9000AI.

Methods: This cross-sectional study was conducted in 36 healthy Mexican individuals (20 men and 16 women). The cSBP, SYS2, and AIx were measured using the HEM 9000AI device. All values are expressed as mean ± SD; the correlation was analyzed using Pearson’s correlation coefficient and the Bland Altman method for agreement. All p-values were two-tailed, and p<0.05 was accepted as statistically significant.

Results: The mean age of participants was 20.6 ± 1.6 years, mean BMI was 23.5± 3.5 and mean waist circumference was 82.5 ± 9.3 cm. Good correlation between the first and second measurements was exhibited by cSBP (r² Pearson = 0.680), SYS2 (r² Pearson = 0.680), and AIx (r² Pearson = 0.675) (p<0.001 for all). The Bland–Altman plots of the first and second SYS2 and AIx measurements also demonstrated good agreement (respective mean differences: 2.4±6.07 mmHg and 0.029±8.39%).

Conclusion: The results obtained by the OMRON HEM-9000AI device demonstrate that there are strong and significant correlations between the first and second measurements of cSBP, SYS2, and AIx, respectively.

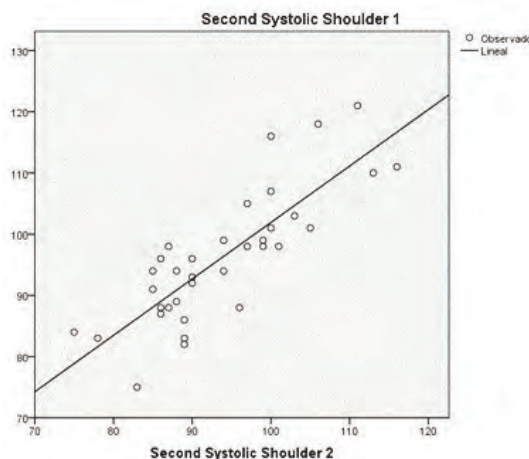


Figure 1. Second systolic shoulder 1, first measurement, vs second systolic shoulder 2, second measurement, Pearson’s r²=0.680, p<0.001

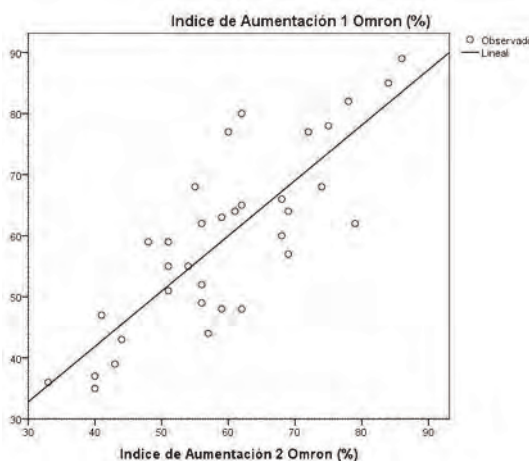


Figure 2. Augmentation Index 1, first measurement, vs Augmentation Index 2, second measurement, Pearson’s r²=0.675, p<0.001

Altered Vessel Hemodynamics at Rest and After Acute Physical Stress in Young Smokers

Alexandra B. Cooke, Department of Medicine, McGill University; Andrew F. Mutter, Department of Medicine, McGill University; Yessica-Haydee Gomez, Department of Medicine, Research Institute of the McGill University Health Centre; Robert J. Doonan, Department of Medicine, McGill University; Simon L. Bacon, Department of Exercise Science, Concordia University; Stella S. Daskalopoulou, Department of Medicine, Research Institute of the McGill University Health Centre

Objectives: Long-standing smokers have stiffer arteries at rest; however the extent of the underlying vascular dysfunction in young healthy smokers has not been fully established. We aimed to examine the acute and chronic effect of smoking and nicotine exposure, on arterial stiffness at rest and in response to acute physical stress in young healthy individuals.

Methods: Young healthy smokers (n=43) and non-smokers (n=80) underwent the 'arterial stress test': blood pressure and arterial stiffness before and after (2, 5, 10, 15, 20 minutes) an exercise test to exhaustion on a treadmill. Several indices were assessed: central and peripheral systolic blood pressure (SBP) and pulse pressure (PP), PP amplification (PPA), augmentation index corrected for a heart rate (HR) of 75 beats/min (AIx75), subendocardial viability ratio (SEVR), as well as carotid-femoral and carotid-radial pulse wave velocity (cfPWV and crPWV). Smokers were assessed under 3 conditions: a) after 12h smoking abstinence (chronic smoking), b) immediately after smoking one cigarette (acute smoking), and c) immediately after chewing nicotine gum.

Results: Smokers at rest had elevated AIx75 ($p<0.001$), and decreased PPA ($p<0.001$) compared to non-smokers. Smoking a single cigarette increased central SBP, HR, and PPA, and lowered SEVR (all $p<0.001$). In response to acute maximal exercise, smokers failed to achieve comparable exercise time ($p=0.015$) and maximal HR ($p<0.001$) as non-smokers. Furthermore, smokers on all 3 conditions demonstrated lower exercise-induced changes in AIx75 (all $p<0.001$) and SEVR (chronic $p=0.003$, acute and nicotine $p<0.001$) compared to non-smokers. After acute smoking, the exercise-induced increase in cfPWV was lower when compared to the chronic condition ($p=0.010$).

Conclusions: These findings demonstrate that acute and chronic smoking lead to an altered vessel hemodynamic response even in young healthy smokers. Therefore, the 'arterial stress test' may serve as a useful tool to identify vascular impairment in young smokers at an early subclinical stage.

Arterial Stiffness and Central Systolic Blood Pressure Response to Dietary Sodium in Young and Middle-Aged Adults

Bryce J. Muth¹, Michael S. Brian¹, Evan L. Matthews¹, Meghan G. Ramick¹, Shannon Lennon-Edwards^{1,2}, William B. Farquhar¹, David G. Edwards¹

¹Departments of Kinesiology & Applied Physiology and ²Behavioral Health and Nutrition, University of Delaware, Newark, DE

High dietary sodium intake has been associated with the development of hypertension and increased incidence of cardiovascular disease.

OBJECTIVE: The aim of this study was to determine the effect of short-term dietary sodium loading on arterial stiffness and central blood pressure in young (YG; 22-40 years old, n=49, 27±1 yrs) and middle-aged (MA; 41-60 years old, n=36, 52±1 yrs) normotensive adults.

METHODS: Subjects were randomized to 7 days of low sodium (LS: 20 mmol/d) and 7 days of high sodium diet (HS: 300 mmol/d). On the last day of each diet, carotid-femoral pulse wave velocity (PWV), central aortic pressure waveform (synthesized by radial artery applanation tonometry and generalized transfer function), and wave separation analysis were assessed.

RESULTS: In comparison to the LS diet, the HS diet elicited an increase in central systolic blood pressure (cSBP) in both YG (LS: 96±1 vs. HS: 99±1 mmHg, p < 0.05) and MA (LS: 106±2 vs. HS: 115±3 mmHg, p < 0.05). The increase in cSBP was greater in MA (YG: 4±1 vs. MA: 9±2, p < 0.05). In MA, HS elicited greater central forward wave amplitude (LS: 25±1 vs. HS: 29±1 mmHg, p < 0.05), central reflected wave amplitude (LS: 19±1 vs. HS: 23±1 mmHg, p < 0.05), and PWV (LS: 7.1±0.3 vs. HS: 7.7±0.5 ms, p < 0.05) whereas these were not different in the YG.

CONCLUSION: These data suggest that high sodium intake is associated with a greater increase in cSBP in MA that may be the result of increased arterial stiffness and forward and reflected wave amplitudes.

Supported by NIH Grant R01 HL104106.

Hemodynamic Responses Following 12 Weeks of Home-Based Exercise in Individuals with Multiple Sclerosis: Wave Separation Analysis

Sang Ouk Wee¹, Garrett Griffith¹, Rachel E. Klaren², Robert W. Motl², Tracy Baynard¹, Bo Fernhall¹, FACSM, ¹University of Illinois at Chicago, Chicago, IL, ²University of Illinois at Urbana-Champaign, Champaign, IL.

Increased inflammation, which is related to cardiovascular disease and altered hemodynamics, is a common condition in individuals with multiple sclerosis (MS). Low to moderate intensity aerobic exercise has shown beneficial effects in reducing inflammation and blood pressure (BP) in individuals without MS. Low to moderate intensity exercise may benefit BP and pulse wave characteristics, in individuals with MS.

Purpose: To investigate the chronic responses to low to moderate intensity home-based exercise vs. stretching on blood pressure and pulse wave characteristics in individuals with MS.

Methods: 29 individuals (16 cycle exercise group, 13 stretching group) with MS participated in 12 weeks of home-based exercise. Both groups received weekly Skype counseling sessions to monitor for exercise compliance. Central BP was estimated and pulse waveforms were measured using applanation tonometry and separated into forward and reflected waves at: 1) baseline; 2) mid-point (6 weeks of training); and 3) after 12 weeks of training.

Results: Neither exercise nor stretching altered peripheral or central BP indices. However, aortic DBP approached a significant decrease after training ($p = 0.073$). Wave separation analysis did not show any change following 12 weeks of exercise or stretching. Forward and reflected pressure did not differ between groups.

Conclusions: The results suggest that moderate intensity home-based exercise did not change BP and had no effect on wave separation variables. The lack of change may be due to the subjects being normotensive. This population may need higher intensity exercise to elicit the beneficial hemodynamic changes including wave separation indices.

Variables	Exercise			Stretching		
	Baseline	Mid-Point	Post	Baseline	Mid-Point	Post
Aortic SBP (mmHg)	107 ± 5.1	112 ± 4.3	107 ± 4.3	105 ± 4.2	104 ± 3.8	102 ± 3.8
Aortic DBP (mmHg)	73 ± 2.8	78 ± 3.1	75 ± 3.1	72 ± 3.3	73 ± 2.9	69 ± 3.1
Brachial SBP (mmHg)	116 ± 4.8	123 ± 4.4	117 ± 4.0	116 ± 4.3	114 ± 3.5	112 ± 3.8
Brachial DBP (mmHg)	72 ± 2.8	77 ± 3.1	74 ± 3.1	71 ± 3.3	72 ± 2.8	69 ± 3.1
MAP (mmHg)	87 ± 3.3	92 ± 3.4	90 ± 3.7	86 ± 3.3	86 ± 2.9	83 ± 3.1
Forward Pressure (mmHg)	24.8 ± 1.63	26.1 ± 1.55	25.2 ± 1.47	24.9 ± 1.55	26.4 ± 1.47	26.9 ± 1.93
Reflected Pressure (mmHg)	16.5 ± 1.67	14.7 ± 1.82	16.1 ± 1.52	15.9 ± 1.36	15.7 ± 0.91	17.7 ± 1.53
Mean ± SEM						

Arterial Stiffness throughout Pregnancy in Women Who Conceive Via In Vitro Fertilization

Kim Phan¹, Laura Elbaz¹, Yessica-Haydee Gomez¹, Jessica Gorgui¹, Amira El-Messidi², Robert Gagnon², Stella S Daskalopoulou¹

¹ *Department of Medicine, Faculty of Medicine, McGill University, Montreal, Quebec, Canada*

² *Department of Obstetrics and Gynecology, Royal Victoria Hospital, McGill University, Montreal, Quebec, Canada*

Objectives: In vitro fertilization (IVF) is an increasingly common method of conception among women. Studies have reported a higher risk in IVF pregnancies for certain cardiovascular-related outcomes, such as pregnancy-induced hypertension. The effect of IVF on arterial structure as a potential underlying mechanism for this increased risk has yet to be explored. Applanation tonometry allows the non-invasive assessment of arterial stiffness, a composite predictor of cardiovascular risk. The objective of this study was to explore the association between IVF and arterial hemodynamic and stiffness parameters in pregnancy.

Methods: In this prospective longitudinal study, women with singleton pregnancies were recruited from high-risk obstetrical clinics. Arterial stiffness was measured using applanation tonometry (Sphygmocor; AtCor, Australia) and compared between women who conceived via IVF and those who conceived spontaneously. Arterial hemodynamics and stiffness were assessed, starting in the first trimester, every four weeks throughout pregnancy and again at six weeks post-partum.

Results: Of the 69 participants recruited (median maternal age = 37 years [IQR = 35-39]), 23 conceived by IVF. Analyses adjusted for both maternal age and body mass index showed there were no significant differences in peripheral and central blood pressures of women who conceived via IVF as compared to those who conceived spontaneously throughout pregnancy or at six weeks post-partum. In addition, carotid-femoral pulse wave velocity, carotid-radial pulse wave velocity, and augmentation index at a heart rate of 75 beats per minute did not differ significantly between these two populations (all p-values > 0.05).

Conclusion: Conception via IVF did not have significant effects on the hemodynamic parameters, nor on central, peripheral, and systemic arterial stiffness throughout pregnancy of women in this population. Nor were differences found six weeks post-partum.

The Influence of Resting Heart Rate on Low- and Very Low-Frequency Blood Pressure Variability

Bunsawat, K, White, DW, Shafer, BM, Marqui, PA, Wang Y, Fernhall B, and Baynard, T.

Integrative Physiology Laboratory. Department of Kinesiology and Nutrition, University of Illinois at Chicago, Chicago, IL.

Low-frequency (LF) blood pressure variability (BPV) is associated with sympathetic modulation of vascular tone, whereas very low-frequency (VLF) BPV reflects myogenic vascular responsiveness to blood pressure oscillations. Heart rate (HR) can affect blood pressure (BP) and is also modulated by sympathetic tone, but whether an equal change in HR affects measures of BPV in individuals with different resting HR levels remains unclear. **Purpose:** To compare measures of BPV in young adults with low (LowHR; HR<70bpm) and high (HighHR; HR>70bpm) resting HR. **Methods:** Subjects were divided into 2 groups: 12 LowHR (female=2; age 25±1 yrs; BMI 24.1±0.7 kg/m²) and 15 HighHR (female=9; age 25±1 yrs; BMI 23.1±0.7 kg/m²) who were matched on HR change (10±1 bpm) during upright leg cycling. Respiration was paced, and beat-to-beat BP and BPV were assessed for 5 min. Natural log-transformation (Ln) was performed on BPV data. **Results:** The LowHR group had a lower resting HR than HighHR (62±2 bpm vs. 81±2 bpm, *P*<0.05). Both LowHR and HighHR had similar BP increases from rest when matched on HR change to a small exercise stimulus (*P*<0.05). However, LowHR had an overall lower BPV_{LnLF} than HighHR (*P*<0.05). No condition or group differences were observed for BPV_{LnVLF} (*P*>0.05). **Conclusions:** A stimulus that caused an equal change in HR also caused an equal change in BP in both groups. The lower BPV_{LnLF} in individuals with low resting HR suggests an overall lower sympathetic modulation of BP, with no differences in myogenic responsiveness between groups.

Table 1. BP and BPV responses to a change in HR of 10 bpm.

		Rest	EX10	Condition	P Value Interaction	Group
SBP	LowHR	117±4	133±4	0.000	0.509	0.450
	HighHR	113±2	131±3			
DBP	LowHR	66±3	69±2	0.002	0.542	0.507
	HighHR	66±2	72±2			
MAP	LowHR	86±3	94±2	0.000	0.332	0.556
	HighHR	86±2	97±2			
BPV _{LF}	LowHR	6.52±1.91	8.67±1.81	0.314	0.783	0.129
	HighHR	9.92±1.71	11.15±1.62			
BPV _{VLF}	LowHR	9.18±5.23	9.88±4.09	0.822	0.635	0.151
	HighHR	18.96±4.7	17.03±3.66			
BPV _{LnLF}	LowHR	1.56±0.22 *	1.92±0.20	0.060	0.515	0.032
	HighHR	2.12±0.16	2.30±0.13			
BPV _{LnVLF}	LowHR	1.54±0.30	1.90±0.29	0.596	0.257	0.111
	HighHR	2.42±0.30	2.29±0.32			

Data are mean±SE. BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; BPV_{LF}, blood pressure variability-low frequency; BPV_{VLF}, blood pressure variability-high frequency; BPV_{LnLF}, blood pressure variability-naturally log transformed low frequency; BPV_{LnVLF}, blood pressure variability-naturally log transformed very low frequency. *significantly different than baseline based on an independent t-test (*P*<0.05).

A New Pathway to Increase Arterial Flexibility: Investigating Oils With Respect To Arterial Flexibility Using Photoplethysmography (The IOWA Study)

Brian S. Peskin

Peskin Pharmaceuticals, Houston, Texas (USA)

OBJECTIVE: The precursor (PSK002) to an investigational new drug (PSK003) was used in a screening experiment (IOWA) to test the ability of a new botanical lipids-based medicament to reduce arterial stiffness utilizing photoplethysmography. Effects were tested following long-term (48 months, N=34) or short-term (8 months, N=16) exposure and in volunteers previously using fish oil who were converted to the medicament short-term (6 months, N=15).

HYPOTHESIS: Supplementation with the essential fatty acids (EFAs) linoleic acid (LA) and alpha-linolenic acid (ALA) and the LA metabolite gamma-linolenic acid (GLA) will reduce arterial stiffness.

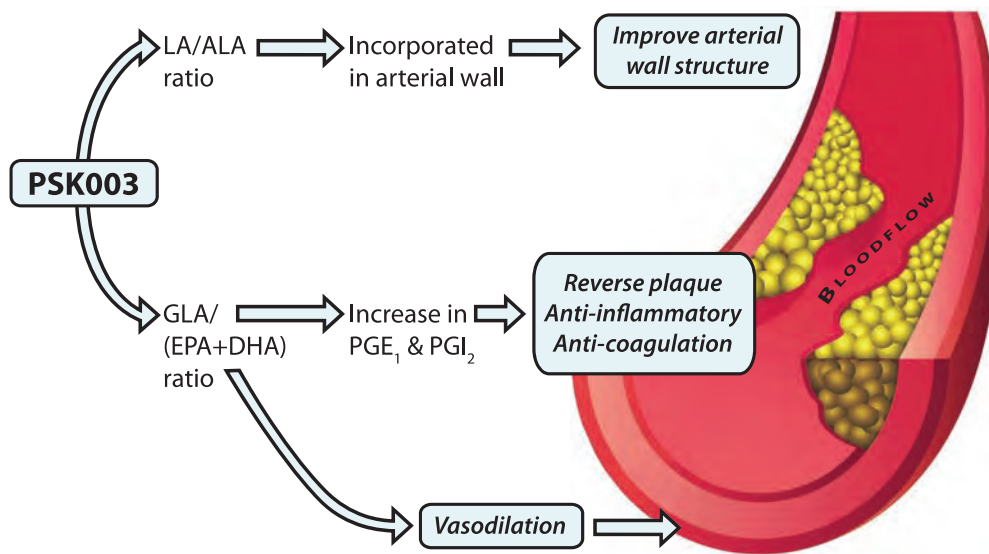


Figure 1. Primary metabolic pathways increasing arterial flexibility

METHODS: Daily dosage was 2,900 mg/day of the PSK002. The effect of treatment on the arterial pressure waveform was measured with photoplethysmography (PPG) with a Meridian® Digital Pulse Analyzer (DPA) focusing on “biological age” derived by the device as a measure of arterial wall stiffness. Error of the mean in “biological age” is stated as ± 5 years. Measurements were taken at a local health store in Des Moines, Iowa.

RESULTS: Change in biological age (mean ± SD) was -7.2±10.2 years (p=0.01) for short-term, -8.8±14.8 years (P=0.0015) for long-term and -11.1±8.4 years (P=0.006) for volunteers who were converted from fish oil.

CONCLUSIONS: The data support the hypothesis that supplementation with the EFAs (LA, ALA) and the LA metabolite (GLA) reduces arterial “biological age.” A randomized, double-blind, placebo-controlled clinical trial utilizing PSK003 and more direct measures of arterial stiffness is warranted.

Longitudinal Strain Is Not Associated With Clinic Central Systolic Blood Pressure in a Young Normotensive Population

Kearney M,^a Thomas J,^a Chapman N,^a Watkeys L,^a Munnery M,^b Cockcroft J,^b Shave R,^a Stöhr EJ,^a McDonnell B.^a

^aCardiff Metropolitan University (UK), ^bWales Heart Research Institute, Cardiff University (UK).

Objectives: Increased central systolic blood pressure (cSBP) is associated with target organ damage (TOD) of the heart as traditionally quantified by left ventricular mass (LVM).¹ However, longitudinal strain (LS), a sensitive marker of left ventricular (LV) function, was recently reported as reduced in hypertensive patients with normal LVM.² This marker may be a more sensitive indicator of sub-clinical TOD than LVM. The purpose of this study was to determine if cSBP was more strongly associated with LS than LVM in a young normotensive population.

Methods: Seventy-four young healthy normotensive adults (23 ±4yrs, 45% male) took part in the study. LVM and LS were measured using 2-D and speckle tracking echocardiography respectively (Vividq, GE Healthcare) with LVM indexed to body surface area (LVMI). Supine office peripheral SBP (pSBP) and cSBP were measured using a validated automated device which was calibrated using peripheral diastolic and mean BP (Mobil-O-Graph, IEM). The relationships between cSBP and LVMI and LS were explored using Pearson product moment correlation.

Results: Participants had normal pSBP (120 ±11 mmHg) and LVMI (84 ±17g/m²) values. Central SBP averaged 133 ±21 mmHg while mean LS was -18 ±2 %. Central SBP was significantly associated with LVMI ($r=0.486$, $P<0.0001$) but not associated with LS ($r=0.137$, $P=0.252$).

Conclusion: Despite a strong association with LVMI, cSBP was not associated with LS in the current study population. Normotension may be an insufficient haemodynamic stimulus to impact LV LS in healthy young adults. Future studies should investigate how incremental chronic increases in cSBP affect LS. This may elucidate at what point cSBP becomes a negative influence on LS and whether changes to this physiological marker precede clinically relevant changes to LVMI.

¹ Protogerou AD et al. J Hypertens 2007; 2: 265-72.

² Imbalzano E et al. Echocardiography 2011; 6: 649-657.

Resting Heart Rate Is a Factor in Acute Blood Pressure Variability Responses to Cycling

Daniel W. White, Paul A. Marqui, Yiyu Wang, Bo Fernhall, and Tracy Baynard

Integrative Physiology Laboratory, Department of Kinesiology and Nutrition, University of Illinois at Chicago, Chicago, IL 60612

Blood pressure variability (BPV) is an indicator of the ability to maintain blood pressure over time and is an indicator of arterial and autonomic health. What is not known is if steady state heart rate (HR) would affect acute measures of BPV. Resting HR is associated with overall sympathetic and vagal modulation. An individual with a lower resting HR should be able to increase sympathetic modulation to a greater extent which peripherally should increase BPV.

Purpose: To determine if a lower resting steady state HR would result in a greater increase in BPV when cycling at a low workload.

Methods: Forty young healthy subjects had HR, blood pressure and respiration measured during upright rest (5 min), followed by cycling at 20W@60rpm. Participants were instructed to breathe at a pace of 12 breaths·min⁻¹ during both the resting and exercise phases. Groupings were based on Low and High resting HRs of <70 or >70 beats·min⁻¹, respectively. Due to non-compliance with paced breathing only 21 subjects were included.

Results and Conclusions: Data are shown in the table. We expected the similar increase in HR between the groups would equate to an increase in BPV, but there was a divergent change in beat-to-beat deviations of SAP (SAP_{dev}). This indicates that resting HR is important in acute BPV. The light workload cycling may have caused a mild exercise pressor reflex or central command induced sympathoexcitation, but this is unlikely, as shown by no increase in diastolic BP with cycling. The increase in systolic pressure (SAP) is likely due to muscle pump effects increasing venous return.

	Low Rest	High Rest	Low W20	High W20
<i>n</i> (female)	10 (1)	11 (7)		
Age	25 ± 1.3	25 ± 1.3		
Height (cm)	177.7 ± 2.02	168.7 ± 2.70*		
Weight (Kg)	77 ± 3.5	67 ± 3.3*		
BMI (Kg·M ⁻²)	24.3 ± 0.82	23.4 ± 0.56		
VO _{2max}	45.1 ± 2.19	33.4 ± 1.62*		
HR (beats·min ⁻¹) ‡	61 ± 2.1	81 ± 2.1*	71 ± 2.7	89 ± 2.6*
SAP (mmHg) ‡	117 ± 4.2	112 ± 3.0	129 ± 4.5	131 ± 2.8
DAP (mmHg)	66 ± 2.9	66 ± 1.6	69 ± 2.5	70 ± 1.8
SAP _{dev} (mmHg) †	5.9 ± 0.51	6.1 ± 0.47	5.0 ± 0.25	6.7 ± 0.37*
DAP _{dev} (mmHg) ‡	2.8 ± 0.22	3.5 ± 0.27	3.3 ± 0.16	4.2 ± 0.21
LF _{SAP} (mmHg ²) ‡	5.7 ± 2.10	3.0 ± 0.91	6.6 ± 1.31	6.2 ± 1.88
VLF _{SAP} (mmHg ²)	10.0 ± 5.91	13.4 ± 4.05	6.3 ± 1.17	11.0 ± 3.30

*group difference; †group x condition interaction; ‡cycling effect

The Blood Pressure Variability Responses to Light Cycling in Individuals With Different Resting Heart Rates

Yiyu Wang, Daniel W White, Paul A Marqui, Bo Fernhall, and Tracy Baynard

Integrative Physiology Laboratory, Department of Kinesiology & Nutrition, University of Illinois at Chicago, Chicago, IL

Blood pressure variability (BPV) is a non-invasive measure that includes the influence of sympathetic modulation. There are also other numerous factors contributing to the variability, some of which are not fully understood, such as resting or exercising heart rates.

Purpose: To determine if the relative workload needed to bring the steady state heart rate (HR) to 80bpm affects BPV differently in individuals with different resting HR.

Methods: Forty young healthy subjects had HR, blood pressure, and respiration measured for 5 min during upright rest, followed by cycling at relative workload to sustain the heart rate at 80 beats×min⁻¹ (Ex80). Participants were instructed to breathe at a pace of 12 breaths×min⁻¹ during both the resting and exercise phases. Groupings were based on Low and High resting HRs of <70 or >70 beats·min⁻¹, respectively. Due to non-compliance with paced breathing or the inability to attain a steady state HR of 80 beats·min⁻¹, only 21 subjects were included.

Results and conclusions: Data are shown in the table. We did not find any group differences in response to exercise. However, exercise affected in frequency analysis of systolic arterial pressure (SAP) (LF_{SAP}) and the beat-to-beat measurements of diastolic arterial pressure deviations (DAP_{dev}) in both groups similarly. This might indicate that LF_{SAP} and DAP_{dev} are susceptible to cyclic changes in external vascular pressure due to muscle pump activity. The increase in SAP with exercise is likely due to increases in venous return, as there were no increases in DAP, indicating no significant sympathoexcitation.

	Low Rest	High Rest	Low Ex80	High Ex80
<i>n</i>	10	11		
Age	25.2 ± 4.1	25 ± 4.4		
Height*	178 ± 6.3	168 ± 8.9		
Weight*	77 ± 11	67 ± 10.8		
BMI	24 ± 2.5	23 ± 1.8		
VO _{2MAX} *	45 ± 6.9	33 ± 5.3		
Rel WL*			14.9 ± 8.53	6.3 ± 2.53
HR†‡	61 ± 6.5	81 ± 6.8*	79 ± 2.1	82 ± 4.5
SAP†	117 ± 13.2	112 ± 9.8	132 ± 17.3	127 ± 5.1
SAP _{dev}	5.8 ± 1.61	6.1 ± 1.61	5.9 ± 1.5	5.7 ± 0.8
DAP	66 ± 9.3	65 ± 5.3	66 ± 5.7	69 ± 3.9
DAP _{dev} ‡	2.7 ± 0.69	3.4 ± 0.91	3.5 ± 0.6	3.8 ± 0.55
LF _{SAP} ‡	5.6 ± 6.63	7.7 ± 3.03	9.4 ± 6.3	10.3 ± 5.58
VLF _{SAP}	9.9 ± 18.7	16 ± 13.4	11.3 ± 6.3	8.3 ± 4.82

*: Group difference P<0.05 ‡: Exercise effect P<0.05; †: interaction P<0.05

Independent and Combined Effects of Aerobic and Resistance Training on Blood Pressure (Art-B)

Elizabeth C. Schroeder, Warren D. Franke, Rick L. Sharp, Duck-chul Lee

Iowa State University, Ames, IA

Objective: To compare the effects of aerobic training only, resistance training only, and a combination of both on blood pressure (BP) and other cardiovascular disease risk factors compared with a non-exercising control group.

Methods: Pre-to-stage 1 hypertensive, overweight/obese, and sedentary adults (ages 58±7 years) were randomized to one of three 8-week exercise programs (aerobic only, resistance only, or a combination of both), or a non-exercise control group. Participants exercised 3 days/week for 60 minutes/session. BP was measured with the SphygmaCor device. Cardiovascular fitness, strength, and body composition were assessed using the Balke treadmill protocol; upper and lower body 1-repetition maximums; and bioelectrical impedance (InBody720), respectively.

Results: At baseline, the mean (SE) for systolic and diastolic BP was 131(13) mmHg and 91(9) mmHg, respectively. Eight weeks of exercise did not significantly change systolic BP in any of the groups ($p > 0.05$), and only the combination group had a significant decrease in diastolic BP (-3.7 mmHg, 95% CI -6.8, -0.6). Significant increases [mean (95% CI); p value] were reached in treadmill time at 85% age-predicted maximal HR for the aerobic [72 seconds (38, 107); $p < 0.01$] and combination groups [51 seconds (17, 86); $p < 0.01$], whereas lower body strength gains were observed in the resistance [29.4 lbs (9.1, 49.7); $p = 0.01$] and combination groups [24.4 lbs (4.7, 44.2); $p = 0.02$]. Improvements [mean (95% CI); p value] in body composition were obtained for all three exercise groups: aerobic [weight: -1.0 kg (-1.9, -0.1; $p = 0.03$) & fat mass -0.9 kg (-1.5, -0.2; $p = 0.01$)], resistance [waist circumference: -1.7 cm (-3.3, -0.1; $p = 0.04$)], and combination [weight 0.9 kg (0.02, 1.8; $p = 0.04$) & lean body mass 0.8 kg (0.0, 1.5; $p = 0.04$)].

Conclusion: The combination of aerobic and resistance exercise training was the most effective in improving blood pressure and other cardiovascular disease risk factors.

JOIN OUR EXCITING NEW 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: _____

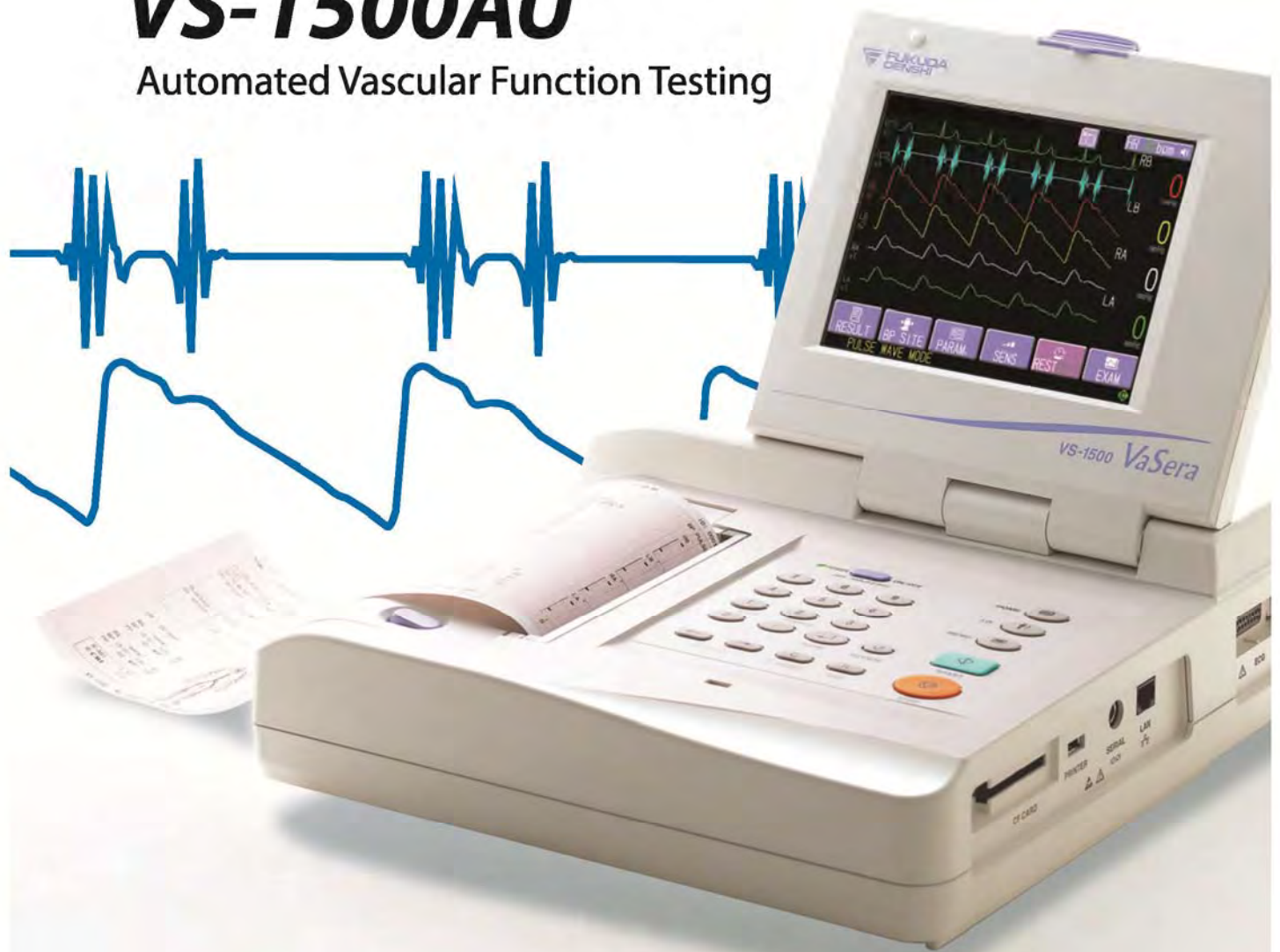
Return this form to: North American Artery Society, c/o Hansen Global Event Management, LLC, 68 Carlton Terrace, Stewart Manor, NY 11530 or fax the form to 866-383-6027.

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

VaSera™

VS-1500AU

Automated Vascular Function Testing



- Bring Quality to your Clinical Practice and Research
- Easy, Office-Based Measurements
- Automated Arterial Stiffness (CAVI), ABI and TBI
- High Reproducibility; Low Operator Dependence



FUKUDA DENSHI USA, INC
17725-C NE 65th Street Redmond, WA 98052 USA
Toll Free: (800) 365-6668 / Local: (425) 881-7737 / Fax: (425) 869-2018
www.fukuda.com

The information contained in this document is subject to change without notice.
© 2014 FUKUDA DENSHI USA, INC. All rights reserved.

USRB-0072-00



NORTH AMERICAN ARTERY

North American Artery

68 Carlton Terrace

Stewart Manor, NY 11530

Email: info@naartery.org

Website: www.naartery.org