



June 2–4, 2011 Seaport Hotel

Meeting Program

BOSTON MASSACHUSETTS, USA

SVM 22nd Annual Scientific Sessions



This meeting is jointly sponsored by
University of Colorado School of Medicine and SVM.

Welcome to Boston

President's Message

Dear Members of SVM, Colleagues and Friends,

Welcome to the 22nd Annual Scientific Sessions of the Society for Vascular Medicine. We are pleased you are here to support your colleagues and your professional society. It is great to be in Boston and I hope you will join me in thanking our Boston hosts, the staff at Brigham and Women's Hospital, Massachusetts General Hospital and Boston University School of Medicine. They are our "boots on the ground" and have helped tremendously with the wonderful programming being offered at these Scientific Sessions.

Dr. Joshua A. Beckman and the scientific program committee have developed an outstanding program for this year's meeting to cover a wide variety of topics in the vascular field. The Scientific Session program reviews many of the latest developments in vascular medicine; the "Year in Review" covers recently published literature. Taking advantage of the vast experience of our members, we're looking forward to:

- **Scientific Sessions** that provide updates on current trends in vascular medicine, information on clinical trials and new treatment options for managing vascular disease.
- **Hands-on Workshops** offer concurrent hands-on sessions covering diagnosis and treatment for diseases and complications of the arteries and veins, and also features techniques for wound care, compression and sclerotherapy and varicose vein ablation.
- **Workshop on Novel Anticoagulants** offers information on the new medications replacing warfarin, covering the pharmacology, how the drugs should be used in venous thromboembolism and atrial fibrillation, the impact on hospital P&T committees and how the new agents will affect anticoagulation clinics.

The Society continues to grow and work for all of us. Your SVM Board of Trustees has stepped up to lead SVM to membership growth. The SVM staff stands ready to help at every turn. Their partnership with the Board of Trustees is shaping the future of the organization. The leadership — committee chairs and members — have worked tirelessly this past year to help create educational materials and provide more opportunities for our members and friends to grow and learn in our profession.

We are happy you are here. Enjoy the sessions, enjoy Boston and have a wonderful stay!

Thom W. Rooke, MD, FSVM

President, Society for Vascular Medicine

About SVM

The Society for Vascular Medicine is dedicated to improving the recognition and care of vascular diseases; fostering education and research in vascular medicine; and providing leadership to government, industry and the profession regarding issues related to vascular health.

SVM includes among its membership the major investigators and opinion leaders in vascular medicine. Membership categories include fellow, doctorate, advanced practitioner, vascular care team and associate members.

The field of vascular medicine encompasses a broad spectrum of arterial, venous and lymphatic disorders, as well as the associated medical disorders of hypertension, hyperlipidemia, insulin resistance and diabetes mellitus. This diversity and complexity of vascular diseases requires knowledgeable practitioners of vascular medicine to provide cost effective care. SVM is a community of vascular clinicians who believe in the benefits of multidisciplinary discussion of critical evolving issues in vascular medicine.

Continuing Medical Education

This activity has been planned and implemented in accordance with the Essential Areas and Policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint sponsorship of University of Colorado School of Medicine (UCSM) and SVM. UCSM is accredited by the ACCME to provide continuing medical education for physicians. UCSM designates this live activity for a maximum of 19 *AMA PRA Category 1 Credit(s)*[™]. Physicians should only claim credit commensurate with the extent of their participation in the activity.

Disclosure

University of Colorado Affiliated Course Director Dr. James B. Froehlich, MD, MPH, FSVM, asks all individuals involved in the development and presentation of continuing medical education (CME) activities to disclose all relationships with commercial interests. Faculty disclosure information will be available in meeting materials.

Accreditation for Pharmacists Attending the Novel Anticoagulant Workshops

TG Medical Education, LLC is accredited by the Accreditation Council for Pharmacy Education as a provider of continuing pharmacy education. This activity provides four hours (0.4 CEUs) of continuing pharmacy education credit.

Registered Vascular Technologists' CME

The American Registry for Diagnostic Medical Sonography (ARDMS) accepts *AMA PRA Category 1 Credit(s)*[™] for registered vascular technologists and sonographers to meet their continuing education requirements. SVM is approved by University of Colorado School of Medicine to issue these credits. Complete the CME application form to submit to ARDMS to obtain verification of credits.

Nursing CME

Most boards of nursing accept a wide variety of CE/CME activities in fulfillment of CE requirements. Because each board has a slightly different way of determining approved programs, you will need to check with your state board of nursing to determine whether or not a specific activity will satisfy its requirements. SVM can stipulate that this educational activity conforms to the MA Board of Nursing Regulation 244 CMR 5.04 (2) (b).

Program Overview

Vascular diseases represent the single most important cause of death and disability in our nation and will remain so for the decades ahead. SVM's meetings provide an important arena to help health care providers maintain their focus on tangible cardiovascular health threats.

An essential component of every year's meeting is to feature internationally recognized clinician investigators, clinician educators and renowned vascular scientists to keep our attendees abreast of issues that impact their patients, their practice and their livelihood.

Disclaimer: THESE MATERIALS AND ALL OTHER MATERIALS PROVIDED IN CONJUNCTION WITH CME ACTIVITIES ARE INTENDED SOLELY FOR PURPOSES OF SUPPLEMENTING CME PROGRAMS FOR QUALIFIED HEALTH CARE PROFESSIONALS. ANYONE USING THE MATERIALS ASSUMES FULL RESPONSIBILITY AND ALL RISK FOR THEIR APPROPRIATE USE. TRUSTEES OF UNIVERSITY OF COLORADO SCHOOL OF MEDICINE MAKE NO WARRANTIES OR REPRESENTATIONS WHATSOEVER REGARDING THE ACCURACY, COMPLETENESS, CURRENTNESS, NONINFRINGEMENT, MERCHANTABILITY OR FITNESS FOR A PARTICULAR PURPOSE OF THE MATERIALS. IN NO EVENT WILL TRUSTEES OF UNIVERSITY OF COLORADO SCHOOL OF MEDICINE BE LIABLE TO ANYONE FOR ANY DECISION MADE OR ACTION TAKEN IN RELIANCE ON THE MATERIALS. IN NO EVENT SHOULD THE INFORMATION IN THE MATERIALS BE USED AS A SUBSTITUTE FOR PROFESSIONAL CARE.

Scientific Program Committee and Speaker Disclosures

Committee Member/ Speaker	Name of Corporate Organization	Type of Affiliation/ Financial Interest
Viken Babikian, MD	Boston Scientific	Consultant
Ken Bauer, MD	GlaxoSmithKline	Consultant
	Johnson & Johnson	Consultant
	Pfizer	Consultant
	Bristol-Myers Squibb	Consultant
Deepak Bhatt, MD, MPH, FACC, FAHA, FSCAI	AstraZeneca	Grant/Research Support
	Bristol-Myers Squibb	Grant/Research Support
	Eisai	Grant/Research Support
	Sanofi-aventis	Grant/Research Support
	The Medicines Company	Grant/Research Support
Eugene Braunwald, MD	CVRX, Inc.	Consultant
Leslie T. Cooper, MD, FSVM	Sanofi Pasteur	Consultant
	Crucell Holland	Consultant
	Asahi Kansei	Consultant
Paul F. Dellaripa, MD	Kowa Pharmaceuticals	Other: Steering Committee
	Gilead	Grant/Research Support
	Genentech	Grant/Research Support
John Fanikos, PharmD, MBA	Blogen	Grant/Research Support
	Bristol-Myers Squibb	Consultant
	Baxter	Consultant

Committee Member/ Speaker	Name of Corporate Organization	Type of Affiliation/ Financial Interest
James B. Froehlich, MD, MPH, FSVM	Ortho-McNeil	Consultant
	Sanofi-aventis	Consultant
	Merck/Schering-Plough	Speakers Bureau
Samuel Z. Goldhaber, MD, FSVM	Bristol-Myers Squibb	Grant/Research Support, Consultant
	Boehringer Ingelheim	Grant/Research Support, Consultant
	Eisai	Grant/Research Support, Consultant
	EKOS	Grant/Research Support, Consultant
	Johnson & Johnson	Grant/Research Support
	Sanofi-aventis	Grant/Research Support, Consultant
	Medscape	Consultant
	Pfizer	Consultant
Heather L. Gornik, MD, MHS, FSVM	Summit Doppler Systems	Grant/Research Support
	Zin Medical	
Michael R. Jaff, DO, MSVM	Abbott Vascular	Consultant
	Boston Scientific	Consultant
	Covidien	Consultant
	Medtronic	Consultant
	I C Sciences	Consultant
	Harvard Clinical Research Institute	Consultant
	Arsenal Medical	Other: Advisory Board
	Micell	Other: Advisory Board

Committee Member/ Speaker	Name of Corporate Organization	Type of Affiliation/ Financial Interest
Michael R. Jaff, DO, MSVM (continued)	Neocure	Other: Advisory Board
	Nexeon	Other: Advisory Board
	Primacea	Other: Advisory Board
	Access Closure	Other: Equity
	Embolitech	Other: Equity
	Hotspur	Other: Equity
	Icon Interventional	Other: Equity
	Sadra Medical	Other: Equity
	TMI	Other: Equity
	Vascular Therapies	Other: Equity
	VIVA Physicians	Other: Board Member
John A. Kaufman, MD, MS	Teneo	Other: Medical Board
	Bio2	Other: Medical Board
	Crux Medical	Other: Medical Board
	Hatch	Other: Investor
	ev3	Consultant
	NIH	Consultant
Esther Soo Hyun Kim, MD, MPH, FACC	Philips Ultrasound	Consultant
	GE	Grant/Research Support
Raghu Kolluri, MD, RVT, FSVM	Advanced Biohealing	Speakers Bureau
Paul A. Monach, MD, PhD	Genetech	Consultant, Other: Advisory Board
	Bristol-Myers Squibb	Grant/Research Support
	EMD-Serono	Consultant

Committee Member/ Speaker	Name of Corporate Organization	Type of Affiliation/ Financial Interest
Manish Mehta, MD	ev3 Endovascular Inc. Cordis Corp W.L. Gore Trivascular Inc. Medtronic Inc.	Consultant
	Abbott Vascular Cordis Corp Terumo Cardiovascular System Trivascular Inc. W.L. Gore Lombard Medical Tech. Bolton Medical Inc. Medtronic Inc. Aptus Endosystems ev3 Endovascular Inc. Maquet Cardiovascular Harvest Technologies	Grant/Research Support
Patrick O'Gara, MD	Lantheus Medical Imaging	Other: DSMB Chair
Elizabeth V. Ratchford, MD, FSVM	Merck	Other: Stockholder
Marc S. Sabatine, MD, MPH	AstraZeneca	Grant/Research Support
	Bristol-Myers Squibb/ Sanofi-aventis joint venture	Grant/Research Support, Consultant
	Daiichi-Sankyo	Grant/Research Support
	Sanofi-aventis	Grant/Research Support, Consultant
	Schering-Plough	Grant/Research Support
	Daiichi-Sankyo/Lilly partnership	Consultant

Learning Objectives

Scientific Sessions

- Describe new developments regarding vascular disease in medical therapy, intervention and management.
- Identify issues and developments in diagnosing and treating arterial diseases.
- Integrate and interpret aspects of interesting cases and new research.

Hands-On Workshops

- Identify issues in diagnosing and addressing carotid artery diseases.
- Explain keys to diagnosing renal artery occlusive disease.
- Apply TCD basics to diagnosis and patient care.
- Develop skills for diagnosing and treating venous reflux and ablation.
- Demonstrate skills for diagnosing and treating arterial complications.
- Develop skills for diagnosis of upper extremity arterial and venous disease.
- Implement techniques for wound care, tactics for use of compression and sclerotherapy.
- Employ skills for the proper methods of varicose vein ablation.

Novel Anticoagulants Workshop, Part 1

- Identify new drug treatments and explain the mechanisms of action.
- Cite emerging treatments for atrial fibrillation.
- Describe new science in venous thromboembolism.
- Analyze the impact of these new therapies on anticoagulation clinics.

Novel Anticoagulants Workshop, Part 2

- Identify issues and potential treatments for cases presented.
- Evaluate elements of and impact of the cost of agents and treatment on the institution.
- Summarize the impact of these novel agents on hospital pharmacy policy.
- Discuss and assess the impact of novel anticoagulants on the health care system in the United States.
- Use novel anticoagulants in cost-effective circumstances.

Program

The schedule is subject to change.
All educational sessions are in the **Amphitheater** unless otherwise noted.

Scientific Sessions Wednesday, June 1

4:00 p.m. – 7:00 p.m. *Atrium Lobby*
Registration

5:30 p.m. – 7:30 p.m.
Satellite Symposium (*corporate sponsors TBD*)

Thursday, June 2

7:00 a.m. – 5:00 p.m. *Atrium Lobby*
Registration

7:00 a.m. – 7:00 p.m. *Atrium Lobby*
Poster Viewing

7:00 a.m. – 8:00 a.m. *Cityview Ballroom*
Continental Breakfast and
Exhibit Viewing

7:00 a.m. – 8:00 a.m. *Skyline Room*
New Member Continental Breakfast

8:00 a.m. – 11:50 a.m. *Amphitheater*
Scientific Sessions 1-3
Presidential Address

8:00 a.m. – 8:05 a.m.
Welcome to the SVM 22nd Annual Scientific
Sessions

Joshua A. Beckman, MD, FSVM, *Brigham and Women's Hospital, Scientific Program Committee Chair and President-Elect, SVM*



VASCULAR INTERVENTIONAL ADVANCES
THE NATIONAL EDUCATION COURSE FOR VASCULAR INTERVENTION AND MEDICINE



OCTOBER 18-21, 2011
WYNN LAS VEGAS

8:05 a.m. – 8:55 a.m.

Session 1: Keynote Address — Health Care in the New World: The Academic Medical Center



ELIZABETH G. NABEL, MD, is president of the Brigham and Women's Hospital (BWH) and professor of medicine at Harvard Medical School. BWH is one of the nation's leaders in academic health care and one of the

largest recipients of National Institutes of Health (NIH) research funding. As president, Dr. Nabel is responsible for patient care, research, education and community missions.

She attended Weill Cornell Medical College in New York City and conducted her internal medicine and cardiovascular training at BWH, followed by faculty positions at the University of Michigan Medical School, where she directed the division of cardiology and the Cardiovascular Research Center. Before assuming her position at BWH in January 2010, Dr. Nabel was director of the NIH's National Heart, Lung and Blood Institute (NHLBI), where she oversaw an extensive national research portfolio with an annual budget of approximately \$3 billion. Her signature efforts included raising awareness for heart disease in women; launching a global health program to combat non-communicable diseases; creating new scientific programs to pursue the promise of genomics and stem cells, stem and progenitor cell biology, and translational research; in addition, Dr. Nabel continues to nurture the careers of young investigators.

8:55 a.m. – 9:55 a.m.

Session 2: Joint Session with VIVA— Vascular InterVentional Advances

Moderator: Michael R. Jaff, DO, MSVM, *Massachusetts General Hospital*

When to place and retrieve Vena Cava filters
John A. Kaufman, MD, MS, *Dotter Interventional Institute/OHSU*

Endovascular aortic stent-graft repair for ruptured abdominal aortic aneurysm Manish Mehta, MD, *The Institute for Vascular Health and Disease*

Drug/device combinations in PAD Kenneth Rosenfield, MD, FSVM, *Massachusetts General Hospital*

Modern management of critical limb ischemia
Manish Mehta, MD, *The Institute for Vascular Health and Disease*

The next wave in device regulations in the United States Gary M. Ansel, MD, FSVM, *MidOhio Cardiology & Vascular Consultants*

Panel Discussion

9:55 a.m. – 10:25 a.m. *Cityview Ballroom*
Refreshment Break and Exhibit Viewing

10:25 a.m. – 11:30 a.m.

Session 3: Thrombolytic Therapy in Pulmonary Embolism

Speakers will help session attendees identify the symptoms that indicate thrombolysis in pulmonary embolism. Treatment will also be discussed: best medical management, how to choose the best option for treatment, and when surgery is the appropriate treatment option.

Moderator: Samuel Z. Goldhaber, MD, FSVM, *Brigham and Women's Hospital*

What are the indications for thrombolysis in pulmonary embolism? Douglas Drachman, MD, FACC, *Massachusetts General Hospital*

Is low intensity anticoagulation (INR 1.5-2) equivalent to standard anticoagulation for indefinite secondary prophylaxis? Ken Bauer, MD, *Beth Israel Deaconess Medical Center*

Is catheter-based lysis better than intravenous administration? John A. Kaufman, MD, MS, *Dotter Interventional Institute/OHSU*

When should surgical pulmonary embolectomy be considered? Samuel Z. Goldhaber, MD, FSVM, *Brigham and Women's Hospital*

Question-and-Answer Discussion**11:30 a.m. – 11:50 a.m.**

Presidential Address

SVM President will provide information on current organizational happenings.

Speaker: Thom W. Rooke, MD, FSVM, *Mayo Clinic, President, SVM*

11:50 a.m. – 1:20 p.m. *Harborview 2 Ballroom*
Annual Business Meeting Lunch
(SVM Fellow Members only)

The agenda for the Annual Business Meeting will be distributed to participants at the meeting. This lunch is open to SVM Fellows only.

11:50 a.m. – 1:20 p.m. *Cityview Ballroom*
Exhibit Viewing and Delegate Lunch Break (on your own)

1:20 p.m. – 5:30 p.m.

Ampitheater

Scientific Sessions 4–7

1:20 p.m. – 2:20 p.m.

Session 4: Case-based Vascular Medicine: How Experts Approach Common Chief Complaints

Each lecture will provide an overview of common and rare causes, case examples, important considerations, diagnostic approach, treatment options, prognosis and what knowledge is lacking in the field.

Moderator: Robert D. McBane, MD, FSVM, *Mayo Clinic*

I am on Coumadin, but need surgery in two weeks Esther Soo Hyun Kim, MD, MPH, FACC, *The Cleveland Clinic Foundation*

I have classic claudication but my ABI is normal Robert D. McBane, MD, FSVM, *Mayo Clinic*

Why do I have an ulcer on my heel with a normal ABI? Raghu Kolluri, MD, RVT, FSVM, *Prairie Vascular Institute*

My mom has Factor V Leiden and a DVT, but I only have FVL Suman W. Rathbun, MD, FSVM, *University of Oklahoma Health Sciences Center*

2:20 p.m. – 3:20 p.m.

Session 5: The Inflamed Vessel

Speakers will demonstrate the use of imaging techniques to determine vasculitis disease activity and how to diagnose the type of vasculitis using vessel size. This session will also describe the key components of emerging therapies for vasculitis, particularly in the small vessels.

Moderator: Steven M. Dean, DO, FSVM, *The Ohio State University College of Medicine*

How to use vessel size to make the diagnosis Paul F. Dellaripa, MD, *Brigham and Women's Hospital*

Novel imaging techniques to determine large vessel disease activity Heather L. Gornik, MD, MHS, FSVM, *The Cleveland Clinic Foundation*

Emerging therapies for vasculitides Paul A. Monach, MD, PhD, *Boston University School of Medicine*

Skin manifestations of vasculitis Steven M. Dean, DO, FSVM, *The Ohio State University College of Medicine*

Question-and-Answer Discussion

3:20 p.m. – 3:35 p.m.

Session 6: Award Presentations – Jess Young Outstanding Educator Award and Master of SVM Award

Presenters: Thom W. Rooke, MD, FSVM, *Mayo Clinic*, President, SVM and Joshua A. Beckman, MD, FSVM, *Brigham and Women's Hospital*, President-Elect, SVM

3:35 p.m. – 4:05 p.m.

Cityview Ballroom

Refreshment Break and Exhibit Viewing

4:05 p.m. – 5:30 p.m.

Session 7: Jay D. Coffman Young Investigator Presentations

In honor of Jay D. Coffman (1928 – 2006), SVM past president and distinguished internist and researcher of vascular medicine and clinical cardiology, SVM sponsors an annual award in vascular medicine and biology. The top six finalists will make an oral presentation regarding their research.

Moderator: Diane J. Treat-Jacobsen, RN, PhD, FSVM, *University of Minnesota School of Nursing*

Poster 1. Activation of Aldehyde Dehydrogenase Type 2 (ALDH2) by Alda-1 Increases Maximum Running Distance in a Hindlimb Ischemia Model of Peripheral Arterial Disease **Presenter:** Porama Thanaporn, MD,

Stanford University

Poster 5. Therapeutic Treatment of Critical Limb Ischemia Using Human Induced Pluripotent Stem Cell-Derived Endothelial Cells **Presenter:** Ngan F. Huang, PhD, *Stanford University*

Poster 7. Trends in Hospitalization for Lower Extremity Thrombosis: A 20-year (1988-2007) Analysis of Incidence, Mortality, and Procedure Outcomes **Presenter:** Ravikiran Korabathina, MD, *Tufts Medical Center*

Poster 20. Acute Exposure to Diesel Particle Matter Impairs NO-mediated Microvascular Function **Presenter:** Aurelien Wauters, MD, Department of Cardiology, *Hospital Erasme, Belgium*

Poster 27. Improvement in Calf Muscle Energetics After Percutaneous Intervention in Patients with Symptomatic Peripheral Arterial Disease as Measured with MRI **Presenter:** Amy M. West, MD, *University of Virginia Health System*

Poster 32. The Utility of 18F-FDG-PET/CT for the Diagnosis of Occult Malignancy in Patients With Acute, Unprovoked Venous Thromboembolism **Presenter:** Matthew T. Rondina, MD, *University of Utah*

5:30 p.m. – 6:30 p.m.

Atrium Lobby

Poster Presentations

6:30 p.m. – 8:00 p.m.

Harborview Ballroom

Celebrating Vascular Medicine Reception

Friday, June 3

7:00 a.m. – 5:00 p.m. *Atrium Lobby*
Registration

7:00 a.m. – 1:30 p.m. *Atrium Lobby*
Poster Viewing

7:00 a.m. – 8:00 a.m. *Cityview Ballroom*
Continental Breakfast, Exhibit Viewing

7:00 a.m. – 8:00 a.m. *Harborview 1 Ballroom*
Women in Vascular Medicine Breakfast Meeting
Supported by JUZO and SIGVARIS



Speaker GALIT LAHAV, PHD, *Harvard Medical School*, discusses “How to survive and thrive in the mother-mentor marathon.” This interactive breakfast allows fellowship and discussion among women pursuing a career in vascular medicine.

Dr. Lahav received her PhD in Biology from the Technion (Israel Institute of Technology) and completed a postdoctoral training at the Weizmann Institute of Science in Israel. She is currently an Associate Professor in the department of Systems Biology, Harvard Medical School. Her lab at Harvard combines experimental and theoretical approaches to study the dynamics and function of the tumor suppressor protein p53, and its relationship to cell fate decisions of cancer and healthy cells. Dr. Lahav is also a dedicated mentor to new faculty and committed to furthering the advancement of women in science.

Moderators: Heather L. Gornik, MD, MHS, FSVM, *The Cleveland Clinic Foundation* and Susan M. Begelman, MD, FSVM, *Genentech, Inc.*

8:00 a.m. – 11:55 a.m. *Ampitheater*
Scientific Sessions 8-11 and Keynote Address

8:00 a.m. – 8:45 a.m.
Session 8: Year in Review

This session, an annual favorite, recaps the most important developments in vascular medicine over the past year. The focus will be on describing new developments in medical therapy and will identify new issues related to vascular intervention and surgery. A discussion of the development in accreditation and board certification for vascular medicine is also on the agenda.

Moderator: Elizabeth V. Ratchford, MD, FSVM, *Johns Hopkins University School of Medicine*

Medical Therapy Reena L. Pande, MD, *Brigham and Women’s Hospital*

Intervention and Surgery Christopher J. Abularrage, MD, *The Johns Hopkins Hospital*

Accreditation Joshua A. Beckman, MD, FSVM, *Brigham and Women’s Hospital*

8:45 a.m. – 8:50 a.m.
Presentation of the Jay D. Coffman Young Investigator Awards

Presenter: Diane J. Treat-Jacobsen, RN PhD FSVM, *University of Minnesota School of Nursing*

8:50 a.m. – 9:50 a.m.

Session 9: Keynote Address — Hypertension: The Past, the Present, the Future



EUGENE BRAUNWALD, MD, is the Distinguished Hersey Professor of Medicine at Harvard Medical School, and the founding chair of the TIMI Study Group at the Brigham and Women’s Hospital.

He received his medical training at New York University and completed his medical residency at The Johns Hopkins Hospital. He served as the first chief of the cardiology branch and as clinical director of the National Heart, Lung and Blood Institute, founding chair of the Department of Medicine at the University of California, San Diego. From 1972 to 1996, he was chair of the Department of Medicine at the Brigham and Women’s Hospital. He was a founding trustee and chief academic officer of Partners HealthCare System. Dr. Braunwald’s first major paper was published in *Circulation Research* in July 1954, and he has been a major force in cardiology in the past half century. According to Science Watch, Dr. Braunwald is the most frequently cited author in cardiology; he has an H index of 177. The living Nobel Prize winners in medicine voted Dr. Braunwald as “the person who has contributed the most to cardiology in recent years.”

9:50 a.m. – 10:20 a.m. *Cityview Ballroom*
Refreshment Break and Exhibit Viewing

10:20 a.m. – 11:20 a.m.

Session 10: Antiplatelet Therapy

Learn about the biology of platelets and their participation in atherosclerosis, and when the implementation of aspirin (versus aspirating) treatment is appropriate for patients with PAD. This session includes a discussion and appraisal of the elements related to the emerging issue of genetics and platelet inhibition.

Moderator: William R. Hiatt, MD, MSVM, *University of Colorado*

Inflammation and platelets in atherothrombosis

Peter Libby, MD, *Brigham and Women's Hospital*

Should PAD patients routinely be treated with aspirin?

William R. Hiatt, MD, MSVM, *University of Colorado*

Genetics and platelet inhibition: Ready for prime time?

Marc S. Sabatine, MD, MPH, *Brigham and Women's Hospital*

Point of care platelet reactivity testing: 2011

Deepak L. Bhatt, MD, MPH, FACC, FAHA, FSCAI, *Brigham and Women's Hospital*

11:20 a.m. – 11:55 a.m.

Session 11: Large Vessel Dissection

How does one assess the implications of endovascular therapy aortic dissection? This session will cover the proper management strategies for visceral artery dissection and when to use biomarkers in aortic dissection diagnosis.

Moderator: James B. Froehlich, MD, MPH, FSVM, *University of Michigan Medical School*

Does endovascular therapy relax indications for type β aortic-dissection intervention?

James B. Froehlich, MD, MPH, FSVM, *University of Michigan Medical School*

What is the proper approach to visceral artery dissection? Patrick O'Gara, MD, *Brigham and Women's Hospital*

The role of biomarkers in aortic dissection diagnosis Kim Eagle, MD, *University of Michigan Cardiovascular Center*

11:55 a.m. – 1:30 p.m. Harborview Ballroom
Vascular Jeopardy and Lunch

Join your colleagues for an interactive lunch featuring a little friendly competition that will test your knowledge of vascular medicine.

Moderators: Joshua A. Beckman, MD, FSVM, *Brigham and Women's Hospital* and Michael R. Jaff, DO, MSVM, *Massachusetts General Hospital*

1:30 p.m. – 4:30 p.m.

Concurrent Live Demonstration Workshops

Nine live demonstration sessions covering diagnosis and treatment for diseases and complications of the arteries and veins, featuring techniques for wound care, compression and sclerotherapy, and varicose vein ablation.

Workshop 1 Back Bay Room

1:30 p.m. and 3:00 p.m.

Carotid Pitfalls Marie Gerhard-Hermann, MD, FSVM, *Brigham and Women's Hospital*

2:00 p.m. and 3:30 p.m.

Renal Pearls Michael R. Jaff, DO, MSVM, *Massachusetts General Hospital*

2:30 p.m. and 4:00 p.m.

TCD Basics Viken Babikian, MD, *Boston Medical Center*

Workshop 2

Federal Room

1:30 p.m. and 3:00 p.m.

Venous Reflux & Ablation Lucy LaPerna, DO, RVT, RPVI, FSVM, *Riverside Interventional Consultants*

2:00 p.m. and 3:30 p.m.

Arterial Complications Raghu Kolluri, MD, RVT, FSVM, *Prairie Vascular Institute*

2:30 p.m. and 4:00 p.m.

Physiologic Maneuvers in the Vascular Lab Paul Wennberg, MD, FSVM, *Mayo Clinic*

Workshop 3

Skyline Room

1:30 p.m. and 3:00 p.m.

A Wound Care Workshop: From scalpel to growth factors Vickie Driver, DPM, MS, FACFAS, *Boston University Medical Campus*

2:00 p.m. and 3:30 p.m.

Compression Primer Cindy Felty, MSN, RN, CNP, FSVM, *Mayo Clinic*

2:30 p.m. and 4:00 p.m.

Sclerotherapy Margaret O'Byrne, MD, RVT, *The Vein Clinic* and Robert M. Schainfeld, DO, FSVM, *Massachusetts General Hospital*

4:30 p.m. – 6:00 p.m.

Harborview Ballroom

Cases Over Cocktails

Supported by Canyon Pharmaceuticals

Step out of your clinic, hospital or training program to join your colleagues for a rousing discussion of "Best Vascular Cases" while enjoying cocktails. This session is sure to be one of the highlights of this meeting. Challenging cases will be presented, discussed by an esteemed panel and considered – with particular good cheer – by the audience as a whole.

Moderators: Joshua A. Beckman, MD, FSVM,

Brigham and Women's Hospital and Michael R. Jaff, DO, MSVM, *Massachusetts General Hospital*

Speakers:

An external carotid artery that really matters, Natalia F. Mahlay, MD, *The Cleveland Clinic Foundation*

Median Arcuate Ligament Syndrome: a non-vascular, vascular diagnosis, Nedaa Skeik, MD, FACP, *Mayo Clinic*

Hand ischemia in a young healthy female, Tony Nguyen, *Boston University Medical Center*

54-year-old smoker with painful fingertip ulceration, Billy G. Chacko, MD, *Wake Forest University Baptist Medical Center*

Lumbar Artery Pseudo caused by a Gunther Tulip Inferior Vena Cava Filter, Nedaa Skeik, MD, FACP, *Mayo Clinic*

Definitive management of rapidly expanding hematoma in a 500 lb. man . . . without surgery or endovascular intervention! Keith E. Swanson, MD, *The Cleveland Clinic*

6:15 p.m. – 7:45 p.m.

Satellite Symposium (corporate sponsors TBD)

Saturday, June 4

7:00 a.m. – 10:30 a.m.

Atrium Lobby

Registration

7:00 a.m. – 8:00 a.m.

Mezzanine Level Lobby

Continental Breakfast

8:00 a.m. – 10:00 a.m.

Ampitheater

Novel Anticoagulants, Part 1

A complete course on the new novel anticoagulant medications replacing warfarin, covering the pharmacology, how the drugs should be used in venous thromboembolism and atrial fibrillation, the

impact on hospital P&T committees and how the new agents will affect anticoagulation clinics.

Moderator: Jerry Bartholomew, MD, FSVM, *The Cleveland Clinic Foundation*

Pharmacology of the New Agents Speaker TBD

New Therapies in Atrial Fibrillation Jonathan L. Halperin, MD, MSVM, *The Cardiovascular Institute*

New Directions in Venous Thromboembolism Samuel Z. Goldhaber, MD, FSVM, *Brigham and Women's Hospital*

10:00 a.m. – 10:15 a.m. *Mezzanine Level Lobby*
Refreshment Break

10:15 a.m. – 12:15 p.m. *Ampitheater*
Novel Anticoagulants, Part 2

Moderator: Jerry Bartholomew, MD, FSVM, *The Cleveland Clinic Foundation*

Case Discussions Panelists: Jerry Bartholomew, MD, FSVM, *The Cleveland Clinic Foundation*; Robert D. McBane, MD, FSVM, *Mayo Clinic*; Matthew T. Rondina, MD, *University of Utah Health Sciences Center*

Impact of novel agents on a hospital's budget and anticoagulation service Julie K. Atay, PharmD, MBA, *Brigham and Women's Hospital*

The P&T committee response to the \$10/day pill, locally and nationally John Fanikos, PharmD, MBA, *Brigham and Women's Hospital*

Question-and-Answer Discussion with Panel

12:15 p.m.

Meeting Adjourns

Upcoming Events



P.A.D. Coalition, Venous Disease Coalition and Vascular Disease Foundation

September 14-15

Ritz Carlton Hotel

Tyson's Corner, Virginia

Pre-session Meeting – Attend the P.A.D. Coalition, Venous Disease Coalition and Vascular Disease Foundation for their annual meeting: Current Issues in Vascular Disease Meeting and Awards Dinner. The event will take place September 14-15, at the Ritz Carlton Hotel, Tyson's Corner, Virginia. See what is happening in patient education and disease awareness programs. www.vdf.org/professionals/annualmeeting.php.

The Society for Vascular Medicine is a partner in the Vascular Disease Foundation and a member of the P.A.D. Coalition and the Venous Disease Coalition.



Veins, Vessels and Vascular Updates September 16-18, 2011

Ritz Carlton Hotel

Tyson's Corner, Virginia

Hear this inclusive, in-depth review of some of the most challenging issues and critical decision

Upcoming Events (cont.)

making that PAs encounter on a daily basis. Topics include diagnosis and treatment of peripheral artery disease, treatment and prevention of DVT, treatment options for limb ischemia, stroke prevention and treatment and hands-on vascular skills lab. September 16-18, 2011 • Ritz Carlton Hotel • Tyson's Corner, Virginia. www.aapa.org, click on Upcoming Events, Calendar of Events.

The Society for Vascular Medicine is a meeting co-sponsor.



The 3rd Annual Vascular Imaging Symposium

September 17, 2011

Philadelphia, Pennsylvania

The 3rd annual Vascular Imaging Symposium aims to provide the tools necessary for the development of better technical and interpretive skills in cerebrovascular, renal, peripheral arterial and venous ultrasound techniques. Using didactic lectures, case studies and hands-on imaging, the experienced faculty will provide thoughtful reviews, and will be readily accessible for technical tips, pearls, and caveats. In addition, participants will have opportunities to discuss laboratory logistical issues, including accreditation and billing. The event will take place September 17, 2011, in Philadelphia, Pennsylvania. www.asecho.org

The Society for Vascular Medicine is an educational partner for this event.



The Veins Chicago 2011 National Venous Interventional Summit

September 23-25, 2011

Chicago, Illinois

This comprehensive venous program will provide participants with the most current and future strategies for venous treatment, as well as the knowledge and tools required to implement a venous program at one's own institution. Keynote lectures from nationally known faculty will kick-start each day; and subsequent topics of discussion will provide specific educational opportunities of practical interest. Breakout sessions will include hands on venous imaging and anatomy, as well as "how to establish a venous practice", and individual case presentations will provide an array of tips and tricks. The event will take place September 23-25, 2011, Chicago, Illinois. www.TheVEINS.org. This event is co-sponsored by the Society for Vascular Medicine.



SAWC Fall — Symposium on Advanced Wound Care

October 13-15, 2011

Rio Hotel

Las Vegas, Nevada

The Symposium on Advanced Wound Care (SAWC) provides a venue at which the interdisciplinary wound care community can gather, greet, learn, and play. We work toward a common

goal: to decrease the number and severity of chronic wounds. The SAWC Fall 2011 will offer up to 40 new clinical sessions and host a major exhibition of products and services for wound care professionals. The SAWC is committed to providing the highest level of clinical education and information on the advancement of wound care and healing. www.fall.sawc.net.

This event is endorsed by the Society for Vascular Medicine.



The 9th Annual Vascular InterVentional Advances (VIVA) Conference

October 18-21, 2011

Wynn Las Vegas, Las Vegas, Nevada

The 9th annual Vascular InterVentional Advances (VIVA) conference will be held at Wynn Las Vegas from October 18-21, 2011. In addition to the four-day conference, VIVA and SVM are collaborating for the 2nd year to provide the only comprehensive Vascular and Endovascular Board Review Course in the nation. Please visit www.VIVAPVD.com for more details.

The 9th annual conference is endorsed by the Society for Vascular Medicine.

Invited Speakers

Christopher J. Abularrage, MD, *The Johns Hopkins Hospital*

Gary M. Ansel, MD, FSVM, *MidOhio Cardiology & Vascular Consultants*

Julie K. Atay, PharmD, MBA, *Brigham and Women's Hospital*

Viken Babikian, MD, *Boston Medical Center*

Jerry Bartholomew, MD, FSVM, *The Cleveland Clinic Foundation*

Ken Bauer, MD, *Beth Israel Deaconess Medical Center*

Joshua A. Beckman, MD, FSVM, *Brigham and Women's Hospital*

Deepak L. Bhatt, MD, MPH, FACC, FAHA, FSCAI, *Brigham and Women's Hospital*

Eugene Braunwald, MD, FRCP, *Harvard Medical School and Brigham & Women's Hospital*

Steven M. Dean, DO, FSVM, *The Ohio State University College of Medicine*

Paul F. Dellaripa, MD, *Brigham and Women's Hospital*

Douglas Drachman, MD, FACC, *Massachusetts General Hospital*

Vickie Driver, DPM, MS, FACFAS, *Boston University Medical Campus*

Kim Eagle, MD, *University of Michigan Cardiovascular Center*

John Fanikos, PharmD, MBA, *Brigham and Women's Hospital*

Cindy Felty, MSN, RN, CNP, FSVM, *Mayo Clinic*

James B. Froehlich, MD, MPH, FSVM, *University of Michigan Medical School*

Marie Gerhard-Hermann, MD, FSVM, *Brigham and Women's Hospital*

Samuel Z. Goldhaber, MD, FSVM, *Brigham and Women's Hospital*

Heather L. Gornik, MD, MHS, FSVM, *The Cleveland Clinic Foundation*

Jonathan L. Halperin, MD, MSVM, *The Cardiovascular Institute*

William R. Hiatt, MD, MSVM, *University of Colorado*

Michael R. Jaff, DO, MSVM, *Massachusetts General Hospital*

John A. Kaufman, MD, MS, *Dotter Interventional Institute/OHSU*

Esther Soo Hyun Kim, MD, MPH, FACC, *The Cleveland Clinic Foundation*

Raghu Kolluri, MD, RVT, FSVM, *Prairie Vascular Institute*

Galit Lahav, PhD, *Harvard Medical School*

Lucy LaPerna, DO, RVT, RPVI, FSVM, *Riverside Interventional Consultants*

Peter Libby, MD, *Brigham and Women's Hospital*

Robert D. McBane, MD, FSVM, *Mayo Clinic*

Manish Mehta, MD, *The Institute for Vascular Health and Disease*

Paul A. Monach, MD, PhD, *Boston University School of Medicine*

Elizabeth G. Nabel, MD, *Brigham and Women's Hospital*

Margaret O'Byrne, MD, RVT, *The Vein Clinic*

Patrick O'Gara, MD, *Brigham and Women's Hospital*

Reena Pande, MD, *Brigham and Women's Hospital*

Elizabeth V. Ratchford, MD, FSVM, *Johns Hopkins University School of Medicine*

Suman W. Rathbun, MD, FSVM, *University of Oklahoma Health Sciences Center*

Thom W. Rooke, MD, FSVM, *Mayo Clinic*

Matthew T. Rondina, MD, *University of Utah Health Sciences Center*

Kenneth Rosenfield, MD, FSVM, *Massachusetts General Hospital*

Marc S. Sabatine, MD, MPH, *Brigham and Women's Hospital*

Robert M. Schainfeld, DO, FSVM, *Massachusetts General Hospital*

Paul W. Wennberg, MD, FSVM, *Mayo Clinic*

SAVE
THE DATE!

SVM
23rd ANNUAL
SCIENTIFIC SESSIONS

Join the Society for Vascular
Medicine for its 23rd Annual
Scientific Sessions

June 14-16, 2012
Minneapolis, MN

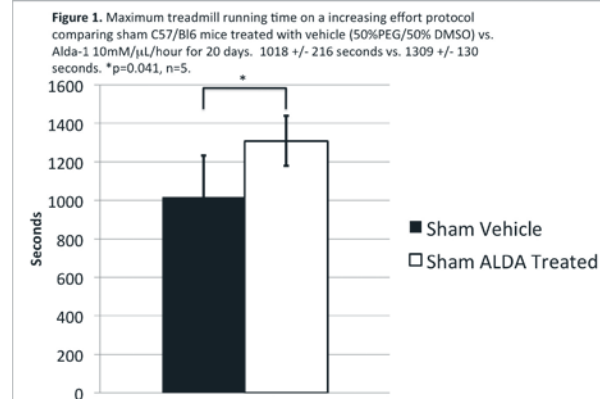
Poster Abstracts

Basic Science — Free Radicals,
Ischemia and Hypoxia



Poster 1. Activation of Aldehyde Dehydrogenase Type 2 (ALDH2) by Alda-1 Increases Maximum Running Distance in a Hindlimb Ischemia Model of Peripheral Arterial Disease

Background: We hypothesize that chronic ischemia-reperfusion injury leads to myopathy caused by reactive oxygen species (ROS). The ROS form aldehydic adducts that damage mitochondrial enzymes and mitochondrial DNA (mtDNA). In patients with PAD, phosphocreatine recovery after exercise is delayed as measured by NMR spectroscopy, and



mtDNA deletions are increased in muscle biopsies as compared to age-matched controls. Our collaborators have discovered an allosteric activator of ALDH2. This mitochondrial enzyme protects the organelle from aldehydic adducts by degrading them. By activating ALDH2, we aim to increase the

elimination of ROS-derived adducts and to improve functioning of mitochondria.

Here, we report that activation of ALDH2 significantly enhances functional capacity (as assessed by running distance and VO₂ max) in both normal mice, and in a mouse model of PAD. In the murine hind limb ischemia model we delivered Alda-1, an allosteric activator of ALDH2, dissolved in vehicle (50% PEG/50% DMSO) via subcutaneous osmotic pump inserted seven days after femoral artery(or sham) ligation. After 20 days of treatment, maximum running distance was significantly increased in sham C57/Bl6 mice treated with Alda-1 vs. sham mice receiving vehicle (* $p=0.041$, $n=5$)(Figure 1), and there was a similar trend in ligated animals treated with Alda-1. This was not due to an increase in blood flow, as there was no difference between groups in perfusion as assessed by laser Doppler spectroscopy. ALDH2*2, a variant with 5% of the catalytic activity of wild-type ALDH2 found in 30% of East Asians, was used to create knock-in mice. Forty week-old heterozygous ALDH2*2 knock-in mice (40% of wild-type activity) had significantly decreased VO₂ maximum as compared to wild-type controls (** $p = 2.25 \times 10E-6$, $n=20$ wild-type and 5 knock-in). These results strongly suggest that mitochondrial ALDH2 activity plays an important role in functional capacity. An allosteric activator of ALDH2 increases physical performance and is a potential new therapeutic approach for PAD.

Presenter: Porama Thanaporn, MD, *Stanford University, Stanford, CA, United States*

Basic Science - Hypertension and Vasoactive Molecules

Poster 2. Discovery of Allosteric Activators of DDAH: a Potential Therapeutic Avenue for Vascular Disorders

Background: Genetic and pharmacological evidence has demonstrated that disruption of the NO synthase pathway induces hypertension (HTN) as well as insulin resistance (IR). On the other hand, overexpression of dimethylarginine dimethylaminohydrolase (DDAH), an enzyme that metabolizes ADMA, in transgenic mice is associated with lower ADMA, increased NO, lower blood pressure and enhanced insulin sensitivity. These animals also have greater endothelial regenerative and angiogenic capacity, and resist vascular lesion formation.

Methods: We hypothesized that an allosteric activator of DDAH might lower plasma ADMA and increase the activity of the NOS pathway, and be useful in treating HTN, IR, and vascular diseases. We developed a high throughput screening (HTS) approach to discover novel small molecules that allosterically activate DDAH. Human DDAH was cloned into an expression vector to generate sufficient amounts of purified recombinant protein for HTS. In parallel, we developed a colorimetric assay compatible with HTS so as to detect enzyme activators by measuring the breakdown of ADMA into citrulline. The primary hits from the biochemical assay were validated using cell-based NO and ADMA assays.

Results: We screened over 132,000 small molecules selected using stringent criteria to maximize diversity and medicinal drug-like

properties. Over 300 compounds gave positive hits. In subsequent validation studies, most of these were found to be false positives, but a small number of compounds were confirmed to be activators. Subsequently, cell based studies indicate that these compounds and their analogs reduce intracellular ADMA levels, and increase cellular NO synthesis.

Conclusions: We have discovered allosteric activators of DDAH using HTS. These activators reduced ADMA levels and increased NO synthesis in endothelial cells. DDAH activators may represent a novel therapeutic approach to the treatment of HTN, IR and other vascular disorders.

Presenter: Yohannes T. Ghebremariam, *Stanford University, Stanford, CA, United States*

Basic Science - Other

Poster 3. Addition of CRP and D-Dimer Enhance the Predictive Value of BNP in Heart Failure

Background: Inflammatory and procoagulant pathways are activated in heart failure. D-dimer and CRP are known to be elevated in dilated cardiomyopathy (DCM). It therefore seemed relevant to study how inclusion of these biomarkers in a panel affected the known predictive value of BNP in the diagnosis of congestive heart failure in dilated cardiomyopathy.

Methods: BNP, CRP, D-dimer, PT, PTT, INR, fibrinogen, thrombin time and platelet count were assessed in eleven controls, eleven well compensated DCM patients and eleven decompensated dilated cardiomyopathy patients (DCM/CHF). For each variable we determined

area under a ROC curve (AUC) using variable level as cutoff between DCM and DCM/CHF. We built a logistic regression (LR) and a support vector machine (SVM) model, using BNP, CRP, D-Dimer: the three variables with the largest AUC.

Results: T-tests reveal that BNP, CRP and D-dimer are the only clinical variables that are significantly different between DCM and DCM/CHF patients. The AUCs for BNP, CRP and D-dimer were 0.81, 0.76, and 0.86, respectively. The AUCs determined using LR and SVM, with BNP and CRP were .9 and .96, and for the three variable models were .89 and .98. The per patient mean BNP and CRP had a Pearson correlation of 0.54 ($p = 0.04$), suggesting that subjects with high BNP tend to have high CRP. Using a multivariate linear regression where we controlled for patient, and removed inter-patient variation, a correlation between BNP and CRP of 0.95 ($p = 0.004$) was noted showing that within a subject an increase in BNP tends to correspond with an increase in CRP.

Conclusions: Addition of CRP and D-dimer to BNP has potential to create a powerful diagnostic biomarker panel and enhance the known predictive values of BNP.

Presenter: Nandini Nair, MD, PhD, *Scott & White Hospital/TAMHSC College of Medicine, Temple, TX, United States*

Poster 4. Effect of Valsartan, Aspirin and Aliskiren on Cholesterol Crystallization and Plaque Rupture

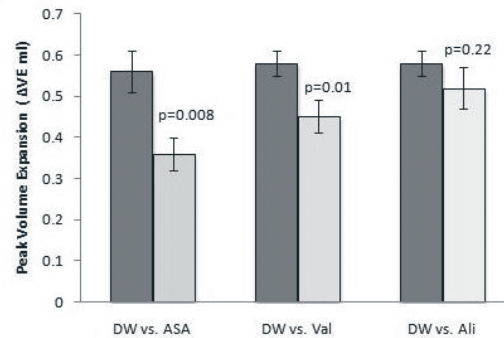
Background: Both angiotensin II receptor blockers (ARBs) and antiplatelet agents are known to be protective of acute cardiovascular events. However, the mechanism for this is unknown. We propose that these agents exhibit their protective effects by decreasing the cholesterol crystal expansion thereby preventing plaque rupture. We also tested the effect of direct renin inhibitors on cholesterol crystallization.

Methods: Normal human plasma concentrations of valsartan (Val: 7 mcg/ml), aspirin (ASA: 0.1 mg/ml) and aliskiren (Ali: 420 ng/ml) were mixed with 3 grams of cholesterol powder in graduated cylinders. The same was repeated using distilled water (DW) as a control. Agent and cholesterol mixture was dissolved by heating. Change in volume expansion was measured for each concentration. Scanning electron microscopy was obtained on human carotid plaques incubated with each agent.

Results: Val and ASA showed significant decrease in cholesterol expansion but not Ali (DW: 0.58 ± 0.10 ml vs. Val: 0.45 ± 0.11 ml, $p=0.01$; ASA: 0.36 ± 0.08 ml, $p=0.008$; Ali: 0.64 ± 0.15 ml, $p=0.22$) (Figure). Scanning electron microscopy demonstrated dissolving of cholesterol crystals mainly with ASA, to a lesser extent with Val and least with Ali.

Conclusions: These data are consistent with known clinical benefits of ASA and ARBs on acute coronary events while Ali is currently unknown. This suggests that the underlying mechanism may

be related to the direct dissolving effect of these agents on cholesterol crystallization.



Presenter: Shaza Khan, MD, Michigan State University, East Lansing, MI, United States

Basic Science - Stem Cells/Tissue Engineering/Regenerative Medicine



Poster 5. Therapeutic Treatment of Critical Limb Ischemia Using Human Induced Pluripotent Stem Cell-Derived Endothelial Cells

Cell-based approaches to restore or regenerate the endothelium so as to enhance angiogenesis hold promise for the treatment of critical limb ischemia. In this study, we examined the therapeutic effect of human induced pluripotent stem cell (iPSC)-derived ECs (iPSC-ECs) for enhancing limb perfusion in a murine model of hindlimb ischemia. Human iPSCs were differentiated towards endothelial lineage in the presence of differentiation media containing BMP4 and VEGF. After 14d, the cells were purified based on the expression of a mature phenotypic EC marker, VE-cadherin, and expanded in vitro. The

iPSC-ECs were characterized for EC phenotype by the expression of known EC phenotypic markers, uptake of acetylated-LDL, and the formation of tube-like structures in matrigel. The iPSC-ECs were lentivirally transduced to enable bioluminescence imaging (BLI) and fluorescence detection. The therapeutic potential of iPSC-ECs was then tested in a murine SCID model of peripheral arterial disease (PAD) in which the cells were intramuscularly delivered by repeated injections on d0 and d7 post-occlusion of the femoral artery (n=11). BLI data showed that iPSC-ECs persisted in the ischemic limb throughout the 14d of assessment. Laser Doppler imaging showed that the mean perfusion ratio was increased by treatment with iPSC-ECs, in comparison to saline. Furthermore, iPSC-ECs significantly enhanced capillary formation. The cells appeared to promote angiogenesis, in part, by producing angiogenic cytokines. This study suggests that iPSC-ECs may be useful in therapeutic strategies to improve ischemic vascular diseases such as PAD. Significant improvement in blood perfusion in the ischemic hindlimb (arrow) after cell transplantation.

Presenter: Ngan F. Huang, PhD, Stanford University, Stanford, CA, United States



Significant improvement in blood perfusion in the ischemic hindlimb (arrow) after cell transplantation

Clinical Science/Epidemiology — Arterial and Aortic Disease

Poster 6. Trends in Utilization of the Ankle-Brachial Index (ABI) in the U.S. Medicare Population: 1998 to 2008

Background: The ankle-brachial index (ABI) has long represented the gold standard for the non-invasive, cost-effective diagnosis of PAD. Evidence of marked under diagnosis of PAD was established in 2001. National evidence-based clinical care guidelines in 2005 defined targeted ABI use as a Class 1B diagnostic tool, followed by an NHLBI supported national PAD public awareness campaign. We assessed the temporal rates of use of physiologic PAD tests in the US Medicare population.

Methods: We analyzed annual Medicare data from a 5% sample of the Part B CMS administrative database. We evaluated annual rates of use of single-level ABI and multi-level segmental pressure (SP) tests to determine trends in utilization of PAD diagnostic tests over the years 1998 to 2002 and 2007 to 2008.

Results: There has been a marked rise in use of ABI and SP diagnostic PAD tests during the eleven year period from 1998 to 2008. Rates of either PAD test increased by 63%, from 131/10,000 in 1998 to 214/10,000 in 2008. The number of individuals having either test rose from 533,940 in 1998 to 1,021,500 in 2008. ABI use has risen faster than SP use over this time period (by 146% and 51%, respectively). The number of ABI or SP tests performed in those tested was similar during the study period (mean 1.3 tests/year). In a Medicare population in which PAD prevalence is 15-20%, in 2008 only 2.1% of this high risk cohort had a PAD diagnostic test performed.

Conclusions: From 1998 through 2008, there was substantial growth in the use of PAD diagnostic testing in the U.S. Medicare population. Despite this increase, these data do not demonstrate “overutilization” as ABI usage rates remain very low compared to the potential Medicare “PAD population at risk.”

Presenter: Sue Duval, PhD, *University of Minnesota, Minneapolis, MN, United States*



Poster 7. Trends in Hospitalization for Lower Extremity Thrombosis: A 20-year (1988-2007) Analysis of Incidence, Mortality, and Procedure Outcomes

Background: Limited data is available regarding the epidemiology of lower extremity thrombosis (LET).

Methods: The National Hospital Discharge Survey (NHDS) database was used for this analysis. ICD-9-CM code 444.22 was used to identify patients with LET. The positive predictive value of this code in identifying patients with LET was 92% at our institution. Descriptive statistics were employed and multivariate regression analysis was performed to find predictors of in-hospital mortality.

Results: Over the twenty year period, there were 1,400,634 cases of LET. The incidence of LET decreased from 44/100,000 cases in 1988 to 15/100,000 in 2007. Similar trends were seen in both males and females. The total case fatality rate (CFR) decreased from 8.32% in 1988-1997 decile to 6.72% in 1998-2007 decile ($p=0.17$). The CFR for males decreased from 7.61% to 5.18%

($p=0.05$) and for females the CFR decreased from 9.14% to 8.46% ($p=0.60$). The overall amputation rate decreased from 10.01% to 8.75% with similar trends in both males and females. Mean length of stay for LET decreased from 10.5 to 8.4 days ($p=0.02$) over the study period. The multivariate regression model showed that acute renal failure (OR 6.1), acute myocardial infarction (OR 4.0), age >65 years (OR 2.7), congestive heart failure (OR 1.6), and female gender (OR 1.2) remained independent predictors of in-hospital mortality. (Figure 1.)

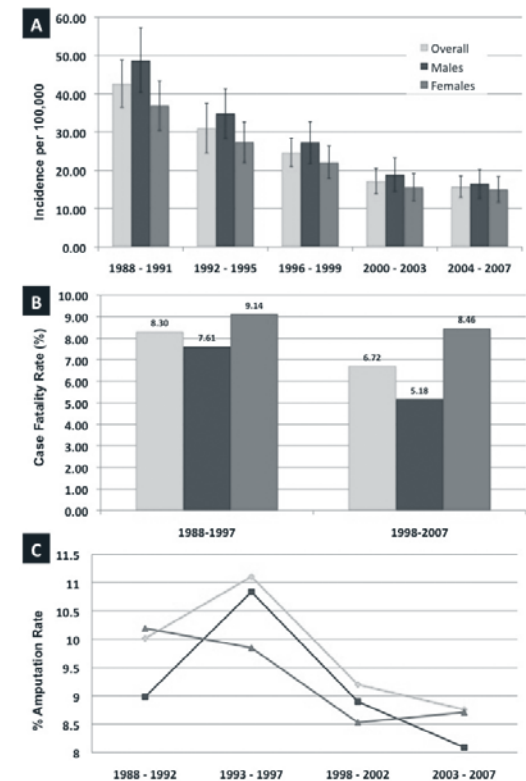


Figure 1: (A) Quintile trends in LET incidence by overall cohort and gender. (B) LET case fatality rates by overall cohort and gender. Decile comparisons for overall cohort ($p=0.17$), male gender ($p=0.05$), and female gender ($p=0.60$). (C) Amputation rates by overall cohort and gender.

Discussion: Over the study period the incidence of admissions with LET has decreased significantly. The in-hospital mortality remained unchanged for females and being female remained an independent predictor of mortality.

Presenter: Ravikiran Korabathina, MD, Tufts Medical Center, Boston, MA, United States

Poster 8. Effect of Socioeconomic Status on Treatment Type and Mortality for Lower Extremity Peripheral Arterial Disease in the United States

Objective: Racial and ethnic disparities in rates of amputations for lower extremity peripheral arterial disease (LE-PAD) have been reported. This study examines the association between socioeconomic status (SES) and treatment type for LE-PAD and in-hospital mortality.

Methods: Hospital discharge data from the Nationwide Inpatient Sample (NIS) for the years 2003-2005 were used. Hospitalizations were identified using ICD-9 diagnosis codes 440.20, 440.21, 440.22, 440.23, 440.24, or 440.29, and ICD-9 procedural codes for amputations and revascularizations (open and endovascular). SES was defined using median household income per patient zip code grouped into quartiles.

Results: Of 896,071 hospitalizations for LE-PAD in subjects >40 years, 30% were in 1st (lowest) income quartile (QL) and 20% in the 4th (highest) income QL. Rates of amputations, open repairs, and endovascular revascularizations varied significantly across income quartiles (chi-square $p < 0.0001$, $p < 0.0001$, and $p = 0.0015$, respectively), with the 1st QL showing higher

rates of major amputations (14% vs 9%) and lower rates of revascularization (open: 22% vs 25%, endovascular: 16% vs 19%) compared to the 4th QL. In multivariable models adjusted for age, gender, race, hospital types, teaching status, and other comorbidities, the 1st QL vs 4th QL showed higher odds of amputation (OR 1.35; 95%CI 1.22-1.49; $p < 0.0001$) and lower odds of open revascularization (OR 0.85; 95%CI 0.77-0.93; $p = 0.0009$). Overall in-hospital mortality was 3.7% (similar in all 4 income QLs). 34% of younger subjects aged 40 to 55, were in the 1st QL (vs 15% in the 4th) and the 1st QL had slightly higher in-hospital mortality compared to the 4th (2% vs 1%). In multivariable model odds of amputation in the younger subset were similar to the overall group.

Conclusions: Lower extremity PAD hospitalizations with low SES have higher odds of major amputations and lower odds of revascularizations both open and endovascular.

Presenter: Hamza Rana, MD, Wake Forest University Baptist Medical Center, Winston-Salem, NC, United States

Poster 10. Serum Bilirubin is Not Associated with Incident Peripheral Artery Disease in the Women’s Health Study

Background: Bilirubin has known anti-oxidant and anti-inflammatory properties, and is currently being considered as a therapy to prevent vascular disease. Several prospective studies have reported an inverse association of bilirubin with coronary

Table: Association between serum bilirubin and incident peripheral artery disease (PAD)

	Quartile 1	Quartile 2	Quartile 3	Quartile 4	
Median total bilirubin levels, mg/dl	0.22 (0.19,0.24)	0.29 (0.28,0.30)	0.35 (0.33,0.38)	0.46 (0.43,0.57)	
Number of cases of PAD	39	22	33	23	
Number of controls	63	54	61	56	
	Odds Ratios (95% confidence intervals) for incident PAD				P for trend
Matched on age and smoking status	1.0	0.65 (0.34-1.24)	0.85 (0.47-1.54)	0.65 (0.33-1.25)	0.3
Multivariable model*	1.0	0.61 (0.30-1.22)	0.86 (0.45-1.66)	0.54 (0.26-1.13)	0.2
	Quartile 1	Quartile 2	Quartile 3	Quartile 4	
Median direct bilirubin levels, mg/dl	0.05 (0.04,0.06)	0.07 (0.07,0.08)	0.10 (0.09,0.11)	0.15 (0.13,0.19)	
Number of cases of PAD	40	29	26	22	
Number of controls	68	68	58	40	
	Odds Ratios (95% confidence intervals) for incident PAD				P for trend
Matched on age and smoking status	1.0	0.72 (0.40-1.30)	0.76 (0.42-1.39)	0.93 (0.48-1.78)	0.7
Multivariable model*	1.0	0.80 (0.42-1.52)	0.73 (0.38-1.41)	0.73 (0.35-1.51)	0.3

*Adjusted for age, smoking status (never, former, current), race, body mass index, exercise frequency, alcohol use, low density lipoprotein cholesterol, high density lipoprotein cholesterol, hormone therapy, history of hypertension, and family history of myocardial infarction

artery disease and stroke. Yet, no prospective data have been available for peripheral artery disease (PAD).

Methods/Results: We assessed the relationship of total and direct bilirubin with incident symptomatic PAD in a prospective, nested case-control study conducted within the Women's Health Study (n=117 cases, n=234 controls, median follow-up 14 years). Median levels of bilirubin were not significantly different among women who developed PAD (cases) compared with those who did not (controls) [0.310 versus 0.315 mg/dl (p=0.5) for total bilirubin and 0.08 versus 0.08 mg/dl (p=0.9) for direct bilirubin]. We observed no significant association between either total bilirubin or direct bilirubin and incident PAD in models matched on age and smoking as well as in multivariable analyses (Table). Adjusted odds ratios (95% confidence intervals) for PAD with increasing quartiles of bilirubin were 1.0, 0.61 (0.30-1.22), 0.86 (0.45-1.66), and 0.54 (0.26-1.13) (p-trend 0.2) for total bilirubin and 1.0, 0.80 (0.42-1.52), 0.73 (0.38-1.41) and 0.73 (0.35-1.51) (p-trend 0.3) for direct bilirubin.

Conclusions: In this population of relatively healthy women, we found no association between baseline bilirubin and incident PAD. Given the interest in therapies aimed at increasing bilirubin levels for systemic atherosclerosis prevention, these data, if confirmed, suggest that the potential benefits of such a strategy may not extend to PAD and that additional longitudinal or clinical trial evidence is needed.

Presenter: Kathryn Britton, MD, *Brigham and Women's Hospital, Boston, MA, United States*

Poster 11. Augmentation Index but not Proximal Aortic Stiffness is Abnormal in BAV Subjects with a Normal Aortic Diameter

Background: The association of ascending aorta dilation with bicuspid aortic valve (BAV) suggests that intrinsic aortic pathology and abnormal stiffness may be present in all BAV subjects regardless of stenosis or regurgitation. To investigate this, we compared aortic stiffness in the setting of BAV to tricuspid aortic valve (TAV) in the absence of aortic dilation.

Methods: Nine BAV subjects (50 ± 14 yr, 7 male) and 13 TAV subjects (44 ± 14 yr, 10 male) were prospectively recruited after echocardiograms (EF $\geq 55\%$). Neither group exhibited severe stenosis or regurgitation. Aortic diameters were similar between groups (p=NS). Characteristic impedance (Z_c) was derived from echocardiographic images and pulse wave Doppler of the left ventricular outflow tract. Applanation tonometry was performed to obtain aortic pulse wave velocity (PWV) and augmentation index (Alx).

Results: There were no differences between BAV and TAV subjects with regard to heart rate, systolic or diastolic blood pressure. Z_c was similar between BAV and TAV subjects (p=NS) as was aortic PWV (p=NS). Carotid Alx was higher in those with BAV than TAV (15.6 ± 13.4 vs -4.0 ± 14.0 %, p=0.004).

Conclusions: Aortic stiffness and impedance are similar between subjects with BAV and TAV with normal aortic dimensions. Thus, ascending aortic stiffness is not always present in BAV despite its association with aortic dilation. The significantly higher carotid Alx in BAV patients may reflect abnormal

arterial stiffness outside of the aorta as Alx is affected by many vascular properties including reflected pressure wave amplitude, location of reflection, arteriolar tone and endothelial function. Future studies should examine diverse arterial stiffness properties in BAV subjects and the potential contribution of increased Alx to ascending aortic dilation.

Presenter: Patrick Warner, *Vascular Function Study Group, Tufts Medical Center, Boston, MA, United States*

Poster 12. Management of Vascular Closure Device Complications

Background: Vascular closure devices (VCD) are frequently used for rapid, reliable hemostasis at the conclusion of arteriography via the common femoral artery (CFA). VCD are rarely associated with complications at the site of vascular access, which are traditionally treated surgically. Percutaneous methods have been described as a means of managing complications associated with VCD, though these typically involve small patient cohorts, short follow up and limited VCD types.

Methods: Complications of VCD application over a 6-year period at two institutions were reviewed. Baseline patient characteristics were collected, as were type of VCD, symptoms, methods of revascularization and follow up.

Results: 24 patients were identified as having vascular complications resulting from VCD, including Angioseal, Perclose and Starclose. 20 of the 24 lesions occurred in the CFA, and the remaining 5 in the external iliac artery (EIA). Four patients were managed surgically (two with infection and abscess) and the remaining

20 patients treated percutaneously. Stents were deployed in seven patients; the remaining 13 were treated with angioplasty, thrombectomy, or a combination of the two. Among the 20 patients managed percutaneously over a median follow-up of 24 months (maximum 67 months), one required repeat revascularization. Among surgically treated patients, none required reintervention or had recurrent symptoms over a median 3 months. All percutaneously treated patients remain symptom-free at last follow up.

Conclusions: Percutaneous management of non-infectious complications associated with VCD application is safe, effective and durable, has similar outcomes to surgery and could be considered first line therapy.

Presenter: Jeffrey M. Sparling, MD, *Brigham and Women's Hospital, Boston, MA, United States*

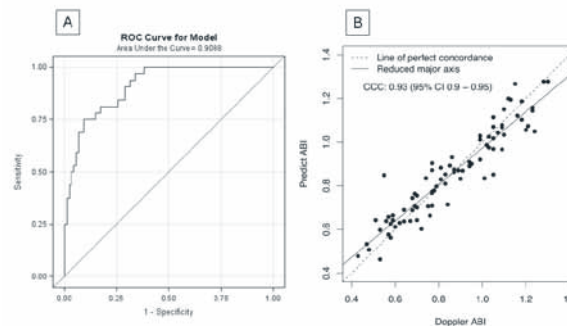
Poster 13. Novel Oscillometric-based Algorithm for Diagnosis of Peripheral Artery Disease (PAD) and Determination of the Ankle-Brachial Index (ABI)

Background: Doppler-based ABI is the diagnostic standard for PAD, but barriers have limited broad detection efforts. Prior studies using oscillometric (OSC) blood pressure (BP) devices for PAD diagnosis have demonstrated limited accuracy, possibly due to application of physiologic assumptions for OSC arm BP measurement (i.e., characteristic ratio, CR) to diseased legs. We developed an oscillometric-based algorithm for PAD diagnosis.

Methods: Patients underwent Doppler BP measurements in the arms and ankles while

simultaneously recording oscillometric waveforms. Spectral analysis using a fast Fourier transform (FFT) was applied to OSC waveforms. Logistic regression of FFT amplitude classified OSC leg waveforms: 1. normal/mild PAD (Doppler ABI > 0.6) or 2. moderate/severe PAD (Doppler ABI < 0.6). Linear regression determined CRs for OSC BP at the ankles.

Results: 60 patients were studied; 68.3% had Doppler ABI < 0.9. Peak FFT amplitude threshold of < 0.7333 accurately classified legs as moderate/severe PAD (Panel A; AUC=0.9088). Optimal CR for normal/mild PAD=0.5698 and moderate/severe PAD=0.7033. Using the CRs, OSC ankle BPs were calculated and OSC ABI determined. Agreement of OSC ABI and Doppler ABI was high (Panel B; concordance correlation coeff.=0.93, 95% CI 0.90-0.95); there was no significant difference between the measures (mean Δ =0.0055+ 0.08, P=0.53). **Conclusions:** We developed an algorithm for diagnosing PAD and determining ABI based on FFT analysis of oscillometric waveforms that will be further validated clinically.



Presenter: Heather L. Gornik, MD, MHS, FSVM, *The Cleveland Clinic Foundation, Cleveland, OH, United States*

Poster 14. Clot Strength by Thromboelastography is Associated With Severity Of Peripheral Arterial Disease

Background: Peripheral arterial disease (PAD) is characterized by increased pro-thrombotic state attributable to platelet activation and aggregation. Degree of platelet contribution to clot strength measured using thromboelastography with maximum amplitude (MA) and time to clot formation (R) hasn't been correlated with severity of symptomatic PAD and presence of diabetes.

Methods: We studied symptomatic PAD pts enrolled in an exercise program. Severity of PAD was assessed using: Ankle brachial index (ABI), maximal walking time (seconds), daily ambulatory activity(steps/day), claudication onset time (seconds), peak oxygen consumption and walking impairment questionnaire (WIQ) % scores for walking distance, walking speed and stair climbing. A Spearman's rank correlation coefficient and non-parametric Wilcoxon rank sum statistical tests were used.

Results: 25 symptomatic PAD pts (56% males, 52% smokers, 48% diabetics and 76% hypertension) were included. Mean age was 63±10 years. Mean ankle brachial index was 0.70±0.19. Mean claudication onset time (seconds), maximal walking time (seconds), peak oxygen uptake (ml/kg/min), daily ambulatory activity (steps/day), and WIQ climbing score were 165.72±149.45, 328.68±195.26, 12.3±3, 3378±1642 and 37.26±20.6 respectively. Mean MA was 65.2±6.3 and mean R was 7.9±2.6. MA correlated with

peak oxygen uptake ($r = -0.67$, $p = 0.0004$), WIQ distance score ($r = -0.47$, $p = 0.01$) and climbing score ($r = -0.69$, $p = 0.0001$) and daily ambulatory activity (steps/day) ($r = -0.42$, $p = 0.04$), whereas R correlated with WIQ speed score ($r = -0.41$, $p = 0.04$). The median MA and R were similar in diabetics and non-diabetics ($p = \text{NS}$).

Conclusions: In symptomatic PAD pts, platelet contribution to clot strength correlates with measures of severity of PAD such as impaired exercise performance and reduced daily ambulatory activity.

Presenter: Tarun Dasari, MD, MPH, *University of Oklahoma Health Sciences Center, Oklahoma City, OK, United States*

Poster 15. Effect of Uremia on Arterio-venous Fistula Maturation and Primary Patency

Background: Arterio-venous fistulas (AVF) are used for hemodialysis (HD) in end stage renal disease (ESRD). Loss of patency remains a significant problem. Literature suggests uremia as a contributing. AVFs are also used for low-density lipoprotein (LDL) apheresis in patients with familial hypercholesterolemia (FH). We describe HD AVF outcomes by comparing to them non-uremic controls with FH.

Methods: Patients with AVFs for LDL apheresis were identified as cases by chart review. Age, gender and fistula location matched controls were sought from a database of patients undergoing HD. The two groups were compared for overall survival, AVF maturation and loss of primary patency.

Results: AVF's were placed for LDL apheresis in 8 patients [(2 female) mean age of 59.3 ± 13.4 yrs]. The mean LDL level was 300 ± 85.1 mg/dL and mean

serum creatinine was 1.32 ± 0.44 mg/dL. Mean follow-up was 282.3 ± 137 days. There were no deaths in the LDL apheresis group while four patients died in the HD group. There was no significant difference in time for maturation and primary patency rates for AVFs between both groups.

Conclusions: Based on our study uremia did not adversely affect maturation or primary patency of AVFs. Individuals undergoing HD have higher mortality rate than similar individuals undergoing LDL apheresis. A larger, prospective study is required to substantiate our result.

Presenter: Rajmony Pannu, *Mayo Clinic, Rochester, MN, United States*

Poster 16. Clostridium Septicum Aortitis

Background: An 84 year-old male with history of coronary artery disease was hospitalized for infectious diarrhea. Computed tomography of the abdomen revealed a thickening of the distal ileum and multiple enlarged ileocolic lymph nodes. At that time he refused colonoscopy for further evaluation and so was treated empirically with ciprofloxacin and metronidazole and dismissed in stable condition. Soon after finishing his antibiotic regimen he presented to the emergency department with abdominal pain, fever, chills and hypotension. A computed tomography of the abdomen revealed new mural gas extending from the distal descending aorta to the infrarenal aorta with associated periaortic fat stranding concerning for bacterial aortitis. Blood cultures grew *Clostridium septicum*.

The patient declined surgical intervention. He initially accepted intravenous (IV) antibiotics, but ultimately chose to go to hospice care with oral antibiotics.

Infectious aortitis due to *Clostridium septicum* is a very rare and lethal condition. Most cases have been reported in patients with ileocecal tumors. Prompt treatment with IV antibiotics is necessary. Aortic resection should be considered in all patients due to high risk of aneurismal formation and rupture.

References:

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Presenter: Fadi Shamoun, MD, *Mayo Clinic Arizona, Phoenix, AZ, United States*

Poster 17. Correlates of ABI Change in Persons With and Without Peripheral Arterial Disease

Background: The ankle-brachial index (ABI) is a well validated and reproducible method of detecting and quantifying peripheral arterial disease (PAD). Abnormal ABI values both low ($< .90$) and high (> 1.40) are associated with worse cardiovascular disease outcomes, but the clinical characteristics associated with changes in ABI are not well characterized. We determined what characteristics

are associated with a change in ABI of 0.15, a clinically meaningful change.

Methods: Participants included 700 persons with PAD and 422 participants without PAD identified from non-invasive vascular laboratories and a large internal medicine practice in Chicago. ABI was measured using established methods. Results: In analyses adjusting for age, sex, race, smoking, and presence of diabetes among persons with PAD, older age (odds ratio (OR) 0.97, 95% confidence interval (CI) 0.95-1.00), current smoking status (OR 0.57, 95% CI 0.32-1.00), and higher baseline ABI values (OR 0.82, 95% CI 0.72-0.94) were inversely significantly associated with increases in ABI >0.15 over up to 8-year follow-up. Sex, race, and presence of diabetes were not significantly associated with increases in ABI. Among persons without PAD, only baseline ABI (OR 0.69, 95% CI 0.51-0.94) was inversely and significantly associated with an increase in ABI. Among persons with PAD, higher baseline ABI (OR 1.39, 95% CI 1.21-1.60) was significantly associated with a decrease in ABI >0.15. In persons without PAD, presence of diabetes (OR 2.10, 95% CI 1.26-3.52) and higher baseline ABI (OR 1.47, 95% CI 1.18-1.83) were significantly associated with decreases in ABI >0.15.

Conclusions: Among persons with and without PAD, lower baseline ABI levels were most consistently associated with increases in the ABI over time. Higher baseline values were most consistently associated with decreases in the ABI over time. There is the possibility that regression to the mean may account for some of the tendency for lower ABIs to increase and higher ABIs to decrease.

Presenter: Natalie S. Evans, MD, *Northwestern University Feinberg School of Medicine, Chicago, IL, United States*

Clinical Science/Epidemiology – Cerebrovascular Disease and Stroke

Poster 18. Distribution of Stroke Risk Factors Among Trial Participants and Urban Blacks with Atrial Fibrillation

Background: Atrial fibrillation (AF) is a potent risk factor for stroke. Current risk stratification schemes and treatment guidelines for stroke prevention are based on trial populations. Randomized trials and published prospective cohorts have included few racial/ethnic minorities (Mayo Clinic-3% black, Framingham-0% black, National Registry of AF-4% black, Kaiser-4% black, SPORTIF III/V-0% black). The validity of extrapolating stroke risk and stroke rates to nonwhite populations remains in question. We sought to define the distribution of stroke risk factors among an urban population with AF with relatively high representation of African Americans and compare it to that of the RE-LY and other landmark RCTs.

Methods: Patients with AF were enrolled from January 2007 to December 2009 from the Boston Medical Center anticoagulation clinic. Demographic data and clinical characteristics were derived from chart review. Stroke risk scores were calculated and compared using the CHADS2 criteria with scores ≥ 3 denoting high risk.

Results: Of 527 AF patients identified, 253 (48%) were white and 196 (37%) were black; mean age 72. The burden of stroke risk factors was significantly greater among BMC urban blacks compared to RE-LY participants (HTN-93% v 79%,

HF-56% v 32%, DM-38% v 23%, CHADS2 score ≥ 3 -48% v 33%). Urban blacks with AF also had a higher prevalence of HTN, HF, and DM compared to urban whites.

Conclusions: Individuals at highest risk for stroke are grossly underrepresented in clinical trials of AF. Concerted efforts are needed to ensure the efficacy and safety of novel therapies in these high risk patient subgroups.

Presenter: Christina L. Cove, MD, *Boston University Medical Center, Boston, MA, United States*

Poster 19. Cerebrovascular Steal Phenomenon in a Patient undergoing Dipyridamole Nuclear Perfusion Cardiac Imaging

Background: Transient ischemic neurological deficits following intravenous dipyridamole administration during pharmacological cardiac stress test is a seldom reported, but serious adverse event. Introduction: Safety of dipyridamole use in the nuclear perfusion cardiac imaging has been well documented. We report a case of transient ischemic attack seen after dipyridamole administration. Case description: 58 year old Caucasian male with high risk cardiovascular disease including coronary artery disease, bilateral carotid disease, peripheral arterial disease and paroxysmal atrial fibrillation was scheduled for an elective dipyridamole-technetium cardiac stress test. After the standard dipyridamole infusion, he developed expressive aphasia and generalized weakness without any focal motor deficits. His blood pressure was 126/80 mmHg with heart rate of 74/min, in sinus rhythm. Intravenous aminophylline was administered to

reverse the effect of dipyridamole. This improved the generalized weakness; expressive aphasia persisted. Basic laboratories were normal and the INR was 1.9. Noncontrast CT scan of the brain showed no intracranial bleed. CT angiogram of the neck revealed a known total occlusion of the right internal carotid artery and 50% stenosis of the left internal carotid artery. His aphasia spontaneously resolved within 24 hours and he was discharged home.

Conclusion: Similar to coronary steal phenomenon, intravenous dipyridamole may result in cerebrovascular steal causing transient or permanent neurologic dysfunction in patients with significant carotid artery stenosis. We suggest caution to the use of intravenous dipyridamole in patients with impaired intracerebral autoregulation or significant carotid artery disease.

Presenter: Manjunath Raju, MD, Michigan State University, East Lansing, MI, United States

Clinical Science/Epidemiology – Endothelial Function and Surrogate Markers



Poster 20. Acute exposure to diesel particle matter impairs NO-mediated microvascular function

Background: Exposure to diesel Particle Matter (PM) was recently identified as an important cardiovascular risk factor. Whether diesel PM exerts acute specific deleterious effects on arterial stiffness, aortic wave reflection and endothelial function are not known.

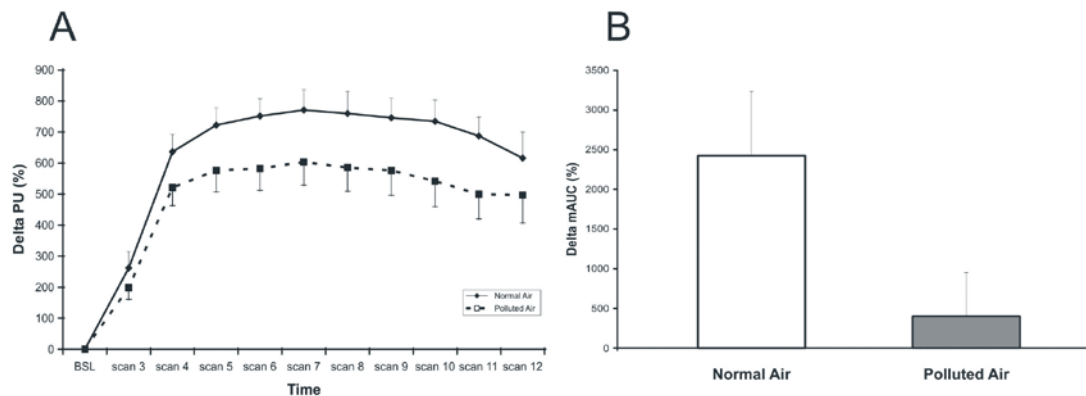
Methods: We tested these hypotheses in a randomized, crossover study design in 13 healthy male. The effects of 2 hours exposure to diesel PM, as compared with normal air, on skin microvascular hyperemia to local heating and iontophoresis of acetylcholine (Ach) and sodium nitroprussiate (SNP), were examined using Laser Doppler Imager system. Before local heating, skin was pre-treated either by an iontophoresis of specific NO synthase inhibitor (L-NAME) or by saline solution (Control). Pulse wave velocity (PWV) and aortic augmentation index (Aix) were also evaluated. Diesel PM exposure was performed in computer-assisted inhalation room, controlling pollutants emitted by motor engine.

Results: The PM_{<2.5} mean concentration was 10.01±0.08µg/m³ on normal air and 127.9±2.8µg/m³ on polluted air (p<0.001). Acute diesel PM exposure increased systolic BP (p<0.05) but had no effect on PWV and Aix. Compared to ambient air, diesel PM exposure reduced skin vasodilatation induced by Ach (p<0.05) (fig.1A),

but did not affect vasodilatation induced by SNP or local heating. However, NO-mediated vasodilatation, assessed by the skin thermal hyperemia difference between control and L-NAME sites; decreased from 2423±809% to 400±552% (p<0.05) (fig.1B) after diesel PM exposure.

Conclusions: In healthy subjects, acute experimental diesel PM exposure, at a level usually encountered during city's pollution peak, impairs microvascular endothelial mediated vasodilatation throughout a decrease in NO bioavailability.

Presenter: Aurelien Wauters, MD, Department of Cardiology, Hospital Erasme, ULB, Brussels, Belgium



Poster 21. The Effects of Resveratrol on Endothelial Function in Patients with Type 2 Diabetes Mellitus

Resveratrol, a polyphenolic compound found in grapes and red wine, improves endothelial function in experimental models of diabetes and obese human subjects. The beneficial effects of resveratrol may be due, in part, to decreased oxidative stress. We completed an open label pilot study to examine the effects of acute and chronic supplementation with two doses of resveratrol (resVida®, DSM Nutritional Products, Kaiseraugst, Switzerland) (90mg/day and 270mg/day for one week each) in 19 patients with Type 2 diabetes mellitus. Plasma levels of resveratrol and metabolites, vascular function, and levels of reactive oxygen species were measured at baseline, one and two hours after acute administration of each dose and after one week of chronic treatment with each dose. Total resveratrol and metabolite levels in plasma increased significantly following acute low-dose ($P=0.002$), acute high-dose ($P<0.001$) and chronic treatment ($P<0.001$). Endothelium-dependent brachial artery flow-mediated dilation measured by high resolution ultrasound, improved significantly with acute high-dose treatment ($P=0.03$) but did not change after acute low-dose treatment or chronic treatment. In peripheral blood mononuclear cells, there was a trend for reduced extracellular hydrogen peroxide levels assessed as Amplex Red fluorescence following chronic high-dose treatment ($P=0.055$). Our pilot study shows that resveratrol (resVida®) improves endothelial function following acute treatment in patients with diabetes mellitus. Resveratrol could have favorable effect on cardiovascular disease risk in patients with diabetes mellitus via this mechanism.

Presenter: Alissa A. Frame, Boston University, Boston, MA, United States

Poster 22. Correlation of Clinical Parameters with Echocardiographic Indices of Diastolic Dysfunction

Background: Four stages of diastolic dysfunction (DD) with increasing severity identified by echocardiography are graded I to IV with grade IV being the most severe. DD is associated with multiple etiologies such as hypertension, ischemic heart disease, older age, obesity and diabetes.

Methods: 400 patients with diastolic dysfunction (stages 1-4) were selected based on echocardiographic parameters of diastolic dysfunction from the echo database. Clinical data on these patients was collected from electronic medical records. Pearson correlations were studied between markers of cardiovascular risk factors such as diabetes, renal failure and lipid profiles and echocardiographic indices of diastolic dysfunction.

Results: Statistically significant but weak positive correlations were noted between serum glucose levels and E velocity, E/Vp and Septal thickness ($p<0.0001$, $p=0.0085$, $p=0.0204$ respectively). Weak negative correlations were noted between serum LDL levels and deceleration time, ($p=0.0133$). Positive but weak correlations were noted between serum creatinine levels and E velocity, left ventricular diastolic diameter, left atrial volume, left atrial diameter, septal and posterior wall thickness ($p=0.0122$, $p=0.0129$, $p=0.0083$, $p=0.0009$, $p=0.0008$, $p=0.0138$ respectively).

Conclusions: The Pearson correlations range from 0-0.2 and hence are weak correlations. However, these results suggest that clinical markers

of cardiovascular risk factors such as diabetes, renal dysfunction and dyslipidemia correlate with echocardiographic parameters of diastolic dysfunction.

Presenter: Nandini Nair, MD, PhD, Scott & White Hospital/TAMHSC College of Medicine, Temple, TX, United States

Clinical Science/Epidemiology – Exercise Physiology

Poster 23. Efficacy and tolerability of the novel fast skeletal muscle troponin activator, CK-2017357, in patients with claudication

Background: CK-2017357 (CK-357), a novel small molecule activator of the fast skeletal muscle troponin complex, slows the rate of calcium release from troponin, resulting in sensitization of fast skeletal muscle fibers to calcium. In preclinical studies, CK-357 increased muscle force and delayed the onset and reduced the extent of muscle fatigue under conditions of hypoxia and muscle ischemia. The present study was designed to evaluate the effect of CK-357 on measures of endurance/fatigue, walking capacity, and tolerability in patients with claudication.

Methods: Patients received single doses of 375 or 750 mg CK-357 or placebo in random order with a 6-10 day wash out between doses. The protocol was amended after 33 patients were dosed due to adverse events reported by 2 patients at 750 mg; the remainder enrolled received a maximum dose of 500 mg. Assessments of muscle function, fatigue, and claudication-limited exercise performance included a novel bilateral heel raise protocol and the six-minute walk test. The number of heel raises, time to onset of claudication, time to intolerable claudication pain or

maximal calf muscle fatigue, total muscle work and distance travelled were assessed. Pharmacokinetic and pharmacodynamic relationships, safety and tolerability were also evaluated.

Results: As of Dec 1st, 36 of the target 54 patients have been enrolled. Final results will be available at presentation.

Conclusions: CK-357 is being investigated as new therapy for improving muscle function and endurance in patients with claudication. CK-357 may benefit these patients by increasing the amount of muscle work performed before reaching symptom-limited exercise tolerance.

Presenter: William R. Hiatt, MD, MSVM, CPC, CPC
Clinical Research, Aurora, CO, United States

Poster 24. Pilot Study of Whether a Walking Intervention Reduces Inflammation in Patients with Diabetes and Peripheral Arterial Disease (PAD)

Hypothesis: The walking intervention will reduce systemic inflammation after 6 months.

Methods: In a larger randomized trial comparing a walking intervention vs. attention control in persons with diabetes and PAD (ankle-brachial index <0.9), we obtained blood samples from 55 consecutive participants (25 control, 30 intervention). Exercise behaviors and walking ability were assessed at baseline and 6 months. Statistical analyses used linear regression.

Results/findings: Among 55 participants (42 men, 13 women; mean age 66.7 years [SD 10.7]), baseline median biomarker levels were soluble intercellular adhesion molecule 245.94,

interleukin-6 3.18, soluble vascular cell adhesion molecule 828.63, monocyte chemoattractant protein-1 414.09, B2-microglobulin 2.80, total cholesterol 168.00, triglycerides 174.00, HDL 40.00, LDL 81.00, CRP 2.82. After 6 months, average change in intervention minus average change in control was: sICAM -5.15 (SE 11.58), VCAM -29.62 (SE 48.99), total cholesterol -9.77 (SE 6.85), triglycerides -10.90 (SE 34.53), HDL -3.61 (SE 2.09), LDL -1.67 (SE 5.83), CRP 0.19 (SE 1.41), $P \geq 0.09$ for all. For every 1 SD change in area under the curve for treadmill walking distance, average changes were: ICAM -1.81 (SE 5.63); VCAM -11.17 (SD 23.85), LDL -0.95 (SE 2.77), and HDL -0.57 (SE 1.04), $P > 0.20$ for all. Changes in biomarkers were similar using as predictors other measures of activity and walking ability including the Walking Impairment Questionnaire and Exercise Behaviors Survey.

Conclusions: These preliminary results suggest that exercise may improve ICAM, VCAM, total cholesterol, triglycerides, and LDL in persons with diabetes and PAD, but surprisingly not HDL.

Presenter: Tracie C. Collins, MD, MPH, *University of Minnesota, Minneapolis, MN, United States*

Poster 25. Bilateral Heel Raise Test: A Novel Functional Endpoint for Early Stage Clinical Trials in Peripheral Artery Disease (PAD)

Background: Patients with peripheral artery disease (PAD) and claudication experience reproducible symptoms of leg pain during walking exercise. The symptom of claudication is due to exercise-induced ischemia of the muscles in the legs, most commonly in the calf muscles, limiting

both walking distance and functional exercise capacity. Peak exercise performance during a standardized graded treadmill test is often used as the primary endpoint in clinical trials. The present report describes the experience and reproducibility of a novel heel raise test of muscle function and claudication-limited exercise performance in patients with PAD and claudication.

Methods: In a multi-center trial, 18 patients performed standing bilateral heel raises at 0.5 Hz until reaching calf muscle fatigue at three visits separated by 1 week. Ankle dorsiflexion was monitored and recorded using a wireless goniometer. The number of heel raises, total work performed, time to the onset of claudication, and maximal exercise time were assessed.

Results: All patients (mean age: 65.7 ± 7.7 yrs) experienced claudication symptoms during heel raise testing. The mean number of heel raises to maximal claudication at baseline was 91.6 ± 48.5 . Intra-class correlations by patient were 0.64 (CI: 0.39-0.80), 0.63 (CI: 0.37-0.80), and 0.77 (CI: 0.56-0.89) for heel raises, time, and total work, respectively.

Conclusion: The bilateral heel raise test is a simple, specific, and reliable method for assessing muscle performance in patients with claudication. The use of the heel raise test in early phase "proof of concept" clinical trials may facilitate demonstration of clinical efficacy in patients with claudication without requiring treadmill procedures.

Presenter: William R. Hiatt, MD, MSVM, *University of Colorado Denver Anschutz Medical Campus, Denver, CO, United States*

Poster 26. Functional Capacity but not ABI Predicts All-cause Mortality

Background: PAD is associated with significant cardiovascular morbidity and mortality. We examined the association between ankle brachial index (ABI), total cardiovascular fitness (functional capacity), and all-cause mortality.

Method: From 01/01/2000 to 01/01/2009, 1560 underwent a symptom limited cardiac exercise stress test and had an ABI study available within 6 months. Functional capacity in metabolic equivalents (METs) was obtained from exercise stress testing and adjusted for age and gender. Pearson correlation was used to assess the association between ABI and functional capacity. We used Cox proportional Hazard models to examine the association between ABI and mortality with and without functional capacity.

Result: Of 1560 patients, 309 (20%) had an ABI ≤ 0.9 . There was no correlation between ABI and functional capacity ($r=0.013$, $p=0.6$). Further

stratification based on age and gender failed to show a significant association between ABI and functional capacity (Figure 1). In the adjusted analysis, ABI was not a predictor ($P>0.2$) while functional capacity remained highly associated ($p<0.0001$) with all-cause mortality.

Conclusion: In a group of patients referred for exercise stress testing, no significant association was seen between ABI and functional capacity. Furthermore, ABI was not predictive of all-cause mortality after adjustment for functional capacity. These data is consistent with previously published association between ABI, 6-minute walk test, and all-cause mortality.

Presenter: Rayan Yousefzai, MD, *Cleveland Clinic, Cleveland, OH, United States*

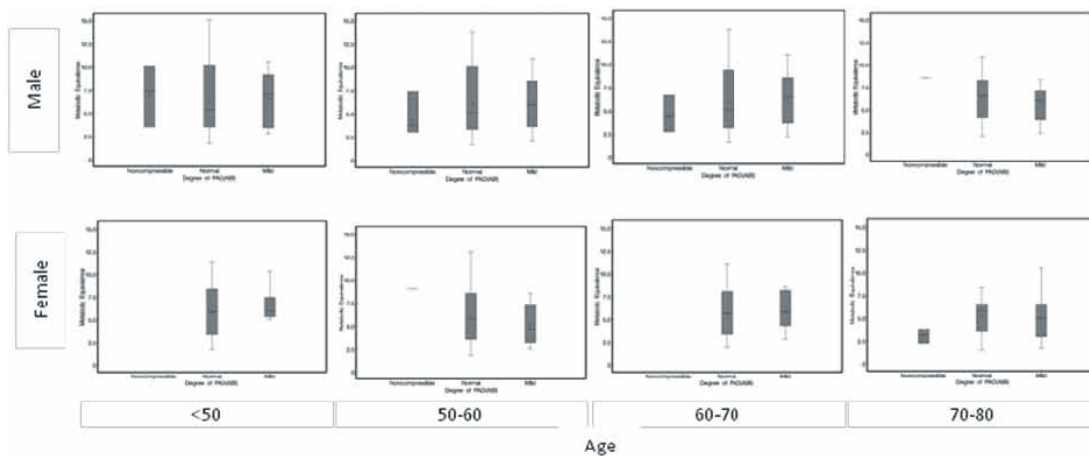


Figure 1. Association between ABI and functional capacity stratified by age and gender.

Clinical Science/Epidemiology — Imaging



Poster 27. Improvement in Calf Muscle Energetics After Percutaneous Intervention in Patients with Symptomatic Peripheral Arterial Disease as Measured with MRI

Background: Prior studies demonstrate improved functional capacity in patients with symptomatic claudication and peripheral arterial disease (PAD) treated with percutaneous intervention of the affected lower extremity artery. We hypothesized that percutaneous intervention would improve calf muscle perfusion and cellular metabolism in PAD as measured by magnetic resonance (MRI) imaging and spectroscopy (MRS).

Methods: 10 patients with symptomatic PAD (mean±S.D. age 57 ± 9 years, ABI 0.62 ± 0.17 , 7 males) were studied before (62 ± 74 days) and (317 ± 98 days) after study leg percutaneous intervention (stenting in iliac ($n=5$), superficial femoral ($n=3$), distal aorta ($n=1$) and 1 superficial femoral angioplasty). Calf muscle phosphocreatine recovery time (PCr) was measured by ^{31}P MRS immediately after symptom-limited exercise on a 1.5T scanner. Calf muscle perfusion was measured using first-pass gadolinium-enhanced MRI at peak exercise. Six minute walk and treadmill with peak V_{O2} were performed. Paired t-test compared changes.

Results: Calf muscle energetics improved significantly following intervention ($91\pm 33s$ to $52\pm 34s$, $p < 0.003$). Rest and post-exercise ABI also improved (0.62 ± 0.17 to 0.93 ± 0.25 , $p < 0.002$)

and 0.33 ± 0.15 to 0.67 ± 0.37 , $p < 0.02$). Despite this, there was no difference in perfusion or exercise parameters (see Table).

Conclusions: Percutaneous intervention in patients with symptomatic PAD is effective at improving calf muscle energetics, suggesting an improvement in mitochondrial function. However, neither tissue perfusion nor exercise parameters improved. Thus, large vessel revascularization improves energetics but not microvascular perfusion, underscoring the need for additional therapies aimed at the microvasculature.

Table. Changes in metabolism, perfusion, and exercise parameters before and after percutaneous stenting (mean \pm SD)

	Pre-intervention	Post-intervention	p-value
PCr, sec	91 \pm 33	52 \pm 34	<0.003
Perfusion index	0.52 \pm 0.18	0.57 \pm 0.17	NS
6 minute walk distance, ft	931 \pm 256	1104 \pm 242	NS
Treadmill time, sec	579 \pm 303	599 \pm 310	NS
Rest ABI	0.62 \pm 0.17	0.93 \pm 0.25	<0.002
Post-exercise ABI	0.33 \pm 0.15	0.67 \pm 0.37	<0.02
V02 peak, ml/kg/min	11.6 \pm 2.5	12.5 \pm 3.1	NS

Presenter: Amy M. West, MD, *University of Virginia Health System, Charlottesville, VA, United States*

Poster 28. Reproducibility of Image Quality Assessment and of Longitudinal Comparisons in the Effectiveness of Intensive Lipid Modification in Preventing the Progression of Peripheral Arterial Disease (ELIMIT) Study

Background: Accurate image quality assessment and co-registration of magnetic resonance image (MRI) scans is important for MRI-based peripheral artery disease [PAD] studies. Therefore, we evaluated an image quality scoring method and co-registration with 3 distinct landmark types in longitudinal proton density weighted [PDW] MRI series of the superficial femoral artery [SFA] (echo time= \sim 30 ms, repetition time=2600 ms).

Methods: Bilateral PDW SFA MRI scans were acquired (3.0T GE EXCITE) in 15 participants at 0, 6, 12 and 24 months. For 12 of the 15 patients, 2 readers, blinded to timepoint, rated series (n=216 scans) from 1 to 4 with 4 criteria: sharpness, blurring, artifacts and noise. Lumen and wall volumes were obtained 2 times by 1 reader for high (mean quality score[QS]=4.0) and low (mean QS=1.6) quality subsets (n=5 scans) to evaluate quality scoring validity. Scans were registered by identifying the best anatomical landmark (artery, vein or muscle) between series. Series were then registered again using only artery or vein or muscle and results were compared with original registration to assess co-registration with each landmark type.

Results: Interreader correlation of quality scoring was excellent (intraclass correlation coefficient [ICC] =0.769.) Lumen and wall volume repeatability was excellent for high quality scans (ICC=0.98 & 0.98) but poor for low quality scans (ICC =-0.14 & 0.57). Intra-reader correlation comparing registration was high for all landmark types (ICC artery 0.966, ICC

vein = 0.974, ICC muscle 0.948).

Conclusions: Our results provide a reliable quality scoring system and registration method that could be used in PAD studies that use MRI as an end point.

Presenter: Tyler O Murray, BA, *Baylor College of Medicine, Houston, TX, United States*

Poster 30. Lower Calf Muscle Density is Associated With Mobility Loss and Mortality in Peripheral Arterial Disease

Background: Men and women with lower extremity peripheral arterial disease (PAD) have adverse calf muscle characteristics, compared to people without PAD. Associations of calf muscle density with mobility loss and mortality are not well established in patients with PAD.

Methods: 418 participants with PAD underwent baseline measurement of calf muscle density using computed tomography. Participants were followed annually for up to four years. The outcome of mobility loss was defined as becoming unable to walk ¼ mile or walk up and down one flight of stairs without assistance, among those without baseline mobility limitations. Cause of death during follow-up was ascertained using death certificates. Results below adjust for age, sex, race, body mass index, the ankle brachial index, smoking, physical activity, and comorbidities. Comorbidities were assessed and confirmed with medical record review and a primary care physician questionnaire and included diabetes, angina, myocardial infarction, heart failure, knee arthritis, hip arthritis, spinal disk disease, pulmonary disease, and cancer.

Results: See Table 1.

Table 1. Adjusted hazard ratios for associations of calf muscle density tertiles with adverse outcomes in PAD (N=418)*

	Tertile 1 (lowest calf muscle density)	Tertile 2	Tertile 3 (highest calf muscle density)	P Trend
All-Cause Mortality	1.69 (1.00-2.87)	0.62 (0.34-1.16)	1.00 (reference)	0.015
Cardiovascular Disease Mortality	3.44 (1.09-10.82)	0.89 (0.24-3.23)	1.00 (reference)	0.007
Mobility Loss	1.85 (0.95-3.59)	0.84 (0.42-1.69)	1.00 (reference)	0.025

*Data shown are hazard ratios and 95% Confidence Intervals.

Conclusions: Lower calf muscle density is associated with higher rates of mobility loss and mortality in people with PAD.

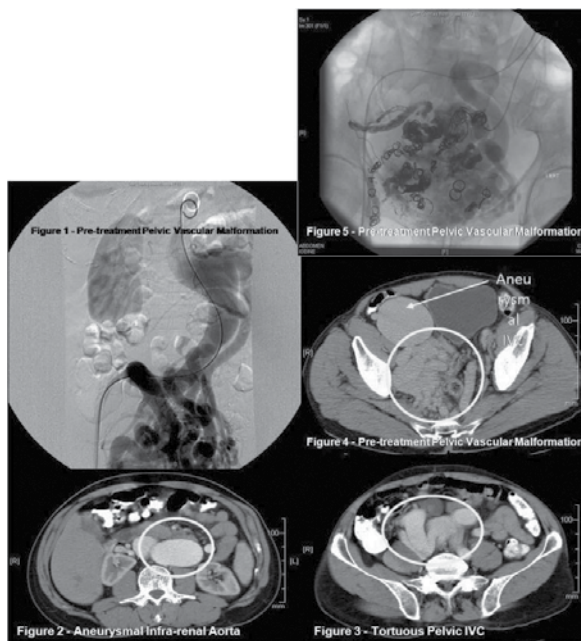
Presenter: Mary M. McDermott, MD, *Northwestern University's Feinberg School of Medicine, Chicago, IL, United States*

Poster 31. Endovascular Management of a Giant Abdominal-Pelvic Vascular Malformation

Background: Pelvic vascular malformations (VM) are rare and present a difficult therapeutic challenge especially when associated with high-output cardiac failure.

Case Report: 59 year old man, history of atrial fibrillation and hypertension, presented with symptomatic heart failure: dyspnea on exertion. Work up revealed giant abdominal-pelvic VM involving aorta, IMA, iliac vessels and IVC (Figure 1-4). Staged embolizations with coils and cyanoacrylate glue were performed over a 3 month period. Therapeutic goals were symptomatic relief by VM flow reduction while maintaining major feeding vessels for access and abdominal (gut) and pelvic circulations.

Results: After serial treatments spanning 3 months, the patient's symptoms and VM appearance on angiography (Figure 5) improved. There were no procedure related complications. At three-year follow up, the patient is symptom free and there is no change on follow up angiography. Echocardiography demonstrates EF 66% with minimal ventricle dilatation, improved from



presenting cardiac failure (EF 45%, moderate-to-severe RV and LV enlargement).

Conclusion: Most pelvic VM are difficult to eradicate and the goal remains symptom relief. This case demonstrates that aggressive multi-modality trans-catheter techniques attenuate even giant, complex abdominal-pelvic VM, and resolve high-output cardiac failure.

Presenter: Christopher J. Smolock, MD, *The Methodist Hospital, DeBakey Heart and Vascular Center, Houston, TX, United States*

Clinical Science/Epidemiology — Thrombosis and Hemostasis

Poster 32. The Utility of 18F-FDG-PET/CT for the Diagnosis of Occult Malignancy in Patients with Acute, Unprovoked Venous Thromboembolism



Background: An association between VTE and occult malignancy has been observed for decades and 7-10% of patients with idiopathic VTE will be subsequently diagnosed with cancer. Increasing attention has been placed on the benefit of comprehensive cancer screening in patients with idiopathic VTE.

Methods: We evaluated the utility of FDG-PET/CT (obtained within 4 weeks after the index VTE) to diagnose occult malignancy in 43 patients with acute, idiopathic VTE. All patients underwent a thorough evaluation for occult malignancy prior to the FDG-PET/CT imaging and were followed at regular intervals for the development of cancer or recurrent VTE.

Results: The average age (\pm SD) was 55 (\pm 14) and 46% of patients were female. Overall, 16 patients (39%) had DVT alone, 21 (51%) had PE, and 4 (10%) had DVT with PE. The average duration (\pm SD) of anticoagulation was 283 (\pm 185) days and 61% of patients remained on anticoagulation at the time of last follow-up. 52.9% had hyperhomocysteinemia while 11.8% were heterozygotes for the Factor V Leiden mutation. During an average follow-up (\pm SD) of 439 (\pm 313) days, two patients died (mortality 4.9%) and two patients had recurrent VTE. FDG-PET/CT imaging demonstrated abnormal findings suspicious for malignancy and requiring additional diagnostic evaluations in 62.5% of patients. The most commonly identified finding was a pulmonary nodule although abnormal appearing mediastinal, abdominal, or pelvic lymph nodes and thyroid nodules were also common. Following completion of these diagnostic evaluations, malignancy was diagnosed in only 1 patient and was evident on traditional CT imaging. Evaluation with FDG-PET/CT and subsequent diagnostic testing was costly, adding an estimated \$57,634 to the diagnostic evaluation (\$1,441 per patient enrolled in the study).

Conclusions: The use of FDG-PET/CT in patients with acute, unprovoked VTE was feasible but resulted in many incidental findings, requiring additional costly and invasive testing, and did not increase the diagnosis of occult malignancy.

Presenter: Matthew T. Rondina, MD, *University of Utah, SLC, UT, United States*

Poster 33. The Effect of Prior Venous Thromboembolism, Diabetes, and Statins on INR Control Among Patients Taking Warfarin

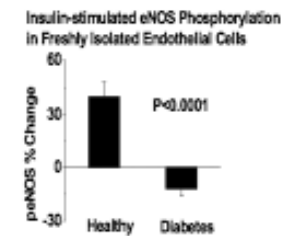
Background: Despite high-quality warfarin management, maintaining therapeutic levels of anticoagulation remains challenging for a significant proportion of patients. This variability may reflect individual thrombogenicity. We sought to explore factors associated with highly variable INR control among a cohort of patients taking warfarin for venous thromboembolism (VTE).

Methods: Patients with VTE were enrolled from January 2007 to December 2009. Demographic data and clinical characteristics were derived from chart review. Eligible patients had \geq 4 months of warfarin therapy. Patients in the highest (best) quartile of INR control were compared to those in the lowest.

Results: Of 460 patients with VTE identified, 411 had at least 4 months of warfarin. Mean age was 69 and 50% were black. We found diabetes and prior VTE to be associated with poorer INR control. The prevalence of diabetes across best to worst quartiles was 18%, 23%, 27% and 28%, and prior VTE: 25%, 27%, 28%, 30%. Fewer patients with poor control were taking a statin, 32% vs 42%.

Conclusions: We found INR control to be associated with diabetes, prior VTE, and statin use. Mechanisms underlying these findings warrant further study including the effects of glycemic control on INR and d-dimer, a marker of thrombogenicity.

Presenter: Andrew J. Cowan, MD, *Boston University Medical Center, Boston, MA, United States*



Clinical Science/Epidemiology – Vascular Cell Biology and Signaling

Poster 34. Insulin-mediated eNOS Activation is Impaired in Freshly Isolated Endothelial Cells from Patients with Type 2 Diabetes Mellitus

Endothelial dysfunction is a prominent feature of diabetes and contributes to atherogenesis and cardiovascular risk. It is well-established that skeletal muscle insulin resistance characterizes Type 2 diabetes. Recent studies using animal models demonstrate that altered insulin signaling at the endothelial level contributes to reduced nitric oxide bioavailability and accelerated atherosclerosis. To gain evidence that similar mechanisms operate in the vasculature in human diabetes, we performed venous endothelial cell biopsy in patients with diabetes ($n=10$) and healthy controls ($n=11$). As determined by quantitative immunofluorescence, expression of eNOS was similar in patients with diabetes compared to controls. Interestingly, basal eNOS phosphorylation was 93% higher in diabetes ($P<0.01$). In healthy controls, treatment of freshly harvested endothelial cells with insulin (10nM) induced a 1.4-fold increase in eNOS phosphorylation. In contrast, patients with diabetes showed marked impairment in insulin-stimulated eNOS activation (see Figure). Patients with diabetes demonstrated reduced flow-mediated dilation compared to controls ($7.5\pm 0.5\%$

vs. $14.5 \pm 1.6\%$, $p < 0.01$) consistent with endothelial dysfunction. Overall, lower insulin-mediated eNOS phosphorylation correlated with reduced flow-mediated dilation ($r = 0.78$, $P = 0.01$). Our findings confirm that human diabetes is associated with endothelial cell insulin resistance and implicate impaired insulin signaling as a mechanism for endothelial dysfunction in diabetes. Furthermore, elevated basal phospho-eNOS levels in patients with diabetes mellitus suggest that chronic activation could contribute to impaired insulin signaling in the diabetic endothelium.

Presenter: Corey E. Tabit, MD, *Boston University School of Medicine, Boston, MA, United States*

Clinical Science/Epidemiology — Vascular Surgery

Poster 35. Clinical Outcomes of Aortic Reconstruction for Vasculitis in the Medicare Population (2004-2007)

Background: Vasculitis is thought to increase the risk of aortic repair due to long-term anti-inflammatory medication use and the chronic inflammatory insult. Given the rarity of such patients and the limited literature, we evaluated outcomes of aortic surgery in patients with vasculitis using a national database.

Methods: Patients undergoing thoracic or abdominal aortic reconstruction were identified using Medicare data (2004-2007), and stratified into those with (VASC) and without (nVASC) vasculitis. Operative morbidity and early and late mortality were analyzed in the cohorts.

Results: 249 VASC and 115,867 nVASC patients underwent aortic reconstruction. Patient age was similar (75 ± 8 VASC vs. 75 ± 7 nVASC; $p = 0.72$).

VASC patients were more likely to be female (60% VASC vs. 24% nVASC; $p < 0.01$), have a chest reconstruction (56% VASC vs. 11%; $p < 0.01$) and have open repair (75% VASC vs. 34%; $p < 0.01$). Hypertension (58% VASC vs. 65%; $p = 0.04$), diabetes (7.2% VASC vs. 14%; $p < 0.01$), coronary artery disease (33% VASC vs. 45%; $p < 0.01$), COPD (25% VASC vs. 34%; $p < 0.01$), and peripheral vascular disease (11% VASC vs. 18%; $p < 0.01$) were less frequent in VASC. Operative mortality was similar (3.2%) in each group ($P = 0.98$). Any complication (34% VASC vs. 24%; $p < 0.01$), bleeding (13% VASC vs. 7%; $p < 0.01$), cardiac complications (12% VASC vs. 6%; $p < 0.01$) and infections (4.8% VASC vs. 2.6%; $p = 0.03$) were more common in VASC patients. Propensity score analysis with 1:30 (VASC: nVASC) matching was performed to appropriately match controls ($N = 7718$). Analysis of outcomes in well matched groups showed no differences in demographics, clinical features, or complications. The one year (87 ± 2 VASC vs. 87 ± 0.3 nVASC), and three year (73 ± 3 VASC vs. 77 ± 0.5 nVASC) survival was similar ($P = 0.14$) between the VASC and propensity matched nVASC controls.

Conclusions: Aortic reconstruction in patients with vasculitis is exceedingly rare and surgery in the setting of vasculitis poses comparable risk of outcomes to appropriately matched patients without vasculitis.

Presenter: Virendra I. Patel, MD, *Massachusetts General Hospital, Boston, MA, United States*
Clinical Science/Epidemiology-Venous Disease

Poster 37. Varicose Veins — A Thirty Five Year Experience in Olmsted County, MN

Background: Varicose veins (VV) are dilated, elongated, tortuous, subcutaneous veins ≥ 3 mm in size that are present in 10-30% of the population, with increasing rates in older individuals. Minimally invasive treatment has been proven to be effective. There is no data on morbidity and mortality burden of this disease.

Methods: This is a retrospective review of all Olmsted county residents who were seen at the Mayo Clinic, Rochester, Minnesota, between January 1980 and December 1985. The database was queried for diagnosis of/or related to VVs and comorbid conditions at the time diagnosis. The identified cohort was followed until end of April 2010. Age and gender matched controls were identified from census data of Caucasian population in state of Minnesota. The survival of the two groups was compared.

Results: Total of 1227 subjects were identified to have VVs; with more than $\frac{3}{4}$ women ($n = 928$ (75.6%)). The mean follow up time was 32.7 ± 3.1 years. Mean age at diagnosis was 51.9 ± 17.3 years. The mean age at diagnosis for men was 55.6 years and 50.6 for women ($p < 0.001$). The median survival time for patients with VVs was 29.4 years. 448 patients died during follow-up. For age and gender matched subjects, with comparable follow-up times, we observed 586 deaths ($p < 0.001$).

Conclusions: Subjects with varicose veins outlive their counterparts from general population. A nested case-control study is warranted to validate our results.

Presenter: Rajmony Pannu, *Mayo Clinic, Rochester, MN, United States*

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Exhibit Hours

Cityview Ballroom

Thursday, June 2

7:00 a.m. – 8:00 a.m.

9:55 a.m. – 10:25 a.m.

11:50 a.m. – 1:20 p.m.

3:35 p.m. – 4:05 p.m.

Friday, June 3

7:00 a.m. – 8:00 a.m.

9:50 a.m. – 10:20 a.m.

American Board of Vascular Medicine

Contact: Micheline Watt

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Phone: +1-440-247-4015

Fax: +1-440-247-6376

E-mail: wattm1@sbcglobal.net

Booth 5

The ABVM is an independent organization dedicated to the certification of qualified vascular medicine physicians. This certification process is based upon formal professional education or practice in vascular medicine with requirements to sit for the examination. This certification will provide evidence of expertise in vascular medicine knowledge and skills to the medical community.

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Booth 13

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Booth 3 and 4

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Booth 27

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Booth 21

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20325 Center Ridge Road #620
Rocky River, OH 44116 USA
Phone: +1-216-834-2410
E-mail: pam.mace@fmdsa.org

Booth 9

FMDSA is a non-profit organization dedicated to improving the lives of those afflicted with fibromuscular dysplasia by raising awareness and developing funds to promote research towards new medical treatments and diagnostic tools.

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E-mail: james.palagonia@ge.com

Booth 24

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Booth 19

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Booth 12

Intersocietal Commission for the Accreditation of Vascular Laboratories (ICAVL)

Contact: Marge Hutchinson
6021 University Blvd., Suite 500
Elliott City, MD 21043 USA
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E-mail: williams@intersocietal.org

Booth 15

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Booth 17

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Booth 20

Summit Doppler Systems, Inc. is a leading manufacturer of high-quality non-invasive diagnostic equipment for monitoring blood flow and performing the ankle-brachial index (ABI) and other arterial exams for the diagnosis of peripheral arterial disease or P.A.D. All Summit Doppler products are made in the United States. The Vista AVS is a full-featured ABI system with three modalities and automated features to expedite the ABI and other exams. The new Vantage ABI is a one-touch, 3 minute ABI system to assist in the diagnosis of P.A.D.

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Saturday, June 4	7:00 a.m. – 10:30 a.m.

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Participation in the SVM 22nd Annual Scientific Sessions is limited to registered attendees. The official badge must be worn for admission to all sessions, exhibits and other meeting activities. Attendees may be asked to present a photo ID corresponding to their name badge for admission to events. We thank you in advance for your understanding and cooperation. If there is an error on a badge, have it corrected at the registration desk.

Spouse/Guest Registration

Spouse/guest registration includes admission to the Celebrating Vascular Medicine Reception on Thursday evening only.

Employment Opportunities

The SVM Web site has an online job bank where you may post your resume or view job opportunities.

View www.vascularmed.org and follow the Job Bank link.

Meeting Evaluation

SVM needs your input to guide planning for future meetings. A link to an online meeting survey will be e-mailed to you shortly after the meeting. Your participation in this survey is greatly appreciated.

Speaker Ready Room *Washington Room*
Audiovisual preview and submission facilities are provided for speakers in the Washington Room (off the Amphitheater Lobby on the Mezzanine Level). If your presentation includes a video segment, it is very important that you visit the Speaker Ready Room and advise the AV techs of the video.

Speaker Ready Room Hours

Wednesday, June 1	5:00 p.m. – 8:00 p.m.
Thursday, June 2	7:00 a.m. – 11:30 a.m. 12:30 p.m. – 4:00 p.m.
Friday, June 3	7:00 a.m. – 1:00 p.m. 3:00 p.m. – 5:00 p.m.
Saturday, June 4	7:00 a.m. – 10:00 a.m.

Americans with Disabilities Act



SVM fully complies with the legal requirements of the ADA and the rules and regulations thereof.

Disclaimer

SVM is not responsible for the opinions expressed by speakers or the content of speaker handout materials.

Solicitations

Sales and promotional activities are restricted to exhibitors and must take place in their assigned exhibit area. Solicitations by unauthorized persons are strictly prohibited.

Celebrating Vascular Medicine Reception

The Celebrating Vascular Medicine Reception will be held Thursday, June 2, from 6:30 – 8 p.m. in the Harborview Ballroom. Your admittance is included in the registration fee for the 22nd SVM Annual Scientific Sessions. If you would like to bring a guest, the cost is \$40.

Continuing Medical Education Credit Claim and Meeting Survey—Online!

This activity has been planned and implemented in accordance with the Essential Areas and Policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint sponsorship of University of Colorado School of Medicine (UCSM) and SVM to provide a maximum of 19 *AMA PRA Category 1 Credit(s)*[™].

For your convenience, it's all online. To claim your credits and obtain your certificate go to <http://www.vascularmed.org/onlineexam/welcome.cfm>, complete the survey and your CME certificate will be delivered to you. This site will be live following the meeting.

Schedule-at-a-Glance

Wednesday, June 1

4:00 p.m. – 7:00 p.m.	Registration <i>Atrium Lobby</i>
5:30 p.m. – 7:30 p.m.	Satellite Symposium (corporate sponsors TBD)

Thursday, June 2

7:00 a.m. – 5:00 p.m.	Registration <i>Atrium Lobby</i>
7:00 a.m. – 7:00 p.m.	Poster Viewing <i>Atrium Lobby</i>
7:00 a.m. – 8:00 a.m.	Continental Breakfast and Exhibit Viewing <i>Cityview Ballroom</i>
7:00 a.m. – 8:00 p.m.	New Member Continental Breakfast <i>Skyline Room</i>
8:00 a.m. – 8:05 a.m.	Welcome to the SVM 22nd Annual Scientific Sessions <i>Amphitheater</i> Joshua A. Beckman, MD, FSVM, <i>Brigham and Women's Hospital and SVM Scientific Program Committee Chair</i>
8:05 a.m. – 8:55 a.m.	Session 1: Keynote Address – Health Care in the New World: The Academic Medical Center <i>Amphitheater</i> Elizabeth G. Nabel, MD, <i>Brigham and Women's Hospital</i>
8:55 a.m. – 9:55 a.m.	Session 2: Joint Session with VIVA – Vascular InterVentional Advances <i>Amphitheater</i> Moderator: Michael R. Jaff, DO, MSVM, <i>Massachusetts General Hospital</i> When to place and retrieve Vena Cava filters John A. Kaufman, MD, MS, <i>Dotter Interventional Institute/OHSU</i> Endovascular aortic stent-graft repair for ruptured abdominal aortic aneurysm , Manish Mehta, MD, <i>The Institute for Vascular Health and Disease</i> Drug/device combinations in PAD , Kenneth Rosenfield, MD, FSVM, <i>Massachusetts General Hospital</i> Modern management of critical limb ischemia , Manish Mehta, MD, <i>The Institute for Vascular Health and Disease</i> The next wave in device regulations in the United States Gary M. Ansel, MD, FSVM, <i>MidOhio Cardiology & Vascular Consultants</i> Panel Discussion

9:55 a.m. – 10:25 a.m.	Break and Exhibit Viewing <i>Cityview Ballroom</i>
10:25 a.m. – 11:30 a.m.	Session 3: Thrombolytic Therapy in Pulmonary Embolism <i>Amphitheater</i> Moderator: Samuel Z. Goldhaber, MD, FSVM, <i>Brigham and Women's Hospital</i> What are the indications for thrombolysis in pulmonary embolism? Douglas Drachman, MD, FACC, <i>Massachusetts General Hospital</i> Is low intensity anticoagulation (INR 1.5-2) equivalent to standard anticoagulation for indefinite secondary prophylaxis? Ken Bauer, MD, <i>Beth Israel Deaconess Medical Center</i> Is catheter-based lysis better than intravenous administration? John A. Kaufman, MD, MS, <i>Dotter Interventional Institute/OHSU</i> When should surgical pulmonary embolectomy be considered? Samuel Z. Goldhaber, MD, FSVM, <i>Brigham and Women's Hospital</i> Q & A and Discussion
11:30 a.m. – 11:50 a.m.	Presidential Address <i>Amphitheater</i> Thom W. Rooke, MD, FSVM, <i>Mayo Clinic</i>
11:50 a.m. – 1:20 p.m.	Annual Business Meeting Lunch (SVM Fellow Members Only) <i>Harborview 2 Ballroom</i>
11:50 a.m. – 1:20 p.m.	Exhibit Viewing and Delegate Lunch Break (on your own)
1:20 p.m. – 2:20 p.m.	Session 4: Case-based Vascular Medicine: How Experts Approach Common Chief Complaints <i>Amphitheater</i> Moderator: Robert D. McBane, MD, FSVM, <i>Mayo Clinic</i> I am on Coumadin, but need surgery in two weeks , Esther Soo Hyun Kim, MD, MPH, FACC, <i>The Cleveland Clinic Foundation</i> I have classic claudication but my ABI is normal , Robert D. McBane, MD, FSVM, <i>Mayo Clinic</i> Why do I have an ulcer on my heel with a normal ABI? Raghu Kolluri, MD, RVT, FSVM, <i>Prairie Vascular Institute</i>

Schedule-at-a-Glance

	My mom has Factor V Leiden and a DVT, but I only have FVL, Suman W. Rathbun, MD, FSVM, <i>University of Oklahoma Health Sciences Center</i>	8:00 a.m. – 8:45 a.m.	Session 8: Year in Review <i>Amphitheater</i> Moderator: Elizabeth V. Ratchford, MD, FSVM, <i>Johns Hopkins University School of Medicine</i>
2:20 p.m. – 3:20 p.m.	Session 5: The Inflamed Vessel <i>Amphitheater</i> Moderator: Steven M. Dean, DO, FSVM, <i>The Ohio State University College of Medicine</i>		Medical Therapy , Reena L. Pande, MD, <i>Brigham and Women's Hospital</i>
	How to use vessel size to make the diagnosis , Paul F. Dellaripa, MD, <i>Brigham and Women's Hospital</i>		Intervention and Surgery , Christopher J. Abularrage, MD, <i>The Johns Hopkins Hospital</i>
	Novel imaging techniques to determine large vessel disease activity , Heather L. Gornik, MD, MHS, FSVM, <i>The Cleveland Clinic Foundation</i>	8:45 a.m. – 8:50 a.m.	Accreditation , Joshua A. Beckman, MD, FSVM, <i>Brigham and Women's Hospital</i>
	Emerging therapies for vasculitides Paul A. Monach, MD, PhD, <i>Boston University School of Medicine</i>	8:50 a.m. – 9:50 a.m.	Presentation of the Jay D. Coffman Young Investigator Awards <i>Amphitheater</i>
	Skin manifestations of vasculitis , Steven M. Dean, DO, FSVM, <i>The Ohio State University College of Medicine</i>		Session 9: Keynote Address – Hypertension: The Past, the Present, the Future <i>Amphitheater</i> Eugene Braunwald, MD, FRCP, <i>Harvard Medical School and Brigham and Women's Hospital</i>
	Q & A and Discussion	9:50 a.m. – 10:20 a.m.	Refreshment Break and Exhibit Viewing <i>Cityview Ballroom</i>
3:20 p.m. – 3:35 p.m.	Session 6: Award Presentations – Jess Young Outstanding Educator Award, Master of SVM <i>Amphitheater</i>	10:20 a.m. – 11:20 a.m.	Session 10: Antiplatelet Therapy <i>Amphitheater</i> Moderator: William R. Hiatt, MD, MSVM, <i>University of Colorado</i>
3:35 p.m. – 4:05 p.m.	Refreshment Break and Exhibit Viewing <i>Cityview Ballroom</i>		Inflammation and platelets in atherothrombosis , Peter Libby, MD, <i>Brigham and Women's Hospital</i>
4:05 p.m. – 5:30 p.m.	Session 7: Jay D. Coffman Young Investigator Presentations <i>Amphitheater</i>		Should PAD patients routinely be treated with aspirin? William R. Hiatt, MD, MSVM, <i>University of Colorado</i>
5:30 p.m. – 6:30 p.m.	Poster Presentations <i>Atrium Lobby</i>		Genetics and platelet inhibition: Ready for prime time? Marc Sabatine, MD, MPH, <i>Brigham and Women's Hospital</i>
6:30 p.m. – 8:00 p.m.	Celebrating Vascular Medicine Reception <i>Harborview Ballroom</i>		Point of care platelet reactivity testing: 2011 , Deepak Bhatt, MD, MPH, FACC, FAHA, FSCAI, <i>Brigham and Women's Hospital</i>
Friday, June 3		11:20 a.m. – 11:55 a.m.	Session 11: Large Vessel Dissection <i>Amphitheater</i> Moderator: James B. Froehlich, MD, MPH, FSVM, <i>University of Michigan Medical School</i>
7:00 a.m. – 5:00 p.m.	Registration <i>Atrium Lobby</i>		Does endovascular therapy relax indications for type β aortic-dissection intervention? James B. Froehlich, MD, MPH, FSVM, <i>University of Michigan Medical School</i>
7:00 a.m. – 1:30 p.m.	Poster Viewing <i>Atrium Lobby</i>		
7:00 a.m. – 8:00 a.m.	Continental Breakfast and Exhibit Viewing <i>Cityview Ballroom</i>		
7:00 a.m. – 8:00 a.m.	Women in Vascular Medicine Breakfast Meeting <i>Harborview 1 Ballroom</i> Galit Lahav, PhD, <i>Harvard Medical School</i>		

Schedule-at-a-Glance

	What is the proper approach to visceral artery dissection? Patrick O'Gara, MD, <i>Brigham and Women's Hospital</i>		
	The role of biomarkers in aortic dissection diagnosis Kim Eagle, MD, <i>University of Michigan Cardiovascular Center</i>		
11:55 a.m. – 1:30 p.m.	Vascular Jeopardy and Lunch <i>Harborview Ballroom</i> Joshua A. Beckman, MD, FSVM, <i>Brigham and Women's Hospital</i> and Michael R. Jaff, DO MSVM, <i>Massachusetts General Hospital</i>		4:30 p.m. - 6:00 p.m. Cases Over Cocktails <i>Harborview Ballroom</i> Joshua A. Beckman, MD FSVM, Brigham and Women's Hospital and Michael R. Jaff, DO, MSVM, Massachusetts General Hospital
1:30 p.m. – 4:30 p.m.	Concurrent Live Demonstration Workshops		6:15 p.m. – 7:45 p.m. Satellite Symposium (corporate sponsors TBD)
	Workshop 1 <i>Back Bay Room</i>		
1:30 p.m. & 3:00 p.m.	Carotid Pitfalls , Marie Gerhard-Hermann, MD, FSVM, <i>Brigham and Women's Hospital</i>		
2:00 p.m. & 3:30 p.m.	Renal Pearls , Michael R. Jaff, DO, MSVM, <i>Massachusetts General Hospital</i>		
2:30 p.m. & 4:00 p.m.	TCD Basics , Viken Babikian, MD, <i>Boston Medical Center</i>		
	Workshop 2 <i>Federal Room</i>		
1:30 p.m. & 3:00 p.m.	Venous Reflux & Ablation , Lucy LaPerna, DO, RVT, RPVI, FSVM, <i>Riverside Interventional Consultants</i>		
2:00 p.m. & 3:30 p.m.	Arterial Complications , Raghu Kolluri, MD, RVT, FSVM, <i>Prairie Vascular Institute</i>		
2:30 p.m. & 4:00 p.m.	Physiologic Maneuvers in the Vascular Lab , Paul W. Wennberg, MD, FSVM, <i>Mayo Clinic</i>		
	Workshop 3 <i>Skyline Room</i>		
1:30 p.m. & 3:00 p.m.	A Wound Care Workshop: From scalpel to growth factors , Vickie Driver, DPM, MS, FACFAS, <i>Boston University Medical Campus</i>		
2:00 p.m. & 3:30 p.m.	Compression Primer , Cindy Felty, MSN, RN, CNP, FSVM, <i>Mayo Clinic</i>		
2:30 p.m. & 4:00 p.m.	Sclerotherapy , Margaret O'Byrne, MD, RVT, <i>The Vein Clinic</i> and Robert M. Schainfeld, DO, FSVM, <i>Massachusetts General Hospital</i>		
			Saturday, June 4
			7:00 a.m. – 10:30 a.m. Registration <i>Atrium Lobby</i>
			7:00 a.m. – 8:00 a.m. Continental Breakfast <i>Mezzanine Level Lobby</i>
			8:00 a.m. – 10:00 a.m. Novel Anticoagulants, Part 1 <i>Amphitheater</i> Moderator: Jerry Bartholomew, MD, FSVM, <i>The Cleveland Clinic Foundation</i>
			Pharmacology of the new agents , Speaker TBD
			New Therapies in Atrial Fibrillation , Jonathan L. Halperin, MD, MSVM, <i>The Cardiovascular Institute</i>
			New Directions in Venous Thromboembolism , Samuel Z. Goldhaber, MD, FSVM, <i>Brigham and Women's Hospital</i>
			10:00 a.m. – 10:15 a.m. Break
			10:15 a.m. – 12:15 p.m. Novel Anticoagulants, Part 2 <i>Amphitheater</i> Moderator: Jerry Bartholomew, MD, FSVM, <i>The Cleveland Clinic Foundation</i>
			Case Discussions (panel) , Jerry Bartholomew, MD, FSVM, <i>The Cleveland Clinic Foundation</i> ; Robert D. McBane, MD FSVM, <i>Mayo Clinic</i> ; Matthew T. Rondina, MD, <i>University of Utah Health Sciences Center</i>
			Impact of novel agents on a hospital's budget and anticoagulation service , Julie Atay, PharmD, MBA, <i>Brigham and Women's Hospital</i>
			The P&T committee response to the \$10/day pill, locally and nationally , John Fanikos, PharmD, MBA, <i>Brigham and Women's Hospital</i>
			Panel discussion/audience Q & A
			12:15 p.m. Meeting Adjourns

Floorplan

