AMERICAN VENOUS FORUM
23rd ANNUAL MEETING

February 23–26, 2011

Hilton San Diego Bayfront ▪ San Diego, California

EXECUTIVE COMMITTEE

President	Peter J. Pappas, MD (2011) Brooklyn, New York
President-Elect	Seshadri Raju, MD (2011) Flowood, Mississippi
Secretary	Fedor Lurie, MD (2013) Honolulu, Hawaii
Treasurer	David L. Gillespie, MD (2011) Rochester, New York
Recorder	Peter K. Henke, MD (2012) Ann Arbor, Michigan
Archivist	Marc A. Passman, MD (2011) Birmingham, Alabama
Past-Presidents	Mark H. Meissner, MD (2011) Seattle, Washington
	Joann M. Lohr, MD (2012) Cincinnati, Ohio
	Joseph A. Caprini, MD (2013) Skokie, Illinois
Councilors	B.K. Lal, MD (2011) Newark, New Jersey
	Joesph D. Raffetto, MD (2012) West Roxbury, Massachusetts
	Elna Masuda, MD (2013) Honolulu, Hawaii

COMMITTEES
2010-2011

BY-LAWS COMMITTEE
Patricia Furey, MD (2011), *Chair*
Daniel Monahan, MD (2012)
Mark Iafrati, MD (2013)
Fedor Lurie, MD (2013), *Ex-Officio*

FELLOWS EDUCATION COMMITTEE
Steven Elias, MD (2013), *Chair*
Manju Kalra, MD (2013)
William Marston, MD (2013)
Linda Harris, MD (2013)
Thomas O'Donnell, MD (2013)
Mark Meissner, MD (2013)
Antonios Gasparis, MD (2013)
Daniel Clair, DVM (2013)
John Blebea, MD (2012), *Ex-Officio*
Lowell Kabnick, MD (2012), *Ex-Officio*

GRANTS & AWARDS COMMITTEE
Paul Pittaluga, MD (2011), *Chair*
Amy Reed, MD (2012)
Nicos Labropoulos, MD (2013)
Mark Meissner, MD (2011), *Ex-Officio*
Robert McLafferty, MD (2011), *Ex-Officio*

GUIDELINES COMMITTEE
Peter Gloviczki, MD (2012), *Co-Chair*
Mark Meissner, MD (2012), *Co-Chair*
Bo Eklof, MD (2012)
David Gillespie, MD (2012)
Joann Lohr, MD (2012)
Robert McLafferty, MD (2012)
Frank Padberg, MD (2012)
Peter Pappas, MD (2012)
Joseph Raffetto, MD (2012)
Thomas Wakefield, MD (2012)
Mark Meissner, MD (2011), *Ex-Officio*
Robert McLafferty, MD (2011), *Ex-Officio*

ISSUES COMMITTEE
Audra Duncan, MD (2011), *Chair*
Ashraf Mansour, MD (2012)
John Blebea, M. D. (2013)
Fedor Lurie, MD (2013), *Ex-Officio*

MEMBERSHIP COMMITTEE
Mark Garcia, MD (2011), *Chair*
Anil Hingorani, MD (2012)
Ruth Bush, MD (2013)
Fedor Lurie, MD (2013), *Ex-Officio*

NOMINATING & HONORARY MEMBERSHIP COMMITTEE
Mark Meissner, MD (2011), *Chair*
Joann Lohr, MD (2012)
Joseph Caprini, MD (2013)
Fedor Lurie, MD (2013), *Ex-Officio*

OUTCOMES COMMITTEE
William Marston, MD, *Co-Chair*
Michael Vasquez, MD, *Co-Chair*
Fedor Lurie, MD
Eberhard Rabe, MD
Mark Meissner, MD, *Ex-Officio*
Robert McLafferty, MD, *Ex-Officio*

PATIENT EDUCATION COMMITTEE
Linda Harris, MD (2012), *Chair*
Gary Lemmon, MD (2012)
Paul Gagne, MD (2012)
Steve Zimmet, MD (2012)
John Blebea, MD (2012), *Ex-Officio*
Lowell Kabnick, MD (2012), *Ex-Officio*

PHYSICIAN/ALLIED HEALTH EDUCATION COMMITTEE
Jose Almeida, MD (2012), *Chair*
Timothy Liem, MD (2012)
Antonios Gasparis, MD (2012)
Julianne Stoughton, MD (2012)
Ashraf Mansour, MD (2012)
Ronald Bush, MD (2012)
John Blebea, MD (2012), *Ex-Officio*
Lowell Kabnick, MD (2012), *Ex-Officio*

PROGRAM COMMITTEE
Peter Henke, MD (2011), *Chair*
Nicos Labropoulos, MD (2012)
Antonios Gasparis, MD (2013)
Fedor Lurie, MD (2013), *Ex-Officio*

RESEARCH COMMITTEE
Joseph D. Raffetto, MD (2012), *Chair*
Daniel Myers, MD (2021)
Gregory Landry, MD (2012)
Teresa Carman, MD (2012)
Mark Meissner, MD (2011), *Ex-Officio*
Robert McLafferty, MD (2011), *Ex-Officio*

COMMITTEES 2010-2011

SCREENING COMMITTEE
Marc Passman, MD (2011), *Chair*
Todd Bohannon, MD (2011)
Jennifer Heller, MD (2011)
Armen Roupenian, MD (2011)
Joseph Schneider, MD (2013)
Colleen Johnson-Moore, MD (2013)
Michele Lentz, *Ex-Officio*
John Blebea, MD (2012), *Ex-Officio*
Lowell Kabnick, MD (2012), *Ex-Officio*

WEBSITE COMMITTEE
Marc A. Passman, MD (2012), *Chair*
Antonios Gasparis, MD (2012)
Alessandra Puggioni, MD (2012)
John Blebea, MD (2012), *Ex-Officio*
Lowell Kabnick, MD (2012), *Ex-Officio*

AD-HOC COMMITTEES 2010-2011

GOVERNMENT RELATIONS
Michael Vasquez, MD, *Chair*

ULCER COMMITTEE
Monika Gloviczki, MD, *Chair*
Marc Passman, MD
David Gillespie, MD
William Marston, MD
Fedor Lurie, MD, *Ex-Officio*

VENOUS REGISTRY STEERING COMMITTEE
Brajesh Lal, MD, *Chair*

VENOUS STENTING
Marc Passman, MD
Anthony Gasparis, MD
David Gillespie, MD
Luis Leon, MD

VARICOSE VEINS
Jose Almeida, MD
Lowell Kabnick, MD
Thomas Wakefield, MD

IVC FILTERS
John Rectenwald, MD
Uchenna Onyeachom, *Ex-Officio*

FUTURE MEETINGS

2012
February 8–12
Loews Royal Pacific
Orlando, Florida

AMERICAN VENOUS FORUM FOUNDATION

The American Venous Forum Foundation was organized in 1988 to support the charitable, educational and scientific purposes of the American Venous Forum.

The Foundation provides the **BSN-Jobst Fellowship Award, Servier Fellowship Award** and other significant educational grants to stimulate and recognize excellence in published writing on laboratory and clinical research in the study of venous diseases.

The Foundation also oversees the education and objectives of the Venous Education Institute of North America (VEIN).

AMERICAN VENOUS FORUM FOUNDING MEMBERS

Robert W. Barnes, MD
John J. Bergan, MD
John J. Cranley, MD
W. Andrew Dale, MD
Ralph G. DePalma, MD
James A. DeWeese, MD
Lazar J. Greenfield, MD
Robert W. Hobson, II, MD
Michael Hume, MD
George Johnson, Jr., MD
Robert L. Kistner, MD
John M. Porter, MD
Seshadri Raju, MD
Norman M. Rich, MD
Charles G. Rob, MD
Joseph G. Sladen, MD
D. Eugene Strandness, Jr., MD
David S. Sumner, MD
J. Leonel Villavicencio, MD
James S.T. Yao, MD

AVF FOUNDATION BOARD OF DIRECTORS

President	Mark H. Meissner, MD (2011) Seattle, Washington
Vice-President	Joann M. Lohr, MD (2011) Cincinnati, Ohio
Secretary	Fedor Lurie, MD (2013) Honolulu, Hawaii
Treasurer	David L. Gillespie, MD (2011) Rochester, NY
Directors	Steven Elias, MD (2011) Englewood, New Jersey
	Fedor Lurie, MD (2011) Honolulu, Hawaii
	Lowell Kabnick, MD (2011) New York, New York
	William A. Marston, MD (2011) Chapel Hill, North Carolina
Ex-Officio	Joseph A. Caprini, MD (2011) Chicago, Illinois

THE AMERICAN VENOUS FORUM WAS ORGANIZED IN COOPERATION WITH MEMBERS OF:

Society for Vascular Surgery

American Association for Vascular Surgery

Canadian Society for Vascular Surgery

WITH THE SUPPORT OF MEMBERS OF:

International Union of Phlebology

North American Society of Phlebology

Phlebology Society of America

Austrian Society for Angiology

Benelux Society of Phlebology (Belgium, Netherlands and Luxembourg)

European Chapter of the International Society for Cardiovascular Surgery

German Society of Phlebology and Proctology

Latin American Chapter of the International Society for Cardiovascular Surgery

Swiss Society for Phlebology

Sociedad Mexicana de Angiologia

College Francais de Pathologie

Société Francaise de Phlebologie

Société Francaise d'Angéiologie

Societa Italiana de Patologia Vascolare

Pan American Society of Phlebology and Lymphology

Sociedad Argentina de Flebologia y Linfologia

Australian and New Zealand Society of Phlebology

ANNUAL MEETINGS/PAST PRESIDENTS

1989	February 22-24	John J. Bergan, MD *New Orleans, LA – Fairmont Hotel*
1990	February 21-23	Norman M. Rich, MD *Coronado, CA – Hotel Del Coronado*
1991	February 20-22	Lazar J. Greenfield, MD *Ft. Lauderdale, FL – Marina Marriott Hotel*
1992	February 26-28	Michael Hume, MD *Coronado, CA – Hotel Del Coronado*
1993	February 24-26	George Johnson, Jr., MD *Orlando, FL – Hilton Walt Disney World Village*
1994	February 23-25	James A. DeWeese, MD *Maui, HI – Maui Inter-Continental Resort*
1995	February 23-25	Robert Hobson, MD *Fort Lauderdale, FL – Marriott Harbor Beach*
1996	February 22-24	Robert L. Kistner, MD *San Diego, CA – Hyatt Regency Hotel*
1997	February 20-23	James S.T. Yao, MD *San Antonio, TX – Hyatt Regency Hill Country Resort*
1998	February 19-21	D. Eugene Strandness, Jr., MD *Lake Buena Vista, FL – Walt Disney World Swan Hotel*
1999	February 18-21	Thomas F. O'Donnell, Jr., MD *Dana Point, CA – Laguna Cliffs Marriott Resort*
2000	February 3-6	David S. Sumner, MD *Phoenix, AZ – Hilton South Mountain Resort*
2001	February 22-25	Anthony J. Comerota, MD *Ft. Myers, FL – Sanibel Harbor Resort*
2002	February 21-24	Gregory L. Moneta, MD *La Jolla, CA – Hilton Torrey Pines La Jolla*
2003	February 20-23	Peter Gloviczki, MD *Cancun, Mexico – Hilton Cancun Beach Resort*
2004	February 26-29	Frank T. Padberg, MD *Orlando, FL – Gaylord Palms Resort*
2005	February 9-13	Bo G. Eklöf, MD *San Diego, CA – Loews Coronado Bay Resort*

2006	February 22-26	Thomas W. Wakefield, MD *Miami, FL – InterContinental Hotel*
2007	February 14-17	Michael C. Dalsing, MD *San Diego, CA – Rancho Bernardo Inn*
2008	February 20-23	Mark H. Meissner, MD *Charleston, SC – Charlston Place*
2009	February 11-14	Joann Lohr, MD *Phoenix, AZ – Arizona Grand Resort*
2010	February 10-13	Joseph A. Caprini, MD *Amelia Island, FL – Ritz-Carlton*

D. EUGENE STRANDNESS JR., MD MEMORIAL LECTURE

On January 7, 2002, the American Venous Forum was saddened by the passing of one of its founding members and past presidents, Dr. D. Eugene Strandness Jr. Dr. Strandness was a friend, mentor, colleague and leader in all aspects of vascular surgery. He held several NIH grants and wrote numerous publications on the etiology and non-invasive diagnosis of deep vein thrombosis. One of his most notable accomplishments was the development of duplex ultrasound scanning. His tireless pursuit of knowledge led to a better understanding of the natural history of venous disease and its diagnosis and treatment, for which our patients and we are forever indebted to him.

Each year, the D. Eugene Strandness Jr., MD Memorial Lecture recognizes the significant contributions of an individual in research, education or clinical investigation in the field of venous diseases. The recipient of this distinction, chosen by the president of the American Venous Forum and confirmed by the Forum's Executive Committee, has previously been named to the position of Presidential Guest Lecturer. In honor of the memory of Dr. Strandness, the lectureship was renamed in 2003 and is now known as the "D. Eugene Strandness Jr., MD Memorial Lecture."

This honor, the highest given by the organization, has been bestowed to the following outstanding candidates in past years:

2010 **Manuel Monreal Bosch, MD, Madrid, Spain**
RIETE Database and Multiple Clinical Perspectives

2009 **O. William Brown, MD, Bingham Farms, Michigan**
Venous Disease and Medical Malpractice: A Peek Inside the Playbook of a Plaintiff's Attorney

2008 **Thomas O'Donnell, Jr., MD, Boston, Massachusetts**
What's the Evidence for Treating Perforators in Venous Ulcers

2007 **Robert L. Kistner, MD, Honolulu, Hawaii**
Foresight 2020: Creating the Venous Vision

2006 **Pan Ganguly, PhD, Bethesda, Maryland**
The Challenges in Venous Thrombosis

2005 **Michel R. Perrin, MD, Chassieu, France**
The Importance of International Collaboration for the Development of a Scientific Approach to Venous Disease

2004 **Professor Eberhard Rabe, MD, Bonn, Germany**
Prevalence and Risk Factors of Chronic Venous Diseases: The Bonn Vein Study

2003 **Professor Claudio Allegra, MD, Rome, Italy**
Involvement of the Microcirculation in Chronic Venous Insufficiency

2002 **Professor Alfred Bollinger, MD, Zurich, Switzerland**
Microcirculation in Chronic Venous Insufficiency and Lymphedema

2001 **Professor C.V. Ruckley, MD, Edinburgh, Scotland**
Chronic Venous Insufficiency: Lessons from Scotland

2000 **Professor Sir Norman Browse, MD, FRCS, FRCP, Channel Islands, England**
Forty Years On

1999 **David Robinson, PhD, Bethesda, Maryland**
A Journey to Complexity: The Continuing Evolution in Vascular Research

1998 **David Bergquist, MD, PhD, Uppsala, Sweden**
Chronic Leg Ulcer—The Impact of Venous Disease

1997 **Professor Kevin G. Burnand, London, United Kingdom**
Venous Thrombosis and Natural Thrombolysis

1996 **Ermenegildo A. Enrici, MD, Buenos Aires, Argentina**
The Role of the Perforants' System in Deep Venous Chronic Insufficiency in its Different Stages: Surgical Indications, Tactics and Techniques

1995 **Philip D. Coleridge Smith, MD, FRCS, London, United Kingdom**
Venous Disease and Leukocyte Mediated Microcirculatory Injury

1994 **Andrew W. Nicolaides, MD, FRCS, London, United Kingdom**
Deep Vein Thrombosis—Aetiology and Prevention: The Legacies of the 70's, the Promises of the 80's and the Challenges of the 90's

1993 **Olav Thulesius, MD, PhD, Linkoping, Sweden**
Vein Wall Characteristics and Valvular Functions in Chronic Venous Insufficiency

1992 **G.W. Schmid-Schonbein, MD, La Jolla, California**
Leukocytes as Mediators of Tissue Injury

1991 **Jack Hirsh, MD, Hamilton, Ontario, Canada**
Development of Low Molecular Weight Heparin for Clinical Use

1990 **Hugo Partsch, MD, Vienna, Austria**
Diagnosis of AV Fistulas in Vascular Malformations

2011
D. EUGENE STRANDNESS, JR., MD
MEMORIAL LECTURE

Microcirculatory and Lymphatic Disorders

David C. Zawieja, PhD
Director, Division of Lymphatic Biology
Texas A&M Health Science Center College of Medicine

David Zawieja, PhD received his Bachelor of Science degree from the University of Wisconsin, completed his Graduate program in Biomedical Engineering at Rensselaer Polytechnic Institute, received his Doctorate in Philosophy from the Medical College of Wisconsin and completed his post-doctoral training at Texas A&M University (Department of Medical Physiology).

He has authored almost 200 papers, has lectured and taught extensively and has received honors and major grant support awards (NIH, NHLBI, AHA, etc.) which are too numerous to mention. Dr. Zawieja has also been a journal reviewer for 15 scientific publications. In his "spare" time, his interests include coaching soccer, little league and the Boy Scouts.

My laboratory investigates the microcirculatory movement of fluid and macromolecules. Our interests include the control and regulation of fluid and macromolecular exchange and transport throughout the three microcirculatory compartments: the microvascular compartment, the interstitial compartment and the lymphatic compartment.

We are investigating both the normal physiological control and pathophysiological alterations of these functions. We have focused most of our recent work on the function of the lymphatic system and are investigating the mechanisms responsible for the generation and regulation of lymph flow.

The lymphatic system is vital to body fluid/protein homeostasis, edema prevention, lymphocyte circulation, immune function and lipid absorption. All of these functions require a regulated lymph flow. We are investigating the influence of physical, neural and humoral factors on the generation of lymph flow with particular emphasis on the mechanisms by which these factors alter the active lymph pump. Mammalian lymphatics possess intrinsic phasic contractions that pump lymph throughout the body and tonic contractions that regulate outflow resistance. The cellular mechanisms regulating the lymphatic contractions are unknown and are the subject of our current studies.

Recently we have focused on the role of calcium and the contractile and regulatory proteins involved in the phasic and tonic lymphatic contractile activity. We have also investigated the influences of flow and shear on lymphatic contractile function and found that shear modulates the phasic and tonic contractile activity via a nitric oxide/cGMP based mechanism. These studies also include the development of more accurate models of lymph flow/shear in microlymphatics.

The growth of new lymph vessels, lymphangiogenesis, is another area of interest in our lab. We have developed and characterized the first cultured microlymphatic endothelial and muscle cell lines. We have begun studies of the factors which regulate the proliferation and migration of these cells. To accomplish these studies, my laboratory utilizes a number of different techniques including: 1) in situ studies using intravital video microscopy, 2) isolated microvessel studies using fluorescent video microscopy, 3) dispersed smooth muscle cells, 4) isolated cultured vascular cells, 5) calcium and membrane potential imaging using fluorescent microscopy, 6) confocal microscopy and 7) mathematical simulation of physiological processes.

This lecture will be presented on Saturday, February 26, 2011 at 11:00 am.
Please plan to attend this featured presentation.

BSN-JOBST RESEARCH FELLOWSHIP IN VENOUS AND LYMPHATIC DISEASE

In 1995, the American Venous Forum Foundation announced the establishment of the BSN-Jobst Research Fellowship In Venous and Lymphatic Disease.

The BSN-Jobst Research Fellowship provides a one-year, $50,000 grant to a research fellow chosen through a competitive peer-review selection process. A committee of distinguished vascular physicians, appointed by the American Venous Forum Foundation, determines the fellowship recipient and announces its selection during the opening session of the Annual Meeting.

1995 **Peter J. Pappas, MD**, UMDNJ New Jersey Medical School
1996 **Jae-Sung Cho, MD**, Mayo Clinic, Rochester, MN
1997 **Andrew C. Stanley, MD**, Burlington, VT
1998 **Klaus See-Tho, MD**, Stanford University Medical Center
1999 **Joseph D. Raffetto, MD**, Boston Medical Center
2000 **No Award Given**
2001 **Brajesh K. Lal, MD**, UMDNJ New Jersey Medical School
2002 **Susan O'Shea, MD**, Duke University Medical Center
2003 **Charles Fields, MD**, Mayo Clinic
2004 **John Rectenwald, MD**, University of Michigan
2005 **Allesandra Puggioni, MD**, Mayo Clinic
2006 **Stephanie K. Beidler, MD**, University of North Carolina
2007 **Danny Vo, MD**, Mayo Clinic
2008 **K. Barry Deatrick, MD**, University of Michigan
2009 **Carolyn Glass, MD**, University of Rochester
2010 **Yanjie Qi, MD**, University of Rochester

SERVIER TRAVELING FELLOWSHIP

The Servier Traveling Fellowship provides two fellows an opportunity to travel to the European Venous Forum to present his or her scientific research. Four (4) finalists are identified through a competitive peer-review process, and are invited to present their science during the AVF Meeting. Travel and accommodations for the four finalists are reimbursed as part of the grant. The finalists are judged by an appointed AVF committee. Two winners will be selected to present their work at the European Venous Forum.

2006	**Charles Stonerock, MD**, Indiana University School of Medicine **Gustavo Oderich, MD**, Mayo Clinic
2007	**Brian Knipp, MD**, University of Michigan **Reagan Quan, MD**, Walter Reed Army Medical Center
2008	**David Paolini, MD**, Toledo Hospital **Jorge Martinez, MD**, Toledo Hospital
2009	**Atul Rao, MD**, University of Pittsburgh Medical Center **Axel Thors, MD**, Good Samaritan Hospital
2010	**K. Barry Deatrick, MD**, University of Michigan **Christopher Pannucci, MD**, University of Michigan

BEST POSTERS

Each year, a formal poster session is held where authors are invited to give a 3-minute synopsis of their work followed by a 2-minute Q&A with the audience in attendance. Posters are scored and prizes are awarded to the top presentations.

2010 WINNERS

Jose Diaz
Role of PAI-1 In Deep Vein Thrombosis in a Murine Model

Felizitas Pannier
Risk Factors for Incidence of Varicose Veins, CVI in the Bonn Vein Study II

Hideo Tashiro
Efficacy and Safety of Great Saphenous Vein Trunk Sclerotherapy Under Balloon Occlusion at Sapheno-Femoral Junction

GENERAL INFORMATION

REGISTRATION DESK

The Registration Desk will be located in the **Indigo West Foyer** and will be open during the following hours:

Tuesday, February 22	4:00 pm – 6:00 pm
Wednesday, February 23	7:00 am – 5:00 pm
Thursday, February 24	7:00 am – 5:00 pm
Friday, February 25	7:00 am – 12:00 pm
Saturday, February 26	7:00 am – 5:00 pm

REGISTRATION INFORMATION

Full Registration Fee Includes: The full registration fee includes all scientific sessions, continental breakfast, coffee breaks and boxed lunches. In addition, the registration fee includes entrance to the Exhibit Hall, the Welcome Reception on Wednesday and the Forum Finale on Saturday evening.

Spouse/Guest Registration Fee Includes: The spouse/guest registration fee includes the Welcome Reception, continental breakfast, mid-morning refreshments daily in the Hospitality Suite and Forum Finale on Saturday evening.

ANNUAL BUSINESS MEETING LUNCH (MEMBERS ONLY)

The Annual Business Meeting will be held on Friday, February 25, 2011 at 11:30 am in **Indigo AE**.

INSTRUCTIONS TO AUTHORS

Audio/Visual
All presentations must be formatted using PowerPoint. All presenters must bring their PowerPoint presentations on a USB flash drive to the Speaker Ready Room at least two hours prior to their scheduled presentation.

Manuscripts
The American Venous Forum requires presenting authors of oral presentations to submit the full manuscript for journal publication. The Journal of Vascular Surgery is the official journal of the American Venous Forum, although authors may petition the AVF Recorder in writing to submit their manuscript to an alternate Index-Medicus, peer-reviewed journal. Presenters who fail to submit a manuscript to a recognized journal shall forfeit their right to present any material at two (2) consecutive future meetings of the American Venous Forum.

SCHEDULE-AT-A-GLANCE

23rd Annual Meeting
February 23-26, 2011
Hilton San Diego Bayfront ■ San Diego, California

WEDNESDAY, FEBRUARY 23, 2011

7:00 AM – 8:00 AM **Continental Breakfast**

8:00 AM – 12:00 PM **DAVID S. SUMNER VENOUS SUMMIT**
Introduction: *Peter J. Pappas, MD, President*
Seshadri Raju, MD, President-Elect

Please Note: The annual Postgraduate Course that has preceded the meeting for several years will henceforth be known as the David S. Sumner Venous Summit to honor his monumental contributions that have facilitated understanding of venous hemodynamics.

12:00 PM – 1:30 PM **ACP LUNCH SYMPOSIUM**

Understanding Venous Hemodynamics Through Ultrasound
Moderator: *Nick Morrison, MD*

1:30 PM – 3:10 PM **SCIENTIFIC SESSION I**
Deep Vein Thrombosis I
Moderators: *Peter Pappas, MD*
Antonios Gasparis, MD

1:30 PM – 1:50 PM **1** **Association of Blood Transfusion and Venous Thromboembolism in the Perioperative Period**
E.S. Xenos, D.L. Davenport
University of Kentucky Medical Center and VA Medical Center, Lexington, KY

1:50 PM – 2:10 PM **2** **Down-Regulation of Hypoxia-Inducible Factor 1 Alpha Reduces Venous Thrombus Resolution**
C.E. Evans, J. Humphries, K. Mattock, M. Waltham, A. Wadoodi, P. Saha, B. Modarai, A. Smith
King's College London, London, United Kingdom

2:10 PM – 2:30 PM	**3**	**Catheter-Directed Thrombolysis of Iliofemoral DVT Reduces DVT Recurrence** F. Aziz, J.T. Chen, A.J. Comerota *The Toledo Hospital, Toledo, OH*
2:30 PM – 2:50 PM	**4**	**Creation of a Simple Venous Thromboembolism Risk Score for Outpatient Surgery: Analysis of the NSQIP Database** C.J. Pannucci, A. Shanks, M. Moote, V. Bahl, P. Cederna, N. Naughton, P. Henke, S. Kheterpal, S. Campbell *University of Michigan, Ann Arbor, MI*
2:50 PM – 3:10 PM	**5**	**Vena Caval Filters: Review of Indications and Practices at a University Hospital** R.J. Meisner, N. Labropoulos, A.P. Gasparis, A. Tassiopoulos *Stonybrook University Hospital, Stony Brook, NY*
3:10 PM – 3:45 PM		**Coffee Break**
3:45 PM – 5:15 PM		**LIVE ULTRASOUND** *Panelists:* *Nicos Labropoulos, MD* *Gail Size, BS, RVT, RVS, RPhS* *Steve Elias, MD*
6:00 PM – 7:30 PM		**WELCOME RECEPTION**

THURSDAY, FEBRUARY 24, 2011

7:00 AM – 8:00 AM **Continental Breakfast — Exhibits Open**

8:00 AM – 10:00 AM **SCIENTIFIC SESSION II**
Chronic Venous Disease: Epidemiology & Screening
Moderators: *Nicos Labropoulos, MD*
Bo Eklof, MD

8:00 AM – 8:20 AM **6** **Venous Disease and the Effects of Increasing Body Mass Index: Results from the National Venous Screening Program**
C.J. Moore[1], R.B. McLafferty[1], M. Lentz[2], J.R. Schneider[3], A. Roupenian[4], J. Heller[5], W. Bohannon[6], M. Passman[7]
[1]Southern Illinois University, Springfield, IL, [2]National Venous Screening Program, Baltimore, MD, [3]Central DuPage Hospital, Winfield, IL, [4]Vein and Laser Center NE, Plymouth, MA, [5]Johns Hopkins Vein Center, Baltimore, MD, [6]Scott & White Memorial Hospital and Clinic, Temple, TX, [7]University of Alabama, Birmingham, AL

8:20 AM – 8:40 AM **7** **Prospective Multi-Center Study of Reliability in Vascular Laboratory Testing of Venous Reflux**
F. Lurie
Kistner Vein Clinic and University of Hawaii, Honolulu, HI

8:40 AM – 9:00 AM **8** **Reflux Time on Air Plethysmography Is Shortened in Patients with Worsening Chronic Venous Insufficiency**
C.R. Lattimer, M. Azzam, E. Kalodiki, G. Geroulakos
Ealing Hospital & Imperial College, London, United Kingdom

9:00 AM – 9:20 AM **9** **Incidence and Risk Factors for Development of Varicose Veins in the General Population: Edinburgh Vein Study**

L.A. Robertson[1], S. Boghossian[1], C.J. Evans[2], A.J. Lee[3], P.L. Allan[1], V. Ruckley[1], F.G.R. Fowkes[1]

[1]University of Edinburgh, Edinburgh, United Kingdom, [2]NHS Lothian, Edinburgh, United Kingdom, [3]University of Aberdeen, Aberdeen, United Kingdom

9:20 AM – 9:40 AM **10** **Progression of Chronic Venous Disorders—Results from the Bonn Vein Study**

F. Pannier[1], E. Rabe[2]

[1]University of Cologne, Cologne, Germany, [2]University of Bonn, Bonn, Germany

9:40 AM – 10:00 AM **AMERICAN VENOUS REGISTRY**

B.K. Lal, MD

10:00 AM – 10:45 AM **Coffee Break — Visit Exhibits**

10:45 AM – 12:00 PM **SCIENTIFIC SESSION III**

Quick Shot

Moderators: *Marc Passman, MD*
Robert McLafferty, MD

Q1 **Trends in Patient Reported Outcomes of Conservative and Surgical Treatment of Primary Chronic Venous Disease Contradict Current Practices**

F. Lurie, R.L. Kistner

Kistner Vein Clinic and University of Hawaii, Honolulu, HI

Q2 **Hyperlipidemia and Deep Vein Thrombosis: The Role of PAI 1**

J.A. Diaz, A.H. Hawley, N. Ballard-Lipka, D.M. Farris, A.L. Rodriguez, S.K. Wrobleski, D.D. Myers, Jr., P.K. Henke, D.A. Lawrence, T.W. Wakefield

University of Michigan, Ann Arbor, MI

Q3 Non-Interruption of Warfarin Therapy Is Safe and Does Not Compromise Outcome in Patients Undergoing Endovenous Laser Therapy (EVLT)

P.J. Riesenman, S.G. Konigsberg, K. Kasirajan
Emory University, Atlanta, GA

Q4 Do We Still Need to do Stripping and Phlebectomy?

S.M. Belentsov
City Cllinic Hospital #40, Yekaterinburg, Russian Federation

Q5 Great Saphenous Vein (GSV) Diameter Does Not Correlate with Worsening Quality of Life Scores in Patients with GSV Incompetence

K. Gibson[1], D. Wright[2]
[1]Lake Washington Vascular Surgeons, Bellevue, WA, [2]BTG, London, United Kingdom

Q6 Vena Cava Filter Practices: Survey Results from a Large Regional Vascular Surgery Society

M. Friedell[1], P. Nelson[2]
[1]Orlando Health, Orlando, FL, [2]University of Florida College of Medicine, Gainesville, FL

Q7 Activation of Hypoxia-Inducible Factor Pathway in Varicose Veins

C.S. Lim[1], S. Kiriakidis[1], A. Sandison[2], P. Singh[2], E. Paleolog[1], A.H. Davies[1]
[1]Imperial College London, London, United Kingdom, [2]Imperial College Healthcare NHS Trust, London, United Kingdom

Q8 Characteristics of Temporary Inferior Vena Cava (IVC) Filters Non-Retrieval

J. Stevens, J. Cho, M. Makaroun, B. McDaniel, E. Dillavou, L. Marone, R. Rhee, R.A. Chaer
UPMC, Pittsburgh, PA

Q9 The Use of Intravascular Ultrasound for Diagnosis and Treatment of Innominate Vein and Superior Vena Cava Obstruction

C. Glass, A. Doyle, M. Dugan, K. Illig, D. Gillespie
University of Rochester Medical Center, Rochester, NY

Q10 Ultrasound Enhanced Thrombolysis for the Management of Deep Venous Thrombosis

C.J. Smolock, J.E. Anaya-Ayala, I. Birnbaum, Z.F. Cheema, F.A. Syed, J.J. Naoum, A.B. Lumsden, E.K. Peden, M.G. Davies
Methodist DeBakey Heart & Vascular Center, Houston, TX

Q11 Novel Repair of An External Iliac Vein Aneurysm

A. Jayaraj, M. Meissner
UW, Seattle, WA

Q12 Efficacy and Safety of Foam Sclerotherapy: Is Ultrasound-Guided Foam Sclerotherapy Always Necessary?

T. Yamaki, A. Hamahata, D. Fujisawa, H. Konoeda, K. Kubo, M. Nozaki, H. Sakurai
Tokyo Women's Medical University, Tokyo, Japan

Q13 C6 Clinical Class Chronic Venous Diseases: Minimally Invasive Approaches, Immediate Results and Follow-Up

S.M. Belentsov
City Clinic Hospital #40, Yekaterinburg, Russian Federation

Q14 Advantages of Tumescent Local Anesthesia with Bicarbonate for Pain, Bleeding, and Quality of Life During Surgery for Varicose Veins: A Prospective Study

P. Pittaluga, S. Chastanet
Riviera Veine Institut, Nice, France

Q15 Cyanoacrylate Adhesive for the Closure of Truncal Veins: 60-Day Swine Model Results

J.I. Almeida[1], R.J. Min[2], R. Raabe[3], D.J. McLean[4], M. Madsen[5]
[1]Miami Vein Center, Miami, FL, [2]Weill Cornell Medical College, New York City, NY, [3]Sacred Heart Medical Center, Spokane, WA, [4]Washington State University, Pullman, WA, [5]Inland Imaging, Spokane, WA

12:00 PM – 1:30 PM **Lunch on Own**

1:30 PM – 3:00 PM **BEST OF NON-JVS PAPERS**
Moderator: Gregory Moneta, MD

3:00 PM – 3:30 PM **Coffee Break — Visit Exhibits**

3:30 PM – 4:50 PM **SCIENTIFIC SESSION IV**
Compression
Moderators: Peter Neglen, MD
William Marston, MD

3:30 PM – 3:50 PM **11** **Effect of Compression Therapy on Leg Veins Anatomy: Quantification by 3D Vectorial Modelling from MRI Slices**
J. Uhl[1], H. Partsch[2], G. Mosti[3]
[1]Université Paris Descartes, Paris, France, [2]University of Vienna, Vienna, Austria, [3]Hospital, Lucca, Italy

3:50 PM – 4:10 PM **12** **Compression Therapy in Mixed Ulcers: Search for a Safe Pressure Range Not Affecting Arterial Inflow**
G. Mosti[1], H. Partsch[2]
[1]Clinica MD Barbantini, Lucca (LU), Italy, [2]Private Practice, Wien, Austria

4:10 PM – 4:30 PM **13** **Venous Lymphedema**
S. Raju[1], B. Furrh, IV[2], P. Neglén[2]
[1]University of Mississippi Medical Center/River Oaks Hospital, Jackson, MS, [2]River Oaks Hospital, Flowood, MS

4:30 PM – 4:50 PM **14** **Reduced Expression of Soluble Urokinase Receptor Fragment DII-III Predicts Venous Ulcers that Fail to Heal**
A. Ahmad[1], M. Waltham[1], G. Høyer-Hansen[2], T.T. Sørensen[2], K. Mattock[1], P. Saha[1], B. Modarai[1], H. Zayed[1], A. Smith[1]
[1]King's College London, London, United Kingdom, [2]Finsen Laboratory, Copenhagen, Denmark

4:50 PM – 5:10 PM **VENOUS FORUM/ROYAL SOCIETY OF MEDICINE (BEST PAPER)**

Randomised Clinical Trial Comparing VNUS® ClosureFAST™ Versus Laser for Varicose Veins (VALVV): Duplex and Quality of Life Outcomes as 6 Months

A.C. Shepherd, M.S. Gohel, L.C. Brown, A.H. Davies
Imperial College, Academic Section of Vascular Surgery, London, England

5:15 PM – 6:30 PM **POSTER SESSION**
Moderator: Joseph Raffetto, MD

FRIDAY, FEBRUARY 25, 2011

7:00 AM – 7:30 AM **Continental Breakfast — Exhibits Open**

7:30 AM – 9:00 AM **SCIENTIFIC SESSION V**
Chronic Venous Disease (Ulcers)
Moderators: *Harold Welch, MD*
Michael Vasquez, MD

7:30 AM – 7:50 AM **15** **Failure of Microvenous Valves in Small Superficial Veins—A Key to the Development of Venous Ulcers**
A.M. van Rij, J. Vincent, G. Hill, G.T. Jones
Department of Surgery, University of Otago, Dunedin, New Zealand

7:50 AM – 8:10 AM **16** **A Comparison of the Villalta and Venous Clinical Severity Scoring Instruments in the Assessment of Post Thrombotic Syndrome**
A. Jayaraj, C. Natiello, S. Nicholls, M. Meissner
University of Washington, Seattle, WA

8:10 AM – 8:30 AM **17** **The Need for an Intersociety Consensus Guideline for Venous Ulcer**
T.F. O'Donnell, Jr.
Tufts Medical Center, Boston, MA

8:30 AM – 8:50 AM **18** **Axial Transformation of the Profunda Vein Sustains Ilio-Caval Stenting in Postthrombotic Limbs**
P. Neglén, B. Furrh, IV, S. Raju
River Oaks Hospital, Flowood, MS

8:50 AM – 9:10 AM **19** **Role of Vein Tissue Nitric Oxide and Hyperpolarization in Venous Relaxation: Implications in Venous Insufficiency Disease**
J.D. Raffetto[1], O.M. Reslan[2], R.A. Khalil[2]
[1]VA Boston HCS, West Roxbury, MA, [2]Brigham and Women's Hospital, Boston, MA

9:10 AM – 9:20 AM **ACP PLATINUM ABSTRACT**

Combined Use of Pretest Clinical Probability Score and Latex Agglutination D-Dimer Testing in Excluding Acute Deep Vein Thrombosis
T. Yamaki, A. Hamahata, M. Nozaki, H. Sakurai
Tokyo Women's Medical University, Tokyo, Japan

9:20 AM – 10:00 AM **Coffee Break — Visit Exhibits**

10:00 AM – 10:25 AM **PRESIDENT'S SESSION**
Moderators: Peter J. Pappas, MD
Seshadri Raju, MD

10:00 AM – 10:15 AM **2010 SERVIER TRAVELING FELLOWSHIP REPORTS**
Christopher Pannucci, MD
University of Michigan

K. Barry Deatrick, MD
University of Michigan

10:15 AM – 10:25 AM **2010 BSN JOBST RESEARCH WINNER – INTERIM REPORT**

A Novel in Vitro Model of Chronic Venous Insufficiency
Yanjie Qi, MD
University of Rochester

10:25 AM – 10:40 AM **Presidential Address Introduction**
Introduction By: Seshadri Raju, MD
President-Elect

10:45 AM – 11:30 AM **PRESIDENTIAL ADDRESS**
Peter J. Pappas, MD

11:30 AM – 12:30 PM **MEMBER BUSINESS LUNCHEON**

12:30 PM **Free Afternoon**
Golf/Tennis Tournaments

SATURDAY, FEBRUARY 26, 2011

7:00 AM – 7:30 AM **Continental Breakfast — Visit Exhibits**

7:30 AM – 9:50 AM **SCIENTIFIC SESSION VI**
CVD – Treatment of Superficial Venous Disease
Moderators: *David Gillespie, MD*
M. Ashraf Mansour, MD

7:30 AM – 7:50 AM **20** **Change in Venous Outflow Patterns of the Leg After High Ligation and Stripping of Great Saphenous Vein and Phlebectomies**
T. Ogawa, S. Hoshino
Fukushima Daiichi Hospital, Fukushima, Japan

7:50 AM – 8:10 AM **21** **Validation of a New Duplex Derived Effectiveness Score in Quantifying Varicose Vein Treatments**
C.R. Lattimer[1], E. Kalodiki[1], M. Azzam[2], P. Trueman[3], G. Geroulakos[1]
[1]Ealing Hospital & Imperial College, London, United Kingdom, [2]Ealing Hospital, Middlesex, United Kingdom, [3]Brunel University, Middlesex, United Kingdom

8:10 AM – 8:30 AM **22** **The State of Endovenous Ablation for Venous Insufficiency in Florida**
M.S. Hong, K. Butler, T.D. Fischer, P.R. Nelson
University of Florida, Gainesville, FL

8:30 AM – 8:50 AM **23** **Randomized Controlled Trial of Ultrasound Guided Foam Sclerotherapy Combined with Sapheno-Femoral Ligation Compared to Surgical Treatment of Varicose Veins: Five-Year Results**
E. Kalodiki[1], M. Azzam[2], C.R. Lattimer[1], E. Shawish[2], N. Zambas[2], G. Geroulakos[1]
[1]Ealing Hospital & Imperial College, London SW7, [2]AZ, United Kingdom, [2]Ealing Hospital, Middlesex, United Kingdom

8:50 AM – 9:10 AM **24** **A New Approach to the Genetics of Varicose Veins: A Genome Wide Association Study**
A.M. van Rij, J. Krysa, G.T. Jones
University of Otago, Dunedin, New Zealand

EUROPEAN VENOUS FORUM BEST PAPER 1

Withdrawn

EUROPEAN VENOUS FORUM BEST PAPER 2

Withdrawn

9:15 AM – 10:00 AM **Coffee Break — Last Chance to Visit Exhibits**

10:00 AM – 11:00 AM **UPDATE SESSION**
Moderator: Peter Pappas, MD

10:00 AM – 10:15 AM **PVS Ulcer Initiative**
Peter Henke, MD

10:15 AM – 10:30 AM **AVF Website Launch**
Marc Passman, MD

10:30 AM – 10:40 AM **AVF National Screening Program**
Marc Passman, MD

10:40 AM – 10:50 AM **Fellows' Courses in Venous Disease**
William Marston, MD

10:50 AM – 11:00 AM **Attendings' Course in Venous Disease**
Antonios Gasparis, MD

11:00 AM – 11:45 AM **D. EUGENE STRANDNESS MEMORIAL LECTURE**

Microcirculary and Lymphatic Disorders
David C. Zawieja, PhD
Director, Division Division of Lymphatic Biology, Texas A&M Health Science Center College of Medicine
Introduction By: Peter Pappas, MD

12:00 PM – 1:15 PM **LUNCH SYMPOSIUM**

Changing Concept in Lymphedema
B.B. Lee, MD

1:30 PM – 3:05 PM **SCIENTIFIC SESSION VII**
Deep Vein Thrombosis II
Moderators: Peter Henke, MD
Seshadri Raju, MD

1:30 PM – 1:50 PM **25** **Thrombolytic Therapy with Tissue Plasminogen Activator: Why Prolonged Continuous Infusion Is Not the Best Approach**
R. Chang, J.N. Lozier, M.K. Horne, III
NIH, Rockville, MD

1:50 PM – 2:10 PM **26** **Postoperative Deep Vein Thrombosis in Total Knee or Hip Replacement Operation Is Associated with Preoperative Increased Calf Muscle Deoxygenation**
T. Yamaki, A. Hamahata, D. Fujisawa, H. Konoeda, K. Kubo, M. Nozaki, H. Sakurai
Tokyo Women's Medical University, Tokyo, Japan

2:10 PM – 2:30 PM **27** **Patient Characteristics, Referral Patterns, and Associated Risk Factors in Patients Referred to an Outpatient Vascular Laboratory to Rule Out Deep Venous Thrombosis (DVT)**
K. Gibson[1], N.L. Polissar[2], M.B. Neradilek[2]
[1]Lake Washington Vascular Surgeons, Bellevue, WA, [2]The Mountain-Whisper-Light Statistics, Seattle, WA

2:30 PM – 2:50 PM **28** **Mode of Thrombolytic Therapy and Residual Obstruction Do Not Affect Valve Function**
D. Vogel[1], E. Walsh[1], J.T. Chen[2], A.J. Comerota[1]
[1]The Toledo Hospital, Toledo, OH, [2]Bowling Green State University, Bowling Green, OH

2:50 PM – 3:05 PM **Coffee Break**
(Foyer)

3:05 PM – 5:05 PM **ASK THE EXPERTS**

Post Thrombotic Syndrome
Moderators: *Peter Henke, MD*
Robert McLafferty, MD
Peter Neglen, MD
Anthony Comerota, MD
Mark Meissner, MD

6:45 PM – 7:15 PM **Cocktail Reception**

7:15 PM – 10:30 PM **THE FORUM FINALE**
Awards, Dinner, Entertainment & More

AMERICAN VENOUS FORUM
23rd ANNUAL MEETING
February 23–26, 2011

Hilton San Diego Bayfront ■ San Diego, California

WEDNESDAY, FEBRUARY 23, 2011

7:00 AM – 8:00 AM **Continental Breakfast**

8:00 AM – 12:00 PM **DAVID S. SUMNER VENOUS SUMMIT**

Introduction: *Peter J. Pappas, MD, President*
Seshadri Raju, MD, President-Elect

Please Note: The annual Postgraduate Course that has preceded the meeting for several years will henceforth be known as the David S. Sumner Venous Summit to honor his monumental contributions that have facilitated understanding of venous hemodynamics.

Educational Objectives: Most vascular surgeons are at least superficially familiar with flow principles in arteries. The fluid dynamics in veins are radically different because they are collapsible tubes. Clinical venous problems are so interconnected with fundamental venous flow dynamics, that meaningful advances in analysis and treatment of these conditions are not possible without an understanding of basic principles. At the conclusion of this session, participants will have a better understanding of these principles.

8:05 AM – 8:50 AM **1. Flow in Collapsible Tubes**

Speaker: *Roger D. Kamm, PhD*
Massachusetts Institute of Technology

Tube Law: Non Linear Volume-Pressure Relationship; Steady, Quasi-Steady, Unsteady and

Time Variant Flows: Which One for Leg Veins?; Wave Speed: Why it Is Important in Venous Flow?; Differences Between Normal and Stiff (Post-Thrombotic) Veins: Subcritical and Supercritical Flow Regimens; The Starling Resistor; Water Hammer; Pump Theory and Modeling

8:50 AM – 9:35 AM **2.** **Venous Energetics**
Speaker: *Sheldon Magder, MD*
McGill University

Static, Dynamic and Gravitational Pressure in Veins: Supine and Erect; Corresponding Changes in Arterial Inflow; The Veno-Arterial Reflux (Bayliss); Lateral and Total Pressure – Bernouli Equation; Interchangeability of Pressure and Velocity; Is Velocity Head Important in Veins? Minimal Energy Loss in Veins Due to Absence of Peripheral Resistance Equalant; Implications for Determining Critical Venous Stenosis; What Is Hip? Zero Pressure (Gauge) Level in the Circulation; Tissue (Perivenous) Pressure: Postural Differences in Abdomen and Leg Veins and Why They are Different; Interstitial Fluid and Edema; Gel and Free Fluid; Protective Mechanisms; Proteoglycan Tissue Segmentation

9:35 AM – 10:05 AM **3.** **Biomechanical Applications in Venous Flow**
Speaker: *Aleksander Popel, PhD*
Johns Hopkins University

Gravity Flow in Open and Closed Systems; IVC Flow in the Abdomen and Thorax; Is it a Starling Resistor?; Vascular Waterfall; Is Venous Flow Effectively "Disconnected" from Arterial?

10:05 AM – 10:20 AM **Coffee Break**

10:20 AM – 11:00 AM **4.** **Flow Dynamics in Capillaries and Veins**
Speaker: *Geert Schmid-Schönbein, PhD*
University of California, San Diego

Principles of Network Flow; The 3 Reservoir Problem; Flow Modeling in Microcirculation; Network Adaptation to Shear Stress; Microcirculatory Damage

11:00 AM – 11:35 AM **5. Microcirculation**

Speaker: *F.E. Curry, PhD*
University of California, Davis

The New Modified Starling Forces Equation and the Lymphatic Paradox; Reynolds Numbers in Venous Flow; Viscosity in Microcirculation and Veins and Related Energy Losses; Capillary Recruitment and Vasomotion

11:35 AM – 12:00 PM **Panel Q&A**

12:00 PM – 1:30 PM **ACP LUNCH SYMPOSIUM**

Understanding Venous Hemodynamics Through Ultrasound

Moderator: *Nick Morrison, MD*

Educational Objectives: At the completion of this session, participants should be able to:

1. Interpret duplex hemodynamic information and avoid misdiagnosis.
2. Identify the best image for endovenous ablation, accommodate concomitant deep and superficial venous disease, and differentiate incompetent and pathologic perforator veins.

Role of the Vascular Lab Pre- and Post-Venous Intervention
John Mauriello, MD

Optimized Imaging for Endovenous Ablation
Joe Zygmunt, RVT

Concomitant Deep/Superficial Venous Disease
Stephen F. Daugherty, MD

Venous Hemodynamics: Practical Guide for Best Outcomes
Diana Neuhardt, RVT

Consequences of Venous Ultrasound Misdiagnosis
Stephanie Dentoni, MD

Patient Communication Regarding Ultrasound Findings
Helane Fronek, MD, FACP, FACPh

Perforators—Innocent or Pathologic?
Nick Morrison, MD

1:30 PM – 3:10 PM **SCIENTIFIC SESSION I**
Deep Vein Thrombosis I
Moderators: *Peter Pappas, MD*
Antonios Gasparis, MD

Educational Objectives: At the end of this session, the attendee will have an understanding of the importance of thrombolysis on decreasing recurrence of deep vein thrombosis, recognize risk factors for deep vein thrombosis and be able to create a venous thromboembolism (VTE) risk score for outpatient surgery.

1:30 PM – 1:50 PM **1** **Association of Blood Transfusion and Venous Thromboembolism in the Perioperative Period**

E.S. Xenos, D.L. Davenport
University of Kentucky Medical Center and VA Medical Center, Lexington, KY

BACKGROUND: Red blood cell (RBC) transfusion is a common event in the perioperative course of patients undergoing surgery. Transfused blood can disrupt the balance of coagulation factors and modulates the inflammatory cascade. Since inflammation and coagulation are tightly coupled we postulated that RBC transfusion may be associated with the development of venous thromboembolic phenomena. We queried the American College of Surgeon's National Surgical Quality Improvement Program (ACS NSQIP) database to examine the relationship between intraoperative blood transfusion and development of venous thromboembolism (VTE) in patients undergoing colorectal resection for cancer.

METHODS: We analyzed the data from 2005 to 2008 for patients undergoing colorectal resections for cancer based on the primary procedure CPT-4 code and operative ICD-9 diagnosis code. The primary outcome was 30-day deep vein thrombosis (DVT) and/or pulmonary embolism (PE). Intraoperative transfusion of RBC's was categorized as: none, 1–2 units, 3–5 units and 6 units or more. DVT/PE occurrences were analyzed by multivariable forward stepwise regression (p for entry < .05, for exit > .10) to identify independent predictors of DVT.

RESULTS: The database contained 21943 colorectal cancer resections. The DVT rate was 1.4% (306/21943) and the PE rate was 0.8% (180/21943). Patients were diagnosed with both only 40 times and the combined DVT or PE rate was 2.0% (446/21943). After adjusting for age, gender, race, ASA (American Society of Anesthesiologists) class, emergency procedure, operative duration and complexity of the procedure (based on Relative Value Units, RVU's), along with six clinical risk factors, intraoperative blood transfusion was a significant risk fact for the development of VTE as shown in the following table. Preoperative hematocrit did not enter the multivariable model as an independent predictor of VTE, nor did open versus laparoscopic resection. The risk for the outcome increased with increasing number of units transfused.

Intraoperative Transfusion PRBCs	30-Day VTE Rate	Multiv. Adj. Odds Ratio	95% Confidence Interval		P-Value
0 U (n = 19588, 89.3%)	1.8%	Reference			
1–2 U (n = 1751, 8.0%)	3.7%	1.393	1.047	1.854	.023
3–5 U (n = 466, 2.1%)	4.9%	1.883	1.187	2.985	.007
6+ U (n = 138, 0.6%)	9.4%	3.189	1.704	5.966	.000

CONCLUSION: In this study of 21943 patients undergoing colorectal resection for cancer blood transfusion is associated with increased risk of VTE. This increased thrombotic risk may be related to dilution of anticoagulant factors, viscosity changes, immunologic effects as well as the formation of microaggregates which are composed of degenerating platelets, granulocyte debris and fibrin strands; these from rapidly during blood storage. Malignancy and surgery are known prothrombotic stimuli. The subset of patients receiving intraoperative RBC transfusion are even more at risk for VTE emphasizing the need for sensible use of transfusions and rigorous thromboprophylaxis regimens.

1:50 PM – 2:10 PM **2** **Down-Regulation of Hypoxia-Inducible Factor 1 Alpha Reduces Venous Thrombus Resolution**

C.E. Evans, J. Humphries, K. Mattock, M. Waltham, A. Wadoodi, P. Saha, B. Modarai, A. Smith
King's College London, London, United Kingdom

BACKGROUND: Hypoxia-inducible factor 1 (HIF1)-mediated angiogenic factors such as vascular endothelial growth factor (VEGF) are induced within venous thrombus during its resolution, but the primary stimulus for VEGF production and thrombus resolution is unknown. Our aim was to determine whether downregulating HIF1α in the thrombus and vein wall reduces angiogenic factor expression, inflammatory cell infiltration, and thrombus resolution.

METHODS: Thrombus was induced in the inferior vena cava (IVC) of 40 mice. The mice were treated with the HIF1α inhibitor 2-methoxyestradiol (2ME, i/p, 150 mg/kg/day) or vehicle control (n = 20/group). HIF1α, VEGF, and placental growth factor (PLGF) expression in the thrombus and IVC were measured at days 1 and 10 (n = 7/group) by enzyme-linked immunosorbent assay (ELISA). Thrombus size, neovascularisation, recanalisation, and macrophage and neutrophil infiltration were also measured at day 10 by image analysis (n = 6/group).

RESULTS: The levels of HIF1α ($P < 0.001$), VEGF ($P < 0.001$), and PLGF ($P < 0.001$), and neutrophil ($P < 0.005$) and macrophage ($P < 0.05$, Figure 1) infiltration were decreased in the thrombus of mice treated with 2ME compared with vehicle control. The levels of HIF1α ($P < 0.005$), VEGF ($P < 0.005$), and PLGF ($P < 0.001$), and neutrophil ($P < 0.01$) and macrophage ($P < 0.005$, Figure 2) infiltration were also decreased in the IVC wall surrounding the thrombus of 2ME-treated mice compared with controls. Thrombus weight ($P < 0.001$, Figure 3) and size ($P < 0.02$, Figure 4) were increased, while thrombus neovascularisation ($P < 0.005$, Figure 5) and vein recanalisation ($P < 0.005$, Figure 6) were decreased in 2ME-treated mice compared with controls.

Figure 1

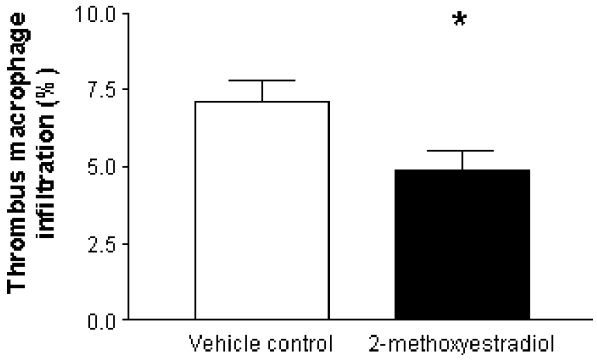


Figure 2

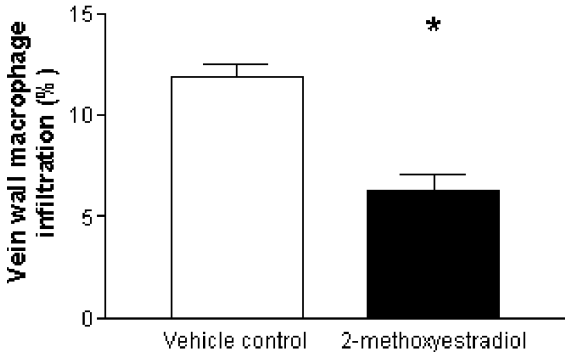


Figure 3

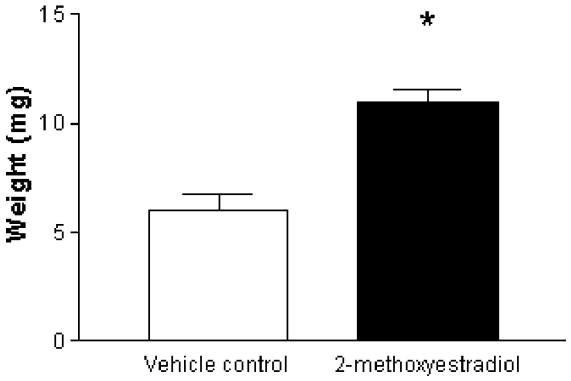


Figure 4

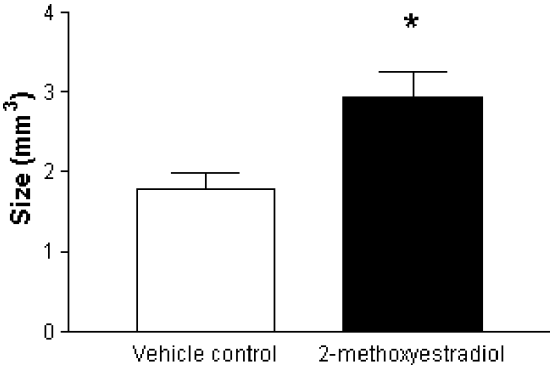


Figure 5

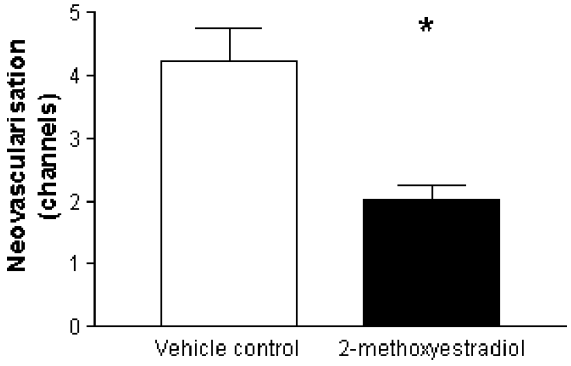
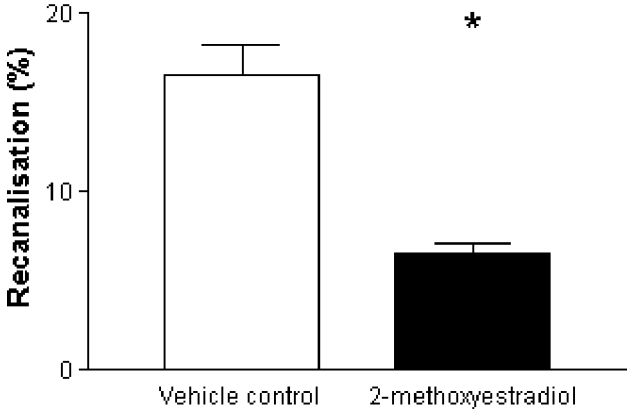


Figure 6



CONCLUSIONS: Reducing HIF1α expression in the thrombus and vein wall reduces angiogenic growth factor expression, inflammatory cell infiltration, and thrombus resolution. These data suggest that HIF1α activity is an important regulatory mechanism in thrombus resolution.

2:10 PM – 2:30 PM **3** **Catheter-Directed Thrombolysis of Iliofemoral DVT Reduces DVT Recurrence**

F. Aziz, J.T. Chen, A.J. Comerota
The Toledo Hospital, Toledo, OH

BACKGROUND: Iliofemoral DVT is a result of a major thrombotic stimulus that results in substantial postthrombotic morbidity in patients treated with anticoagulation alone. It has been shown that iliofemoral DVT is a powerful and an independent risk factor for recurrent thromboembolism. Furthermore, recurrent DVT escalates postthrombotic morbidity since recurrent DVT has been correlated with residual venous thrombus. Eliminating thrombus in high-risk patients might reduce the risk of recurrence. We reviewed the entire cohort of patients with iliofemoral DVT treated with catheter-directed thrombolysis to determine their risk of recurrence and whether there was a relationship to lytic success.

METHODS: All patients who underwent catheter-based thrombus removal for iliofemoral DVT had their degree of lysis assessed by comparison of pre- and post-procedural phlebography and were classified according to the percentage of residual thrombus. Recurrence was defined as a symptomatic presentation with image verification of new or additional thrombus.

RESULTS: Sixty-two patients underwent catheter-directed thrombolysis for iliofemoral DVT. Mean age was 45 ± 17 years (range 16–79), and 35 patients (56%) were male. Mean follow-up was 18.9 months and 7 patients (11%) were lost to follow-up. Forty-eight patients (87%) had no recurrence and 7 patients (13%) developed recurrent DVT. Recurrence developed in 67% of patients who had >60% residual thrombus but occurred in 10% of those who had <60% residual thrombus (P = .0253). The 7 patients who developed recurrent DVT were anticoagulated at the time of recurrence.

CONCLUSION: Patients who underwent either catheter-directed thrombolysis of iliofemoral DVT have lower incidence of recurrent DVT as compared to historic groups who are treated by anticoagulation alone. Furthermore, there is a direct correlation between the amount of residual thrombus following catheter-based thrombolysis and recurrence. As lytic success improves, the risk of recurrent DVT decreases. These data raise the hypothesis that successful thrombolysis reduces recurrence rates, which requires validation in prospective studies.

2:30 PM – 2:50 PM **4** **Creation of a Simple Venous Thromboembolism Risk Score for Outpatient Surgery: Analysis of the NSQIP Database**

C.J. Pannucci, A. Shanks, M. Moote, V. Bahl, P. Cederna, N. Naughton, P. Henke, S. Kheterpal, S. Campbell
University of Michigan, Ann Arbor, MI

BACKGROUND: Factors which contribute to venous thromboembolism (VTE) risk after outpatient surgery are unknown. We used the National Surgical Quality Improvement Program (NSQIP) database to examine risk factors for VTE after outpatient surgery and empirically derive a VTE risk-scoring model.

METHODS: NSQIP is a prospective database of surgical patients with 30-day outcomes. Inclusion criteria for this analysis were age ≥18, surgery classified as "outpatient", and length of stay equal to zero days.

Independent variables included known VTE risk factors (Table 1). Age, operative time, and body mass index were transformed to categorical variables to facilitate risk-score creation.

Trained NSQIP clinical nurses collect risk factor and adverse event data using medical record review. Mandatory nurse-patient contact on post-operative day 30 identifies complications treated at other hospitals. NSQIP defines deep vein thrombosis (DVT) as venous clots requiring either systemic anticoagulation or IVC filter. Pulmonary embolus (PE) is defined as an obstructing pulmonary arterial clot. Imaging is required for DVT or PE diagnosis. The primary study outcome was VTE, generated as a composite of DVT and/or PE.

Multivariable logistic regression identified independent risk factors. ß-coefficients for independent predictors were used to derive a weighted risk-scoring model; this was compared to the unweighted risk-scoring model using the c-statistic.

RESULTS: 168,518 patients met inclusion criteria. DVT incidence was 0.1% (172 patients), PE was 0.04% (38 patients) and VTE was 0.12% (210 patients). Of patients with VTE, 1 in 10 (0.013% overall) had both DVT and PE.

Independent predictors of VTE included arthroscopic surgery, current pregnancy, active cancer, and invasive venous procedure. When compared to the reference group, age 41–60, age > 60, BMI > 40, and operative time >120 minutes were also independent predictors (Table 1). The model accounted for 80% of the variability in VTE (c-statistic 0.800). The average time-to-event for both DVT and PE was post-operative day 10 ± 7.

C-statistic for weighted risk scores (0.76 ± 0.02) was significantly higher than the unweighted risk score (0.72 ± 0.02). The weighted risk-score model is shown in Figure 1.

Table 1

Risk Factor	Adjusted Odds Ratio (95% CI)	p-Value
Male gender	1.04 (0.77–1.40)	0.819
General anesthesia	1.38 (0.95–2.00)	0.091
Arthroscopic surgery	**4.87 (2.88–8.21)**	**<0.001**
Abdominal laparoscopy	1.15 (0.72–1.82)	0.555
Current pregnancy	**8.91 (1.11–71.24)**	**0.035**
Active cancer	**5.38 (2.33–12.41)**	**<0.001**
Congestive heart failure	3.73 (0.43–32.21)	0.231
Chronic obstructive pulmonary disease	1.24 (0.49–3.13)	0.648
Diabetes requiring medication	0.86 (0.51–1.45)	0.567
Central vascular disease	1.40 (0.84–2.33)	0.192
Peripheral vascular disease	0.73 (0.17–3.14)	0.642
Current smoker	0.99 (0.67–1.46)	0.940
Renal failure on dialysis	0.70 (0.16–3.09)	0.636
Prior operation within 30 days	1.09 (0.40–2.99)	0.872
Invasive venous procedure	**13.42 (9.56–18.84)**	**<0.001**
Age		
<40 years	Reference	---
41–60 years	**1.98 (1.24–3.16)**	**0.004**
>60 years	**2.57 (1.57–4.20)**	**<0.001**
Body mass index		
<25	Reference	---
25–40	1.04 (0.74–1.45)	0.836
>40	**1.85 (1.10–3.09)**	**0.019**
Total operative time		
<60 minutes	Reference	---
60–120 minutes	1.12 (0.82–1.52)	0.494
>120 minutes	**1.68 (1.02–2.77)**	**0.043**

Figure 1

Two Point Factors	Three Point Factors	Four Point Factors
☐ OR time >120 minutes ☐ BMI >40	☐ Age 41-60	☐ Age >60
Six Point Factors	**Eight Point Factors**	**Ten Point Factors**
☐ Active cancer ☐ Arthroscopic surgery	☐ Current pregnancy	☐ Invasive venous procedure

TOTAL SCORE _____

Total Score	30-day VTE Rate	Risk Level
0-2	<0.1%	Low
3-5	0.1%	Moderate
6-10	0.2-0.4%	High
≥ 11	Up to 1.3%	Very High

CONCLUSIONS: 30-day VTE risk can be quantified in the outpatient surgery population using a simple risk-scoring model. Aggressive chemoprophylaxis may be considered in patients with higher risk. However, further research is necessary to examine the risks, benefits, and cost of chemoprophylaxis for outpatient surgery.

2:50 PM – 3:10 PM **5** **Vena Caval Filters: Review of Indications and Practices at a University Hospital**

R.J. Meisner, N. Labropoulos, A.P. Gasparis, A. Tassiopoulos
Stonybrook University Hospital, Stony Brook, NY

BACKGROUND: Vena cava filter (VCF) use has been increasing in recent years particularly after the advent of retrievable filters. A significant number of VCFs appear to be utilized outside the defined indications for this procedure. This, so-called, prophylactic VCF placement, is particularly controversial, as it is not supported by solid clinical data but is applied liberally in "high-risk" patients (multiple trauma, morbid obesity). The aim of this study was to investigate the current practice on VCF use at our own institution.

METHODS: Consecutive patients with VCF placement over a 2-year period (2007 to 2009) at a university hospital were reviewed. Patient demographics, filter type, retrieval, complications, indications for the procedure, and department performing the procedure were collected in all patients.

RESULTS: A total of 244 patients underwent VCF placement of which 159 were retrievable and 84 were permanent. Fifty four percent of patients had the VCF placed for an absolute indication, 14% for a relative indication and 32% for prophylaxis. Of those patients with a retrievable filter only 14 (9%) had it removed. Eight patients had a complication of VCF placement while there were no complications of filter retrieval. Two of the 8 patients had a major complication: death and right ventricle injury. The other 6 complications were considered minor such as hematoma, filter migration, and misdeployment. The department of trauma and surgical critical care (TSCC) placed the majority of VCFs (n = 107) followed by vascular surgery (n = 77) and interventional radiology (IR) (n = 60). VCF placement for prophylaxis alone without absolute indication was 57% from the TSCC, 18.3% from IR ($p < 0.0001$) and 5.2% from vascular surgery ($p < 0.0001$ compared to TSCC and $p = 0.025$ compared to IR).

CONCLUSIONS: The results of this study indicate that as many as 32% of VCFs placed are placed for prophylaxis. A very low percent of VCFs is retrieved. The majority of VCFs at this university were placed by the trauma critical care department. These practices are not in accordance to strict evidence based guidelines for VCF placement. This is likely the practice at many other large US university based hospitals necessitating strategies for reducing their placement.

3:10 PM – 3:45 PM **Coffee Break**

3:45 PM – 5:15 PM **LIVE ULTRASOUND**

Panelists: *Nicos Labropoulos, MD*
Gail Size, BS, RVT, RVS, RPhS
Steve Elias, MD

Educational Objectives: At the completion of the session, participants should be able to:

1. Understand the technical principles of non-invasive venous diagnosis.
2. Develop a patient treatment plan based on diagnostic imaging.
3. Recognize post thrombotic and post procedural Duplex imaging findings.

6:00 PM – 7:30 PM **WELCOME RECEPTION**

NOTES

THURSDAY, FEBRUARY 24, 2011

7:00 AM – 8:00 AM **Continental Breakfast — Exhibits Open**

8:00 AM – 10:00 AM **SCIENTIFIC SESSION II**
Chronic Venous Disease: Epidemiology & Screening
Moderators: *Nicos Labropoulos, MD*
Bo Eklof, MD

Educational Objectives: At the completion of the session, participants should be able to:

1. Describe the role of body mass on prevalence of chronic venous disease.
2. Define the variance and factors associated with variance for venous reflux testing, as well as ways to incorporate standardization.
3. Describe the relationship of saphenous vein diameter and venous reflux testng, in the setting of chronic venous disease.
4. List 3 factors associated with varicose veins, and 3 factors associated with chronic venous disease progression.

8:00 AM – 8:20 AM **6** **Venous Disease and the Effects of Increasing Body Mass Index: Results from the National Venous Screening Program**

C.J. Moore[1], R.B. McLafferty[1], M. Lentz[2], J.R. Schneider[3], A. Roupenian[4], J. Heller[5], W. Bohannon[6], M. Passman[7]
[1]Southern Illinois University, Springfield, IL, [2]National Venous Screening Program, Baltimore, MD, [3]Central DuPage Hospital, Winfield, IL, [4]Vein and Laser Center NE, Plymouth, MA, [5]Johns Hopkins Vein Center, Baltimore, MD, [6]Scott & White Memorial Hospital and Clinic, Temple, TX, [7]University of Alabama, Birmingham, AL

OBJECTIVE: To determine differences in venous disease across a spectrum of body-mass index (BMI) from participants in the National Venous Screening Program (NVSP).

METHODS: Utilizing the prospectively maintained database from the NVSP, statistical analysis was performed to examine differences between participants according to standard BMI group designations. Data points for comparison

included demographics, thromboembolic (VTE) risk assessment, venous quality of life (CIVIQ2), duplex evaluation, CEAP classification, and venous clinical severity score (VCSS). A p-value of less than 0.01 was considered statistically significant.

RESULTS: From 2005 to 2010, the NVSP has screened 7227 Americans. Body mass index (BMI) category distribution included underweight (UW: BMI < 18.5)—1.3%; normal weight (NW: BMI 18.5–24.9)—34.9%; overweight (OW: BMI 25–29.9)—34.9%; obese (OB: BMI 30–34.9)—16.6%; morbidly obese (MOB: BMI 35–39.9)—7.8%; and super morbidly obese (SMOB: BMI > 40)—4.7%. Significant increases in BMI occurred incrementally in diabetes (NW: 4.9% to SMOB: 25.2%) and hypertension (NW: 22.9 to SMOB: 54.3%). Mean VTE risk assessment scores significantly increased incrementally (NW: 3.32 to SMOB: 4.12). Mean quality-of-life scores significantly increased incrementally (NW: 20.3 to SMOB: 29.02). This observation was related to differences in limitations of activity. Mean CEAP scores demonstrated significant incremental increases (NW: 1.49, OW: 1.54, OB: 1.64, MOB: 1.86, SMOB: 2.01). Mean VCSS scores significantly increased incrementally (NW: 2.80 to SMOB: 4.58). Duplex data demonstrated a significantly higher percentage of common femoral vein (CFV) reflux in the UW group compared to the overweight classes (UW: 26.04% to SMOB: 18.69%)

CONCLUSIONS: Americans participating in the NVSP show worsening of venous disease across most objective measures as BMI increases. The presence of CFV reflux appears to be less with increasing BMI, and brings in to question the appropriateness of this limited duplex exam in this population versus calf-muscle pump dysfunction. As obesity rates climb at a rapid rate, the morbidity and mortality from venous disease could markedly worsen. This information remains vital across all levels of health care and further provides objective data to promote programs to prevent obesity.

8:20 AM – 8:40 AM **7** **Prospective Multi-Center Study of Reliability in Vascular Laboratory Testing of Venous Reflux**

F. Lurie
Kistner Vein Clinic and University of Hawaii, Honolulu, HI

PURPOSE: To define basic properties of the duplex ultrasound diagnostic test for reflux in veins of lower extremities, and to examine if some of the elements of this test can be standardize in order to improve reproducibility.

METHODS: This is a prospective multi-center study sponsored by the American venous Forum Foundation. Vascular laboratories from 11 centers participated in protocol development, educational intervention and data collection.

Repeatability studies were performed as a duplicate test within 2 weeks between replicates performed by same technologist, at the same time of the day, using same reflux provoking maneuver, and with patient at the same position. Repeatability was separately examined for different combinations of patient position, reflux-inducing maneuvers, and time of the test.

Reproducibility was examined by two different technologists performed test at the same time of the day, using same reflux provoking maneuver, and with patient at the same position.

Facilitated Reproducibility was studied by examining the same patients by two different technologists immediately after an educational intervention at the central laboratory.

In order to examine potential for decreasing in variability of results, limits of agreement between two duplex scan were studied by changing three elements of the test: time of the day (morning vs. afternoon), patient's position (standing vs. supine), and reflux initiation (manual vs. automatic compression-decompression).

RESULTS: A total of 51 patients were examined by different technologists during four sessions at the central laboratory. At the time of this abstract submission, additional 68 patients were examined at participating laboratories.

Overall repeatability was 97.7% with higher values for superficial veins and lower for deep veins. Reproducibility was lower, which indicates a potential for improvement by standardization. This was confirmed by better reproducibility after educational intervention (Facilitated Reproducibility). The element of the test contributing to most significant variability was the time of the test.

CONCLUSIONS: Duplex ultrasound test for venous reflux is repeatable, but the reproducibility can be improved by standardization and training.

8:40 AM – 9:00 AM **8** **Reflux Time on Air Plethysmography Is Shortened in Patients with Worsening Chronic Venous Insufficiency**

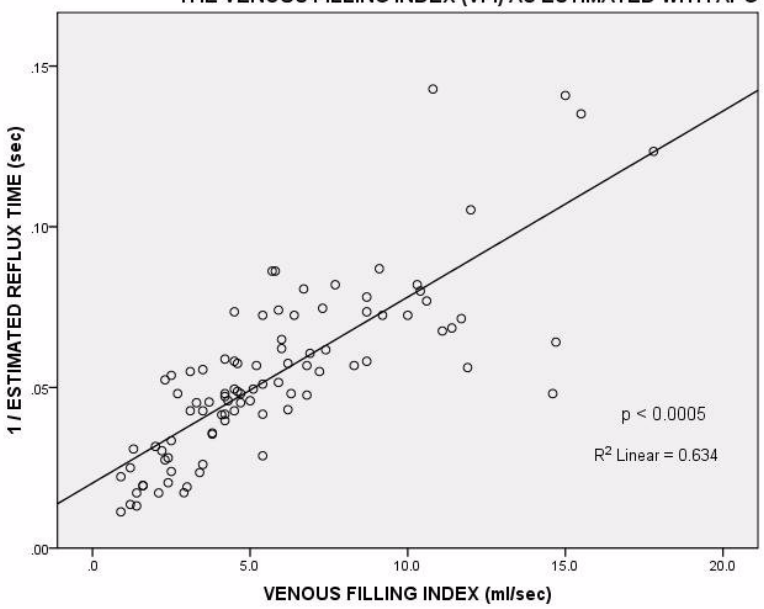
C.R. Lattimer, M. Azzam, E. Kalodiki, G. Geroulakos
Ealing Hospital & Imperial College, London, United Kingdom

BACKGROUND: The venous filling index (VFI), when elevated, is a measure of global venous reflux in the calf. Treatments which successfully abolish reflux normalize the value to under 2 ml/sec. Duplex derived reflux time (RT) at the sapheno-femoral junction (SFJ) has been shown to increase with disease severity and its reduction has been used as an indicator of treatment success. Our hypothesis is that the stimulus to arrest reflux occurs when the leg approaches venous capacity. The shorter the RT the sooner the leg is subjected to the effects of maximal venous hypertension. Our aim was to investigate the direction of the relationship between RT estimated from APG and the VFI, a validated indicator of disease severity.

METHODS: Ninety-three consecutive patients with primary SFJ reflux (>0.5 sec) and great saphenous vein (GSV) reflux (>0.5sec) awaiting endovenous treatment were included into the study. Patients with sapheno-popliteal junction incompetence, deep venous reflux or a history of a deep vein thrombosis were excluded. Baseline parameters (median, range) included age (48, 22–78), averaged GSV diameter at three sites (7.5 mm, 4–12) and the clinical component of the CEAP classification (C3, C2–C6). A gravitational challenge was applied to an 'emptied' leg with the venous reservoir supported with a sensor cuff at an initial pressure of 6 mmHg. Reflux time was estimated using the software (ACI MEDICAL®, SAN MARCOS, CA, 92078) as the time taken to reach 90% of the total venous volume (90TVV), from the rapid filling phase seen on the monitor.

RESULTS: Median and interquartile range (IQR) of RT and VFI were 20 sec (14) and 4.7 ml/sec (4.3) respectively. Using APG, estimated RT is inversely related to VFI in patients with chronic venous insufficiency ($p < 0.0005$, Spearman's rho, linear $R^2 = 0.634$) (GRAPH 1). Furthermore shorter RT's were observed in patients with worsening C scores ($p = 0.001$) and increasing GSV diameters ($p < 0.0005$).

GRAPH 1: THE INVERSE RELATIONSHIP BETWEEN REFLUX TIME (RT) AND THE VENOUS FILLING INDEX (VFI) AS ESTIMATED WITH APG



CONCLUSIONS: Increasing GSV diameters may contribute to a faster rate of reservoir refilling (VFI). This results in a shorter RT with a worsening C score. The effects of maximal venous hypertension occur after the end of RT when the reservoir is full, so RT measured by APG may indicate more severe disease. Simultaneous duplex and APG studies are required to support these findings.

9:00 AM – 9:20 AM **9** **Incidence and Risk Factors for Development of Varicose Veins in the General Population: Edinburgh Vein Study**

L.A. Robertson[1], S. Boghossian[1], C.J. Evans[2], A.J. Lee[3], P.L. Allan[1], V. Ruckley[1], F.G.R. Fowkes[1]

[1]University of Edinburgh, Edinburgh, United Kingdom, [2]NHS Lothian, Edinburgh, United Kingdom, [3]University of Aberdeen, Aberdeen, United Kingdom

BACKGROUND: Numerous studies have reported on the frequency of varicose veins in the general population (prevalence) and associated lifestyle factors but very few have investigated, longitudinally, the development of new varicose veins (incidence). The aim of this study was to measure the incidence of varicose veins in the adult population and identify factors that increase the future risk of an individual acquiring varicose veins.

METHODS: The study was a population cohort in which a random sample of 1566 men and women aged 18–64 years examined at baseline in the Edinburgh Vein Study, were invited to have a 13 year follow up examination. Assessment included: clinical classification of venous disease using CEAP, duplex scanning to assess incompetence of venous valves, body mass index (BMI), and questionnaire on venous history and lifestyle factors.

RESULTS: 880 study participants took part in the follow up study and underwent clinical examination (response rate = 60%). Of the 555 participants who had no trunk varicose veins at baseline, 101 developed C2 trunk varicose veins in any leg during the 13 year follow up. The annual incidence rate of trunk varicose veins was 1.35%. The incidence in men and women was similar with respective rates of 1.31% and 1.39% per year ($p = 0.45$). The risk of developing new varicose veins appeared to increase with age (incidence rate = 0.73% per annum in 18–34 years; 1.23% in 35–44 years; 1.62% in 45–54 years and 1.93% in 55–64 years). Increased BMI was also associated with incidence of varicose veins (incidence rate = 1.06% per annum in underweight; 1.28% normal weight; 1.41% overweight and 1.54% obese). The incidence rate in those with no family history of varicose veins was 1.07% compared to 1.60% in those with a family history but this finding was not statistically significant. Number of pregnancies was a significant risk factor with an incidence rate of 1.24% per year in women who had never been pregnant increasing to 2.11% per year in those who had been pregnant four times ($p = 0.02$).

CONCLUSION: The Edinburgh Vein Study is one of the first to measure the adult incidence of trunk varicose veins and to examine, longitudinally, factors which increase the risk of varicose veins. The associations found between risk factors and incident disease provides stronger evidence of causality than those in prevalence studies. Further analysis of other risk factors, including venous incompetence, and the impact of changing risk factors over time will be presented.

9:20 AM – 9:40 AM **10** **Progression of Chronic Venous Disorders—Results from the Bonn Vein Study**

F. Pannier[1], E. Rabe[2]
[1]*University of Cologne, Cologne, Germany,*
[2]*University of Bonn, Bonn, Germany*

BACKGROUND: Chronic venous disorders are among the most common diseases in Germany. In the Bonn Vein Study I (BVS I), conducted in 2000, 3072 participants of the general population of the city of Bonn and two rural townships, aged 18–79 years were took part in this study (1350 men, 1722 women). Participants were selected via simple random sampling from the registries of residents. In this follow-up study 6.6 years later, the same population was investigated again to. The aim was to identify the incidence and risk factors of progression of pre-existing CVD. In addition incidence and progression of venous symptoms were documented.

METHODS: From May 2007 to September 2008, we contacted all participants of BVS I and invited them for a reinvestigation. The participants answered a standardized questionnaire and were examined by clinical means and by duplex ultrasound in the same way as in BVS I.

RESULTS: The response at follow-up after 6.6 years was 84.6%. We reinvestigated 1978 participants. The prevalence for varicose veins rose from 22.7 to 25.1% and for CVI from 14.5 to 16%. Participants with C-Class C2 as a maximum at BVS I increased to higher C-classes in 19.8% (nonsaphenous VV) and in 31.8% (saphenous VV). In a multivariate analysis the main risk factors for were age, obesity and arterial hypertension.

CONCLUSIONS: These results show a high incidence of progression of CVD to higher C-classes.

9:40 AM – 10:00 AM **AMERICAN VENOUS REGISTRY**
B.K. Lal, MD

10:00 AM – 10:45 AM **Coffee Break — Visit Exhibits**

10:45 AM – 12:00 PM **SCIENTIFIC SESSION III**
Quick Shot
Moderators: *Marc Passman, MD*
Robert McLafferty, MD

Educational Objectives: At the completion of the session, participants should be able to:

1. Understand options for management of anticoagulation in patients undergoing endovenous ablation.
2. Evaluate impact of tumescent anesthesia techniques on varicose vein operations.
3. Differentiate role of different venous operation options for treatment of saphenous vein insufficiency and associated varicose veins, comparing newer minimally invasive options to traditional therapy.

Q1 Trends in Patient Reported Outcomes of Conservative and Surgical Treatment of Primary Chronic Venous Disease Contradict Current Practices

F. Lurie, R.L. Kistner
Kistner Vein Clinic and University of Hawaii, Honolulu, HI

OBJECTIVE: To analyze patient-reported quality of life (QOL) and symptoms in a prospective cohort of CVD patients who was managed within the framework of existing policies.

STUDY DESIGN: Prospective cohort study of 150 patients with C_{2-4} clinical class of primary chronic venous disease (CVD). Management consisted of initial conservative measures, following which, the patients were given a choice of continuing conservative therapy, or surgical treatment. Patients completed SQOR-V (Specific Quality of Life and Outcome Response-Venous) tool prior to initial visit, after completion of conservative treatment, and 1 and 12 month follow up visits after surgical treatment. Management consisted of initial conservative measures. QOL score and symptom score (SS) part of this instrument was analyzed separately.

RESULTS: Conservative treatment resulted in improvement of symptom score in 85 (57%) patients, and the QOL in 111 (74%) patients. Despite this improvement, the majority of patients (121) chose surgical option. At the one-month follow up after surgical treatment 97 (80%) patients reported significant improvement of their symptoms and 114 (94%) in the QOL compare to their status after conservative therapy. The QOL improvement was due mainly to improvement in symptom score. Patients who improved after conservative therapy were more

than 15 times more likely to have symptoms relief at one month (RR = 15.6, 95% CI 4.3–56.5), and 21 times higher at one year after surgery (RR = 21.3, 95% CI 4.7–96.9) compared to those who did not change the SS.

CONCLUSIONS: Surgical treatment resulted in a better relief of symptoms compare to conservative therapy. The relief of symptoms after conservative therapy predicts better outcomes of surgical treatment. These findings suggest that success of conservative therapy should be considered as an indication, and the failure of conservative therapy should not be an indication to surgical treatment.

Q2 Hyperlipidemia and Deep Vein Thrombosis: The Role of PAI 1

J.A. Diaz, A.H. Hawley, N. Ballard-Lipka, D.M. Farris, A.L. Rodriguez, S.K. Wrobleski, D.D. Myers, Jr., P.K. Henke, D.A. Lawrence, T.W. Wakefield

University of Michigan, Ann Arbor, MI

BACKGROUND: Hyperlipidemia increases the levels of plasminogen activator inhibitor 1 (PAI 1) which regulates fibrinolysis by inhibiting urokinase (uPA) and tissue plasminogen activator (tPA). While this fibrinolytic pathway is well known, the role of PAI 1 in venous thrombosis (VT) in hyperlipidemia has not been fully established. We sought to determine the effects of PAI 1 in an *in vivo* hyperlipidemic model of VT.

METHOD: C57BL/6 (WT) and ApoE-/- mice (with cholesterol levels of 4-fold elevation) were used. Inferior vena cava (IVC) ligation below the level of the renal veins was performed to create a stasis VT. Mice were harvested at acute (day 2 after surgery) and chronic (days 6 and 14 after surgery) time points. At euthanasia, blood samples were collected for total cholesterol level, plasmin activity assay, and PAI 1 activity. In addition, the IVC and its thrombus were evaluated for thrombus weight (TW), total uPA antigen, differential leukocyte count and ELISA for monocyte chemoattractant protein 1 (MCP 1), matrix metalloproteinase (MMP) 2 and MMP-9.

RESULTS: Acute VT: ApoE-/- demonstrated a statistically significant increase in TW, and a significant increase in circulating PAI 1 activity, while showing a non significant decrease in circulating plasmin activity compared to WT mice at day 2 (Table 1).

Chronic VT: ApoE-/- demonstrated undetectable levels of uPA in both vein wall and thrombus compared to WT mice at both day 6 and day 14. MMP-2 and MMP 9 were significantly decreased at chronic time points compared to WT mice (Table 1).

In addition, in ApoE-/- mice, MCP 1 was significantly decreased at both acute (day 2) and chronic (day 6) time points compared to WT mice. As expected, following a decrease MCP 1, monocyte recruitment was significantly decreased at days 6 and 14.

CONCLUSIONS: In this model of hyperlipidemic mice undergoing VT, increased PAI 1 contributes to an early increase in TW due to impaired fibrinolysis. Additionally, the increase in PAI 1 results in undetectable levels of uPA, decreased MMP 2 and 9, and lower levels of MCP 1 with decreased monocyte recruitment, thus impairing thrombus resolution. Taken together, in hyperlipidemic mice, PAI 1 is elevated, drives thrombogenesis and initiates a sequence of biological events that result in impaired thrombus resolution.

Supported by NIH 1PO1HL089407

Q3 Non-Interruption of Warfarin Therapy Is Safe and Does Not Compromise Outcome in Patients Undergoing Endovenous Laser Therapy (EVLT)

P.J. Riesenman, S.G. Konigsberg, K. Kasirajan
Emory University, Atlanta, GA

BACKGROUND: Oral anticoagulation with warfarin is routinely discontinued prior to venous surgery due to concerns for potential bleeding complications. Discontinuation of warfarin therapy may necessitate transition anticoagulation, before and after the surgical procedure, in order to minimize the risk of thrombotic complications from preexisting medical conditions. The practice of transition anticoagulation adds cost and complexity to the planning of the patient's intervention. No formal guidelines currently exist regarding the need or advisability of withdrawing warfarin therapy for patients undergoing minimally invasive endovenous laser therapy (EVLT) procedures.

METHODS: Between September 2004 and July 2010, 518 patients underwent 770 EVLT procedures on the lower extremity at our institution. Of these patients, 5 underwent a total of 12 separate lower extremity EVLT procedures for the treatment of symptomatic reflux in the greater and/or small saphenous veins without interruption of therapeutic warfarin therapy. The great saphenous vein was the target in 8 of these procedures, and the small saphenous vein was the target in 4 procedures. Concomitant phlebectomies were preformed during five of these interventions, and ultrasound-guided sclerotherapy was performed during an additional five procedures. Duplex ultrasound of the treated venous segments was performed in all patients within 1 week, and at approximately 8-weeks post-intervention.

RESULTS: No bleeding complications were observed during the procedure or in early follow-up. No patients developed a deep venous thrombosis. Complete ablation of the target vessel was observed in all patients on follow-up Duplex ultrasounds. One patient reported persistent severe lower extremity pain in follow-up at 7 weeks. The remaining 4 patients experienced significant resolution of their symptoms.

CONCLUSION: Our data indicated that EVLT can be safely performed in patients taking warfarin and that target vessel ablation is not compromised by oral anticoagulation. The minimally invasive nature of EVLT may make routine cessation of warfarin therapy unnecessary. Examination of additional EVLT cases is needed to determine whether clinical guidelines can safely assert that warfarin therapy should not be routinely interrupted in patients undergoing this procedure.

Q4 Do We Still Need to do Stripping and Phlebectomy?

S.M. Belentsov
City Cllinic Hospital #40, Yekaterinburg, Russian Federation

BACKGROUND: There is still an opinion that a surgery is the main method of treatment of patients with varicose veins, despite new opportunities such as Ultrasound Guided Sclerotherapy (UGFS), Radiofrequency Ablation (RFA), etc.

METHODS: We have analyzed our experience of treatment of patients with C2-C6 (CEAP). 1st group: 1417 pts (1648 Great Saphenous Veins (GSV)) who were underwent high flush ligation, GSV stripping and phlebectomy of side branches. 2nd–354 pts (421 GSVs) with high flush ligation, UGFS of GSV and compression sclerotherapy of varicose veins (VV). 3rd–654 pts (811 GSVs) with UGFS of GSV and sclerotherapy of VVs. 4th–110 pts (131 GSVs) were treated with the using RFA, UGFS of incompetent perforators and compression sclerotherapy of VVs. The groups were comparable on basic parameters.

RESULTS: All the patients of the four groups were released from varicose veins. Mean hospital length of stay in the first group was 5.1 ± 0.82 days, the mean sick leaves were 21.0 ± 3.41 days. Mean hospital length of stay in the second group was 1.1 ± 0.13 days, the mean sick leaves were 12.0 ± 5.53 days. The treatment of patients of 3rd and 4th groups was on outpatient basis and they weren't in need of sick-list.

There were 13.5% complications in the first group and no intact GSVs. The recurrence was 20.8% three years after surgery. There were 4% complications in the second group. Immediate results showed 99.5% occluded GSVs. The rate was 95.2% after 6 months, 100% after 1 year, 94.7% after 2 years, and 100% after 3 years. The third group: there were 4 (0.5%) complications, 98.8% GSVs were occluded 2 weeks after the procedure, 93.3% 6 months after, 95.2% 1 year after, 92.9% 2 years after, and 93.3% 3 years after. The recurrence was 2.1%, 11.9% and 21.4% after 1, 2 and 3 years accordingly. The forth group: all GSVs were occluded immediately after RFA (in one case we obtained a partly occlusion). The occlusion rate after 1, 2 and 3 years was 100% (in one case after 1 year we found a partly recanalization). The recurrence was 2.2%, 3.9% and 7.8% after 1, 2 and 3 years accordingly.

CONCLUSIONS: Our experience let us conclude the surgery is not the method of choice of varicose vein treatment now. The treatment should be based on minimally invasive approaches provided better results on an outpatient basis.

Q5 Great Saphenous Vein (GSV) Diameter Does Not Correlate with Worsening Quality of Life Scores in Patients with GSV Incompetence

K. Gibson[1], D. Wright[2]
[1]Lake Washington Vascular Surgeons, Bellevue, WA, [2]BTG, London, United Kingdom

BACKGROUND: Previous studies have correlated increasing GSV diameter with increasing CEAP classification. Venous disease specific questionnaires and quality of life measures (QOL) have demonstrated clinically meaningful improvement following elimination of saphenous incompetence. Currently, specific GSV diameters are being used by some insurance carriers to determine coverage for treatment of axial venous insufficiency. There are no previous studies correlating patient QOL measures with GSV diameters in patients with varicose veins.

METHODS: Data was collected from the charts of 91 patients prospectively enrolled in two varicose vein trials. The patients had symptomatic varicose veins with saphenofemoral junction and proximal GSV reflux. Maximum GSV diameter was measured on duplex ultrasound with the patient in the upright position within five centimeters of the saphenofemoral junction. Chronic Venous Insufficiency Questionnaires (CIVIQ-2, Servier, Neuilly-sur-Seine, France) and Venous Clinical Severity Scores (VCSS) were completed prior to any vein treatment. Demographic information, patient weight, height, and BMIs were collected. Correlations between pairs of data were carried out using Pearson product moment correlation coefficients.

RESULTS: Of the 91 patients, 19 were men and 72 were women. The mean age was 45 (range 18–65) and the mean GSV diameter was 6.7 mm (range 2.2–14.1 mm). VCSS (score ranges from 0–30, with 30 being most severe) ranged from three to twelve with a mean score of 7.8. CIVIQ (score ranges from 0–100, with 100 being most severe) scores ranged from 20 to 85 with a mean score of 42. There was a weak correlation between increasing GSV diameter and BMI ($r = 0.24$) and weight ($r = 0.26$), but not patient height ($r = 0.07$).

There was no correlation between GSV diameter and CIVIQ score ($r = 0.02$) or VCSS ($r = 0.13$).

CONCLUSION: GSV diameter is a poor surrogate marker for assessing the impact of varicose veins on patients' quality of life or predicting potential benefit. As such, it is inappropriate to use GSV diameter as a sole criterion for determining medical necessity for the treatment of GSV reflux. Further correlations between GSV diameter and other patient QOL measures will be investigated.

Q6 Vena Cava Filter Practices: Survey Results from a Large Regional Vascular Surgery Society

M. Friedell[1], P. Nelson[2]
[1]Orlando Health, Orlando, FL, [2]University of Florida College of Medicine, Gainesville, FL

BACKGROUND: Vena cava filter (VCF) use has increased dramatically with the availability of low profile, retrievable devices. To our knowledge, practitioners have never been surveyed regarding filter placement safety and practice patterns.

METHODS: A 17 question online VCF survey was offered to all 276 members of a large regional vascular surgery society. The responses were analyzed using Chi-square goodness of fit.

RESULTS: 126 (46%) members responded and 117 (93%) indicated that they placed filters in their practice. Highly significant differences were identified with each question (at least $p < 0.002$). Regarding the inferior vena cava (IVC), the preferred permanent filter was the Greenfield (31%) and a variety of retrievable devices (49%). Fifty percent of the respondents placed retrievable filters selectively, 26% always placed them and 24% never did. The preferred retrievable filter was the Bard (45%). Despite the fact that 52% and 46% of respondents placed VCFs in trauma and bariatric patients respectively, filters were placed for prophylactic indications less than 50% of the time by 63% of respondents. In trauma and bariatric patients, a retrievable filter was commonly used. Overall, retrievable filters (when not used as permanent filters) were removed less than 25% of the time by 64% of respondents and less than 50% of the time by 78% of respondents. The femoral vein was the preferred access for 84% of respondents. IVC filters were rarely placed at the bedside. Major complications were few, but included: migration to the atrium (1), atrial perforation (1), abdominal pain with filter legs outside the IVC (3), IVC thrombosis (11:4 with OptEase/TrapEase filters), strut fractures (4 Bard filters) and severe tilting making percutaneous retrieval impossible and efficacy questionable (8:7 with Bard filters). Regarding superior vena cava (SVC) filters, 60% of respondents had never placed one and 29% had placed five or less. No procedural complications were reported and only 9% of respondents had ever retrieved one.

CONCLUSIONS: In the IVC, the Greenfield filter was the single most utilized permanent filter. A 49% use of retrievable filters as permanent filters may reflect a preference for a lower profile device. The use of filters for prophylactic reasons was low, except in trauma or bariatric cases. SVC filter placement was extremely rare. VCF insertion is safe with few major complications or long-term problems being reported. However, certain complications appear to be specific to retrievable filters and, given the low removal rate, their use should be questioned until the long-term safety of these relatively new devices is proven.

Q7 Activation of Hypoxia-Inducible Factor Pathway in Varicose Veins

C.S. Lim[1], S. Kiriakidis[1], A. Sandison[2], P. Singh[2], E. Paleolog[1], A.H. Davies[1]
[1]Imperial College London, London, United Kingdom, [2]Imperial College Healthcare NHS Trust, London, United Kingdom

BACKGROUND: Various structural and biochemical changes in varicose vein wall have been reported, and are likely to contribute to varicose veins formation. The causes of these changes remain unknown and stresses including hypoxia are likely to contribute. Hypoxia-inducible factors (HIFs) are nuclear transcriptional factors that regulate the expression of genes of oxygen homeostasis and other cellular stresses. This study aimed to assess the expression of HIF-1alpha, HIF-2alpha, and their target genes in varicose and non-varicose veins.

METHODS: Varicose and non-varicose veins were surgically retrieved from patients with and without varicosities, and immediately snap frozen or stored in formalin. The mRNA and protein expression of HIF-1alpha, HIF-2alpha, and their target genes in varicose and non-varicose veins was analysed with Q-PCR, Western blot and immunohistochemistry. The mRNA expression was calculated relative to one individual non-varicose vein. Data represent mean ± SEM. The differences between varicose and non-varicose veins were tested with unpaired t-test and Mann-Whitney U test. $P < 0.05$ was considered significant.

RESULTS: HIF-1alpha and HIF-2alpha mRNA were significantly up-regulated in varicose compared to non-varicose veins (89.8 ± 18.6, n = 11 versus 10.4 ± 7.2, n = 5; P = 0.012) and (384.9 ± 209.4, n = 11 versus 8.1 ± 4.2, n = 5; P = 0.008), respectively. Increased HIF-1alpha and HIF-2alpha protein expression was also observed in varicose veins. HIF target gene mRNA expression was significantly elevated in varicose compared to non-varicose veins; namely glucose transporter-1 (8.7 ± 2.1, n = 20 versus 1.0 ± 0.3, n = 10; P < 0.001), carbonic anhydrase-9 or CA9 (8.5 ± 2.1, n = 20 versus 2.8 ± 1.2, n = 10; P = 0.006), vascular endothelial growth factor (7.5 ± 2.1, n = 20 versus 0.9 ± 0.2, n = 10; P = 0.001), BNIP-3 (4.5 ± 0.7, n = 20 versus 1.4 ± 0.3, n = 10; P = 0.004), enolase-1 (11.2 ± 2.1, n = 11 versus 3.1 ± 1.9, n = 5 ; P = 0.019), prolyl-hydroxylase domain (PHD)-2 (5.6 ± 1.1, n = 11 versus 1.7 ± 0.7, n = 5; P = 0.034), and PHD-3 (9.9 ± 2.2, n = 11 versus 2.4 ± 1.2, n = 5; P = 0.047). The up-regulation of HIF target genes in varicosities was also reflected at the protein level. Immunohistochemistry demonstrated that HIF-1alpha was only expressed in some endothelial cells of varicose (n = 8) and non-varicose veins (n = 8). Meanwhile, HIF-2alpha and target genes (CA9 and PHD-2) were extensively expressed in endothelial and smooth muscle cells of all varicose (n = 8) and non-varicose veins (n = 8).

CONCLUSIONS: HIF-1alpha, HIF-2alpha, and HIF target genes were up-regulated in varicose compared to non-varicose veins. HIF-1alpha was only expressed in some endothelial cells, whereas HIF-2alpha and target genes were expressed extensively in endothelial and smooth muscle cells. Our data suggest that HIF pathway is activated and may be an important contributor to various structural and biochemical changes in varicosities. Furthermore, HIF-2alpha rather than HIF-1alpha may be the key regulator of the HIF pathway in varicose and non-varicose veins. Therefore, the HIF pathway may be an important therapeutic target in the treatment of chronic venous insufficiency.

Q8 Characteristics of Temporary Inferior Vena Cava (IVC) Filters Non-Retrieval

J. Stevens, J. Cho, M. Makaroun, B. McDaniel, E. Dillavou, L. Marone, R. Rhee, R.A. Chaer
UPMC, Pittsburgh, PA

BACKGROUND: Non-retrieval is an ongoing problem with removable IVC filters. This study examines patient characteristics and anatomic findings associated with non-retrieval.

METHODS: A retrospective single institutional review of all retrievable IVC filters (Tulip, G2, Celect, Optease) placed between 2004 and 2009. Caval anatomy was reviewed from procedural venograms. Angulation (degrees) at the lowest renal vein was categorized as straight, <30, 30–60, and >60. Filter tilt (degrees) at retrieval was classified as none, ≤45, and 45–90. Adjunctive maneuvers for difficult retrieval included dual femoral and jugular access to straighten the filter and disengage the hook from the caval wall. Fisher's exact test, Chi Square test and logisitic regression were used for analysis when applicable.

RESULTS: 401 patients had a temporary filter placed (Table 1). 236 (59%) were retrieved within a mean dwell time of 29.6 days, 26 (11%) of which were difficult to remove and required adjunctive maneuvers. The most common reasons for non retrieval (N = 165, 41%) included oversight (38%), patient non compliance (21%), mechanical inability to retrieve (12%), medical decision to leave as permanent (11%), and death (10%). In patients with attempted retrieval (N = 259), 26% had a minor amount (<33%) of thrombus in the filter which did not preclude removal, but 5% had a significant amount of thrombus necessitating

Patient Characteristics (Total N = 401)

Age	Less than 25 years 13.2% 26–50 years 40.7% 51–75 years 37.9% 76 years and older 8.2%
Sex	Male 61.4% Female 38.7%
Indication	Prophylactic 83.0% Therapeutic 17.0%
Indication note	Prophylactic/unable to A/C 57% DVT/unable to A/C 22% PE/unable to A/C 12.8% DVT/PE/unable to A/C 6.4%
Traumatic injuries	Trauma patient 65.4% Non-trauma patient 34.6%
A/C =	Anticoagulation

abortion of the retrieval. Patients with a non-retrieved filter were more likely to be middle age males (age 26–50) with a therapeutic indication for insertion ($p < 0.0001$). Overall, gender and the presence of traumatic injuries were similar among the retrieved and non-retrieved groups (p = NS). In patients with an attempted but failed retrieval (N = 20), the dwell time was higher (67 vs. 30 days, p = 0.08), but filter tilt, insertion site and caval angulation were comparable to patients with successful retrieval (p = NS). Predictors of a failed or difficult retrieval combined (N = 49) included dwell time (OR = 1.02, p = 0.004), any filter tilt and caval angulation at the renal veins (OR = 3.99, p = 0.003). Caval penetration by a filter strut, although common (45%), was not a predictor of failed or difficult retrieval (p = NS).

CONCLUSIONS: Caval angulation and filter tilt complicate IVC filter retrieval. Consideration should be made to deployment in a straight segment of the IVC even if not flush with the renal veins in order to optimize retrieval. Dwell time adversely affects retrieval success, and overall retrieval rate continues to be moderate, suggesting physician prompts and patient follow up reminders as possible future targets for improvement.

Q9 The Use of Intravascular Ultrasound for Diagnosis and Treatment of Innominate Vein and Superior Vena Cava Obstruction

C. Glass, A. Doyle, M. Dugan, K. Illig, D. Gillespie
University of Rochester Medical Center, Rochester, NY

BACKGROUND: Avoiding catheterization of the subclavian vein to prevent central venous obstruction is a well recognized and practiced KDOQI guideline for hemodialysis patients. Contrast venography and endovascular intervention with percutaneous balloon angioplasty and/or stent placement has emerged as first line treatment for central venous stenosis. The aim of this study is to compare the diagnostic efficacy of intravascular ultrasound (IVUS) to contrast venography for proximal central venous obstruction, and report preliminary treatment outcomes of IVUS guided innominate vein and SVC stenting.

METHODS: All patients diagnosed with venous outflow obstruction of the innominate or SVC using IVUS between February 2009 and June 2010 were retrospectively reviewed. All patients also underwent contrast venography in the same operative period. A previous cohort of dialysis patients with proximal central venous obstruction was used as a historical control to compare clinical outcomes.

RESULTS: Sixteen proximal central venous lesions (8 SVC, 8 innominate) were identified and confirmed by both IVUS and contrast venography. Contrast venography initially revealed obstruction of the SVC in 75% and innominate vein in 67%. However, 42% of the central lesions were reported as "questionable" after venography alone. Mean contrast dose administered to identify the lesions was 20.3 ml. Subsequent IVUS exam of the "questionable lesions" identified 60% of them as "moderate" lesions which prompted intervention, and the remaining "questionable" lesions showed no pathology, avoiding further intervention. Therapeutic endovascular interventions guided by IVUS included angioplasty (7 SVC, 6 innominate) and stent placement (5 SVC, 1 innominate). IVUS was used to adjust stent size in 60% of the SVC stents after venography alone. Stents placed using IVUS had diameters and lengths of 14–22 mm and 45–70 mm respectively, which were considerably larger in diameter compared to the historical control using venography guided stent placement (diameter 8–12 mm, length 42–94 mm). Primary patency at 3 months for IVUS guided stent placement was 100%, markedly greater than the previously reported 46% for venography guided stent placement, and all patients with SVC syndrome had resolution of symptoms. In the subset of patients with threatened fistulas, the hemodialysis salvage rate was 89%, compared to 63% previously reported with venography guided stenting.

CONCLUSIONS: Proximal central venous obstruction remains a problem despite avoidance of subclavian vein catheterization. IVUS appears to be very useful for recognition of innominate and SVC obstruction, as supported by the 40% of lesions that were left undiagnosed after contrast venography alone. Preliminary results also show IVUS assisted stenting provides immediate symptom relief and may have superior early outcomes compared to venography guided stent placement for threatened dialysis access.

Q10 Ultrasound Enhanced Thrombolysis for the Management of Deep Venous Thrombosis

C.J. Smolock, J.E. Anaya-Ayala, I. Birnbaum, Z.F. Cheema, F.A. Syed, J.J. Naoum, A.B. Lumsden, E.K. Peden, M.G. Davies
Methodist DeBakey Heart & Vascular Center, Houston, TX

BACKGROUND: Ekos catheter (Endowave, Ekos Corporation) utilizes a low-energy, high frequency ultrasound (2 MHz) to alter the thrombus structure and allow the thrombogenic drug to be more effective. We evaluated retrospectively the efficacy of combined, Ekos and catheter directed thrombolysis (CDT) for the treatment of acute Deep Venous Thrombosis (DVT).

METHODS: From January 2008 to July 2009, 23 patients (70% males mean age 43 yrs) diagnosed with DVT underwent ultrasound enhanced CDT with tissue Plasminogen Activator (tPA). Patient demographics, symptoms, comorbidities, risk factors and periprocedural data was obtained from records. Technical success was classified as defined by the National Venous Registry.

RESULTS: Fifteen patients had limb swelling, four had pulmonary embolisms (PE) and two had phlegmasia cerulean dolens. Seventeen (74%) patients had involvement of the ilio-femoral veins, 1 (4%) in the IVC, 3 (13%) in the femoro-popliteal vein, 1 (4%) in the popliteal-tibial vein, and 2 (9%) in the subclavian/axillary vein. Mean time to lysis was 26 hours. 43% (10 patients) experienced Grade III or complete lysis, 35% (8 patients) experienced Grade II or partial lysis and 22% (5 patients) experienced Grade I or ineffective lysis. For note, two patients developed a non fatal PE during therapy (11%). There were no bleeding or access-related complications.

CONCLUSION: The adjunctive role of the Ekos system facilitates rapid lysis but is associated with a higher PE rate than those reported for conventional venous thrombolysis.

Q11 Novel Repair of An External Iliac Vein Aneurysm

A. Jayaraj, M. Meissner
UW, Seattle, WA

BACKGROUND: Venous aneurysms are rare with approximately twenty-five iliac venous aneurysms reported in the literature. The etiology of venous aneurysms may be primary, including congenital disorders such as Klippel Trenaunay, Parkes-Weber and Mafucci syndromes, or may be secondary to trauma, proximal flow obstruction or conditions that increase flow or pressure within a venous territory. Although precise criteria for repair have not been established, such aneurysms do pose a theoretical risk of pulmonary embolism. We present a novel surgical approach to the treatment of external iliac vein aneurysms.

METHODS: A 37 year old female presenting with complaints of L buttock pain was found to have bilateral iliac vein aneurysms on abdominal U/S and MRI. CT venography and venous duplex subsequently demonstrated venomegaly involving the IVC, bilateral common iliac, external and internal iliac veins in addition to fusiform aneurysms of bilateral external iliac veins (3.6 cm on the right and 2.1 cm on the left). She additionally had a high grade left common iliac vein stenosis (80–90%).

RESULTS: The patient initially underwent stenting of the L common iliac vein to exclude central venous obstruction as a cause for the external iliac aneurysm (18 x 60 mm and 18 x 40 mm Wallstents, Boston Scientific; Natick, MA) [Figure 1]. Follow-up after 6 months of anticoagulation demonstrated no change in aneurysm diameter. She subsequently underwent operative repair of her aneurysm via left lower quadrant and groin incisions. After percutaneous placement of a 16 x 4 mm angioplasty balloon as a mandrel, a 60 mm endoGIA stapler (Covidien, Dublin, Ireland) was advanced through the left groin incision into the retroperitoneum. The stapler was fired longitudinally over the balloon mandrel, resecting the excess aneurysm wall, and the staple line was oversewn with 5-0 prolene (Ethicon, Somerville, NJ) [Figure 2]. Post operative imaging at 16 weeks demonstrated patent stents with the external iliac vein measuring 17 mm compared to 36 mm at baseline.

CONCLUSIONS: Although rare, venous aneurysms may pose a risk of embolization and warrant consideration of repair. We report successful repair of an iliac vein aneurysm with a simple approach of staple plication and resection over a balloon mandrel.

Figure 1

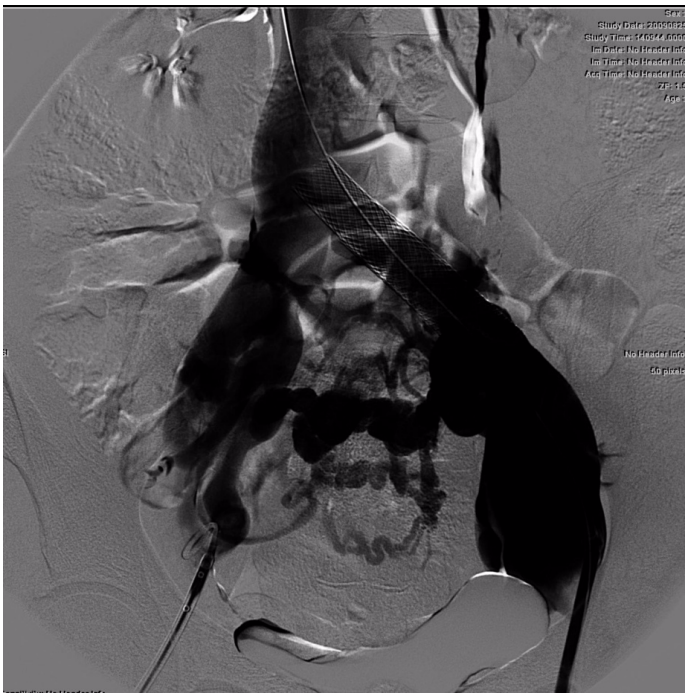
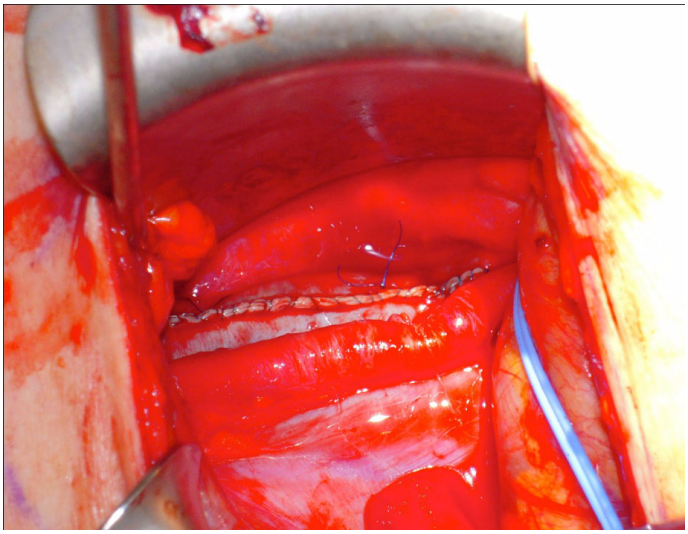


Figure 2



Q12 Efficacy and Safety of Foam Sclerotherapy: Is Ultrasound-Guided Foam Sclerotherapy Always Necessary?

T. Yamaki, A. Hamahata, D. Fujisawa, H. Konoeda, K. Kubo, M. Nozaki, H. Sakurai
Tokyo Women's Medical University, Tokyo, Japan

BACKGROUND: To compare ultrasound-guided foam sclerotherpy (UGFS) for great saphenous vein (GSV) combined with foam sclerotherapy (FS) for varicose tributary veins and FS for varicose veins alone in treating GSV reflux.

METHODS: One-hundred and three limbs in 97 patients with GSV reflux randomized to receive either UGFS combined with FS or FS alone. 1% polidocanol-foam was used for both UGFS and FS. Ultrasonographic inspection of the foam in the GSV was carried out during 5 minutes before compression was applied. Post-sclerotherapy surveillance was done at 1 month, 3, and 6 months. The primary endpoint of the study was elimination of reflux in the GSV at 6 months.

RESULTS: Fifty-one limbs in 48 patients were treated with UGFS combined with FS and the remaining 52 limbs out of 49 patients were treated with FS alone. There were no significant differences in age, men/women ratio, CEAP clinical manifestation and venous clinical severity score. The mean diameter of GSV was 0.6 cm for UGFS + FS group and 0.57 cm for FS group ($p = 0.419$). The mean injected volume of foam for varicose tributary veins 3.9 ml for UGFS + FS group and 5.8 ml for FS group, and significantly higher amount of foam was used for FS group ($p < 0.001$). However, the mean total amount of foam was significantly higher in limbs treated with UFGS + FS than these treated with FS (6.5 ml, 5.8 ml, $p = 0.017$, respectively). Ultrasonographic inspection revealed complete spasm of GSV in 37 (72.5%) limbs in UGFS + FS group and 29 (55.8%) in FS group during sclerotherapy ($p = 0.097$). At 6-month follow-up point, reflux was absent in 30 limbs (58.8%) treated with UGFS + FS and in 37 (71.2%) treated with FS, and there was no significant difference between the groups ($p = 0.190$). No serious complications were found in both groups.

CONCLUSIONS: These results show equivalent efficacy for UGFS + FS and FS in the treatment of GSV reflux despite lower amount of foam in FS group. The amount of foam injected into varicose tributary veins may be a key to both reducing total amount of foam and obtaining better results.

Q13 C6 Clinical Class Chronic Venous Diseases: Minimally Invasive Approaches, Immediate Results and Follow-Up

S.M. Belentsov
City Clinic Hospital #40, Yekaterinburg, Russian Federation

BACKGROUND: The treatment of patients with C6 class chronic venous disease (CVD) is a difficult problem because of the ulcers is an additional risk factor for surgery, and the most of the patients have serious concomitant diseases.

METHODS: 68 consecutive patients (mean age 59 ± 11.5 years) with C6 primary CVD limited to superficial venous reflux were included in the study. All the patients have had non-effective compression therapy for at least 2 months and up to 14 months prior to the surgery.

- 9 patients (group I) were randomized into a standard of care treatment i.e., Great Saphenous Vein (GSV) ligation and stripping with phlebectomies of side branches and perforator ligation under general or spinal anesthesia.
- 25 patients were treated minimally invasive (group II) i.e., high flush ligation of the GSV in combination with local phlebectomies, perforator interruption under local anesthesia. Five patients who had the Small Saphenous Vein (SSV) incompetence were treated with SSV ligation.
- Group III included 30 patients in whom we performed Ultrasound Guided Foam Sclerotherapy (UGFS) of incompetent GSVs or SSVs and (or) incompetent perforators.
- Group IV (4 patients) in whom GSVs (3 pts) or SSV (1 pt) incompetence were treated by Radiofrequency Ablation, and perforator incompetence (3 pts) with the help of UGFS.

All the patients of the second, third and the forth groups were underwent compression sclerotherapy of Varicose Veins followed by surgery, or UGFS, or RFA.

RESULTS: Mean hospital length of stay in the second group was 0.8 ± 0.2 days, compare to 4.4 ± 0.8 days in the first group ($p < 0.05$). Treatment of the patients of the third and the forth groups was on outpatient basis. UGFS closed 100% of GSVs and SSVs, and 96.2% of incompetent perforators immediately after the procedures. Ulcer healing was achieved in all patients with no significant difference between the four groups. During four-year follow-up all but 4 ulcers remained healed. The cases of recurrent ulceration were associated with recurrence of perforator incompetence.

CONCLUSIONS: Minimally-invasive treatment of patients with C6 clinical class CVD has early advantages and equally effective compare to standard treatment.

Q14 Advantages of Tumescent Local Anesthesia with Bicarbonate for Pain, Bleeding, and Quality of Life During Surgery for Varicose Veins: A Prospective Study

P. Pittaluga, S. Chastanet
Riviera Veine Institut, Nice, France

BACKGROUND: The developement of mini-invasive surgical procedures for varicose veins is facilitated by the practice of tumescent local anesthesia initially described by Klein. This study reports the advantages of a tumescent local anesthesia in which isotonic bicarbonate substitutes the saline solution for the dilution of lidocaine.

METHODS: All patient whom underwent surgery for varicose veins using a tumescent local anesthesia with bicarbonate (TLAB) were prospectively included during 4 months.

We asked patient for the pain level during the surgery and at D8 postop by a visual analog scale (VAS). The extent of ecchymosis and hematomas was measured by a tracing paper at D8 postop. The quality of life (QoL) has been evaluated by a non specific SF-12 questionnaire at D8 and D30 postop.

RESULTS: A total of 160 limbs have been operated on in 156 patients (122 females, 34 males) aged from 21 to 85 years (median 52 years). The CEAP class C classification was: CO–C1 0%, C2 85.0%, C3 4.0% and C4–C6 11.0%. That was a reccurence of varicose veins after a saphenous stripping in 16.2% of the cases. Symptoms were present in 70% of the limbs. The surgical treatment has consisted in a stripping of the saphenous vein in 12.5% of the cases and isolated phlebectomy in 87.5%. The TLAB was done with a high dilution of lidocaine (0.003%), and was used alone in 28.8% of the procedures while the TLAB was associated with a slight intravenous sedation in 71.2%. The mean volume of TLAB injected was 425 cc (60 to 1100 cc). None postoperative general or local complication has been observed. The average VAS pain score was 1.52 during the surgery and was not significantly different with or without a slight parental sedation (1.63 vs 1.26). The average extent of ecchymosis and hematomas measured was respectively 10.1 cm^2 et 5 cm^2, with none ecchymosis in 29.4% of the cases and none hematoma in 78.8%. The physical and mental components of QoL were non significantly different at D8 and at D30 (96.8 vs 97.3 and 36.6 vs 36.9).

The VAS pain score was not correlated to the extent of the ecchymosis. There was no significant difference in term of pain and Qol according to the kind of surgical procedure which has been done.

CONCLUSIONS: The TLAB has enabled us to do all kind of surgical treatments using a very high dilution of local anesthetics with a very low pain level. In addition, the postoperative extent of ecchymosis and hematomas was very limited and the postoperative QoL was not affected.

Q15 Cyanoacrylate Adhesive for the Closure of Truncal Veins: 60-Day Swine Model Results

J.I. Almeida[1], R.J. Min[2], R. Raabe[3], D.J. McLean[4], M. Madsen[5]
[1]Miami Vein Center, Miami, FL, [2]Weill Cornell Medical College, New York City, NY, [3]Sacred Heart Medical Center, Spokane, WA, [4]Washington State University, Pullman, WA, [5]Inland Imaging, Spokane, WA

BACKGROUND: The introduction of cyanoacrylate (CA) within a blood vessel triggers polymerization, followed by an inflammatory reaction. Cyanoacrylates were used in the 1960s to stop bleeding and seal wounds, but more recently, CA have been employed to treat varicoceles and other vascular malformations. We report results of a 60-day animal study of endovascular superficial vein closure utilizing a proprietary CA glue.

METHODS: Both superficial epigastric veins from two swine were used; in terms of diameter and length, they resemble human great saphenous veins. Percutaneous access with a micropuncture kit was followed by placement of a 5F sheath under ultrasound control. The sheath was positioned 2.0 cm caudad to the junction of the superficial epigastric and abdominus rectus veins. A 5F delivery catheter was inserted, followed by withdrawal of the sheath, exposing the tip of the delivery catheter. A dispenser gun with a 3 mL syringe was then locked to end of the delivery catheter followed by injection of 0.16 mL of CA glue. Immediately after glue delivery, the catheter delivery system was pulled back 3 cm and manual compression was employed to the vein for 30 seconds. Subsequently, another 0.16 mL injection was delivered, with immediate 3 cm pullback of the catheter delivery system and manual compression of the treated vein for 30 seconds. This process was repeated until the delivered CA reached the level of the access site. At 60 days post-implantation, the treated veins were harvested surgically and examined histologically.

RESULTS: Grossly, the treated veins were totally occluded and of a pale color. Histologically, spindle cells with dense eosinophilic matrix replaced the tunica intima and the tunica media. The vein walls were disrupted by aggregates of histiocytes, multinucleated giant cells, lymphocytes, plasma cells, and eosinophils. These aggregates projected into the vein lumen. The inflammation extended into the tunica media and tunica adventitia, and segmental wall thickening by fibrous tissue was observed. The lumen also contained well demarcated, thin bands of granular material (foreign) that occasionally entrapped lysed erythrocytes. The histologic changes were consistent with a chronic foreign-body type inflammatory response.

CONCLUSION: Results of this animal study demonstrated that intravascular injection of cyanoacrylate is feasible for closure of superficial veins in animal models. The histologic results at 60 days post-procedure suggested that veins treated with this formulation developed the onset of fibrosis. Additional studies of this proprietary cyanoacrylate are planned in order to assess efficacy, safety and its effects on perivenous structures.

12:00 PM – 1:30 PM **Lunch on Own**

1:30 PM – 3:00 PM **BEST OF NON-JVS PAPERS**
Moderator: *Gregory Moneta, MD*

Educational Objectives: At the completion of the session, participants should be able to:

1. List the indications and efficacy of the new anticoagulants for venous thromboembolism and superficial venous thrombosis, including novel non anticoagulants.
2. Describe the factors related to improved quality of life after effort thrombosis.
3. Define the recent advances in prediction of venous thromboembolism risk and recurrence, including genetics and venous duplex testing.
4. Describe the factors associated with the successful long term iliofemoral thrombectomy.
5. List patient factors associated with vena caval filter occlusion and outcomes.

3:00 PM – 3:30 PM **Coffee Break — Visit Exhibits**

3:30 PM – 4:50 PM **SCIENTIFIC SESSION IV**

Compression

Moderators: *Peter Neglen, MD*
William Marston, MD

Educational Objectives: At the completion of the session, participants should be able to:

1. Understand the effect of compression therapy on leg vein anatomy and arterial inflow.
2. Be aware of the connection between venous disease and lymphatic dysfunction.
3. Know one factor predicting failed healing of leg ulcer.

3:30 PM – 3:50 PM **11** **Effect of Compression Therapy on Leg Veins Anatomy: Quantification by 3D Vectorial Modelling from MRI Slices**

J. Uhl[1], H. Partsch[2], G. Mosti[3]
[1]*Université Paris Descartes, Paris, France,*
[2]*University of Vienna, Vienna, Austria,*
[3]*Hospital, Lucca, Italy*

BACKGROUND: Direct mechanical compression of the veins seems to be the main mechanism of action of compression therapy in chronic venous disease.

New imaging techniques allow for a quantitative evaluation of the biophysical impact of compression on the 3D anatomy of the leg, particularly on the venous system.

OBJECTIVE: To use 3D modeling and volume quantification in order to better understand the anatomical effects of compression therapy on the venous system.

MATERIAL AND METHODS: A total of 15 individuals were studied by T2 weighted MRI of the calf or thigh in different body positions (supine, prone, upright) before and after application of different stockings and bandages.

In every case the interface pressure was measured by the use of Picopress® pressure transducer. Compression devices producing different pressures and stifness were assessed.

3D vectorial models were built with Winsurf® software from cross sectional pictures by manual segmentation of all important anatomical structures (bone, muscles, skin, superficial and deep veins). A realistic interactive 3D vectorial model of the extremity was obtained for each leg showing the influence of compression on the leg's anatomy not only in a single cross-sectional slice but for the whole calf.

RESULTS: Even low external pressure is able to induce deformations of the underlying muscle compartments. These shifts of tissue go along with changes of venous caliber and are sometimes unrelated to the balance between intravenous pressure and external compression on the skin. Discrepant findings concerning the narrowing of superficial and deep veins are obtained depending on the body position.

CONCLUSIONS: 3D modeling renders clear graphic images of segments of the lower extremity demonstrating the effect of different kinds of compression on the configuration of the underlying tissue structures including superficial and deep veins.

3:50 PM – 4:10 PM **12** **Compression Therapy in Mixed Ulcers: Search for a Safe Pressure Range Not Affecting Arterial Inflow**

G. Mosti[1], H. Partsch[2]
[1]*Clinica MD Barbantini, Lucca (LU), Italy,*
[2]*Private Practice, Wien, Austria*

BACKGROUND: About 15–20% of patients with venous leg ulcers have a reduced ankle brachial pressure index (ABPI) causing retarded healing. Compression is able to improve venous haemodynamics in mixed ulcers but needs to be applied with caution in order not to reduce arterial inflow.

AIM: to define a safe range of compression pressure that does not impede arterial flow.

METHODS: In 25 patients with mixed ulcers (10 males, 15 females aged 76,4 ± 10 years), presenting with a mean ABPI: 0,57 ± 0,09 and a systolic ankle pressure of 91,8 ± 18,3 mmHg, skin flow was assessed in the peri-wound area and in the plantar surface of the first toe by means of LaserDoppler flowmetry* and toe pressure was measured simultaneously. The measurements were carried out in baseline conditions and after inelastic bandage** from the base of the toes to the popliteal area, applied with different pressure ranges of 20–30, 30–40 and 40–50 mmHg. The pressure exerted by the bandage was continuously measured by a pneumatic device*** with its flat probe placed next to the LaserDoppler probe. The flat, periwound LaserDoppler probe remained under the bandage whereas the toe probes were placed distally to the bandage.

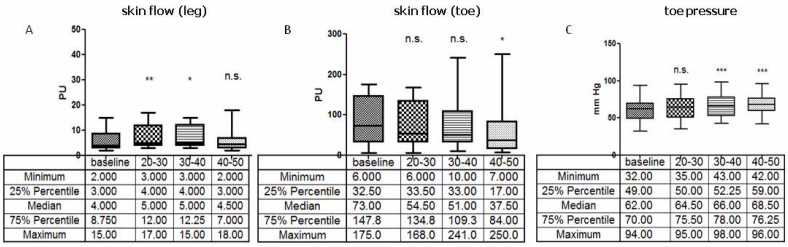
RESULTS: Compared to baseline conditions skin perfusion increases significantly with a bandage pressure of 20–30 and 30–40 mmHg and returns to the baseline level with 40–50 mmHg (Figure 1A). Toe perfusion shows a minor, not significant decrease with 20–30 and 30–40mmHg, but a significant reduction with 40–50 mmHg (Figure 1B). Toe pressure increases with every pressure step, showing significant differences compared to baseline with 30–40 and 40–50 mmHg (Figure 1C).

---

*Periflux®System 5000, Perimed, (Jarfalla, Stockholm), Sweeden, with pressure device PF 5050®, flat probe 404; thermostated probe 457.

**Mollelast Haft® short stretch cohesive bandage and Cellona® as padding material (Lohmann&Rauscher, Rengsdorf, Germany).

***Picopress® (Microlabitalia, Padua, Italy).



	baseline	20-30	30-40	40-50
Minimum	2.000	3.000	3.000	2.000
25% Percentile	3.000	4.000	4.000	3.000
Median	4.000	5.000	5.000	4.500
75% Percentile	8.750	12.00	12.25	7.000
Maximum	15.00	17.00	15.00	18.00

	baseline	20-30	30-40	40-50
Minimum	6.000	6.000	10.00	7.000
25% Percentile	32.50	33.50	33.00	17.00
Median	73.00	54.50	51.00	37.50
75% Percentile	147.8	134.8	109.3	84.00
Maximum	175.0	168.0	241.0	250.0

	baseline	20-30	30-40	40-50
Minimum	32.00	35.00	43.00	42.00
25% Percentile	49.00	50.00	52.25	59.00
Median	62.00	64.50	66.00	68.50
75% Percentile	70.00	75.50	78.00	76.25
Maximum	94.00	95.00	98.00	96.00

Fig. 1: skin flow in Perfusion Units (PU) at leg (A) and toe (B) level and toe pressure (C). p<0.05=*; p<0.01=**; p<0.001=***; not significant: n.s.

CONCLUSIONS: External compression of 20–30 or 30–40 mmHg increases the arterial flow, even in patients with very low ABPI and does not affect the toe pressure as long the individual systolic ankle pressure is not exceeded. Absolute ankle pressure values are more reliable than ABPI to assess the individual risk concerning compression pressure.

4:10 PM – 4:30 PM **13** **Venous Lymphedema**

S. Raju[1], B. Furrh, IV[2], P. Neglén[2]
[1]*University of Mississippi Medical Center/River Oaks Hospital, Jackson, MS,* [2]*River Oaks Hospital, Flowood, MS*

BACKGROUND: Lymphatic dysfunction found in swollen limbs with chronic venous disease (CVD) including iliac venous outflow obstruction (venous lymphedema) is often mistaken for primary lymphedema because of inability to differentiate the etiology by present investigations. The diagnosis of primary lymphedema is often based solely on clinical features, as radioisotope lymphangiogram may be abnormal in both types.

METHODS: Radioisotope lymphangiography was performed in 1608 limbs in 819/1658 patients with symptoms of CVD over a 13 year period, which underwent IVUS-diagnosed/guided iliac vein stenting for iliac venous outflow obstruction. Patients with leg swelling and normal or abnormal lymphangiography were assessed clinically and compared post-operatively in regards to swelling (Grade 0–3) and quality of life (QoL, CIVIQ score, 20–100).

RESULTS: Lymphangiography was abnormal in 251 limbs in 201 patients (25%), bilateral in 25/201 patients (12%) (no node visualization in 48/251 (19%) limbs; delayed visualization/reduced flow in 203 (81%)). Abnormal lymphangiograms occurred in 72 of 443 swollen limbs (16%) (median age 55 years (range: 19–91); female/male = 9/1; left/right limbs = 3/2, Group AbL) and were compared to 240 limbs with normal lymphangiograms (Group NL). Median follow-up was 10 months (range: 2–133).

Clinical features thought to be characteristic of primary lymphedema (early onset, bilateral involvement, swelling of dorsum of foot, squaring of toes, Stemmer's sign) were present in some limbs of both groups. After iliac vein stenting grade of swelling improved significantly in both Groups AbL and NL (median: 3 (range: 1–3) to 2 (range: 0–3) and 3 (range: 1–3) to1 (range: 0–3), $p < 0.0001$, respectively). Complete relief of swelling was found in 9/59 limbs (15%) and improvement ≥1 grade in 17/59 (29%) of stented limbs as compared to 104/240 limbs (43%) and 54/240 limbs (23%) in Groups AbL and NL, $p = 0.0000$ and $p = 0.003$, respectively. In 4/23 (17%) limbs in Group AbL, lymphangiography normalized after stenting.

QoL scores in Group AbL showed significant improvement in the work-related leg swelling category, but the other 4 categories were unimproved. QoL scores in Group NL showed significant improvement in the work-related leg swelling, pain and sleep categories and in the cumulative score. These outcome scores were significantly greater in Group NL as compared with Group AbL.

CONCLUSIONS: Clinical features and abnormal lymphangiography in swollen limbs with CVD can not reliably differentiate primary from venous lymphedema. IVUS-guided iliac venous stenting in limbs with abnormal lymphangiograms provides substantial relief of leg swelling in almost half of the cases, but the outcome is superior in patients with normal lymphangiograms. Despite abnormal lymphangiogram, it appears to be beneficial to diagnose and stent underlying iliac vein obstruction.

4:30 PM – 4:50 PM **14** **Reduced Expression of Soluble Urokinase Receptor Fragment DII-III Predicts Venous Ulcers that Fail to Heal**

A. Ahmad[1], M. Waltham[1], G. Høyer-Hansen[2], T.T. Sørensen[2], K. Mattock[1], P. Saha[1], B. Modarai[1], H. Zayed[1], A. Smith[1]
[1]King's College London, London, United Kingdom, [2]Finsen Laboratory, Copenhagen, Denmark

BACKGROUND: The plasminogen activator system may be critical for venous ulcer healing. Urokinase plasminogen activator receptor (uPAR), which is composed of 3 domains (DI, DII, and DIII) is expressed in the epidermal layers of the ulcer edge. This receptor may be cleaved in the linker region between DI and DII yielding two separate fragments (DI & DII-III), exposing a highly chemotactic area on the DII-III domain that is a potent inducer of cell migration. This study compares levels of soluble uPAR (suPAR, DI-III) and its fragments in exudates from healing and non-healing venous ulcers.

METHODS: Patients with venous ulcers (CEAP C6 disease) were recruited from a dedicated leg ulcer clinic. Venous aetiology was confirmed on venous duplex. Ulcer exudates were aspirated from Opsite™ covered ulcers at recruitment. Acute wound exudates were collected from split skin graft donor sites to act as controls. All exudates were centrifuged at 16000 g for 10 min at 4°C and supernatants aliquoted, snap frozen and stored at –80°C until assayed. All patients were treated with standard compression dressings and prospectively followed for ulcer healing, defined as complete re-epithelialisation of the ulcer within 6 months. Time-resolved fluorescence immunoassays were validated and used to measure levels of suPAR and its fragments, DI and DII-III in wound exudates. Levels were normalised against soluble protein concentration (mg/ml). Statistical analysis was carried out using unpaired t test.

RESULTS: Exudates were collected from twenty-five patients with venous ulcers (13 females, 12 males; median age 68 yrs range 34–92 yrs). Nine patients were defined as healers. Control (acute) wound exudates were obtained from seven patients (4 females, 3 males; median age 78 yrs range 47–88 yrs). Healers had significantly higher levels of DII-III (138 ± 19 fmol/mg) compared with non-healers (47 ± 7 fmol/mg, $P < 0.0001$) and controls (41 fmol/mg ± 19, $P < 0.005$). Soluble uPAR levels were higher in both healers (19 ± 5 fmol/mg) and controls (32 ± 3 fmol/mg) compared with non-healers (8 ± 1 fmol/mg, $P < 0.05$ for both). There was no significant difference in the levels of DI fragment between any of the groups.

CONCLUSIONS: This is the first study to show that suPAR and its fragments are present in venous ulcer exudates. Levels of suPAR and its DII-III fragment were significantly lower in poorly healing ulcers, with the latter providing a better discrimination between the groups. Low levels of the non-proteolytic DII-III fragment, known to stimulate cell migration, could be a useful predictor of ulcers that would benefit from early skin grafting. This fragment may also represent a novel target for treatment to promote venous ulcer healing.

4:50 PM – 5:10 PM **VENOUS FORUM/ROYAL SOCIETY OF MEDICINE (BEST PAPER)**

Randomised Clinical Trial Comparing VNUS® ClosureFAST™ Versus Laser for Varicose Veins (VALVV): Duplex and Quality of Life Outcomes as 6 Months

A.C. Shepherd, M.S. Gohel, L.C. Brown, A.H. Davies
Imperial College, Academic Section of Vascular Surgery, London, England

AIMS: Early results from the VALVV trial showed outcomes following segmental radiofrequency ablation (RFA) and laser ablation (EVLA) were comparable at 6 weeks. The aim of this study was to compare technical success, quality of life and disease severity at 6 months following RFA and EVLA.

METHODS: Consecutive patients with great saphenous vein (GSV) reflux were randomised to EVLA or RFA. After 6 months, technical success was assessed using colour duplex. GSV ablation was classified as successful if completely ablated above the knee or failed ablation if the above knee GSV was not ablated or re-canalised. Clinical disease severity was assessed using the VCSS and quality of life was evaluated using the Aberdeen Varicose Vein Questionnaire (AVVQ).

RESULTS: Duplex scans performed at a median (IQR) of 27 (25–29) weeks following the procedure were available for 107/131 randomised patients (82%) (RFA n = 55, EVLA n = 52). Successful ablation was seen in 49/55 (89%) and 49/52 (94%) for RFA and EVLA groups respectively. Improvements in quality of life and VCSS seen at 6 weeks were maintained at 6 months in both groups. Mean (SD) scores from 6 weeks–6 months in the RFA and EVLA groups respectively were: AVVQ 10.9 (9.2)–10.2 (9.4), VCSS 1.7 (1.7)–1.4 (1.8) and AVVQ 10.8 (8.9)–10.8 (8.7), VCSS 1.5 (1.8–1.4 (1.7). There were no significant differences at 6 months between the groups for the AVVQ (p = 0.286), and the VCSS (p = 0.239) respectively (ANCOVA).

CONCLUSION: Both RFA and EVLA resulted in good technical success rates at 6 months. Improvements in quality of life and clinical disease severity were maintained from the 6 week results and comparable between the two groups.

Registration number ISRCTN66818013

5:15 PM – 6:30 PM **POSTER SESSION**

Moderator: *Joseph Raffetto, MD*

Educational Objectives: At the completion of the session, participants should be able to understand:

1. Changes in lymphatic structure in incompetent saphenous veins.
2. Vein wall fibrosis and deep venous thrombosis.
3. Venous thromboembolism risk factors.
4. Deep venous thrombosis in surgical intensive care unit patients.
5. Epidemiology of trunk varices.
6. Contractile mechanism of varicose veins.
7. Evaluation of patients with cerebrospinal venous insufficiency with plethysmography.
8. Inferior vena cava filter fractures.
9. The role of plasminogen activator and inhibitors on thrombotic wall remodeling.
10. Complications after phlebectomy.
11. Deep venous thrombosis and socioeconomic impact.
12. Retrieving inferior vena cava filters.
13. Thrombophilia and thrombotic disease during pregnancy.
14. Measuring skin changes in chronic venous disease.
15. Relation of great saphenous vein diameter and endovenous energy delivered.

P1 **Loss of Vasa Lymphatic Vessels and Changes in Lipid Accumulation in Incompetent Greater Saphenous Vein**

H. Tanaka, N. Unno, N. Zaima, T. Sasaki, N. Yamamoto, M. Suzuki, M. Nishiyama, M. Sano, H. Konno, M. Setou
Hamamatsu University School of Medicine, Hamamatsu, Japan

BACKGROUND: We previously determined the characteristic distribution of lipid molecules in incompetent valve tissue by using imaging mass spectrometry (IMS). Recent studies suggests that along with valvular tissue degeneration, biological changes occur in the vein wall in non-valvular regions. However, the pathogenesis of these biological changes is yet to be elucidated. In this study,

we utilized IMS to analyze the incompetent greater saphenous vein (GSV) in varicose vein (VV) patients in order to assess the distribution of lipid molecules.

METHODS: We obtained GSV tissue from 40 limbs of 25 VV patients (VV) who underwent GSV stripping and from 10 limbs of 10 peripheral artery occlusive disease (PAD) patients as a bypass graft during the surgery (control veins: CVs). Conventional and immunostaining were performed for histopathological examination. Lipid contents in vein tissue was determined by colorimetric method after total lipid was extracted from vein tissue. The localization of each lipid molecule in the vein wall was assessed by IMS.

RESULTS: The VV wall was 1.7-fold thicker than the CV wall. The medial cell number in the VV wall was 2.2-fold higher than that in CV wall, although there was no difference in cell number in the intima and adventitia of the VV compared to the CV. Only the VV adventitia was positive for lipid staining. The amounts of phospholipid and triglyceride (TG) in the VV wall were 1.8, and 2.0-fold higher than those in the CV wall, respectively. IMS revealed abnormal accumulation of lyso-phosphatidylcholine (lyso-PC) (1-acyl 16:0) and PC (1-acyl 36:4) in the VV intima and media. TG was specifically found in VV adventitia. (Figure 1) On the other hand, the number of lymphatic vessels, as measured by staining with a lymphatic vessel-specific marker (D2–40), was significantly lower in the VV adventitia than in the CV adventitia (Figure 2), suggesting that lymphatic drainage in the VV adventitia was improper, and may biologically influence both the intima and media.

Fig.1

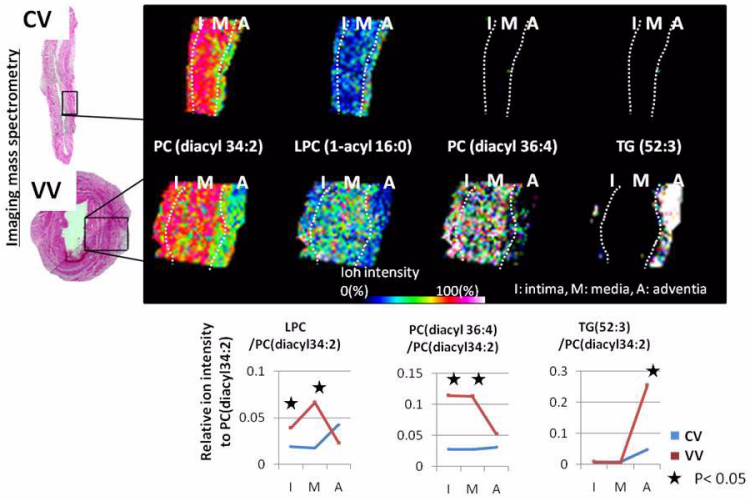
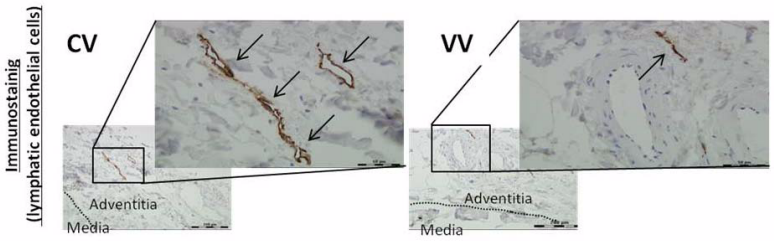


Fig.2



CONCLUSIONS: The accumulation of LPC (1-acyl 16:0) and PC (1-acyl 36:4) in the VV intima and media may be associated with chronic inflammation, leading to VV tissue degenation. Furthermore, the loss of vasa lymphatic vessels in the VV adventitita may disrupt lymphatic drainage in VV tissue.

P2 Neutralizing Interleukin-6 Reduces Vein Wall Fibrosis in a Deep Vein Thrombosis Mouse Model

B.M. Wojcik, S.K. Wrobleski, A.E. Hawley, T.W. Wakefield, D.D. Myers, Jr., J.A. Diaz
University of Michigan, Ann Arbor, MI

BACKGROUND: Deep Vein Thrombosis (DVT) and its associated sequelae, post-thrombotic syndrome (PTS), are a significant US health care problem. It is estimated that up to 60% of patients diagnosed with DVT may develop PTS, which is characterized by extensive perivenous and mural fibrosis. Interleukin-6 (IL-6) has been linked to fibrosis and high circulating plasma levels increase the risk of developing DVT. The aim of this study was to elucidate the role of IL-6 in the progression of vein wall fibrosis using a mouse model of DVT.

METHODS: C57BL/6 mice were treated with either anti-IL-6 mAb (anti-IL6) or control rat-IgG. Thrombus was induced using an inferior vena cava (IVC) ligation model. IVC and thrombus were harvested at days 2, 6, or 14 for thrombus weight, gene expression of IL-6, C-C motif chemokine ligand 2 (CCL2), inflammatory cell recruitment, and morphometric analysis of vein wall fibrosis.

RESULTS: Mice treated with anti-IL6 had smaller thrombus weights at day 2 (Figure 1), decreased vein wall gene expression and protein concentration of CCL2 at day 2 (Figure 2), and impaired vein wall influx of monocytes from day 2 to day 6 vs. controls (Figure 3). Intimal thickness was reduced by 44% ($p = 0.0230$) and vein wall collagen deposition was decreased by 30% at day 14 in the anti-IL6 group ($p < 0.0001$) (Figure 4).

Figure 1

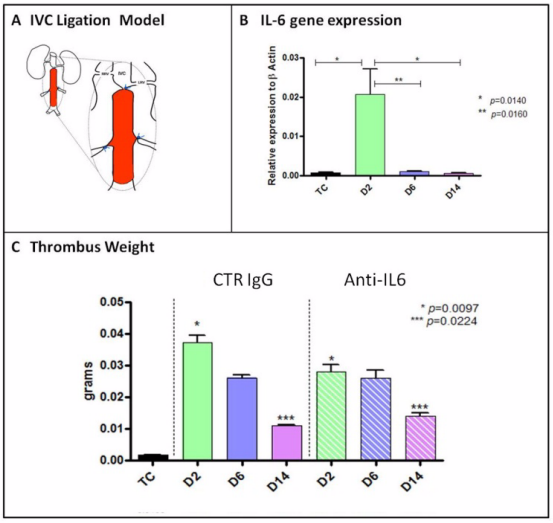


Figure 2

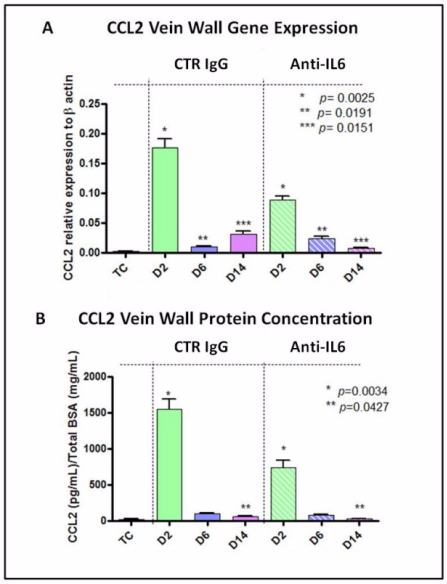
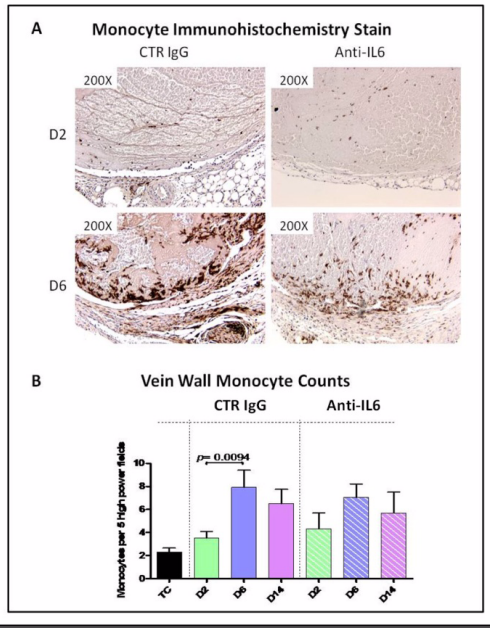
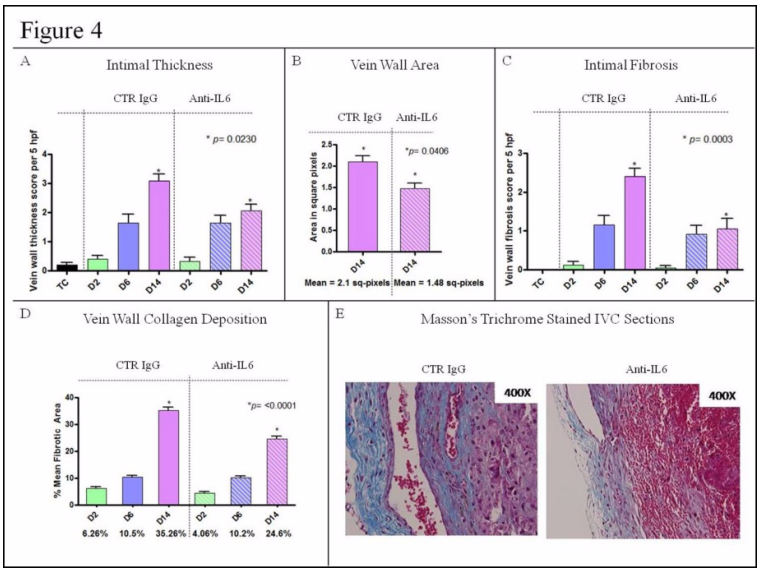


Figure 3





CONCLUSIONS: Neutralizing IL-6 throughout venous thrombogenesis decreased the production of CCL2, reduced monocyte recruitment, and decreased vein wall intimal thickness and fibrosis. These results suggest that IL-6 may serve as a therapeutic target to prevent the fibrotic complications seen in PTS.

P3 An Observational Study for VTE Risk Assessment Among Hospitalized Patients in General Surgery Clinics Across Turkey

Mehmet Kurtoglu[1], Ismail Akgun[2], Mustafa Ateş[3], Salih Mujdat Balkan[4], Ilyas Baskonus[5], Ugur Bengisun[6], Adnan Calik[7], Nuh Zafer Cantürk[8], Halil Iyikosker[9], Servet Karahan[10], Mustafa Kısakurek[11], Ferda Nihat Koksoy[12], Mahir Özmen[13], Alper Öztürk[1], Melih Paksoy[14], Mustafa Sare[15], Atakan Sezer[16], Halil Ibrahim Tacyildiz[17], Mustafa Tercan[18], Koray Topgül[19], Yılmaz User[20]

[1]Istanbul University, Istanbul Faculty of Medicine, Istanbul; [2]Sisli Etfal Training and Research Hospital, Istanbul; [3]Malatya State Hospital, Malatya; [4]Gülhane Military Medical Academy, Ankara; [5]Gaziantep University Faculty of Medicine, Gaziantep; [6]Ankara University Faculty of Medicine, Ankara; [7]Karadeniz Technical University, Faculty of Medicine, Trabzon; [8]Kocaeli University Faculty of Medicine, Izmit; [9]Gaziantep State Hospital, Gaziantep; [10]Sisli Okmeydani Training and Research Hospital, Istanbul; [11]Elazig State Hospital, Elazig; [12]Taksim Training and Research Hospital, Istanbul; [13]Ankara Numune Training and Research Hospital, Ankara; [14]Istanbul University, Cerrahpasa Faculty of Medicine, Istanbul; [15]Gazi University Faculty of Medicine, Ankara; [16]Trakya University Faculty of Medicine, Edirne; [17]Dicle University Faculty of Medicine, Diyarbakır; [18]Malatya Beydagi State Hospital, Malatya; [19]University Faculty of Medicine, Samsun; [20]Haydarpaşa Numune Training and Research Hospital, Istanbul, Turkey

BACKGROUND: Venous thromboembolism (VTE) still remains a significant public health problem due to gaps between recommendations and clinical practice in VTE prophylaxis. This is the first clinical study designed to evaluate the applicability of a standard "VTE prophylaxis and risk factor assessment form (VTE-PRAF)" and prescription of VTE prophylaxis among hospitalized patients in the daily practice of general surgeons in Turkey.

METHODS: Adult general surgery in-patients (n = 1472) across Turkey were included with respect to cross-sectional (n = 537), first longitudinal (n = 452) or the second longitudinal (n = 483) registry phases lasting for 11 months. Data on demographics, hospitalization, surgical intervention and prophylactic measures were collected during cross-sectional phase, whereas utilization of VTE-PRAF provided following cross-sectional phase was evaluated during longitudinal phases.

RESULTS: Mean age of all patients was 52.4 ± 16.9 years, and 49.5% (n = 729) were male. Among 1227 patients (83%) evaluated for VTE risk regardless of the VTE-PRAF, 62.1% were identified to be at "high+ highest" risk with use of prophylaxis only in 65.9%. Utilization of VTE-PRAF in the second longitudinal phase (74.1%) was higher than the first one (60.6%; $p < 0.001$). However, there was no relation between implementation of the VTE-PRAF and the use of prophylaxis. VTE-PRAF was completed for 70.6% and 84.8% of patient who received prophylaxis while it was completed for 50.8% and 50.4% of patients with no prophylaxis, in the first and second longitudinal phases, respectively. Prophylaxis was administered in 58.6% and 62.6% of patients with completed VTE-PRAF in the first and second longitudinal phases, respectively. Patients lacking VTE-PRAF and prophylaxis application composed 68% of the population. "Two doses of Low Molecular Weight Heparin" was the most frequently used prophylaxis regimen regardless of the application of VTE-PRAF in both longitudinal phases. "Suggested" and "used" prophylaxis regimens were significantly more consistent for the cases evaluated with VTE-PRAF ($p < 0.001$).

CONCLUSION: Based on the use of prophylaxis only for 65.9% of hospitalized patients at high risk for VTE in general surgery clinics across Turkey, low use of prophylaxis is assumed to remain a significant threat to public health despite the availability of effective and safe prophylactic measures and treatments. While consistency of "suggested" and "used" prophylactic measures and treatments were determined to be increased with the application of VTE-PRAF, treatment options which are not based on the guidelines are still common in daily practice in surgery clinics. Inclusion of a standard VTE-PRAF in the hospital protocol seems to raise clinical awareness of VTE risk assessment and appropriate management in VTE which otherwise well-known to be associated with significant mortality and morbidity. Impact of e-VTE-PRAF is worth investigating.

P4 Prospective Surveillance for Lower Extremity Deep Vein Thrombosis in Surgical Intensive Care Unit Patients

N.A. Urban, P.A. Fong, P. Bendick, F.A. Ivascu
William Beaumont Hospital, Royal Oak, MI

BACKGROUND: Despite the increased emphasis on prevention of venous thromboembolism (VTE), it remains a significant cause of morbidity and mortality in the intensive care setting. A retrospective review at our institution showed the incidence of occult deep venous thrombosis (DVT) in our surgical intensive care unit to be much higher than previously thought, despite the use of prophylaxis in this population. The purpose of this study was to prospectively evaluate the prevalence of lower extremity DVT in critically ill surgical patients to confirm the findings of the retrospective study.

METHODS: During the period of March 1st to June 30th, 2010, duplex ultrasound was used to prospectively screen all patients admitted to the surgical intensive care unit (SICU) with an anticipated length of stay over five days. Patients were screened initially on average at day six of their stay in the SICU, followed by repeat screening examinations at four to six day intervals regardless of initial results. Demographic data, co-morbidities, acute physiology and chronic health evaluation (APACHE) III score, admitting service and diagnosis, VTE risk factors, and type of VTE prophylaxis were also recorded.

RESULTS: There were 196 patients evaluated with an average age was 67.8 years, APACHE III score of 62, and an ICU length of stay of 20.5 days. A 31% prevalence of DVT was found; 14 patients had isolated intramuscular calf DVT, 14 had posterior tibial or peroneal DVT, and 33 had femoro-popliteal DVT. Twelve of 61 patients were found to be positive for DVT after an initially normal examination; the average number of ICU days to development of DVT was 10 days. Age, gender, APACHE III score, admitting diagnosis and service were not associated with presence of DVT. The only significant risk factor for DVT was the type of prophylaxis; patients receiving only sequential compression devices as prophylaxis had a 45% prevalence of DVT, while patients receiving pharmacologic prophylaxis in addition to SCDs had a 27% prevalence of DVT. ($p = .021$) Congestive heart failure ($p = .045$) renal failure ($p = .019$) were significantly associated with femoro-popliteal DVT; patients with sepsis were also noted to have a relatively high incidence of femoro-popliteal DVT ($p = .055$).

CONCLUSIONS: Deep vein thrombosis remains a significant, potentially life-threatening complication in patients with extended surgical intensive care unit stays despite aggressive prophylaxis. Prospective duplex ultrasound surveillance may be warranted in these patients, particularly in those with congestive heart failure, renal failure and/or sepsis given their increased incidence of femoro-popliteal disease.

P5 Deterioration in Trunk Varicosities in the General Population Over a 13-Year Period: Edinburgh Vein Study

S. Boghossian[1], L.A. Robertson[1], C.J. Evans[2], A.J. Lee[3], P.L. Allan[1], V. Ruckley[1], F.G.R. Fowkes[1]

[1]University of Edinburgh, Edinburgh, United Kingdom, [2]NHS Lothian, Edinburgh, United Kingdom, [3]University of Aberdeen, Aberdeen, United Kingdom

BACKGROUND: Epidemiological studies of venous disease in the general population have investigated the frequency and risk factors but almost none have reported on the natural history. The few studies on progression are limited to those carried out in clinical practice. The aim of this study is to describe the progression of trunk varicose veins in the general population in the Edinburgh Vein Study over a 13 year follow-up.

METHODS: The study design is a population based cohort study in which a randomly assigned sample of 1566 men and women aged 18 to 64 years already examined at baseline, underwent a 13 year follow-up examination. The clinical investigation of subjects comprised the following measurements: self-administered questionnaire of lifestyle and other factors, height and weight, leg observation to determine Basle and CEAP classification, and duplex ultrasound to measure venous incompetence. Deterioration in varicosities was defined as a worsening of grade or change from unilateral to bilateral disease.

RESULTS: Of the 1566 studied at baseline, 880 took part in the follow-up (response rate = 60.4%), 325 subjects had trunk varicose veins at baseline: 154 had trunks in one leg only, and 171 subjects had trunks in both legs. According to Basle and CEAP classification, 271 (83.4%) had Grade I, 51 (15.7%) Grade II, 3 Grade III (0.9%).

Of the 325 with trunks at baseline, during the 13 year follow-up, 154 subjects deteriorated (47.4%) (95% CI 42.0%–52.8%), 62 subjects stayed the same (19.1%) (95% CI 15.4%–24.0%), and 109 subjects showed improvement (33.5%) (95% CI 28.4%–38.7%). The rate of progression was 3.54% per annum. Of those 154 deteriorating subjects, 59 progressed in both legs (38.3%), 95 deteriorated in one leg (61.7%) equally in right and left. The number of subjects who progressed from unilateral to bilateral disease (one leg to both legs) was 39 (25.3%).

Of those subjects who showed improvement in any leg during the follow-up, 16.6% had undergone surgery or sclerotherapy (18 surgery, 4 sclerotherapy). Of the remaining 87 subjects, 85 changed from Grade I at baseline to Grade 0 at follow-up. It is likely that much of this minor change was due to observer variability.

CONCLUSION: Little is known of the natural history of varicose veins in the general population. Our findings show that over a 13 year period, almost half will progress to more severe varicose veins and around one fifth will remain the same. Analysis of risk factors associated with this progression will also be presented.

P6 Functional Adaptation of Varicose Veins Contractile Mechanisms to Excessive Wall Stretch

V. Mam[1], J.D. Raffetto[2], O.M. Reslan[1], R.A. Khalil[1]
[1]Brigham and Women's Hospital, Boston, MA, [2]VA Boston HCS, West Roxbury, MA

BACKGROUND: Varicose veins are characterized by valve dysfunction and excessive vein wall dilation and tortuosity. The wall dilation in varicose veins has been partly explained by decreased wall contraction mechanisms and inability to contract in response to increasing venous pressure. The purpose of this study was to test whether the varicose veins ability to contract decreases with progressive vein wall stretch.

METHODS: Specimens of control greater saphenous vein were obtained from patients undergoing lower extremity bypass (n = 5). Varix segments were obtained from patients undergoing varicose vein stripping (n = 4). Circular vein segments were incubated in a tissue bath filled with Krebs solution 95% O_2 5% CO_2 at 37˚C in preparation for measurement of isometric contraction. Both control and varicose veins were subjected to increasing wall stretch (0.5 g to 8 g) at 1 g increments, and equilibrated under the specific basal tension for 30 min. The veins were then stimulated with 96 mM KCl depolarizing solution and the steady-state contraction was measured (g/mg tissue weight).

RESULTS: Vein specimens produced significant contraction to KCl that reached steady-state in 15 min. In control veins, stepwise increases in basal tension were associated with corresponding increases in KCl contraction (max 0.09 ± 0.03 at 2 g basal tension). Additional increases in basal tension did not cause significant increases in KCl contraction, which was maintained at 0.13 ± 0.04 at 7 g basal tension. Further stretch of control veins to 8 g tension was associated with significant reduction in KCl contraction (0.05 ± 0.01, $p < 0.05$). In contrast, in varicose veins: 1) Stepwise increases in wall stretch were associated with greater increases in KCl contraction (0.25 ± 0.04 at 2 g basal tension), 2) KCl contraction did not reach a maximum until 6 g of basal tension, 3) The maximum KCl contraction (0.49 ± 0.06 achieved at 6 g basal tension) was significantly greater than maximum contraction in control veins (achieved at 2 g basal tension, $p < 0.05$), and 4) Further stretch to 7 g and 8 g basal tension was not associated with any significant decrease in KCl contraction (0.49 ± 0.07 at 7 g, and 0.56 ± 0.05 at 8 g).

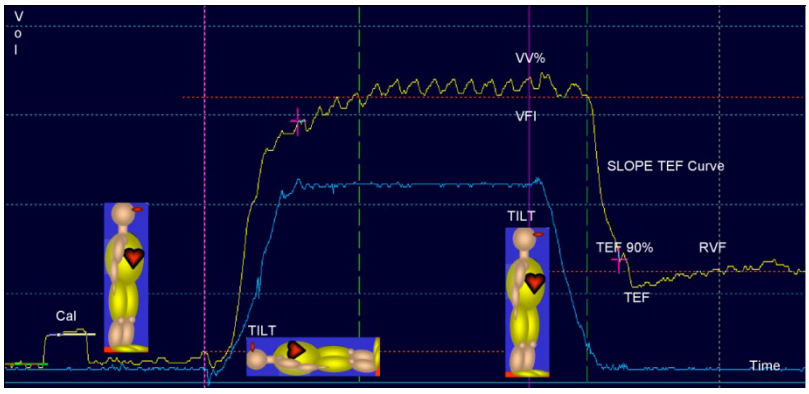
CONCLUSIONS: Control human saphenous veins demonstrate increases in contraction with moderate stretch that reach a maximum, then undergo reduction with excessive stretch (Hook's law). Contrary to our stated hypothesis, varix segments of varicose veins demonstrate greater increases in contraction in response to wall stretch and do not show reduction in contraction even at excessive 8 g basal tension. The persistent increase in varicose veins contraction even under excessive vein wall stretch may represent a functional adaptation mechanism to maintain vein wall contractility and venous return against excessive venous pressure.

P7 Screening of Chronic Cerebrospinal Venous Insufficiency by Cervical Strain-Gauge Plethysmography

E. Menegatti[1], M. Morelli[1], G. Bergamo[1], M. Zuolo[1], S. Gianesini[1], F. Salvi[2], P. Zamboni[1]
[1]University of Ferrara, Ferrara, Italy, [2]Bellaria Neurosciences, Bologna, Italy

BACKGROUND: Chronic cerebrospinal venous insufficiency (CCSVI) is a syndrome characterized by venous flow blockages at the level of the jugular and/or azygous veins, compensated by activation of collateral circulation. Blocked outflow is due to truncular stenosing malformation, mainly intraluminal defect like malformed valve, septum, web, etc., or more rarely, vein hypoplasia and agenesis. It has been described a strong association between CCSVI and multiple sclerosis (MS). The CCSVI condition can be diagnosed by vascular Doppler sonography and/or catheter venography. The former is operator dependent and the latter is of course invasive. MR venography does not represent a valid alternative, since diagnostic accuracy is still low. We experimented strain-gauge plethysmography as a screening device for CCSVI. Aim of the test is to assess the gravitational mechanism of venous outflow from the brain.

METHODS: 40 healthy controls (HC) matched for age and gender with 29 CCSVI-MS patients were screened for CCSVI by means of vascular Doppler sonography by an expert operator. The entire cohort blindly underwent a protocol using an original strain-gauge collar connected with a volume transducer and dedicated software. After calibration, the subject is tilted from the upright to the supine posture (Figure 1). The redistribution of blood volume permits to obtain a volume-time curve from which extrapolates the venous volume (VV%), corresponding to the highest point of the filling plateau, the 90% VV and the venous filling index (VFI). The subject is tilted to up again, obtaining a reduction in venous volume defined as tilt ejection fraction (TEF and TEF 90%), with a slope curve proportional to the time of emptying. Finally, the residual volume fraction (RVF) corresponds to the cervical volume after tilting up (Figure 1).



RESULTS: VV% measured respectively in HC 5.3 ± 2.5 and in CCSVI-MS 6.7 ± 2.5 ($p < 0.0002$); VFI 0.9 ± 0.5 and 1.3 ± 0.8 ($p < 0.0001$); TEF 90% 1.8 ± 0.7 and 2.8 ± 1.1 ($p < 0.0001$); TEF slope 2.6 ± 1.7 and 1.8 ± 1.1 ($p < 0.0001$); RVF 0.6 ± 1.5 and 1.7 ± 1.7 ($p < 0.0001$). No significant variations were found for VV 90% and TEF between the two populations.

CONCLUSIONS: Cervical strain-gauge plethysmography showed several parameters significantly different in CCSVI respect to HC. It is a novel tool for non-invasive, non-operator dependent screening of CCSVI. Imaging techniques remains indispensable for defining location and morphology of venous outflow obstructions.

P8 Frequent Fracture of TrapEase Inferior Vena Cava Filters: Long-Term Follow-Up Assessment

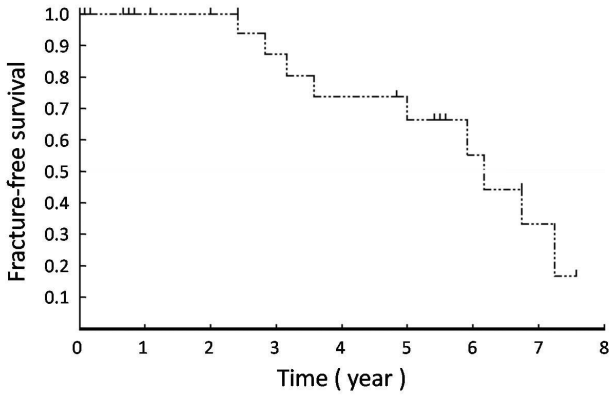
M. Sano, N. Unno, N. Yamamoto, H. Tanaka, M. Nishiyama, Y. Mano, D. Sagara, M. Suzuki, H. Konno
Hamamatsu University School of Medicine, Hamamatsu, Japan

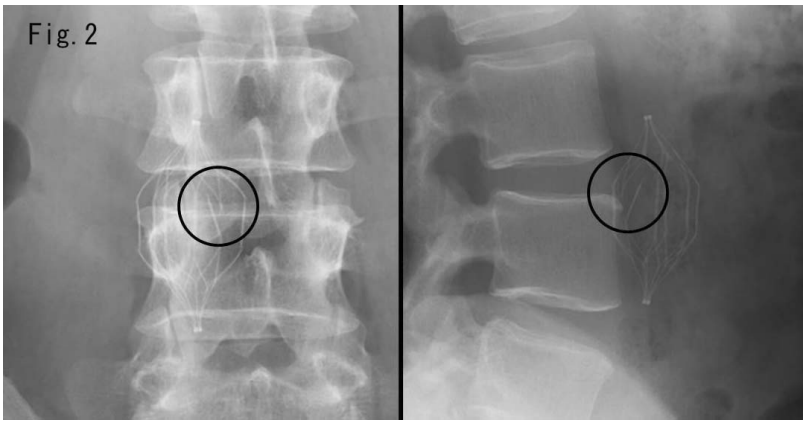
BACKGROUND: Inferior vena cava (IVC) filters are often used to prevent pulmonary thromboembolism when anticoagulation has failed or is contraindicated. The TrapEase filter (Cordis, Bridgewater, NJ) was introduced in 2000, and is currently one of the most popular filtration devices, although only a few studies have been conducted on this device. The current study analyzes the incidence of TrapEase filter fractures during long-term follow-up assessment.

METHODS: Between Novermber 2002 and July 2006, 25 Trap-Ease IVC filters were used for 25 patients (10 men, 15 women; average age, 63 years, range, 18–84 years). Follow-up sessions were conducted at our outpatient clinic every 6 months after insertion. X-ray scans of the IVC filters were obtained in 2 views: the anteroposterior and lateral views. All scans were reviewed and analyzed specifically to detect filter fractures. Data were analyzed in a retrospective manner. Fracture-free survival of the IVC filter was analyzed using Kaplan Meier statistics.

RESULTS: The mean follow-up time was 43 months (range, 1–93 months). Assessment of the X-ray scans showed that 36.0% (9/25) filter fractures had occurred (Figure 1). The lateral-view x-ray scans showed that the dorsal-connected straight struts were fractured in all cases. The cumulative stent fracture-free survival was estimated at 20.0% (3/15), and 10.0% (9/10) at 36- and 84-months of follow up, respectively (Figure 2). At the time at which the fractures were detected, none of the patients showed any filter-related adverse symptoms.

Fig.1





CONCLUSIONS: The incidence of TrapEase filter fractures is quite frequent, especially at 3 years after insertion. In all cases in this study, the filter fractures occurred at the dorsal-connected straight struts, which suggests the effect of compression by the vertebral osteophyte when the patient is in the supine position. Although no clinical adverse effects of the fractures were noted, careful observation over a long period is necessary.

P9 The Role of Urokinase Plasminogen Activator and Plasmin Activator Inhibitor-1 on Vein Wall Remodeling in Experimental Deep Vein Thrombosis

J. Baldwin, V. Sood, M. Elfline, C. Luke, N. Dewyer, J. Diaz, D. Myers, T. Wakefield, P. Henke

University of Michigan, Ann Arbor, MI

BACKGROUND: Deep vein thrombosis (DVT) resolution instigates a strong inflammatory response, resulting in vein wall damage. Urokinase plasminogen activator (uPA) and its inhibitor, plasminogen activator inhibitor-1 (PAI-1), are integral components of the fibrinolytic system, but their role in vessel injury is not known. This study examined the effects of altered plasmin activity on the vein wall in the setting of DVT .

METHODS: A mouse inferior vena cava (IVC) ligation model in uPA -/- or PAI-1 -/- mice and their genetic WTs (B6SVEV and C57BL/6, respectively) were used to create stasis thrombi, with tissue harvest at either 8 days (d) or 21d. Tissue analysis included gene expression of contractile state (normal) vascular smooth muscle cells (alpha SMA [ASMA], SM22) and endothelial markers (CD31, endothelial nitric oxide synthase [eNOS]) by real time PCR, antigen analysis by ELISA, matrix metalloproteinase(MMP)-2 and -9 activity by zymography, and vein wall collagen sirus red histologic analysis.

RESULTS: Thrombi were 28–70% larger in both 8d and 21d uPA -/- when compared with WT ($P < .05$, $n = 13–20$) and were 21–31% smaller in both 8 and 21d PAI-1 -/- when compared to WT ($P < .01$, $n = 11–17$). Correspondingly, 8d plasmin levels were reduced 2.7 fold in uPA -/- and increased 3 fold in PAI-1-/- when compared to respective WT thrombi ($P < .05$, $n = 4–6$). Evaluation of vein wall contractile state VSMC genes showed that 8d and 21d PAI-1 -/- had 2.3 and 3.8 fold more SM22 and 1.8 and 2.3 fold more ASMA expression than respective WT ($P < .05$, $n = 5–7$), with no significant differences in endothelial gene expression. Conversely, CD31 expression was 2.5 fold greater in WT than uPA -/- ($P = .02$, $N = 5–6$), suggesting less endotheliazation. No significant difference in MMP2 or 9 activity was found in the PAI-1 -/- mice compared with WT, while 5.4 fold more MMP9 activity was present in 21d WT than 21d uPA -/- ($P = .03$, $N = 5$). Lastly, while vein wall collagen significantly increased over time in each group, no significant differences in vein wall collagen were found between uPA -/-, PAI-1-/- and their respective WTs at 8d or 21d.

CONCLUSIONS: In experimental stasis DVT, vein wall remodeling was positively affected by lack of PAI-1, with preservation of VSMC contractile phenotype, while deletion of uPA was associated with less endothelialization, but little other significant alteration. Whether PAI-1 inhibition could preserve vein wall VSMC integrity awaits further study.

P10 Postoperative Complications of Trans-Illuminated Powered Phlebectomy: A Review of 188 Surgeries

A. Kussman, F.C. Vandy, S. Blackburn, J. Bloom, A. Clay, E. Fellows, M. Kantola, W. Laforge, C. Stabler, T.W. Wakefield
University of Michigan, Ann Arbor, MI

BACKGROUND: Trans-illuminated powered phlebectomies (TIPPS) is an alternative to stab phlebectomies for elimination of venous varicosities. However, TIPPS remains underutilized and thus, outcomes are underreported.

METHODS: We reviewed our prospectively collected venous procedural database from January 2008 to July 2010. All patients who underwent TIPPS with or without saphenous vein ablation were included for analysis. Limbs with a CEAP classification of 5 or 6 were combined into one group due to small sample size. Follow up was conducted at early (0–30 days) intermediate (31–90 days) and later (91–180 days) intervals.

RESULTS: 188 limbs in 156 patients underwent TIPPS. Demographically, the cohort was 72% female and the average age was 49.9 years with a body mass index of 29.4. Combined radiofrequency or laser ablation was done 63% and 8% of the time respectively. The majority of limbs were had a CEAP classification of 2 (C_2–60%; C_3–28%; C_4–9%; $C_{5/6}$–3%). Average pre-operative Caprini risk score was 5.3 and the average pre-operative venous clinical severity score was 7.0. The mean number of incisions per patient was 8. Venous ultrasound at the first postoperative visit (average, 11.9 days) detected 9 (5.3%) cases of deep vein thrombosis (DVT). Of these, 5 (2.7%) cases were attributed to the ablation procedure and consisted of a tail of thrombus at the saphenofemoral junction. The other 4 (2.1%) cases were attributed to TIPPS. Pulmonary embolism occurred only once in this series and was not associated with a DVT. Cellulitis occurred 17 times (9%) and hematomas developed 12 times (6%), 3 requiring evacuation. Bruising, noted at the first postoperative visit, was seen in 57% of cases (mild, 30 cases; moderate, 74 cases; severe, 4 cases). At intermediate follow up (average, 3.2 months), paresthesias persisted in 35 cases (19%) of which the majority were peri-incisional.

CONCLUSIONS: TIPPS can be done with minimal risk. In favor of TIPPS, the number of incisions with TIPPS (8) is much lower than the number of incisions with our previously reported stab phlebectomy (31). However, patients must be advised of persistent paresthesias and transient bruising, and monitored for cellulitis.

P11 DVT Clinical Scoring Criteria: The Socioeconomic Impact

M. Esco, Y. Rits, M. Mattos, O. Brown, J. Rubin
Detroit Medical Center/Wayne State University, Detroit, MI

BACKGROUND: To create a scoring system for the diagnosis of acute DVT (aDVT) for comparison between urban (UN) vs. suburban (SBN) settings to eliminate unnecessary ultrasounds.

METHODS: 400 patients were evaluated for aDVT in a 4-month period. Prospectively collected, 29 variables (SVS/ISCVS plus 17 reported in literature) were compared between two socioeconomically distinct tertiary hospitals.

RESULTS: Acute DVT was detected in 15.7% of all ultrasounds. The urban setting had more African Americans (71%) and no insurance (32%). In the UN setting, 5 of 12 SVS/ISCVS criteria were predictors of aDVT: obesity, heart disease, immobility, postpartum and prothrombotic state; 6 of 17 clinical criteria: thigh and calf circumference, venous stasis, gender, duration of symptoms and insurance status. Conversely, in the suburban setting, 2 of 12 SVS/ISCVS criteria were individual predictors for aDVT: prior DVT and hormonal therapy. In addition, 2 of 17 clinical criteria: surveillance examination and suspected DVT were predictive. African Americans with obesity, malignancy, symptoms > 7 days, trauma, postpartum, recent surgery, prothrombotic, lower extremity swelling and coronary artery disease were individual predictors of acute DVT. In comparison, Caucasians with a history of prior DVT, immobility, hormonal therapy and female gender were found to be individual predictors of acute DVT.

CONCLUSIONS: To improve pretest probability, clinical predictive value, elimination of unnecessary and costly US evaluations of a suspected acute DVT, clinical scoring criteria should be adjusted to the socioeconomic composition of the particular institution.

P12 Improving Retrieval Rates in Temporary Inferior Vena Cava Filters

A.P. Gasparis, G. Spentzouris, D. Elitharp, N. Labropoulos, A. Tassiopoulos
SUNY Stony Brook, Stony Brook, NY

OBJECTIVE: Most studies have shown that the rate of inferior vena cava filter (IVCF) retrieval rarely exceeds 20%. A review of practices in our own institution revealed similar results (14%). This prompted us to develop a program dedicated to improve retrieval rates. We report the preliminary results of an ongoing study following the development of this program.

METHODS: Consecutive patients who had IVCF placed by the vascular service over a 6 month period (1/10–6/10) were followed prospectively. A dedicated nurse practitioner was responsible in developing a database, maintain contact with all patients and ensure that arrangements were made for retrieval when indications for IVCF protection were no longer present. Demographics, indication for filter placement, timing to filter retrieval and complications during placement and retrieval were prospectively collected. Retrieval rate was compared to the baseline institution data.

RESULTS: During the study period, 30 patients had IVCF placed. There were 23 men and 7 women with a mean age of 58 (25–88). Seven IVCF s (23%) were placed as permanent and 23 as temporary. The indications for IVCF included acute deep vein thrombosis and contraindication to anticoagulation (57%), perioperative interruption of anticoagulation (27%) while 5 patients (17%) had an IVCF placed prophylactically as they were considered high risk for pulmonary embolism. During follow-up 1 patient died from terminal cancer and 4 IVCFs were made permanent (one refused to have the filter removed, 2 could not receive anticoagulation and one had IVC thrombus). Therefore retrieval was attempted in 18 patients with a 100% success rate and no complications. Median time to retrieval was 21 days ranging from 4–140 days. Retrieval rate for IVCFs designated as temporary was 82% (18/22), which was significantly higher compared to our baseline data ($p < 0.001$).

CONCLUSION: Initial data show that a dedicated follow up program that closely monitors patients with temporary IVCFs for ongoing need of filter prophylaxis can result in high retrieval rates. The endurance and long term success of such a program need to be further validated.

P13 Inherited Thrombophilia and Thrombotic Venous Disease in Pregnancy

A. Wiazniewski, P. Szopiski, K. Bykowska
Institute of Haematology and Transfusion Medicine, Warsaw, Poland

BACKGROUND: Pregnant women are known to be at an increased risk of venous thromboembolism (VTE). The risk of symptomatic VTE during pregnancy is between 0,5 and 3,0 per 1000 women. Women with inherited thrombophilia (IT) have an increased risk of VTE during pregnancy and the puerperium.

The aim of our study was to present dependency between prevalence of deep vein thrombosis (DVT) and inherited thrombophilia (IT) in pregnant women.

METHODS: In period June 2000–August 2010, 41 pregnant women with proximal DVT (28 in pregnancy, 13 in puerperium) were treated. DVT was diagnosed by providing ultrasound colour Doppler. Treatment and next prophylaxis of DVT in those pregnant women included use of low molecular weight heparin (LMWH). Only in case of one patient presence of risk factors of IT were known before hospitalization. The defect was combined: protein C and S deficiencies. In the rest of women examinations towards IT were performed approx. 6 months after delivery and/or six months after finished treatment of DVT.

RESULTS: We found the following risk factors of VTE during pregnant women hospitalization: prolonged immobilization, obesity, smoking, varicose veins, surgery (cesarean section), previous thromboembolism and the presence of an inherited hypercoagulable state.

9/41 (22%) women had in history of VTE and 27/41 (65,8%) had chronic venous disease: C1 and/or C2 accoding CEAP. In 13/41 (31,7%) pregnant women was detected at least one risk factor of inherited thrombophilia and six of them had in history VTE. The following defects of IT were detected: in three patients factor V Leiden mutation, in four protein C deficiency, in three prothrombin G20210A mutation and in two combined defects: protein C and S deficiencies and protein S deficiency and prothrombin G20210A mutation. Only in patient where presence of IT was diagnosed before pregnancy, antithrombotic prophylaxis using LMWH was given during pregnancy and postpartum.

CONCLUSIONS: Pregnant women with a history of idiopathic venous thrombosis which can suggest presence of inherited hypercoagulable defects (IT) should be offered, during pregnancy and postpartum, LMWH and/or antithrombotic stockings as a prophylaxis against VTE.

P14 Clinimetry of Skin Changes in CVD: The Potential of Clinical Image Analysis

F. Becker[3], P. Fourgeau[2], P.H. Carpentier[1]
[1]Grenoble University Hospital, Grenoble, France, [2]Laboratoires Innothera, Arcueil, France, [3]University Hospital of Geneva, Geneva, Switzerland

BACKGROUND: Quantitative assessment of the cutaneous complications of chronic venous disorders (CVD) is necessary for therapeutic trials as well as for natural history studies. Digital image analysis of the physical signs makes theoretically possible such a clinimetric approach; however, no such method has been validated yet. The aim of this study was to evaluate the clinical validity and the reproducibility of the quantification of the ankle blue telangiectases (corona phlebectatica) and the pigmentation surface area obtained through a standardized photographic technique and a dedicated digital image analysis system.

METHODS: Pictures are obtained through a specially designed photographic stand aiming at a reproducible positioning of the medial aspect of the patients' leg, using a Panasonic camera with a 14–50 mm objective and a 10 Mp resolution and a standardized illumination system. Digital image analysis was performed by means of a specially developed system using optimized algorithms for the detection of blue telangiectases and pigmentation, and quantifying them as the relative surface area of these abnormalities compared to the surface area of the region of interest. The clinical validity was studied as the correlation between the measured parameters and the CEAP C classes in a series of 32 subjects (25 females and 7 males, median age: 69 years) undergoing a spa treatment for CVD. The inter- and intra-observer reproducibility was tested in a subgroup of 10 subjects, and explained as the median value and 9^{th} decile of the relative variation (difference over mean value) for each parameter.

RESULTS: Both pigmentation ($r = .56$; $p < .001$) and telangiectases ($r = .36$; $p < .005$) surface areas increased significantly with the severity of the CVD as expressed by the CEAP C class. With regard to the reproducibility, for the telangiectases, median intra-observer variation was 4% (9^{th} decile: 14%) and median inter-observer variation was 12% (9^{th} decile: 23%). Regarding the pigmentation median intra-observer variation was 17% (9^{th} decile: 34%) and median inter-observer variation was 10% (9^{th} decile: 29%).

CONCLUSION: These results show that the quantification of CVD related skin disease by our technique produce parameters that are both clinically valid, and show a relatively low variability, allowing relevant comparative measurements. The long-term reproducibility, depending on physiological variability of the pigmentation and telangiectases, remains to be assessed before using this technique in longitudinal studies.

P15 Effect on the Great Saphenous Vein Diameter of Two Different Doses Delivered Energy with Radiofrquency VNUS Closurefast™

C. Garcia-Madrid[1], J. Pastor[2], E. Sala Planell[2]
[1]*Hospital Clínic, University of Barcelona, Spain,* [2]*Institut Vascular Sala-Planell (Teknon Medical Center), Barcelona, Spain*

BACKGROUND: The aim of the VNUS Closure radiofrequency ablation (RFA) is the occlusion of the treated vein. This study was designed to analyze how two differents doses of supplied energy affects the retraction of the great saphenous vein (GSV).

METHODS: Prospective, comparative study. 67 consecutive extremities were treated by means of the radiofrequency VNUS Closure*FAST* in patients with varicose veins in CEAP 2–6 secondary to reflux of the GSV. Two different doses of energy were supplied along the GSV: group I (n = 22) received 1 treatment cycle/segment and group II (n = 45) received 2 cycles/segment. Patients underwent clinical and duplex follow-up at 4 day, 1, 3, 6, 12 months and yearly. Main outcomes were different GSV diameters (maximum and medium) rate of occlusion, presence of varicose veins and reflux. Analysis of the different diameters was performed using T-test and linear mixed model. Intraobserver variability was assessed by means the method of Bland & Altman, Lin's coefficient and intraclass correlation coefficient (ICC).

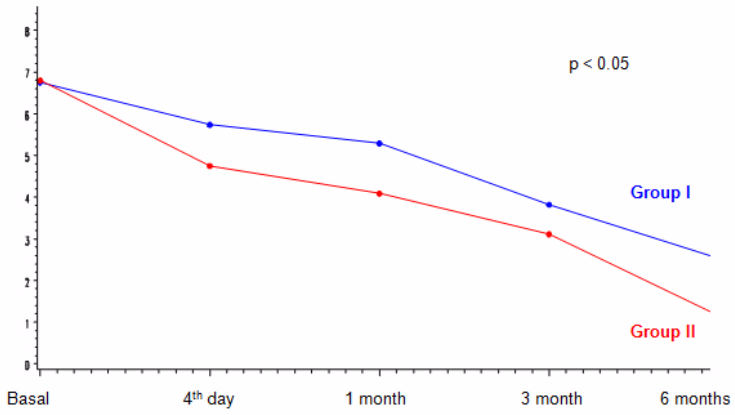


Fig 1.- Average reduction of GSV medium diameter (D med) along the study (T-test and linear mixed model)

RESULTS: Both groups were comparable so for demographic variables (sex, age, BMI, side) as for specific study variables (different basal diameters, length of treated vein, GSV stump). Intraobserver variability showed excellent for the maximum (Dmax) and medium (Dmed) diameters. Inmediate occlusion rate

was 100% for both groups. Mean length of segment vein treated was 31 ± 8 cm (range 14–45 cm) and mean distance to the junction was 13.8 ± 5.08 mm. The average reduction of the Md and md at 6 months were respectively 5.1 ± 2.0 mm (range 2.1–10 mm) and 5.3 ± 1.8 mm (range 1.1–7.8 mm). The group II showed a quicker and higher reduction of both diameters Md (54.4% vs 44.6%) and md (83.3% vs 62.5%) ($p < 0.05$) at 6 months, with statistically significant differences at 4º day for Md at 4º day and 1 month for md. Not skin burns, paresthesia-neuritis, or deep vein thrombosis appeared.

CONCLUSIONS: Closure*FAST* radiofrequency of the GSV induces 100% occlusion rate and progressive reduction of its diameter. An increase × 2 of the dose energy supplied carries a quicker and higher reduction in the diameters without an increase of side effects. This could be important to reach an earlier dissapareance of the treated vein and also in order to increase the efficacy in the treatment of large veins.

NOTES

FRIDAY, FEBRUARY 25, 2011

7:00 AM – 7:30 AM **Continental Breakfast — Exhibits Open**

7:30 AM – 9:00 AM **SCIENTIFIC SESSION V**
Chronic Venous Disease (Ulcers)
Moderators: *Harold Welch, MD*
Michael Vasquez, MD

Educational Objective: At the completion of the session, participants should be able to:

1. Define the involvement of microvenous valves in the development of CVD.
2. Understand scoring instruments used to assess post-thrombotic syndrome.
3. Determine the need for best practice guidelines for venous ulcers.
4. Understand the need to identify profunda femoral vein axial transformation as an outflow source in patients with severe femoral-ilio-caval obstruction.
5. Gain knowledge in the cellular mechanisms of vein tissue relaxation and the potential impact in management of CVD.

7:30 AM – 7:50 AM **15** **Failure of Microvenous Valves in Small Superficial Veins—A Key to the Development of Venous Ulcers**

A.M. van Rij, J. Vincent, G. Hill, G.T. Jones
Department of Surgery, University of Otago, Dunedin, New Zealand

BACKGROUND: While some patients who develop gross varicose veins with marked reflux fail to go on to develop the skin changes of venous insufficiency and ulceration, other patients with similarly severe varicose veins do develop these complications. Why this is so is not understood. Differences in compliance in the varicose veins have been suggested. This study using retrograde resin caste venography explores another possible factor.

METHODS: Resin castes were made of the superficial venous system in amputated lower limbs using retrograde filling from the GSV (similar in concept to retrograde venography to show valve incompetence in the deep venous system). Resin was injected into the GSV at the level of the medial malleolus. Outflow vessels were ligated directing resin into the small superficial veins. This could only occur if valves guarding these regions were either absent or incompetent.

Following hardening of the resin and chemical maceration of the tissues the remaining caste was examined with a dissecting microscope for the presence of valves as identified by their unique imprint in the resin. Valves were mapped to display their (1) competence, (2) diameter, and (3) position in the branching network extending to the GSV.

Two groups of limbs were examined: a) those where duplex ultrasound prior to amputation had shown no reflux in the GSV, b) where reflux was present along with skin changes of venous insufficiency.

RESULTS: Variable levels of reflux were demonstrated, from the 7 limbs with normal GSV, through several generations of small veins even out to the small venular networks in the skin. Most of the 247 microvalves identified were in the third generation of small veins from the GSV and these appeared to be most critical as their failure most often lead to reflux directly into the skin network. There was no such reflux seen in the leg of the youngest subject.

In the 4 limbs with venous insufficiency with venous ulcer formation there was dramatic extensive incompetence of the microvenous valves and appearance of resin into tortuous varicose networks in the area and into the distended capillaries.

CONCLUSIONS: Reflux and valvular incompetence occurs in the small superficial veins of the normal lower leg in the absence of reflux in the GSV. This may increase with age and with loss of tissue support around these small veins.

We suggest that varicose veins only go on to damage the skin when they are associated with areas of failure of microvenous valves.

7:50 AM – 8:10 AM **16** **A Comparison of the Villalta and Venous Clinical Severity Scoring Instruments in the Assessment of Post Thrombotic Syndrome**

A. Jayaraj, C. Natiello, S. Nicholls, M. Meissner
University of Washington, Seattle, WA

BACKGROUND: Post-thrombotic syndrome is a common chronic complication of acute deep venous thrombosis (DVT), with as many as two-thirds of patients developing symptoms of pain, edema, hyperpigmentation, or ulceration. There exist multiple instruments to assess post thrombotic syndrome including the commonly used scoring systems put forth by Villalta et al and the American Venous Forum's Venous Clinical Severity Score (VCSS). At present studies comparing the two in their ability to identify and grade the severity of post thrombotic syndrome (PTS) do not exist. This is important to enable comparison of studies that have used different instruments to assess PTS. The purpose of this study is to compare the two instruments as part of a larger randomized controlled study that assessed the impact of graduated compressive stockings in the prevention of post thrombotic syndrome.

METHODS: 138 extremities in 69 consecutive patients with an acute deep venous thrombosis documented by duplex ultrasonography were randomized to treatment with graduated compressive stockings (GCS) that provided compression of 30–40 mm Hg or no stockings to assess impact of GCS on the prevention of PTS. As part of this study, these patients were sequentially followed at months 1, 3, 6, 12, 18, and 24 following diagnosis of DVT. Post-thrombotic syndrome scores as defined by Villalta et al (PTSV) and the Venous Clinical Severity Score (VCSS) were assessed at these follow up visits. The PTSV was scored as Absent (Score < 3 or = 3 without objective criteria), Mild to Moderate (score ≥ 3 with one objective criteria) or Severe (score ≥ 4) while the VCSS score was assessed as Absent (score ≤ 3), Mild to Moderate (score 4–7) and Severe (score ≥ 8) based on performance characteristics of the venous clinical severity score. Each extremity was considered separately for analysis. The two instruments were compared using Pearson's Chi square analysis at various time points mentioned above. Additionally correlational statistics including Spearman correlation and Gamma statistic were computed.

RESULTS: A significant difference was not detected in the ability of PTSV and VCSS instruments to detect mild to moderate disease. (Spearman correlation: 0.41 to 0.73, Gamma statistic: 0.71 to 0.98, $p < 0.05$). For severe disease, the Chi square test suggests a difference in the ability of the two instruments to detect disease although there exists good correlation (Spearman correlation: 0.20 to 0.59, Gamma statistic: 0.71 to 1.0, $p < 0.05$) between the two instruments.

CONCLUSIONS: Both PTSV and the VCSS scoring systems are important tools in the identification and follow up of post thrombotic syndrome. There exists agreement between the two instruments for detecting both mild to moderate and severe disease.

8:10 AM – 8:30 AM **17** **The Need for an Intersociety Consensus Guideline for Venous Ulcer**

T.F. O'Donnell, Jr.
Tufts Medical Center, Boston, MA

BACKGROUND: Due to their recurrence and prolonged healing time, venous ulcers (VU) consume considerable resources in healthcare systems-up to 1% of healthcare budgets in some industrialized countries. Best practice guidelines (GLs) incorporate evidence-based diagnostic and therapeutic recommendations in a cost-effective manner and have been associated with improved and effective outcomes for many diseases, e.g., DVT/PE.

OBJECTIVES: In order to develop a more universal GL we determined whether there are common elements in GLs for VU and their evidentiary strength.

METHODS: A systematic analysis of GLs for VU that were identified through clinicaltrials.gov, a government-sponsored web site, and from experts outside the U.S.

RESULTS: Ten of 12 GLs on VU (7 North America and 5 Europe) were evidence-based, with the majority using the GRADE method. Only 2 had been developed or updated within the last 3 years. Venous duplex and ankle ABIs were recommended in all. Debridement was suggested in 2, while simple nonadherent wound dressings were favored in 9, and hydrocolloid in 2. Only 1 GL discussed a range of dressing options, dependent on the condition of the VU. High pressure multi-layer compression bandages were favored in 10. Only 2 focused on the importance of improving ankle joint mobility.

CONCLUSIONS: While there are numerous evidence-based GLs for VU, the majority may lag recent developments in the field. There is agreement on two elements- dressings and compression, among the various GLs, which should facilitate the development of a consensus GL, similar to that for DVT/PE. To improve patient care and reduce wasted resources, it is imperative for specialty societies to join together and develop this consensus document.

8:30 AM – 8:50 AM **18** **Axial Transformation of the Profunda Vein Sustains Ilio-Caval Stenting in Postthrombotic Limbs**

P. Neglén, B. Furrh, IV, S. Raju
River Oaks Hospital, Flowood, MS

BACKGROUND: The profunda femoris vein (PFV) provides an important collateral pathway when the femoral vein is obstructed by thrombosis. There is a special subset of limbs with severe obstructive postthrombotic disease in which the PFV enlarges to a variable extent (axial transformation) to compensate for severe chronic postthrombotic obstruction of the femoral vein. In extreme cases the profunda femoris vein completely replaces the femoral vein as the main outflow source for the limb. This study aims to assess patency and clinical outcome after stenting of the femoro-ilio-caval venous outflow in presence of femoral vein obstruction with axial transformation of the profunda vein.

METHODS: Limbs with ilio-caval obstruction combined with varying degrees of axial transformation and obstruction of the femoral vein were identified. The patent proximal profunda vein was usually image by duplex ultrasound scanning, but the complete transformation and the popliteal vein connection frequently required visualization by ascending venography. The profunda vein was accessed by direct puncture in the proximal thigh or selective catherization of the profunda-popliteal connection following popliteal vein cannulation. Stents were also placed through the obstructed femoral vein after puncture in the thigh area ensuring that the stent extended caudally to just above the profunda vein. Stent patency was followed by venogram or ultrasound imaging. Symptoms of pain (Visual Analogue Scale, 0–10), swelling (grade 0–3) and ulcer healing were recorded prospectively.

RESULTS: Thirty-two limbs in 31 patients (median age 50 years, range: 22–77; female/male ratio = 3/1; left/right limb ratio = 2.6/1; C5–6 = 37%; obstruction combined with reflux in 68%) were included in this study. Stents could be placed in all but 2 patients (failed recanalization). No major complications occurred. Twenty eight limbs were followed for 17 months (median, range: 1–133). The cumulative primary, assisted primary, and secondary patency rates at 4 years were 38%, 88% and 88%. Leg ulcer was found in 4 patients, 3 ulcerated limbs healed. Cumulative complete relief and improvement of pain (VAS drop ≥3) and swelling (≥1) at 4 years were 62% and 70%, and 39% and 84%, respectively.

CONCLUSIONS: It may appear to be impossible to stent patients with extensive postthrombotic obstructive disease because of perceived poor inflow. It is worthwhile to identify the presence of an axial profunda vein transformation in these limbs. Caudad extension of stenting of the femoro-ilio-caval venous outflow to just above or into a patent profunda vein transformation in the presence of an obstructed femoral vein, results in satisfactorily high patency rates and substantial symptomatic relief. Additional non-visualized axial collateralization may also contribute to these results.

8:50 AM – 9:10 AM **19** **Role of Vein Tissue Nitric Oxide and Hyperpolarization in Venous Relaxation: Implications in Venous Insufficiency Disease**

J.D. Raffetto[1], O.M. Reslan[2], R.A. Khalil[2]
[1]VA Boston HCS, West Roxbury, MA, [2]Brigham and Women's Hospital, Boston, MA

BACKGROUND: Vein wall dilation may play a role in varicose veins. However, the cellular mechanisms involved in vein tissue relaxation are not clearly understood. We have previously demonstrated that MMP-2 induces venous relaxation and hyperpolarization. The purpose of this study was to further characterize the venous relaxation pathways and the K^+-channels involved in hyperpolarization.

METHODS: Circular segments of inferior vena cava (IVC) were isolated from male rats, and suspended between two wires in a tissue bath for measurement of isometric contraction/relaxation. Following contraction to 96 mM KCl and phenylephrine (PHE, 10^{-5} M), veins were treated with acetylcholine (Ach, 10^{-9} to 10^{-5} M) and venous relaxation was measured. To measure the nitric oxide (NO)- and prostacyclin (PGI_2)-dependent relaxation, veins were treated with L-NAME (3×10^{-4} M) and indomethacin (10^{-5} M), respectively. To measure the hyperpolarization pathway, the tissues were treated with tetraethylammonium (TEA, 10^{-3} M), a nonselective blocker of K^+ channels. To test for the contribution of a specific K^+-channel, the effects of K^+-channel blockers on Ach-induced IVC relaxation were tested: apamin (small conductance Ca^{2+}-dependent, 10^{-7} M), iberiotoxin (IbTx, large conductance Ca^{2+}-dependent, 10^{-8} M), 4-aminopyridine (4-AP, voltage-dependent, 10^{-3} M), glibenclamide (GLB, ATP-dependent, 10^{-5} M). Relaxation data are presented as % means ± sem.

RESULTS: Ach caused concentration-dependent relaxation of PHE contraction (max 48.3 ± 5.4). In the presence of L-NAME, Ach-induced relaxation was reduced (max 28.8 ± 5.7, p = 0.009). Addition of sodium nitroprusside (10^{-5} M) caused further relaxation to 87.7 ± 2.5, indicating that the VSM relaxation mechanisms are intact. ODQ (inhibitor of guanylate cyclase and cGMP production, 10^{-5} M) inhibited Ach-induced relaxation (34.9 ± 12.2), supporting a role of NO-cGMP relaxation pathway. In the presence of L-NAME and indomethacin, Ach still produced significant relaxation (45.3 ± 5.4), that was abolished in the presence of TEA (0.7 ± 0.5). Cromakalim, activator of K^+ channels, caused dose-dependent relaxation (max 94.2 ± 0.8), that was inhibited in IVC precontracted with 96 mM KCl (1.6 ± 0.1), indicating that the IVC has functional K^+-channels involved in relaxation. In veins precontracted with PHE, specific K^+-channel blockers caused significant inhibition of Ach relaxation: Apamin 4.8 ± 0.3, IbTx 7.5 ± 2.5, 4-AP 0.0 ± 0.0, GLB 9.2 ± 0.8.

CONCLUSIONS: A significant component of venous relaxation involves the NO-cGMP pathway. An additional component of venous relaxation involves hyperpolarization and activation of various types of K^+ channels. Increased vein tissue NO-cGMP activity and membrane hyperpolarization could promote venous dilation and varicose vein formation, and localized vein delivery of specific blockers of the NO-cGMP pathway and K^+-channels may be useful in the management of venous insufficiency disease.

9:10 AM – 9:20 AM **ACP PLATINUM ABSTRACT**

Combined Use of Pretest Clinical Probability Score and Latex Agglutination D-Dimer Testing in Excluding Acute Deep Vein Thrombosis

T. Yamaki, A. Hamahata, M. Nozaki, H. Sakurai
Tokyo Women's Medical University, Tokyo, Japan

BACKGROUND: Currently, latex agglutination D-dimer assay is widely used but is considered less sensitive to exclude deep vein thrombosis (DVT) in comparison with ELISA based D-dimer test. The purpose of study was to determine if a combination of different cut-off points rather than single cut-off point of 1.0 g/mL and pretest clinical probability (PTP) score could reduce the use of venous duplex scanning in patients with suspected DVT using latex agglutination D-dimer assay.

STUDY DESIGN: Ninety hundred eighty-nine consecutive patients with suspected DVT were evaluated using PTP score and D-dimer testing before venous duplex scanning. After calculating clinical probability scores, patients were divided into low risk (0 points), moderate risk (1–2 points), and high risk (3 points) pretest clinical probability groups. The receiver operating characteristic (ROC) curves analysis was used to determine appropriate D-dimer cutoff point in each PTP with a negative predictive value of >98% for a positive duplex scan.

RESULTS: Eight hundred eighty-six patients were enrolled. The prevalence of DVT in this study was 28.9%. Five hundred and eight patients (57.3%) were classified as low risk, 237 (26.8%) as moderate risk, and 141 (14.9%) as high risk PTP. DVT was identified in 29 patients (5.7%) with low risk, 118 (49.8%) with moderate risk, and 109 (77.3%) with high risk PTP. Using ROC curves analysis, D-dimer cut-off points of 2.6, 1.1 and 1.1g/mL were selected for the low, moderate and high PTP groups respectively. In the low PTP group, specificity increased from 48.9% to 78.2% ($P < 0.0001$) with use of the different D-dimer cut-off value. In the moderate and high risk PTP groups, however, the different D-dimer levels did not achieve substantial improvement. Regardless, overall venous duplex scanning could have been reduced by 43.0% (381/886) using different D-dimer cut-off points.

CONCLUSIONS: A combination of a specific D-dimer level and clinical probability score is most effective in the low PTP patients in excluding DVT. In the moderate and high PTP group, however, the recommended cut-off points of 1.0 g/mL may be preferable. These results show that different D-dimer levels for different risk patients is feasible using latex agglutination D-dimer assay in excluding DVT.

9:20 AM – 10:00 AM **Coffee Break — Visit Exhibits**

10:00 AM – 10:25 AM **PRESIDENT'S SESSION**
Moderators: Peter J. Pappas, MD
Seshadri Raju, MD

10:00 AM – 10:15 AM **2010 SERVIER TRAVELING FELLOWSHIP REPORTS**
Christopher Pannucci, MD
University of Michigan

K. Barry Deatrick, MD
University of Michigan

10:15 AM – 10:25 AM **2010 BSN JOBST RESEARCH WINNER – INTERIM REPORT**

A Novel in Vitro Model of Chronic Venous Insufficiency
Yanjie Qi, MD
University of Rochester

10:25 AM – 10:40 AM **Presidential Address Introduction**
Introduction By: Seshadri Raju, MD
President-Elect

10:45 AM – 11:30 AM **PRESIDENTIAL ADDRESS**
Peter J. Pappas, MD

11:30 AM – 12:30 PM **MEMBER BUSINESS LUNCHEON**

12:30 PM **Free Afternoon**
Golf/Tennis Tournaments

NOTES

SATURDAY, FEBRUARY 26, 2011

7:00 AM – 7:30 AM **Continental Breakfast — Visit Exhibits**

7:30 AM – 9:50 AM **SCIENTIFIC SESSION VI**
CVD – Treatment of Superficial Venous Disease
Moderators: *David Gillespie, MD*
M. Ashraf Mansour, MD

Educational Objectives: At the completion of the session, participants should be able to:

1. Describe anatomical changes to the superficial venous system after saphenous phlebectomy.
2. Define the main clinical variables in new duplex venous testing scores after varicose vein treatment.
3. Be able to list the method and 2 candidate genes associated with varicose vein presence.
4. Describe the demographic and practice patterns of patients undergoing endovenous ablation for venous insufficiency.
5. Define outcomes and efficacy of foam sclerotherapy for venous varicosities.

7:30 AM – 7:50 AM **20** **Change in Venous Outflow Patterns of the Leg After High Ligation and Stripping of Great Saphenous Vein and Phlebectomies**

T. Ogawa, S. Hoshino
Fukushima Daiichi Hospital, Fukushima, Japan

BACKGROUND: High ligation and stripping of great saphenous vein (GSV) is still the method of choice for treatment of varicose veins in most parts of the world. Recurrence rate is high and new endovenous modalities are developed where one of the advantages is to avoid the incision in the groin. This study was undertaken to clarify the change of venous outflow after high ligation and stripping of GSV and phlebectomies.

METHODS: 45 patients (50 legs) with primary varicose veins with reflux of GSV (C2-C4b) participated in this study. They were examined before and 1–3 months after surgery using multi-detector CT venography (venous outflow evaluated after dye injection in the medial marginal vein of the foot until appearance of dye in IVC), and air-phlethysmography (venous filling index: VFI and outflow fraction: OF). All participants underwent stripping of GSV from sapheno-femoral junction to knee level with the complete interruption of saphenous confluence and stab avulsion of varicose veins.

RESULTS: CT venography visualized new superficial venous networks at calf and thigh in 40 of 50 legs where the main distribution was 20 legs at calf, 16 legs at thigh and 4 legs at both calf and thigh. Average VFI was significantly reduced from 4.62 ml/sec to 1.85 ml/sec after surgery, OF from 58.8% to 43.8%. Comparing OF in the 40 patients where CT venography showed new superficial venous network after surgery with the 10 patients without this finding, there was a significant decrease in the first group (50.4% to 44.6%) but no change in the second group (47.7% to 48.5%).

CONCLUSIONS: After high ligation and stripping of the incompetent GSV above the knee with phlebectomies, 80% of the legs showed new superficial venous networks draining the venous outflow from the distal GSV using CT venography. This may reflect the decreased venous hypertension or a compensatory development of superficial veins after removal of the GSV outflow.

7:50 AM – 8:10 AM **21** **Validation of a New Duplex Derived Effectiveness Score in Quantifying Varicose Vein Treatments**

C.R. Lattimer[1], E. Kalodiki[1], M. Azzam[2], P. Trueman[3], G. Geroulakos[1]
[1]Ealing Hospital & Imperial College, London, United Kingdom, [2]Ealing Hospital, Middlesex, United Kingdom, [3]Brunel University, Middlesex, United Kingdom

BACKGROUND: Duplex evaluations of success are usually descriptive using terms like abolition of reflux and obliteration. Many patients however, have mixed patterns of treatment effect which are difficult to compare. A simple and flexible treatment scoring system is proposed, the saphenous treatment score (STS), which references the pre- and post-treatment evaluation of the above (AK) and below knee (BK) part of the great saphenous vein (GSV). Analysis of the change in STS may then provide a numerical value of effectiveness which can be used for standardizing treatment comparisons between studies.

METHODS: Sixty-six consecutive patients with GSV reflux (>0.5 sec), received either endo-venous laser therapy (EVLT) with concurrent phlebectomies or ultrasound-guided foam sclerotherapy (UGFS) with up to 12 ml of 1% STD™ foam. Patients with lesser saphenous or deep vein reflux or a history of DVT were excluded. Assessments were performed before and after treatment using the Aberdeen varicose vein questionnaire (AVVQ), the venous clinical severity score (VCSS), the venous filling index (VFI) using air plethysmography and the STS using duplex. The AK and BK segments of the GSV were individually graded: 3, 2, or 1 representing the presence of reflux, patency with no reflux, or occlusion, respectively. Mixed patterns were weighted with a score of 3 having preference over both 1 and 2, and 1 having preference over 2. This gives a final STS of between 6 and 2 for the GSV. The Difference in Mean STS (DMS) before and after treatment is presented.

RESULTS: These results demonstrate the DMS compared against other assessment parameters (Table 1), ongoing treatments (Table 2) and between different treatments (Table 3).

Table 1: Analysis on 66 Patients Undergoing Primary Treatment Before and at 3 Weeks

STS	Median (range)	Mean (95% CI)	
Pre	6 (4–6)	5.7 (5.58–5.82)	
Post	3 (2–6)	3.30 (3.04–3.56)	
Difference	2 (0–4)	2.39 (2.11–2.67)	**DMS** = 2.39
AVVQ	**Median (range)**	**IQR**	
Pre	21.52 (0.86–52.93)	15.48	
Post	18.86 (5.50–66.89)	11.27	p = 0.14 (Wilcoxon)
VCSS	**Median (range)**	**IQR**	
Pre	6 (2–20)	4	
Post	3 (0–10)	4	p < 0.0005 (Wilcoxon)

Table 2: Subgroup Analysis on the 15 Patients Requiring Additional UGFS Treatments

STS	**Median (range)**	**Mean (95% CI)**		
Pre	6 (5 - 6)	5.8 (5.57 - 6.03)		
Post 1	4 (2 - 6)	4.13 (3.58 - 4.68)	**DMS** = 1.67 (Pre - Post1)	
Post 2	3 (2 - 4)	2.6 (2.25 - 2.95)	**DMS** = 1.53 (Post1 - Post2)	p < 0.0005 (Friedman)
VFI (ml/sec)	Median (range)	Mean (95% CI)		
Pre	6.3 (1.4 - 15)	6.9 (4.3 - 9.5)		
Post 2	1.9 (0.3 - 3.7)	1.9 (1.4 - 2.6)	p = 0.001 (Wilcoxon)	

Table 3: Three Week Primary DMS Between 38 EVLT and 28 UGFS Patients

DMS	**EVLT (95% CI)**	**UGFS (95% CI)**	
AK	1.92 (1.83–2.01)	1.57 (1.27–1.88)	
BK	0.87 (0.57–1.17)	0.29 (0.03–0.54)	
TOTAL	2.79 (2.46 - 3.12)	1.86 (1.43–2.29)	p = 0.001 (M-W U-Test)
	p < 0.0005 (Wilcoxon)	p < 0.0005 (Wilcoxon)	

CONCLUSIONS: The STS is based on scoring the presence of reflux, patency/competency and occlusion. It can grade the effects of treatment on the GSV both above and below the knee. We have shown a different numeric score for foam compared to laser in the management of primary GSV reflux at three weeks. Further studies incorporating the lesser saphenous vein into the scoring will provide us with a global score of effectiveness in the management of the saphenous trunks of the extremity.

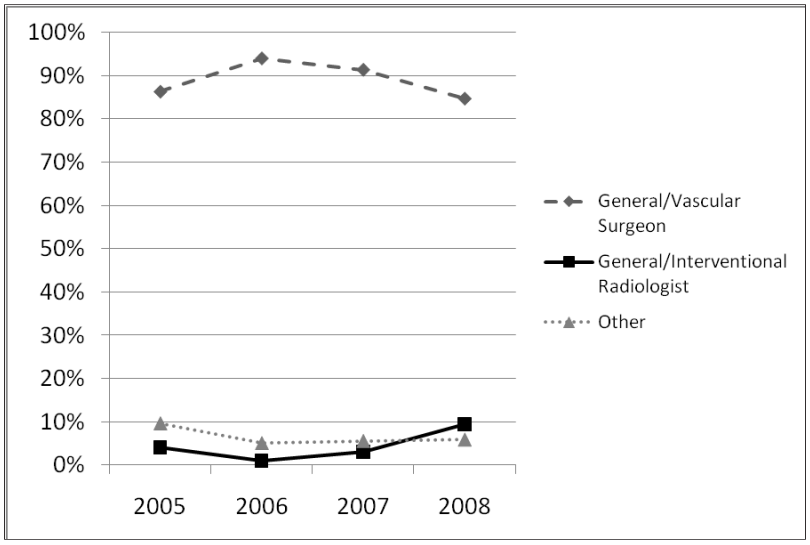
8:10 AM – 8:30 AM **22** **The State of Endovenous Ablation for Venous Insufficiency in Florida**

M.S. Hong, K. Butler, T.D. Fischer, P.R. Nelson
University of Florida, Gainesville, FL

BACKGROUND: Endovenous ablation has recently been adopted for treatment of lower extremity venous insufficiency. We sought to define the current trends in the use of endovenous procedures and the impact on the associated costs in the state of Florida.

METHODS: The Agency for Healthcare Administration (AHCA) database, which contains 100% of encounters from ambulatory centers in Florida, was queried for endovenous laser or radiofrequency ablation procedures, using CPT codes 36478 and 36475 respectively, for the study period of q1 of 2005 to q3 of 2008. ICD-9 diagnostic codes were used to identify severity of the venous disease. The main measures included type of ablation, provider specialty, and total charges. A comparison of practice patterns and associated charges were performed for the two main provider groups, namely General/Vascular surgeons (GS/VS) and Diagnostic/Interventional Radiologists (DR/IR).

RESULTS: Our query consisted of 4,143 encounters during the study period. The mean age was 54 ± 13 years, and 73% were women. Compared to the Florida population, white and white-hispanic race was over-represented (91.4% vs. Florida 81%), and black race was under-represented (4.3% vs. Florida 16%). The most common complaint was edema, pain, or swelling (45.5%) followed by varicose veins with inflammation (17.8%), unspecified



venous insufficiency (12.6%), asymptomatic varicose veins (11.9%), and phlebitis/thrombophlebitis (10.2%). Laser ablation outnumbered radiofrequency ablation each year (mean 697 vs. 416 per year respectively). Vascular and general surgeons comprised the vast majority of providers, consisting of 48.7% and 40.9% of encounters, respectively. Other providers of note include cardiothoracic surgeons (1.2%), and diagnostic/interventional radiologists (3.8%). In total, 21 specialties were represented. GS/VS providers performed secondary ablations in 32.5% encounters, and stab phlebectomies in 18.2%. In contrast, DR/IR performed secondary ablations in only 3.8%, and stab phlebectomy in 5.1%. Total charges overall were $11,644 ± 6,682, $11,696 ± 6,594 for GS/VS, and $6,392 ± 4,820 for DR/IR. Most of this difference was due to charges related to the operating room ($7,858 GS/VS and $3,944 DR/IR), likely reflecting differences in practice patterns.

CONCLUSIONS: Endovenous ablation offers a minimally invasive option for treatment of venous insufficiency. Although providers span a wide range of specialties, the majority of endovenous ablations performed in ambulatory centers are performed by General or Vascular Surgeons. GS/VS performed substantially more secondary procedures compared to DR/IR, resulting in higher total charges. More studies are required to determine whether this disparity in practice patterns result in different outcomes.

8:30 AM – 8:50 AM **23** **Randomized Controlled Trial of Ultrasound Guided Foam Sclerotherapy Combined with Sapheno-Femoral Ligation Compared to Surgical Treatment of Varicose Veins: Five-Year Results**

E. Kalodiki[1], M. Azzam[2], C.R. Lattimer[1], E. Shawish[2], N. Zambas[2], G. Geroulakos[1]
[1]Ealing Hospital & Imperial College, London SW7, [2]AZ, United Kingdom, [2]Ealing Hospital, Middlesex, United Kingdom

BACKGROUND: Up to 5 year results of a prospective randomized controlled trial comparing foam sclerotherapy and surgery to standard surgery, in patients with primary varicose veins.

METHODS: Seventy three patients (82 legs) underwent sapheno-femoral ligation, stripping and multiple phlebectomies under general anaesthesia (n = 39, M:F = 16:23, age 47 [23–76]) (group S) or sapheno-femoral ligation under local anaesthesia and concurrent sclerotherapy (n = 43, M:F = 11:32, age 49 [26–42]) (group F). Assessments included CEAP classification, ultrasound, VCSS, AVVQ and SF36 scores.

RESULTS: CEAP was similar between groups, C_{2-6}. On the 59 legs with completed ultrasound, reflux at 3–5 years is presented (Table 1).

Table 1: Ultrasound Result on 59 Completed Legs (1 = Occluded, 2 = Competent, 3 = Reflux)

	Venous Status	3 Years Above Knee n (%)	5 Years Above Knee n (%)	3 Years Below Knee n (%)	5 Years Below Knee n (%)
Group Surgery n = 26	1	17 (65.4)	14 (53.8)	6 (23.1)	10 (38.5)
	2	2 (7.7)	3 (11.5)	6 (23.1)	7 (26.9)
	3	7 (26.9)	9 (34.6)	14 (53.8)	9 (34.6)
Group Foam n = 33	1	16 (48.5)	19 (57.6)	15 (45.5)	8 (24.2)
	2	6 (18.2)	1 (3.0)	4 (12.1)	11 (33.3)
	3	11 (33)	13 (39.4)	14 (42.4)	14 (42.4)
Mann-Whitney U-Test	p =	p = 0.298	p = 0.194	p = 1.000	p = 0.341

In group S:40% legs required 25 additional foam sessions with a mean volume of 11 ml, total 154 ml. In group F:47.5% legs required 33 additional sessions, mean volume 9 ml, total 207 ml. Preoperatively the VCSS score was equivalent between the 2 groups (median-range-Interquartile Range (IQR), group S:5, 3 to 12, 3 and group F:4.5, 2 to 15, 2, p = 0.359 Mann-Whitney U-test). However, after treatment there was improvement within both groups (median-range-IQR for S:1, 0 to 9–5, p = 0.001, for F:1, 0 to 9–2, p < 0.0005 Wilcoxon). Changes

in VCSS before and at 3 years (p = 0.504) and the absolute VCSS scores (p = 0.313) were similar between both groups. The VSDS score improved in both groups due to treatment (Table 2).

Table 2: Venous Disease Severity Score Up to 5 Years Between Groups on 70 Patients

	Group Surgery Median (IQR)	Group Foam Median (IQR)	Mann-Whitney U-Test
Pre-treatment	1.0 (1.3)	1.0 (1.0)	p = 0.518
3 years	0.5 (1.0)	1.0 (1.0)	p = 0.780
5 years	1.0 (1.0)	0.25 (1.0)	p = 0.388
Friedman	p < 0.0005	p < 0.0005	

The AVVQ score also improved within both groups median-IQR preoperatively vs 3 yrs (S:16.32–4.7 vs 8.94–11.51, p = 0.003, F:12.28–10.37 vs 4.97–6.19 p < 0.0005 Wilcoxon). The improvement on the AVVQ score before and after treatment was similar in both groups, p = 0.703, Mann-Whitney U-Test). The SF36 mental scoring over 3 years improved in S (p = 0.04). However, there was no change in the physical scores in both groups (S:p = 0.361, F:p = 0.889) or the mental score in group F:p = 0.285. Furthermore, there was no difference in the changes on the physical and mental score between the treatment groups due to treatment (physical p = 0.724, mental p = 0.354, Mann-Whitney U-Test).

CONCLUSIONS: At 3–5 years follow up the treatment was equally effective between the 2 groups, as demonstrated with VSDS, VCSS and AVVQ score improvements. The additional foam sessions were also similar. Since surgery may not provide a definitive solution, foam sclerotherapy could be offered like a dental care treatment model i.e., "treat as and when the problem appears."

8:50 AM – 9:10 AM **24** **A New Approach to the Genetics of Varicose Veins: A Genome Wide Association Study**

A.M. van Rij, J. Krysa, G.T. Jones
University of Otago, Dunedin, New Zealand

BACKGROUND: The exact nature of the genetic basis of varicose veins remains unclear. A number of genetic associations have been described. These have however all been relatively small, limited candidate gene studies none of which have been validated. The aim of this study was to consider these current reported genetic associations with varicose veins and to carry out a case control analysis to validate them using the approach of a more comprehensive nonbiased genome wide association study (GWAS).

METHODS: An indirect, in silico, genome wide association study of varicose veins was undertaken. This was based on our abdominal aortic aneurysm GWAS in which the frequency of varicose veins was similar in cases and controls. Genetic polymorphisms associations with venous disease to date were identified through a literature search. All known single nucleotide polymorphisms (SNPs), with >5% allele frequency, in the genes previously implicated were analysed. Genotyping was carried out using Affymetrix Genome-Wide Human SNP Array 6.0.

RESULTS: 349 patients with varicose veins and 857 controls were included. Genes which have been implicated in venous disease so far include FOXC2, HFE C282Y, factor XIII V34L, estrogen receptor B, TNF-A, MTHFR and thrombomodulin. None of the SNPs in these genes have shown to have significant association in this study. However there were a number of other SNPs which were found to be associated with varicose veins. Some of these are quite novel and previously not linked to venous disease. These are being further validated in another larger and more strictly phenotyped cohort.

CONCLUSIONS: This is the first GWAS applied to varicose veins. The previous candidate genes implicated in common venous disease have not been confirmed. A GWAS approach has been shown to be useful in validation and discovery of novel genes in venous disease. Further larger cohorts are required to confirm these.

EUROPEAN VENOUS FORUM BEST PAPER 1

Withdrawn

EUROPEAN VENOUS FORUM BEST PAPER 2

Withdrawn

9:15 AM – 10:00 AM **Coffee Break — Last Chance to Visit Exhibits**

10:00 AM – 11:00 AM **UPDATE SESSION**
Moderator: Peter Pappas, MD

10:00 AM – 10:15 AM **PVS Ulcer Initiative**
Peter Henke, MD

10:15 AM – 10:30 AM **AVF Website Launch**
Marc Passman, MD

10:30 AM – 10:40 AM **AVF National Screening Program**
Marc Passman, MD

10:40 AM – 10:50 AM **Fellows' Courses in Venous Disease**
William Marston, MD

10:50 AM – 11:00 AM **Attendings' Course in Venous Disease**
Antonios Gasparis, MD

11:00 AM – 11:45 AM **D. EUGENE STRANDNESS MEMORIAL LECTURE**

Microcirculary and Lymphatic Disorders
David C. Zawieja, PhD
Director, Division Division of Lymphatic Biology, Texas A&M Health Science Center College of Medicine
Introduction By: Peter Pappas, MD

12:00 PM – 1:15 PM **LUNCH SYMPOSIUM**

Changing Concept in Lymphedema
B.B. Lee, MD

Educational Objectives: At the completion of the session, participants should be able to:

1. Become familiar with new approach on the lymphedema management based on a changing concept.
2. Understand newly recognized physiologic/pathophysiologic interrelationship between the venous and lymphatic system.

General Overview: How Much Did We Learn?
B. B. Lee, MD

Lymphedema: Where Have We Been? Where are We Going?
Stanley Rockson, MD

Revisit to Surgical Treatment: Is it a Viable Option?
Peter Gloviczki, MD

1:30 PM – 3:05 PM **SCIENTIFIC SESSION VII**
Deep Vein Thrombosis II
Moderators: *Peter Henke, MD*
Seshadri Raju, MD

Educational Objectives: At the completion of the session, participants should be able to:

1. Gain an understanding of alternative methods for thrombolysis in DVT and its effect on venous valve function.
2. Learn about the epidemiology of duplex ultrasonography for DVT, and common clinical associations.
3. Learn about new diagnostic methods for diagnosing DVT.

1:30 PM – 1:50 PM **25** **Thrombolytic Therapy with Tissue Plasminogen Activator: Why Prolonged Continuous Infusion Is Not the Best Approach**

R. Chang, J.N. Lozier, M.K. Horne, III
NIH, Rockville, MD

BACKGROUND: Continuous infusion has been assumed to be the optimal way of administering every thrombolytic agent approved for clinical use. However, this simplistic approach fails to take advantage of differences in properties of thrombolytic agents that could be exploited to increase efficacy as well as safety. In particular, continuous infusion is not necessary when using thrombolytic agents with strong fibrin binding and is not the optimal way of administering recombinant tissue plasminogen activator (r-tPA or alteplase). Once given by intraclot injection, alteplase binds to fibrin clot, and once bound to clot, its activity as a plasminogen activator increases several hundred fold,- also known as fibrin selectivity. Once the clot has been laced with tPA, prolonged fibrinolysis ensues, obviating the need for prolonged infusions.

METHODS: Forty five patients with subclavian, jugular, or central venous thrombosis (SJ-CVT) and 56 patients with acute deep vein thrombosis (of the lower extremity (DVT-LE)were treated with once daily intraclot pulse spray injection of tPA without prolonged infusions of tPA, but with full systemic anticoagulation. Initial protocols used high doses of tPA (20–40 mg/day for SJ-CVT, and up to 50 mg/day for DVT-LE) but were reduced 5–10 fold to a maximum of 4 mg tPA /d for SJ-CVT and a maximum of 10 mg tPA/d for DVT-LE after pharmacokinetic studies indicated the higher doses greatly exceeded the amount of tPA that could bind to acute fibrin clot.

RESULTS: Venous patency was restored in 34 of 45 (76%) SJ-CVT patients after an average of 2.1 days/treatments and 51 of 56 (91%) of acute DVT-LE patients after an average of 2.7 days/treatments. There was no loss of efficacy with decrease in dose of tPA. No major bleeding complications requiring transfusion were found at either dosing schedule.

CONCLUSIONS: Elimination of prolonged continuous infusion from thrombolytic regimens using tPA has 2 advantages. When many venous divisions are thrombosed, the catheter can be moved from one division to the next to load each segment of clot with tPA quickly, instead of having to leave the catheter in one division for prolonged infusion. This allows thrombolysis of many divisions in almost parallel fashion instead of the serial fashion required with conventional thrombolytic therapy. The second advantage is safety because with termination of intraclot injection, any tPA which reached the systemic circulation during injection is cleared rapidly due to its short half-life ($T_{\frac{1}{2}} \approx 5min$) shortening the duration of circulating tPA, whereas with conventional thrombolytic therapy, elevated systemic tPA levels and suppressed PAI-1 levels are likely to persist as long as the prolonged infusion continues.

1:50 PM – 2:10 PM **26** **Postoperative Deep Vein Thrombosis in Total Knee or Hip Replacement Operation Is Associated with Preoperative Increased Calf Muscle Deoxygenation**

T. Yamaki, A. Hamahata, D. Fujisawa, H. Konoeda, K. Kubo, M. Nozaki, H. Sakurai
Tokyo Women's Medical University, Tokyo, Japan

BACKGROUND: To assess whether preoperative calf muscle deoxygenated hemoglobin (HHb) level during light-intensity exercise is useful for identifying patients at risk of developing postoperative deep vein thrombosis (post-op DVT).

METHODS: Sixty-three patients receiving either total knee or total hip replacement operation were enrolled. Preoperative screening using compression ultrasound (CUS) of the bilateral lower extremities was performed if study patients already had DVT. The mean flow velocity of the popliteal vein (POPV) was assessed. Moreover, prevalence of venous reflux in the POPV was evaluated preoperatively. Near-infrared spectroscopy (NIRS) was used to measure calf muscle HHb levels. Calf venous blood filling index (HHbFI) was calculated on standing, then the calf venous ejection index (HHbEI) was obtained after one tiptoe movement and the venous retention index (HHbRI) after 10 tiptoe movements. All patients received fondaparinux for postoperative thromboprophylaxis.

RESULTS: There were no preoperative DVTs. Of 63 patients evaluated, post-op CUS confirmed DVT in 13 (20.6%) patients. There were no significant differences in mean age, BMI and gender distributions between patients with post-op DVT and these without. There was no significant difference in the mean flow velocity in the POPV between patients with post-op DVT and these without. ($p = 0.062$). Reflux in the POPV was found in 3 patients with post-op DVT and 12 without post-op DVT, and there was no significant difference between the groups ($p = 0.945$). The preoperative NIRS-derived HHbRI was significantly increased in patients who developed DVT in comparison with those who did not (7.78 ± 8.65, 1.83 ± 2.30, $p = 0.006$, respectively). There were no significant differences in the values of HHbFI and HHbEI between the study groups.

CONCLUSIONS: These results suggest that HHbRI, as measured by NIRS, may be promising parameter for identifying patients at risk of developing post-op DVT despite pharmacological DVT prophylaxis. These findings might be very helpful for physician in detecting patients who require more extensive thromboprophylaxis.

2:10 PM – 2:30 PM **27** **Patient Characteristics, Referral Patterns, and Associated Risk Factors in Patients Referred to an Outpatient Vascular Laboratory to Rule Out Deep Venous Thrombosis (DVT)**

K. Gibson[1], N.L. Polissar[2], M.B. Neradilek[2]
[1]Lake Washington Vascular Surgeons, Bellevue, WA, [2]The Mountain-Whisper-Light Statistics, Seattle, WA

BACKGROUND: Previous studies utilizing National or Regional data demonstrated that a significant proportion of DVTs are diagnosed in the outpatient setting. Little is known, however, about patient characteristics, referral patterns, and percentage of positive DVTs in patients referred to outpatient vascular laboratories (OVL). The study objective was to examine demographics, risk factors, presenting symptoms, and referring physician specialties in patients referred to an OVL and to delineate clot extent in patients diagnosed with DVT.

METHODS: Data was retrospectively collected from an OVL database of 1506 patients referred to rule out DVT over a thirteen-month period. Data collected included patient age, gender, risk factors, presenting symptom(s), and referring physician specialty. In patients with positive findings, the DVTs were categorized (acute or chronic), and extent of DVT was classified on a scale of one to four (4 = gastronemius vein, 3 = tibial vein, 2 = femoral and/or popliteal vein and 1 = common femoral and/or more proximal veins). Logistic regression was used to quantify association of risk factors with the presence of acute DVT.

RESULTS: Of the 1506 patients, 567 (38%) were men and 939 (62%) were women. Mean age was 57 (range 14–96). 30% of patients referred (n = 453) were postoperative. The most common presenting symptoms were pain (n = 600, 40%), edema (n = 425, 28%) and pain and edema (n = 406, 27%). Duplex scans were abnormal in 335 (22%) patients (223 acute DVTs,102 chronic DVTs). In the acute DVTs, extent was classified as 4 in 34 limbs (15%), 3 in 100 limbs (45%), 2 in 66 limbs (30%) and 1 in 23 limbs (10%). The most common referring physician specialties were Family Medicine (n = 601, 40%), followed by Orthopedics (n = 491, 33%), and Internal Medicine Specialties (n = 280, 19%). In acute DVTs, hypercoaguable states ($p < 0.001$), pregnancy ($p = 0.001$) and recent travel ($p = 0.04$) were associated with increased severity of DVT and postoperative state was associated with a decreased severity ($p = 0.03$). In the multivariate model for the presence of acute DVT, postoperative state (OR = 3.08, $p < 0.001$), male gender (OR = 1.94, $p < 0.001$), presentation with pain and edema (OR = 2.39, $p < 0.001$, compared to edema alone), and younger age (OR = 0.88, $p = 0.01$, per 10 years) conferred the greatest risk of acute DVT. Once gender, age, and postoperative status are accounted for, referring physician specialty was not a statistically significant predictor of DVT.

CONCLUSION: In patients referred to an OVL, patient gender, age, postoperative state and presenting symptoms were predictive of a positive scan. When controlling for these factors, the risks for acute DVT were similar across specialties. Outreach and education to referring physicians in regards to risk factors for, and appropriate workup of DVT may assist in efficient utilization of OVL.

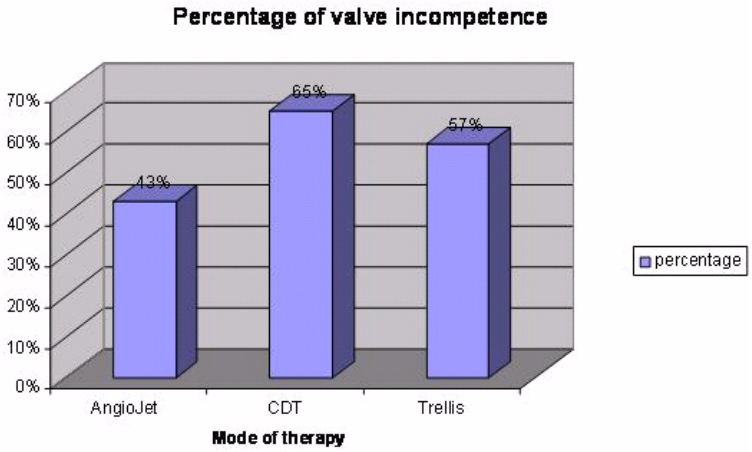
2:30 PM – 2:50 PM **28** **Mode of Thrombolytic Therapy and Residual Obstruction Do Not Affect Valve Function**

D. Vogel[1], E. Walsh[1], J.T. Chen[2], A.J. Comerota[1]
[1]The Toledo Hospital, Toledo, OH, [2]Bowling Green State University, Bowling Green, OH

BACKGROUND: Successful catheter-directed thrombolysis (CDT) for iliofemoral deep vein thrombosis (IFDVT) reduces postthrombotic morbidity and is suggested by the American College of Chest Physicians for treatment of patients of IFDVT. Pharmacomechanical thrombolysis (PMT) is also suggested to shorten the treatment times and reduce the dose of plasminogen activator. However, there is concern that mechanical devices will damage vein valves. The purpose of this study is to examine whether PMT adversely affects venous valve function compared to CDT alone in IFDVT patients treated with catheter-based techniques.

METHODS: Sixty-nine limbs in 54 patients who underwent catheter-based treatment IFDVT form the basis of this study. Lytic success and degree of residual obstruction were analyzed by reviewing post-procedural phlebograms. All patients underwent bilateral postprocedure duplex examinations to evaluate patency and valve function. Patients were divided into three groups based on the mode of lytic therapy: Group1-CDT alone, Group 2-CDT with AngioJet, and Group 3-CDT with Trellis catheter. The validated outcome measures were compared between the three groups.

RESULTS: Sixty-nine limbs underwent CDT with or without pharmacomechanical thrombolysis. Average age was 47 years (range 16–78). Figure 1 demonstrates the correlation between mode of therapy and valve incompetence. Residual venous obstruction did not have an effect on valve function; however, the vast majority of patients had less than fifty percent residual obstruction. Valve function following catheter-based intervention correlated best with valve function of the non-affected limb ($P < 0.05$).



CONCLUSIONS: In patients undergoing catheter-based intervention for IFDVT, PMT does not adversely affect valve function compared to CDT alone. Valve function following catheter-based intervention correlated best with valve function of the unaffected limb.

2:50 PM – 3:05 PM **Coffee Break**
(Foyer)

3:05 PM – 5:05 PM **ASK THE EXPERTS**

Post Thrombotic Syndrome

Moderators: *Peter Henke, MD*
Robert McLafferty, MD
Peter Neglen, MD
Anthony Comerota, MD
Mark Meissner, MD

Educational Objectives: At the completion of the session, participants should be able to:

1. Define the underlying pathophysiology of PTS.
2. Be able to list the primary cellular changes in the vein wall after DVT.
3. Delineate the contemporary medical management of PTS.
4. Describe the appropriate compression therapy to prevent PTS.
5. Describe the role of thrombolysis and endoluminal procedures to decrease the development of PTS.

6:45 PM – 7:15 PM **Cocktail Reception**

7:15 PM – 10:30 PM **THE FORUM FINALE**
Awards, Dinner, Entertainment & More

AMERICAN VENOUS FORUM

Alphabetical Roster

(S) Abbott, William

(A) AbuRahma, Ali
RC Byrd Health Science Center
of WVU
3110 MacCorkle Ave SE
Charleston, WV 25304
P: 304-347-1306
F: 304-556-3823

(A) Adelman, Mark
University Vascular Associates
530 1st Ave, #6F
New York, NY 10016
P: 212-263-7311
F: 212-263-7722

(C) Adler, Grit
Cornell Medical Center
1300 York Avenue
New York, NY 10065

(A) Aguila Marquez, Roberto
Hospital Angeles Lomas
Vialidad de la Barranca No. 22
Col. Valle de las Palmas
Huixquilucan, 52763
Mexico
P: +52.55.19929743

(C) Ahluwalia, Hardeep
Duke University Medical Center
DUMC 3467
Durham, NC 27710
P: 919-681-2915

(H) Allegra, Claudio
S. Giovanni Hospital – Angiology
Department
26 Via Del Colosseo
Rome, 184
Italy
P: +39.06.485527
F: +39.06.77055582

(A) Almeida, Jose Ignacio
Miami Vein Center
1501 South Miami Avenue
Miami, FL 33129
P: 305-854-1555
F: 305-854-1166

(S) Alpert, Joseph
4 Top Gallant Circle
Savannah, GA 31411-2720
P: 912-598-8287

(C) Amin, Rohit
Oschner Clinic Foundation
1514 Jefferson Highway
New Orleans, LA 70121

(A) Anderson, Robert
Vein Centers for Excellence
of Des Moines
1300 37th Street, Suite 3
West Des Moines, IA 50266
P: 515-223-0592
F: 515-223-8316

(A) Angle, Niren
University of California at
San Diego
200 W. Arbor Drive
San Diego, CA 92103
P: 619-543-6980
F: 619-543-2615

(A) = Active (As) = Associate (H) = Honorary (S) = Senior (C) = Candidate (I) = International

(A) Arata, Michael
South Coast Vein Care
1640 Newport Blvd, Suite 200
Costa Mesa, CA 92627
P: 949-221-0128
F: 949-221-8133

(A) Arbid, Elias
Commonwealth Surgical Associates
3640 High Street
Portsmouth, VA 23707
P: 757-397-2383
F: 757-387-5201

(I) Arfvidsson, Berndt
University Hospital of Orebro
Orebro, 70185
Sweden
P: +46.19.6021000
F: +46.19.125439

(A) Ascher, Enrico
Maimonides Medical Center, Vascular Surgery
4802 Tenth Avenue
Brooklyn, NY 11219
P: 718-283-7957
F: 718-635-7050

(I) Balas, Panayiotis
Hiraclitou 4
Athens, GR-1067
Greece
P: +30.16.801209
F: +30.16.712055

(A) Baldwin, John
Texas Tech University Health Sciences Center
3601 4th Street, MS6258
Lubbock, TX 79430-6258
P: 806-743-2900

(A) Balkany, Louis
1614 So. Byrne Road, Suite FF
Toledo, OH 43614-3403
P: 419-382-9425
F: 419-382-9427

(A) Balshi, James
Progressive Physician Assoc, Inc.
3735 Nazareth Rd, #206
Easton, PA 18045
P: 610-252-8281
F: 610-253-5321

(A) Baribeau, Yvon
Cardiothoracic Surgical Associates, PA
100 McGregor Street, Suite B600A
Manchester, NH 03102
P: 603-663-6160
F: 603-663-6822

(S) Barker, Wiley
29129 Paiute Drive
Agoura, CA 91301
P: 818-865-9904
F: 818-865-9901

(S) Baron, Howard
75 Central Park West 13D
New York, NY 10023
P: 212-362-0990

(I) Bass, Arie
Assaf Harofeh Medical Center
Department of Vascular Surgery
Zerifin, 70300
Israel

(A) Bassiouny, Hisham
University of Chicago
5841 So. Maryland St, MC 5028
Chicago, IL 60637
P: 773-702-6128
F: 773-702-0863

(A) Beavers, Frederick
Washington Hospital Center
110 Irving St NW
Washington, DC 20010

(S) Beebe, Hugh
Jobst Vascular Center
2109 Hughes Drive
Toldo, OH 43606
P: 419-291-2088
F: 419-479-6980

(A) Bein, Norman
Vein Specialties
11456 Olive Blvd
Suite 200
Creve Coeur, MO 63141
P: 314-993-8233

(A) Belentsov, Sergey
CKH 40
Volgogradskaya St, 189
Yekaterinburg, 620102
Russia
P: +8.103.432669715
F: +8.103.432669715

(S) Bergan, John
c/o A. Davis
2801 27th Street N.
Arlington, VA 22207

(H) Bergqvist, David
University of Uppsala
Academic Hospital Vascular Surgery
Uppsala, S-751 85
Sweden
P: +46.18.664633
F: +46.18.664632

(S) Bernhard, Victor
3627 Grand Valley Canal Road
Palisade, CO 81526
P: 970-464-4653
F: 970-464-4654

(A) Bernstein, Rick
Advanced Vein Treatment Center
7010 Smoke Ranch Road,
Suite 120
Las Vegas, NV 89128
P: 702-303-3493

(A) Bjarnason, Haraldur
Mayo Clinic – Vascular and Interventional Radiology
200 First Street, SW
Rochester, MN 55902
P: 507-255-8454
F: 507-255-7872

(A) Blebea, John
University Hospital Case Medical Center
Division of Vascular Surgery,
MS LKS7060
11100 Euclid Avenue
Cleveland, OH 44106-7060
P: 216-844-3013
F: 216-844-7716

(A) Blondeau, Benoit
Central Maine Medical Center
12 High St, Suite 401
Lewiston, ME 2410
P: 207-795-2919

(S) Blumenberg, Robert
2259 Algonquin Road
Schenectady, NY 12309
P: 518-393-7700

(A) Bogachev, Vadim
Russian State Medical University
Moscow
Russia

(A) Bohannon, W. Todd
Scott & White Memorial Hospital
& Clinic
2401 South 31st Street
Temple, TX 76508
P: 254-724-0657
F: 254-724-5978

(S) Boland, James
RC Byrd Health Sciences Center
3110 MacCorkle Ave, SE
Charleston, WV 25304
P: 304-347-1333

(H) Bollinger, Alfred
University of Zurich
Trubelstr 31
Strafa, CH-8712
Switzerland

(A) Bonawitz, Cara
Medical Center Radiologists
6330 N. Center Dr, Bldg 13
Suite 220
Norfolk, VA 23502

(A) Bradbury, Andrew
University Department of
Vascular Surgery
Flat 5, Netherwood House
Solihull Hospital
Solihull, B91 2JL
UK
P: +44.121.4245086
F: +44.121.4245086

(A) Brazil, Clark
1508 Tenth Street
Wichita Falls, TX 76301
P: 940-322-6671

(A) Brown, O. William
William Beaumont Hospital
31700 Telegraph Rd, 140
Bingham Farms, MI 48025
P: 248-433-0881
F: 248-433-1628

(A) Brown, Kellie
Medical College of Wisconsin
9200 W. Wisconsin Avenue
Milwaukee, WI 53226
P: 414-805-9160

(H) Browse, Norman
Corbet House
Butes Lane, Alderney
Channel Islands, GY9 3UW
UK

(A) Buchbinder, Dale
Greater Baltimore Medical Center
5601 Loch Raven Blvd, Ste 511
Baltimore, MD 21239-2905
P: 410-849-2393
F: 410-849-3435

(A) Buckman, Jeffrey
Vascular Diagnostics
1600 Dempster, #105
Park Ridge, IL 60068
P: 847-298-7876
F: 847-298-7886

(S) Bulkin, Anatoly
SDIVA
488 E. Valley Pkwy, Suite 404
Escondido, CA 92025
P: 760-739-7666
F: 760-739-7633

(A) Bunke, Nisha
Vein Institute of La Jolla
9850 Genessee Avenue, Suite 410
La Jolla, CA 92037
P: 858-550-0330
F: 858-550-0676

(H) Burnand, Kevin
St. Thomas Hospital, Academic Department of Surgery
1st Flr North Wing, Lambeth Palace Road
London, SE1 7EH
UK
P: +44.207.6339405
F: +44.207.9288742

(As) Bush, Ruth
Scott & White Memorial Hospital & Clinic
2401 South 31st Street
Temple, TX 76508
P: 254-724-9158
F: 254-724-2173

(A) Caggiati, Alberto
University "La Sapienza" – Department of Anatomy
Via Borelli 50
Rome, NY 1-00153
Italy

(A) Calcagno, David
Calcagno and Rossi Vein Treatment Center
2025 Technology Parkway, Suite 304
Mechanicsburg, PA 17050
P: 717-763-0510
F: 717-761-6081

(A) Cambria, Robert
Eastern Maine Medical Center
489 State Street
Bangor, ME 04402
P: 207-973-6670
F: 207-973-5226

(S) Cannon, Jack
25132 Via Pacifica
Dana Point, CA 92629-2049

(A) Cantelmo, Nancy
Massachusetts General Hospital Department of Endovascular Surgery
15 Parkman Street WAC440
Boston, MA 02118
P: 617-726-4464

(S) Caprini, Joseph
North Shore University Health System
9977 Woods Drive
Division of Vascular Surgery
Skokie, IL 60077
P: 847-663-8050
F: 847-663-8054

(A) Carman, Teresa
University Hospitals Case Medical Center
11000 Euclid Ave, LKS 5038
Cleveland, OH 44106
P: 216-844-1261
F: 216-844-8318

(A) Carney, Wilfred
2 Dudley Street
Providence, RI 02905
P: 401-553-8325

(I) Carpentier, Patrick
Grenoble University Hospital
Vascular Medicine Clinic
Grenoble, F38043
France
P: +33.47.6768859
F: +33.47.6768735

(A) Carr, Sandra
Wisconsin Heart & Vascular Institute
2601 W. Beltline Hwy
Madison, WI 53713

(A) Castronuovo, John
York Hospital, Surgery
1001 S. George Street
York, PA 17405
P: 717-851-2474

(A) Cazaubon, Michele
American Hospital Paris
48 Rue St Didier
Paris, 75116
France
P: +33.14.7271063
F: +33.14.7272147

(A) Chaer, Rabih
University of Pittsburgh Medical Center
200 Lothrop Street, Suite A1011
Pittsburgh, PA 15213
P: 412-802-3025

(A) Chaikof, Elliot
Emory University
101 Woodruff Circle
5105WMB
Atlanta, GA 30322
P: 404-727-8413
F: 404-727-3396

(A) Chang, Benjamin
The Vascular Group, PLLC
43 New Scotland Ave, MC 157
Albany, NY 12208
P: 518-262-8720
F: 518-262-6720

(S) Chang, John
Long Island Vascular Center
1050 Northern Blvd
Roslyn, NY 11576
P: 516-484-3430
F: 516-484-3482

(A) Chang, Jeanette
Commonwealth Surgical Associates
91 Montvale Avenue, Suite 208
Stoneham, MA 02180
P: 781-279-1123
F: 781-438-3034

(A) Chang, Richard
National Institutes of Health
9000 Rockville Pike
Clinical Center Bldg 10
Bethesda, MD 20892
P: 301-402-0256

(C) Cheng, Van Le
San Diego Vein Institute
1011 Devonshire Drive, Suite B
Encinitas, CA 92024
P: 760-944-9263
F: 760-944-9295

(A) Cherry, Kenneth
University of VA Hospital
PO Box 800679
Charlottesville, VA 22908
P: 434-243-7052
F: 434-982-1026

(A) Cho, Jae-Sung
Shadyside Medical Building
200 Lothrop St, PUH A1011
Pittsburgh, PA 15232

(I) Christenson, Jan
University of Geneva, Division Cardiovascular Surgery
4 rue Gabriel-Perret-Gentil
Geneva, CH-1292
Switzerland
P: +41.22.3727638
F: +41.22.3727634

(A) Chubak, John
2 Sears Drive, Suite 101
Paramus, NJ 07652
P: 201-261-1772
F: 201-261-1776

(A) Cicci, Christopher
Northeast CardioVascular Clinic
200 Medical Park Drive, Suite 230
Concord, NC 28025
P: 704-783-1349

(I) Cigorraga, Jorge Raul
Av Las Heras 2223 5A
Buenos Aires, 1425
Argentina

(A) Clagett, G. Patrick
University of TX SW Medical Center
5323 Harry Hines Blvd
Dallas, TX 75390-9157
P: 214-648-3516
F: 214-648-2790

(A) Clair, Daniel
The Cleveland Clinic
9500 Euclid Ave, S-40
Cleveland, OH 44195
P: 216-444-3857
F: 216-636-1002

(H) Coleridge Smith, Philip
Thames Valley Nuffield Hospital
Wexham Street
Wexham, SL3 6NH
UK
P: +44.207.6368333
F: +44.207.6799413

(A) Collier, Paul
Greater Pittsburgh Surgical Alliance, PC
701 Broad Street
Sewickley, PA 15143
P: 412-749-9868
F: 412-749-9729

(A) Collins, David
Collins Vein & Laser Care
PO Box 337
126 Trivette Drive
Pikeville, KY 41502
P: 606-478-1407

(A) Collins, Paul
Collins & Hart, MD, FACS, PA
960 7th Avenue N
St. Petersburg, FL 33705
P: 727-821-8101
F: 727-825-1357

(A) Comerota, Anthony
Jobst Vascular Center
2109 Hughes Dr, 400-Conrad Jobst Tower
Toledo, OH 43606
P: 419-291-2088
F: 419-479-6980

(A) Conrad, John Kenneth
LA Vascular and Endovascular
201 South Buena Vista, Suite 300
Burbank, CA 91505
P: 818-558-7700
F: 818-558-7779

(A) Cordts, Paul
Office of the Surgeon General
5201 Brawner Place
Alexandria, VA 22304-8645
P: 703-681-0104
F: 703-681-6568

(I) Cornu-Thenard, Andre
Saint Antoine Hospital
113 avenue Charles de Gaulle
Neuilly/Seine, 92200
France
P: +33.14.7470045
F: +33.14.7451421

(A) Corr, John Price
Vein Clinic @ Albany Surgical
401 Fourth Ave
PO Box 1686
Albany, GA 31701-1915
P: 229-881-0628
F: 229-434-4207

(A) Corrales, Noel Ernesto
Private Vascular Center
6a Avenida 3-22, Zona 10
Edificio C. Medico 2, of. 705
Guatemala City, 1010
Guatemala
P: +50.25.7069842
F: +50.23.3317673

(A) Corson, John
New Mexico VA Healthcare System
1501 San Pedro, SE
Mail Drop 112
Albuquerque, NM 87108

(A) Cranley, Robert
Cranley Surgical Associates
3747 West Fork Road
Cincinnati, OH 45247-7548
P: 513-961-4335
F: 513-961-4227

(A) Criado, Enrique
University of Michigan School of Medicine
5463 Cardiovascular Center
SPC 5867
Ann Arbor, MI 48109
P: 734-763-0250
F: 734-647-9867

(C) Crisostomo, Paul
Indiana University
545 Barnhill Drive
EH 203
Indianapolis, IN 46202
P: 317-274-4966

(A) Cummings, Emily
University of Michigan Livonia Vein Center
19900 Haggerty Road, Suite 105
Livonia, MI 48152
P: 734-432-7662
F: 734-432-7637

(A) Daake, John
The Reno Vein Clinic
9480 Double Diamond Parkway, Suite 100
Reno, NV 89521
P: 775-329-3100
F: 775-329-3199

(A) Dalsing, Michael
Indiana University Medical School
1801 N. Senate Blvd
MPC II, #3500
Indianapolis, IN 46202
P: 317-962-0280
F: 317-962-0289

(C) Danczyk, Rachel
Oregon Health & Science Univeristy
3181 SW Sam Jackson Park Road
Portland, OR 97239

(A) Danylewick, Richard
Clinique RWD
1565, boul. de l'Avenir
Laval, QC
Canada
P: 450-668-8377
F: 450-668-5808

(A) Darling, R. Clement
The Vascular Group, PLLC
43 New Scotland Ave, MC-157
Albany, NY 12208
P: 518-262-8720
F: 518-262-6720

(A) Daugherty, Stephen Franklin
Vein Care Centers of Tennessee
647 Dunlop Lane, Suite 100
Clarksville, TN 37043
P: 931-551-8991
F: 931-551-4053

(I) Davies, Alun Huw
Charing Cross Hospital
Fulham Palace Rd, Surgery,
4th Floor
London, W6 8FR
UK
P: +44.208.8467320
F: +44.208.8467362

(C) Davis, Ross
Wake Forest university Baptist
Medical Center
838 Brent Street
Winston-Salem, NC 27103-3810

(A) Deak, Steven
Deak Vein NJ Clinic
37 Clyde Road, Suite 102
Somerset, NJ 08873-5034
P: 732-873-0200
F: 732-873-0255

(A) Deatherage, Mark Frederick
Grants Pass Surgical Associates
1600 NW 6th Street
Grants Pass, OR 97526
P: 541-474-5533
F: 541-476-2380

(S) Delaria, Giacomo
Scripps Clinic & Res Fnd
10666 N. Torrey Pines Rd
La Jolla, CA 92037
P: 858-554-8122
F: 858-554-6135

(S) Delaurentis, Dominic
209 Sir Thomas Lunsford Drive
Williamsburg, VA 23185
P: 757-220-2592
F: 757-220-2987

(S) Denbo, Howard
45 Castro Street, Suite 138
San Francisco, CA 94114
P: 415-776-9557
F: 415-922-0773

(S) DePalma, Ralph
Department of Veterans Affairs
810 Vermont Ave NW, Rm 111B
Washington, DC 20420
P: 202-273-8505
F: 202-273-9108

(S) DeWeese, James
78 Winding Creek Lane
Rochester, NY 14625
P: 716-248-9412

(I) **di Marzo, Luca**
Department of Surgery
"Pietro Valdoni"
University of Rome "La Sapienza"
Viale del Policlinico, 155
Rome, 161
Italy
P: +39.06.49970794
F: +39.06.49972203

(C) **Dias, Celso**
Stony Brook University Medical Center
100 Nicolls Road
Stony Brook, NY 11794
P: 631-444-2683

(A) **Dickerson, Sandra Dee**
Vein Treatment Center
4102 24th Street, Suite 303
Lubbock, TX 79424
P: 806-725-5120

(A) **Dillavou, Ellen**
University of Pittsburgh Medical Center
Suite A-1011, PUH, 200 Lothrop S.
Pittsburgh, PA 15213
P: 412-802-3333

(A) **Dilling, Emery**
Vein Solutions
6818 austin Center Blvd, Suite 208
Austin, TX 78731
P: 512-452-8346
F: 512-795-8346

(As) **Dion, Yves**
Hopital St-Francois d'Assise
10 de l'Espinay
Quebec, QC G1L 3L5
Canada

(I) **Disselhoff, Ben**
Mesos Medical Center
Department of Vascular Surgery
8605 RP Utrecht
Utrecht, 3527CE
Netherlands
P: +30.32.953737

(C) **Dobzyniak, Christopher**
William Beaumont Hospital
2274 Golfview 101
Troy, MI 48084

(A) **Donaldson, Magruder**
Metro West Medical Center
85 Lincoln Street
Framingham, MA 01702
P: 508-383-1553
F: 508-383-1746

(A) **Donayre, Carlos**
Harbor/UCLA Medical Center
2324 Colt Road
Rancho Palos Verdes, CA 90275
P: 310-222-2704
F: 310-787-1889

(A) **Drougas, James**
Jefferson Surgical Clinic
1234 Franklin Road
Roanoke, VA 24016
P: 540-345-1561
F: 540-342-2112

(A) **Duensing, Robert**
The Vascular Center South
Orange County Surgical Medical Group
350 Health Center Drive, Suite 350
Laguna Hills, CA 92653
P: 949-457-7900
F: 949-583-9148

(S) Duffy, David
4201 Torrance Blvd, #710
Torrance, CA 90503-4511
P: 310-370-5679
F: 310-214-2071

(A) Duncan, Audra
Mayo Clinic
200 First St SW, Gonda 4 South
Rochester, MN 55905
P: 507-284-4751
F: 507-266-7156

(A) Durham, Joseph
10347 So. Longwood Drive
Chicago, IL 60643
P: 708-633-2800
F: 708-799-2261

(A) Edwards, James
Portland VAMC (P-8-VS)
3710 US Veterans Hospital Rd
Portland, OR 97207
P: 503-220-8262
F: 503-220-3415

(S) Eklof, Bo
University of Lund, Sweden
Batteritorget 8
Helsingborg, SE 252-70
Sweden
P: +46.42.260728

(A) Eldrup-Jorgensen, Jens
The Maine Surgical Group
887 Congress St, Ste 400
Portland, ME 04102
P: 207-774-6368
F: 207-774-9388

(A) Elias, Steven
Englewood Hospital & Medical Center
350 Engle Street
Englewood, NJ 07631
P: 201-816-0666
F: 201-894-9951

(S) Elliott, Joseph
3282 Woodview Lake Road
West Bloomfield, MI 48323

(A) Engle, Jennifer
3290 West Big Bear Road,
Suite 410
Troy, MI 48084
P: 248-816-6300
F: 248-816-6335

(A) Ennis, William
University of Illinois @ Chicago
820-840 S. Wood
3rd Floor, Suite 376
Chicago, IL 60612
P: 312-996-8459
F: 312-355-3722

(H) Enrici, Ermenegildo
Remedios de Escalada
2339 (1640) Martinez Bs.As
Buenos Aires, 1123
Argentina
P: +54.11.47425440
F: +54.11.47425440

(S) Ernst, Calvin
1 Greythorne Woods Circle
Wayne, PA 19087
P: 610-688-3445
F: 610-688-6690

(C) Fanicullo, Dustin
University of Rochester
601 Elmwood Avenue
Box 652
Rochester, NY 14642

(I) Farmache, Alejandro
Instituto de Flebolog a
Necochea 350 1 Piso Dpto 12
Ciudad
Mendoza, 5500
Argentina
P: +54.26.14290317
F: +54.26.14210997

(C) Febles, Anthony
Columbia Presbyterian Hospital
113 Stanwich Road
Greenwich, CT 6830

(A) Feied, Craig

(As) Felty, Cindy
Mayo Clinic Medical Center
200 SW First Street
Rochester, MN 55905
P: 507-266-9737
F: 507-266-1617

(A) Fernandez, Bernardo
Cleveland Clinic Florida
2950 Cleveland Clinic Blvd
Weston, FL 33331-3609
P: 954-659-5230
F: 954-659-5292

(A) Ferrier, Frank
Ferrier Management & Consulting
3091 Farmington Drive
Atlanta, GA 30339
P: 404-943-1341
F: 404-943-1830

(S) Ferris, Ernest
University of AR for Medical Sciences
4301 W. Markham, Slot 556
Little Rock, AR 72205
P: 501-686-5744
F: 501-686-6900

(A) Finkelmeier, William
Carmel Medical Center
13450 N. Meridian, Suite 160
Carmel, IN 46032
P: 317-582-7676
F: 317-582-7099

(C) Fiorianti, John
UMDNJ
Department of Vascular Surgery
Middletown, NY 10940
P: 973-518-1346

(C) Fischman, Aaron
Mt. Sinai Medical Center
1 Gustave Levy Place
New York, NY 10029

(A) Fisher, Jay
Easton VeinSolutions
3735 Nazareth Road, 206
Easton, PA 18042

(A) Flanigan, D. Preston
St Joseph Hospital, Orange, CA
1140 W. La Veta Ave, #850
Orange, CA 92868
P: 714-560-4450
F: 714-560-4455

(A) Fleck, Robin
Southwest Skin & Cancer Institute
242 Whipple Street
Prescott, AZ 86301
P: 928-778-0808
F: 928-778-4788

(A) Fleisher, Arlen
Premier Vein Centers of Westchester
280 North Central Avenue, Suite 70
Hartsdale, NY 10530
P: 914-949-8346

(C) Fleming, Shawn
Wake Forest University
Medical Center Blvd
Winston-Salem, NC 27157

(A) Flinn, William
University of Maryland Medical Systems
22 So. Greene St, S10B04
Baltimore, MD 21201
P: 410-328-5840
F: 410-328-0717

(A) Flynn, William
William F. Flynn Jr., MD PC
22 Mill Street, Suite 301
Arlington, MA 02476
P: 781-643-6313
F: 781-643-6316

(A) Fodera, Maria Elena
New York Surgical Assoc P.C.
2235 Clove Road
Staten Island, NY 10305
P: 718-815-8100
F: 718-815-8200

(C) Foegh, Pia
Gentofte Hospital, Department of Surgery
Niels Andersenjvej 65
Hellerup, 2900
Denmark

(S) Fogarty, Thomas
3270 Alpine Road
Portola Valley, CA 94028
P: 650-854-1822
F: 650-854-2778

(A) Forrestal, Mark
Northwest Vein Care
1430 N. Arlington Heights Road, Suite 206
Arlington Heights, IL 60004
P: 847-259-8226

(A) Franz, Randall
Central Ohio Vascular Services
285 E. State Street, Suite 260
Columbus, OH 43215
P: 614-855-0862

(C) Friedman, Joseph
Montefiore Medical Center
1364 Pennington Road
Teaneck, NJ 07666

(S) Fronek, Arnost

(A) Frusha, John
Vascular Surgery Associates
19110 Honors Point Court
Baton Rouge, LA 70810
P: 225-769-4493
F: 225-766-3144

(A) Furey, Patricia
Surgical Care Group, PC
4 Elliot Way, Suite 302
Manchester, NH 03103
P: 603-627-1887

(A) Gagne, Paul
Southern Connecticut Vascular Center
999 Silver Lane
Trumbull, CT 06820
P: 203-375-3927

(A) Gale, Steven
Veinsolutions, Toledo
2109 Hughes Drive, #550
Toledo, OH 43606-3856
P: 419-291-2090
F: 419-479-6135

(A) Garcia, Mark
Christiana Health Care
4755 Ogletown Stanton Road
Newark, DE 19718
P: 301-733-1805

(A) Garcia, Manuel
Veins Plus
3496 E. Lake Lansing Road,
Suite 160
East Lansing, MI 48823
P: 517-371-5515

(C) Garcia-Toca, Manuel
Rhode Island Hospital/Alpert
Medical School of Brown
University
Two Dudley Street, Suite 470
Providence, RI 02905
P: 401-228-0600
F: 401-868-2311

(A) Gardner, Glenn
University of Missouri
Healthcare
One Hospital Drive
Surgery, DC077.00
Columbia, MO 65212

(A) Garner, Scott
Michigan Vascular Center/Vein
Solutions
5151 Gateway Centre, Suite 400
Flint, MI 48507
P: 810-232-3363
F: 810-232-3602

(S) Gaspar, Max
1780 St John Road, #48-C
Seal Beach, CA 90740
P: 562-799-3318
F: 562-429-0807

(A) Gasparis, Antonios
Stony Brook, Surgery
HSC T-18 Rm 040
Stony Brook, NY 0
P: 631-444-1279
F: 631-444-8824

(A) Geraghty, Patrick
Washington University –
Department of Surgery
660 S. Euclid Avenue
Campus Box 8109
St. Louis, MO 63110
P: 314-362-6490

(A) Gibson, Kathleen
Lake Washington Vascular
Surgeons
1135 116th Ave NE, Suite 305
Bellevue, WA 98004
P: 425-453-1772
F: 425-453-0603

(A) Gillespie, David
University of Rochester School of
Medicine
601 Elmwood Avenue, Box 652
Rochester, NY 14642
P: 585-275-6772
F: 585-756-7752

(A) Ginzburg, Enrique
University of Miami, Department
of Surgery
PO Box 016960, (D-40)
Miami, FL 33101
P: 305-585-7529
F: 305-585-3076

(C) Glass, Carolyn
University of Rochester
601 Elmwood Avenue, Box 652
Rochester, NY 14642
P: 585-275-6722

(A) Gloviczki, Peter
Mayo Clinic
200 First St, SW
Rochester, MN 55905
P: 507-284-4652
F: 507-266-7156

(A) Gloviczki, Monika
Mayo Clinic
200 First Street, SW
Rochester, MN 55905
P: 507-284-4695
F: 507-266-7156

(A) Gocke, John
LaGrange Vascular Center
5201 S. Willow Spring Road,
Suite 200
LaGrange, IL 60525
P: 630-829-3835
F: 708-579-4986

(A) Goldman, Mitchell
University of TN Graduate
School of Medicine, Surgery
1924 Alcoa Highway, Box U-11
Knoxville, TN 37920
P: 865-544-9234
F: 865-544-6958

(S) Gomes, Mario

(A) Goodman, Robert
Goodman Vein & Laser Center
66 Morgan Road
PO Box 1163
West Springfield, MA 01090
P: 413-781-1576
F: 413-758-1812

(A) Goodney, Philip
Dartmouth – Hitchcock
1 Medical Center Drive
Lebanon, NH 03456
P: 802-295-7843

(S) Goodson, Spencer
Methodist Hospital of Indiana
1801 North Senate Blvd 755
Indianapolis, IN 46202

(A) Gorski, Yara
Crown Surgical Group
800 Magnolia, Suite 107
Corona, CA 92879
P: 951-736-0696

(A) Gosin, Jeffrey
Jersey Shore Center for Vascular
Health
442 Bethel Road
Somers Point, NJ 08244
P: 609-927-3030
F: 609-927-5293

(A) Gradman, Wayne
Beverly Hills Vein Center
450 N. Roxbury Drive, Suite 275
Beverly Hills, CA 90210
P: 310-278-7710
F: 310-278-2877

(A) Granke, Kenneth
Detroit VA Medical Center
7080 Colony Dr
West Bloomfield, MI 48323
P: 734-740-0461
F: 313-576-1002

(A) Green, Richard
Lenox Hill Hospital
130 East 77th St
13th Floor
New York, NY 10075
P: 212-434-3400
F: 212-434-3410

(S) Greenfield, Lazar
University of Michigan
1327 Jones Drive, #201
Ann Arbor, MI 48105
P: 734-936-6398
F: 734-998-0173

(A) Gruneiro, Laura
HMA
3067 Tamiami Trail, Unit 2
Port Charlotte, FL 33952

(S) Gruss, Jorg
Germany

(A) Gueldner, Terry
Wisconsin Vein Center
940 Maritime Drive
Manitowoc, WI 54220
P: 920-686-7900
F: 920-686-7985

(I) Guex, Jean-Jerome
Angiology Clinic
32, Boulevard Dubouchage
Nice, F-06000
France
P: +33.49.3859926
F: +33.49.3854130

(I) Gupta, Prem
Care Hospital
Road No. 1
Banjara Hills
Hyderabad, 500034
India
P: +91.40.23410522
F: +91.40.20010123

(A) Hallett, John
Roper St. Francis Heart & Vascular Center
316 Calhoun Street
Charleston, SC 29401
P: 843-720-5665
F: 843-727-3370

(A) Hallman, Grady
Texas Heart Institute
1101 Bates, Suite P514
Houston, TX 77225-0345
P: 832-355-4129
F: 832-355-3770

(A) Hammond, Sharon
Colorado Cardiovascular Surgical Associates
6282 So. Netherland Way
Aurora, CO 80016-1326
P: 303-388-6461

(A) Hans, Sachinder
28411 Hoover
Warren, MI 48093
P: 586-573-8030
F: 586-573-2504

(A) Hansen, Henry Andrew
Central Texas Cardiovascular Surgery
1721 Birmingham Drive, Suite 202
College Station, TX 77845
P: 979-764-5700
F: 979-764-4013

(A) Harris, E. John
Stanford University Medical Center
300 Pasteur Dr, H-3637, Vasc
Stanford, CA 94305-5642
P: 650-723-8648
F: 650-498-6044

(S) Harris, Edmund
555 Laurel Ave, Ste #605
San Mateo, CA 94401-4153
P: 650-348-1414
F: 650-348-1414

(A) Harris, Linda
Millard Fillmore Hospital
3 Gates Circle, Department of Surgery
Buffalo, NY 14209
P: 716-887-4807
F: 716-887-4220

(I) Hartung, Olivier
Service de Chirurgie Vasculaire,
CHU Nord
Chemin des Bourrelys
Marseille, 13015
France
P: +33.49.1968704
F: +33.49.1968370

(A) Hasaniya, Nahidh
Loma Linda University Medical
Center
11175 campus Street, Suite 21121
Loma Linda, CA 92354
P: 909-558-4354
F: 909-558-0348

(A) Haser, Paul
UMDNJ – RWJMS, Vascular
Surgery
1 R WJ Place, MEB-541
New Brunswick, NJ 08903-0019
P: 732-235-7816
F: 732-235-8538

(A) Heller, Jennifer
Johns Hopkins Vein Center
4940 Eastern Avenue
Baltimore, MD 21224
P: 410-550-4335
F: 410-550-1274

(A) Henke, Peter
University of MI Health System
1500 E. Medical Center Dr,
2210D Taubman Center
Ann Arbor, MI 48109-0329
P: 734-763-0250
F: 734-647-9867

(A) Hertzman, Phillip
Vein Care of New Mexico
1651 Galisteo Street 8
Santa Fe, NM 87505
P: 505-662-2900
F: 505-662-4333

(A) Hill, Douglas
The Vein Treatment Centre
2004 14th Street NW, #207
Calgary, AB T2M3N3
Canada
P: 403-220-9353
F: 403-210-0593

(A) Hingorani, Anil
Maimonides Medical Center
4802 10th Ave, Admin Bldg
Brooklyn, NY 11219
P: 718-283-7957
F: 718-635-7050

(A) Hirano, Tetsuya
Hirano Clinic
1-3-3 Uemachi
Hakurazaki
Izumisano, 598-0037
Japan

(H) Hirsh, Jack
Hamilton Civic Hospital
Research Center
711 Concession St
Hamilton, ON L8V 1C3
Canada
P: 905-527-2299
F: 905-575-2646

(H) Hobbs, John
4 Upper Wimpole Street
London, W1G 6LF
UK
P: +20.732.32830
F: +20.722.42930

(S) Hobson, Robert

(A) Hoffman, Cheryl
SMH-UCLA
1250 16th Street
2nd Floor Tower – Radiology
Santa Monica, CA 90404
P: 310-319-4033

(A) Hollier, Larry
LSU School of Medicine
533 Bolivar Street
New Orleans, LA 70012
P: 504-568-4009
F: 504-568-4008

(C) Hong, Steve
Carle Foundation Hospital
602 W. University Avenue
Department of Radiology
Urbana, IL 61801

(C) Honig, Shaun
Mount Sinai Medical Center
One Gustave L. Levy Place
Box 1
New York, NY 10029

(I) Hoshino, Shunichi
Fukushima Daiichi Hospital
16-2 Nariide, Kitasawamata
Fukushima-City, 960-8251
Japan
P: +18.12.4557511
F: +11.81.245575064

(A) Housman, Leland
Scripps Clinic Medical Group
4878 Avion Way
San Diego, CA 92115
P: 858-764-9052
F: 858-764-9067

(A) Hovorka, John
Valley Laser Surgical Solutions
909 Jackson Road
McAllen, TX 78501
P: 956-991-9161

(A) Hsiang, York
Vancouver General Hospital
510-943 W. Broadway
Vancouver, BC
Canada
P: 604-876-5882

(C) Huang, Joe
UMDNJ – New Jersey Medical School
150 Bergen Street, Suite F-102
Newark, NJ 07103

(A) Hunter, Glenn
2975 W. Shining Star Drive
Tucson, AZ 85745

(A) Hutto, John
Allouez Health Center
1821 S. Webster Avenue
Green Bay, WI 54301
P: 920-49-64700

(A) Iafrati, Mark
New England Medical Center
750 Washington St, NEMC 1035
Boston, MA 02111
P: 617-636-5019
F: 617-636-8003

(A) Ihnat, Daniel Michael
University of Arizona
1501 N. Campbell Avenue
Room 4404
Tucson, AZ 85724
P: 750-626-6670
F: 750-626-6008

(A) Illig, Karl
University of Rochester Medical Center
601 Elmwood Ave, Box 652
Rochester, NY 14642
P: 716-275-6772
F: 716-273-1077

(C) Impellizzeri, Paul
The Reading Hospital and Medical Center
PO Box 16052
Division of Vascular Surgery
Reading, PA 19612-6052
P: 610-988-8000

(A) Isaacs, Mark
Walnut Creek
1981 N. Broadway, Suite 427
Walnut Creek, CA 94596
P: 925-945-8656
F: 925-945-8818

(I) Ishimaru, Shin
Tokyo Med College, Surgery
6-7-1 Nishi-shinjuku, Shinjuku-ku
Tokyo, 160-0023
Japan
P: +81.33.3426111
F: +81.33.3422827

(A) Isobe, James Hajime
Alabama Vascular and Vein Center
700 Montgomery Highway,
Suite 210
Birmingham, AL 35216
P: 205-823-0151
F: 205-823-5218

(A) Iwai, Takehisa
Tsukuba Vascular Center,
Buerger Disease Research Institute
980-1 Tatsuzawa
Moriya City, Ibaragi-pret, 302-0118
Japan
P: +81.29.7479955
F: +81.29.7454541

(A) Jacobowitz, Glenn
NYU Langone Medical Center
530 First Avenue, Suite 6F
New York, NY 10016
P: 212-263-7311
F: 212-263-7722

(A) Jain, Krishna
Advanced Vascular Surgery
1815 Henson Avenue
Kalamazoo, MI 49048
P: 269-492-6500
F: 269-492-6461

(A) Jamil, Zafar
St. Michael's Medical Center
306 Dr M L King Jr. Blvd, MS-45
Newark, NJ 07102
P: 973-877-5059
F: 973-877-2954

(C) Janzen, Mark
University of Missouri-Columbia
One Hospital Drive
DC077.00
Columbia, MO 65212

(S) Jarrett, Fredric
UPMC-Shadyside
5200 Centre Ave, #716
Pittsburgh, PA 15232-1300
P: 412-681-8720
F: 412-681-8713

(C) Jayaraj, Arjun
University of Washington
1959 NE Pacific Avenue
Seattle, WA 98104

(C) Joglar, Fernando Luis
Boston University Medical Center
88 East Newton
D-506 Collamore Bldg
Boston, MA 02118
P: 787-403-4349
F: 617-638-8409

(A) Joh, Jin-Hyan
Kyung Hee University School of
Medicine
149 Sangil-dong, Gangdong-gu,
East-West Neo Medical Center
Seoul, 134-727
South Korea
P: +82.2.4406261

(A) Johnston, Robert
Vein Clinics of Texas
PO Box 3353
Victoria, TX 77903
P: 361-570-8346
F: 512-582-5780

(A) Jones, Andrew
Inovia, LLC
2200 NE Neff Road, Suite 204
Bend, OR 97701
P: 541-382-8346

(C) Jung, Daniel
Maimonides Medical Center
4802 10th Avenue
Department Vascular Surgery
Brooklyn, NY 11219

(S) Jurnecka, Jan
Central Coast Vein Center
1505 Soquel Drive, Suite 3
Santa Cruz, CA 95065
P: 831-462-9811
F: 831-476-3808

(A) Kabnick, Lowell
New York University Medical School
NYU Vein Center – 530 1st Ave
Suite 6D
New York, NY 10016
P: 212-263-8346

(A) Kalra, Manju
Mayo Clinic
200 First Street, SW
Rochester, MN 55905
P: 507-284-4494
F: 507-266-7156

(A) Kang, Steven
Reiss & Kang, MD, P.A.
6200 Sunset Drive, Suite 505
South Miami, FL 33143
P: 305-668-1660
F: 305-668-1650

(A) Kanter, Alan
Vein Center of Orange County
250 East Yale Loop, Suite D
Irvine, CA 92604-4697
P: 949-551-8855
F: 949-527-8860

(A) Kaplan, Jeff
Jeff Kaplan MD
2505 Samaritan Drive, #508
San Jose, CA 95124
P: 408-358-3540
F: 408-356-7481

(A) Kasirajan, Karthikeshwar
Emory University Hospital
1364 Clifton Road NE,
STE H-122A
Atlanta, GA 30322
P: 404-727-8407
F: 404-727-3316

(A) Kaufman, Steven
Total Vein Care
1136 E. Stuart Street, Suite 4102
Fort Collins, CO 80525
P: 970-498-8346
F: 970-419-8346

(A) Kazmers, Andris
Petoskey Surgeons
560 W. Mitchell, Suite 140
Petoskey, MI 49770
P: 231-487-1900
F: 231-487-2707

(A) Kechejian, Gregory
Milton Hospital
Lintz Medical Office Building
100 Highland Street, Suite 222
Milton, MA 02186
P: 617-696-0660
F: 617-696-6655

(S) Kempczinski, Richard
3435 Golden Ave, Apt 201
Cincinnati, OH 45226
P: 513-321-4724
F: 513-321-7350

(A) Kent, K. Craig
University of Wisconsin
600 Highland Avenue
H4-710D Clinical Sciences Center
Madison, WI 53792
P: 608-265-8854

(A) Kerr, Thomas
The Vein and Vascular Institute of Tampa Bay
2809 West Waters Avenue
Tampa, FL 33614
P: 813-348-9088
F: 813-348-9310

(S) Kerstein, Morris
1214 Valley Road
Villanove, PA 19085
P: 610-527-4316
F: 610-520-9293

(A) Khoury, Thomas
Southern Ohio Medical Center
1711 27th Street
Braunlin Bldg, Suite 306
Portsmouth, OH 45662
P: 740-353-8661

(A) Kikuchi, Rodrigo
Clinica Miyake
Praca Amadeu AMaral, 27 6.Andar
Sao Paulo, 01327-010
Brazil

(A) Killewich, Lois
University of Texas Medical Branch
301 University Blvd, Department of Surgery
Room 6. 136 McCullough
Galveston, TX 77555-0735
P: 409-772-6366
F: 409-747-0966

(I) Kim, Young-Wook
Samsung Medical Center
50, Ilwon-Dong, Gangnam-Gu
Seoul, 135-710
Korea
P: +82.2.34103461
F: +82.2.34100040

(A) King, Ted
Vein Clinics of America
120 Oakbrook Center, Suite 300
Oak Brook, IL 60523
P: 630-57-10022

(A) Kirkland, John Smith
The Harbin Clinic
115 John Maddox Drive, Suite B
Rome, GA 30165
P: 706-232-4122
F: 706-232-4131

(A) Kiser, Robert Cameron
Center for Vein Restoration
718 West Berry Avenue
Mishawaka, IN 46545
P: 269-352-2666

(S) Kistner, Robert
Beretania Medical Plaza
848 So. Beretania Street, Suite 307
Honolulu, HI 96813
P: 808-532-8346
F: 808-532-2240

(C) Klem, Taco
Sint Franciscus Gasthuis
Kleiweg 500
Rotterdam, 3045PM
Netherlands
P: +31.10.4616161

(S) Kloecker, Richard
P: 314-692-9100
F: 314-569-3119

(A) Knight, Jr, Charles
Highland Clinic
1455 E. Bert Kouns
Shreveport, LA 71135
P: 318-798-4691

(I) Komlos, Pedro
Pedro Pablo Komlos Vascular Surgery Clinic
rua Dr Florencio Ygartua St, 131rm605
Porto Alegre- RS, 90430-010
Brazil
P: +55.51.32225065
F: +55.51.33302315

(I) Kompf, Boguslaw
Klinika Zdrowych Nog
ul. Reduty Ordona 54/1
71-202 Szczecin
Poland
P: 48914874598

(S) Konigsberg, Stephen
Highland Park Surgical Associates
31 River Road
Highland Park, NJ 08904
P: 732-846-9500
F: 732-846-3931

(A) Kurtoglu, Mehmet
Istanbul Medical Facility
Emergency Surgery
Capa, Topkapi
Istanbul, 34390
Turkey
P: +90.21.25311246
F: +90.21.25331882

(C) Kwan, Sharon
University of California, San Francisco
505 Parnassus Avenue
Box 0628
San Francisco, CA 94143-0628

(A) Labropoulos, Nicos
Stony Brook University Medical Center
Department of Surgery
HSC Level 19, Rm 090
Stony Brook, NY 11794-8191
P: 631-444-2683
F: 631-444-8824

(A) Lal, Brajesh
University of Maryland Medical Center
22 S. Greene Street, N4W66
Vascular Surgery
Baltimore, MD 21201
P: 410-328-5840

(A) Lalka, Stephen

(A) Lall, Purandath
Mayo Clinic
Department of Vascular Surgery
200 First St, SW
Rochester, MN 55905
P: 507-284-2511
F: 507-284-0161

(S) Lamesch, Alfred
Clinic Dr Bohler
30 Rue de Luxembourg
Goetzingen, L-8360
Luxembourg

(A) Landry, Gregory James
Oregon Health & Science University
3181 SW Sam Jackson Park Road
Portland, OR 97239
P: 503-494-7593
F: 503-494-4324

(A) Lantis, John Carlos
The Vein Center at Roosevelt Hospital
425 W. 59th Street, Suite 7B
New York, NY 10019
P: 212-523-4797

(A) Laredo, James
Georgetown University Hospital
Department of Surgery
3800 Rservoir Rd, NW, 4 PHC
Washington, DC 20007
P: 202-444-2255
F: 202-444-6498

(A) Lauber, Andre
Venenpraxis
Unter Oter Egg No
Lucerne, MD 6004
Switzerland
P: +41.41.3705570
F: +41.41.3705370

(S) Lazarus, Harrison
21st Century Vein Clinic
3584 W. 9000 South, Suite 400
West Jordan, UT 84088
P: 801-263-0778

(S) Lee, Byung-Boong
Georgetown University
1830 Town Center Drive Suite 401
Reston, VA 20190
P: 703-880-9500
F: 703-880-9598

(A) Lemmon, Gary
IU Vascular Surgery
1801 North Senate Blvd, MPC-2
Suite D 3500
Indianapolis, IN 46202-1228

(A) Liasis, Nikolaos
Reference Vascular Center
Mesogion 109-111
Athens, 11526
Greece
P: +30.21.06911668
F: +30.21.06911676

(A) Liem, Timothy
Oregon Health & Science University
3181 SW Sam Jackson Park Blvd
Division of Vascular Surgery OP-11
Portland, OR 97239
P: 503-494-7593

(I) Liew, Ngoh
University of Putra Malaysia
Department of Surgery
Kuala Lumpor, 50586
Malaysia
P: +60.3.20501013
F: +60.3.20501076

(A) Lin, Judith
Henry Ford Hospital
2799 W. Grand Blvd
Detroit, MI 48202
P: 313-916-3156

(A) Lin, Peter
Baylor College of Medicine
HVAMC-112
2002 Holcombe Blvd
Houston, TX 77030
P: 713-794-7892
F: 713-794-7352

(A) Lochridge, Stanley
Varicosity Vein Center
48 Medical Park E. Drive, Suite 151
Birmingham, AL 35235
P: 205-838-3835

(S) Lofgren, Eric

(S) Lofgren, Karl

(A) Lohr, Joann
Lohr Surgical Specialists
6350 Glenway Ave, #208
Cincinnati, OH 45211-6378
P: 513-451-7400
F: 513-451-7888

(A) Long, John
California Pacific Medical Center
3838 California Street
San Francisco, CA 94118
P: 415-221-7056
F: 415-221-3583

(A) Lumsden, Alan
The Methodist Hospital
Cardiovascular Surgery
Department
6560 Fannin Street, Suite 1006
Houston, TX 77030
P: 713-798-8412

(C) Lundgren, Rachel
University of Washington
325 9th Avenue
Department of Vascular Surgery,
Box 3
Seattle, WA 98104

(A) Lurie, Fedor
Kistner Vein Clinic
848 South Beretania Street,
Suite 307
Honolulu, HI 96813
P: 808-532-8346
F: 808-532-2240

(A) Lynch, Thomas
University of NE Medical Center
9721 Spring Street
Omaha, NE 68124
P: 402-391-5811
F: 402-559-6749

(A) Lynn, Richard
Vascular Lab of the Palm Beaches
1411 No Flagler Dr, 9700
West Palm Beach, FL 33401-3413
P: 561-655-1877
F: 561-655-6404

(A) Maharaj, Dale
12 Park View – Trincity
Trinidad
West Indies

(C) Malgor, Rafael
Stony Brook Medical Center
HSC Level 19, Room 090
Stony Brook, NY 11794-8191

(A) Malhotra, Praveen
Vein Care Center
525 North Eastown Road
Lima, OH 45807
P: 419-236-1803

(A) Mansour, M. Ashraf
Michigan State University
4069 Lake Drive SE, Suite 312
Grand Rapids, MI 49546-8816
P: 616-459-8700
F: 616-459-0247

(A) Markel, Arie
Haemek Medical Center
Road Afula-Kfar Saba
Afula, 18101
Israel

(A) Marston, William
University of North Carolina
3023 Burnett-Womack Building
Department of Surgery
Chapel Hill, NC 27599-7212
P: 919-966-3391
F: 919-966-2898

(S) Martin, Alfred
PO Box 4697
Santa Fe, NM 87502
P: 505-820-1544
F: 505-982-0382

(A) Martinez, Jeffrey
Peripheral Vascular Associates
111 Dallas Street, Suite 200A
San Antonio, TX 78205
P: 210-225-7508
F: 210-225-1486

(A) Martinez Trabal, Jorge
604 Mansion Real
Calle Felipe II
Coto Laurel, PR 780
Puerto Rico
P: 787.812.0694

(A) Maru, Sandip
Vascular Associates of Long Island, PC
2500 Nesconset Hwy, Bldg 21C
Stony Brook, NY 111790
P: 631-246-8289
F: 631-246-8294

(A) Masuda, Elna
Straub Clinic & Hospital
888 So. King St, Palma 5
Honolulu, HI 96813
P: 808-522-4469
F: 808-522-4523

(A) Matsumura, Jon
NMFF
201 E. Huron St, Suite 10-105
Chicago, IL 60611
P: 312-695-4857
F: 312-695-4955

(A) Mattos, Mark
Harper Hospital/Detroit Medical Center
Vascular Surgery
3990 John R
Detroit, MI 48201
P: 313-745-0462
F: 313-745-1873

(A) McCarthy, Walter
Rush Presbyterian – St. Luke's Hospital
1725 W. Harrison, Rm 1156
Chicago, IL 60612
P: 312-563-2762
F: 312-829-8680

(S) Mckittrick, James
649 Camino Campana
Santa Barbara, CA 93111-1424
P: 805-967-3282
F: 209-315-5808

(A) McLafferty, Robert
SIU Surgical Clinics
421 North 9th Street
PO Box 19680
Springfield, IL 62794
P: 217-545-7983
F: 217-545-9711

(A) Meissner, Mark
University of Washington
Medical Center
Department of Surgery,
Box 356410
1959 NE Pacific St, Room BB487
Seattle, WA 98915-6410
P: 206-221-7047
F: 206-616-7495

(A) Menzoian, James
University of CT Health Center
263 Farmington Avenue
Farmington, CT 06030
P: 860-679-7650
F: 860-679-4948

(A) Merchant, Robert
The Reno Vein Clinic
9480 Double Diamond Pkwy,
Suite 100
Reno, NV 89521
P: 775-329-3100
F: 775-329-3199

(As) Meretei, Attila
Clinasys LLC
6797 Willow Wood Drive,
#6036
Boca Raton, FL 33434
P: 561-488-0422
F: 561-558-1358

(A) Merli, Geno
Thomas Jefferson University
Hospital
111 S. 11th Street, Suite 2210
Philadelphia, PA 19107
P: 215-955-9558
F: 215-955-2197

(A) Mihranian, Mardiros Haig
1510 S. Central Ave, Suite 630
Glendale, CA 91204
P: 818-240-7001
F: 818-240-7006

(A) Milic, Dragan
Vascular Clinic, Clinical Centre Nis
Bulevar Nemanjica 72A/25
Nis 18 000
Serbia
P: +38.11.8205111
F: +38.11.8205111

(I) Milleret, Rene
Vein Center
2 Rue de Verdun
Montpellier, 34000
France
P: +33.46.7659898

(A) Min, Robert
NewYork-Presbyterian Hospital/
Weill Cornell
525 East 68th Street
Room Starr 8a-37
New York, NY 10065
P: 212-746-2520

(A) Mintz, Bruce
St. Clare's Riverside Medical
Center
16 Pocono Road, 313
Denville, NJ 07834
P: 973-625-0112
F: 973-625-0721

(A) Miskin, Barry
1926 Lenmore Drive
Palm Beach Gardens, FL 33410
P: 561-745-7789
F: 561-745-4470

(A) Monahan, Daniel
Vein Surgery & Treatment Center of No. California
1211 Pleasant Grove Blvd, Suite 120
Roseville, CA 95678-6971
P: 916-791-8346
F: 916-791-8833

(A) Monedero, Javier Leal
Hospital Ruber Internacional
C/ LA Maso N. 38
Madrid, AL 28034
Spain
P: +34.91.3875157
F: +34.91.3875158

(A) Moneta, Gregory
OR Health Sciences University, Vascular
3181 SW Sam Jackson Park Road
Mailcode OP11
Portland, OR 97201-3098
P: 503-494-7593
F: 503-494-4324

(A) Moore, Colleen
SIU Surgical Clinics
421 North 9th Street
PO Box 19680
Springfield, IL 62794
P: 217-545-7983
F: 217-545-9711

(A) Morasch, Mark
Northwestern University Medical School
201 E. Huron St, #10-105,
Vascular Surgery
Chicago, IL 60611
P: 312-695-2716
F: 312-695-4955

(C) Moreira, Barbara D'Agnoluzzo
Harper Hospital
3990 John R – 4th Floor
Vascular Surgery Division
Detroit, MI 48201
P: 313-745-8637

(A) Moritz, Mark
Vein Institute of New Jersey
95 Madison Avenue
Morristown, NJ 07960
P: 973-539-6900
F: 973-588-4115

(A) Morrison, Nick
Morrison Vein Institute
8575 E. Princess Drive, Suite 223
Scottsdale, AZ 85255
P: 480-860-6455
F: 480-860-6679

(A) Muck, Patrick
Good Samartian Hospital
375 Dixmyth Ave, 3rd Floor,
Surgery
Cincinnati, OH 45220
P: 513-232-8181
F: 513-624-2964

(S) Mulcare, Robert
9 Cedarwood Drive
Greenwich, CT 06830
P: 203-661-3295
F: 203-661-2545

(A) Murray, James
Kaiser Permanente
Vascular Surgery
1011 Baldwin Park Blvd
Baldwin Park, CA 91706
P: 626-851-6878
F: 626-851-6802

(A) Mutyala, Manikyam
Wyckoff Heights Medical Center
374 Stockholm Street
Brooklyn, NY 11237
P: 718-486-4159

(A) Myers, Jr., Daniel
University of Michigan Medical School
1150 W. Medical Center Drive, Dock 6
MSRB II A570D
Ann Arbor, MI 48109-0654
P: 734-763-0940
F: 734-763-7307

(A) Najibi, Sasan
LA Vascular & Endovascular
PO Box 16335
Encino, CA 91416
P: 818-558-7700
F: 818-558-7779

(C) Nassiri, Naiem
Lenox Hill Heart & Vascular Institute of New York
100 East 77th Street
New York, NY 10021

(H) Natali, Jean
17 rue Lamennais
Paris, F-75008
France
P: +14.28.95439
F: +14.35.90100

(A) Nath, Ronald
Commonwealth Surgical Associates
91 Montvale Avenue, Suite 208
Stoneham, MA 2180
P: 781-279-1123
F: 781-438-3034

(C) Naughton, Peter
Northwestern Memorial Hospital
512 N. McClurg Court, Apt 4507
Chicago, IL 60611

(As) Navarro, Felipe

(A) Nazzal, Munier MS
Medical College of Ohio, Surgery
3064 Arlington Avenue
Toledo, OH 43614
P: 419-383-6810

(A) Neglen, Peter
River Oaks Hospital
1020 River Oaks Drive, Suite 480
Flowood, MS 39232
P: 601-664-6680
F: 601-664-6694

(C) Nguyen, Tony
Boston University Medical Center
45 East Newton, 706
Boston, MA 02118

(A) Nicholls, Stephen
Southwest Washington Thoracic and Vascular Surgery
200 NE Mother Joseph Place, Suite 300
Vancouver, WA 98664
P: 360-514-1854

(A) Nicholson, Phifer
Surgical Consultants, P.A.
6405 France Avenue South, Suite W440
Edina, MN 55435-2166
P: 952-927-7004
F: 952-927-5146

(H) Nicolaides, Andrew
Vascular Screening and Diagnostic Centre
2 Kyriacou matsi Street
Ayios Dhometios
Nicosia, 1683
Cyprus
P: +35.72.2780543
F: +35.72.2780553

(A) Noppeney, Thomas
Klinik Hallerwiese, Department of Surgery/Praxis fuer Gefaessmedizin
Obere Turnstrasse 8-10
Nuremberg, DE-90429
Germany
P: +49.91.12706170
F: +49.91.12706181

(A) Nypaver, Timothy
Henry Ford Hospital
2799 W. Grand Blvd, Vascular Surgery
Detroit, MI 48202
P: 313-916-3153
F: 313-916-3023

(A) OByrne, Margaret
The Vein Clinic
4765 Carmel Mountain Road, 103
San Diego, CA 92130
P: 619-218-8980
F: 858-793-1632

(A) Oderich, Gustavo
Mayo Clinic
200 First Street SW
Gonda 4 South Vascular Surgery
Rochester, MN 55901
P: 507-284-1575
F: 507-266-7156

(A) O'Donnell, Thomas
New England Medical Center
750 Washington St, Box 259
Boston, MA 02111
P: 617-636-5660
F: 617-636-5936

(I) Ogawa, Tomohiro
CV Disease Center/Fukushima Daiichi Hospital
16-2 Kitasawamata Nariide
Fukushima City, 960-8251
Japan
P: +81.24.5575111
F: +81.24.5575064

(A) Oliver, Mark
Morristown Memorial Hospital
182 South Street
Morristown, NJ 7960
P: 973-538-0165
F: 973-538-9344

(C) O'Neill, Alissa
Englewood Hospital Medical Center

(A) Opie, John
Optima Vein Care
1343 N. Alma School Road, Suite 150
Chandler, AZ 85224
P: 480-963-1853
F: 480-726-0695

(A) Orrego, Alvaro Esteban
Centro Clínico de Especialidades Vasculares
2 Oriente 387
Viña del Mar, Other 2520000
Chile

(A) Ortega, Raul
2220 Lynn Road, Suite 306
Thond Oaks, CA 91360

(I) Osse, Francisco
Venaclinic
Rua Lomas Valentinas, 278
Sao Paulo, 05084-010
Brazil
P: +55.11.41916127
F: +55.11.38359365

(A) Owens, Lewis
CRL Surgical Associates
1490 Pantops Mountain Place,
Suite 100
Charlottesville, VA 22911
P: 434-244-4580
F: 434-244-4579

(A) Padberg, Frank
Doctors Office Center
90 Bergen St, Ste 2300
Newark, NJ 07103
P: 973-676-1000
F: 973-395-7193

(A) Paladugu, Ramesh
Texas Vascular and Vein Center
1325 Pennsylvania Ave, Suite 440
Fort Worth, TX 76104
P: 817-332-8346
F: 817-332-1723

(A) Pamoukian, Vicken
Park Lenox Surgical, PC
130 E. 77th Street
13th Floor
New York, NY 10075
P: 212-434-3420

(C) Pannu, Rajmony
Mayo Clinic
200 1st Street SW
Rochester, MN 55901

(I) Papendieck, CM
Universidad del Salvador
Catamarca 3179 – 1636 Olivos
Buenos Aires, 1636
Argentina
P: +54.11.47907957
F: +54.11.47990740

(A) Pappas, Peter
The Brooklyn Hospital Center
121 DeKalb Avenue
Chair, Department Surgery
Brooklyn, NY 11201
P: 718-250-6751

(S) Paramo, Marcelo
Surasgo 247 – 3er Piso, Col, Rom
Mexico, D.F., 6700
Mexico
P: +52.55.153201
F: +52.55.165362

(H) Partsch, Hugo
Medical University
Baumeisterg 85
Vienna, A1160
Austria
P: +43.14.855853
F: +43.14.800304

(A) Passman, Marc
University of Alabama at
Birmingham
Section of Vascular Surgery
BDB 503 1808 7th Avenue South
Birmingham, AL 35294-0012
P: 205-934-2003
F: 205-934-0024

(A) Patterson, Robert
Providence Surgical Care Group
486 Silver Spring Street
Providence, RI 02904
P: 401-454-0690
F: 401-454-4281

(A) Pavcnik, Dn
Dotter Interventional Institute,
OHSU L342
630 SW Gaines Street
Portland, OR 97239-3098
P: 503-494-3669
F: 503-494-4258

(A) Pavone, Lisa
University of Michigan Livoinia
Vein Center
19900 Haggerty Road, Suite 105
Livonia, MI 48152
P: 734-432-7662

(A) Pearce, William
Northwestern Medical Faculty
Foundation
201 East Huron #10-105,
Vascular Surgery
Chicago, IL 60611
P: 312-926-7775
F: 312-695-4955

(A) Peden, Eric
Methodist Hospital
6550 Fannin Street, Suite 1401
Houston, TX 77030
P: 713-441-9319

(A) Peloso, Ole
Vein Center of New Mexico
801 Encino PL NE, Suite C-12
Albuquerque, NM 87106
P: 505-247-4849

(A) Pennell, Richard
St. Louis Vascular Center
625 South New Ballas Road,
Suite 7063
St. Louis, MO 63141
P: 314-251-4200
F: 314-251-5816

(C) Pennycooke, Owano
UMDNJ – New Jersey Medical
School
150 Bergen Street, Suite F-102
Newark, NJ 7103

(C) Perez, Alejandro
Cleveland Clinic
30 Severance Circle, Suite 601
Cleveland Heights, OH 44118
P: 216-659-1302
F: 216-636-6955

(H) Perrin, Michel
Clinique Du Grand Large
26 Chemin de Decines
Chassieu, 69680
France
P: +33.47.2057266
F: +33.47.2057274

(S) Persson, Alfred
5 Dean Road
Wellesley, MA 02481
P: 781-235-6910
F: 781-431-1632

(A) Pester, Thomas
Center For Venous Disease
1700 Murchison, Suite 211
El Paso, TX 79902
P: 915-533-5100

(I) Pietravallo, Antonio FR
Inst Privado de Flebologia
Av Callao 1243, 1B
Buenos Aires, 1023
Argentina
P: +54.18.135172
F: +54.18.144496

(As) Pietropaoli, John Anthony
Chesapeake Vein Clinic
3904 Chaneyville Road
Owings, MD 20736
P: 410-535-2811

(A) Pittaluga, Paul
Riveriera Vein Institute
6 Rue Gounod 06000
Nice, 6800
France
P: +49.38.56171
F: +49.35.40142

(A) Plaza-Ponte, Mario
Pittsburgh Vein Center
2550 Mosside Blvd 105
Monroeville, PA 15146
P: 412-373-9580
F: 412-373-9582

(A) Polak, Joseph
New England Medical Center
750 Washington St, Radiology
Boston, MA 02111
P: 617-636-0040
F: 617-636-0041

(A) Pounds, Lori
Peripheral Vascular Associates
7950 Floyd Curl Drive
San Antonio, TX 78229
P: 210-692-9700

(A) Procter, Charles
Surgical Specialists of Georgia
1250 Jesse Jewel Pkwy, #300
Gainesville, GA 30501
P: 770-534-0110
F: 770-531-2423

(As) Proctor, Mary
Orthofix
1720 Bray Central Drive
McKinney, TX 75069
P: 214-578-2234

(A) Proebstle, Thomas
Private Practice
Zinkenbergweg 2
Hirschberg, D-69493
Germany
P: +49.17.12065419
F: +49.62.01879660

(A) Puggioni, Alessandra
Scottsdale Vascular Services
8390 E. Via De Ventura,
Suite F114
Scottsdale, AZ 85258
P: 602-910-4331
F: 480-998-7503

(H) Rabe, Eberhard
Klinik und Poliklinik fur
Dermatologie
Sigmund Freud, Suite 25
Bonn, D-53105
Germany
P: +22.82.875370
F: +22.82.874333

(A) Raffetto, Joseph
VA Boston Healthcare System
1400 VFW Prkwy, Surgery 112,
Vasc
West Roxbury, MA 02132
P: 857-203-5572
F: 857-203-5567

(A) Rai, Dinker
555 Prospect Place
Brooklyn, NY 11238
P: 718-499-0202
F: 516-248-1547

(S) Raju, Seshadri
River Oaks Hospital
1020 River Oaks Drive, Suite 420
Flowood, MS 39232
P: 601-939-4230
F: 601-939-5210

(A) Ramnauth, Subhash
Jersey Shore Surgery and
Vein Center
40 Bey Lea Road
Bldg. B – 202
Toms River, NJ 08753
P: 732-240-4466
F: 732-240-4451

(I) Rasmussen, Lars
Kirurgisk Center Naestved
Eskadronvej 4 A
Naestved, 4700
Denmark
P: +45.55.700038
F: +45.55.723038

(S) Ratliff, Jack

(A) Razvi, Syed
Caritas St. Elizabeth's Medical
Center
Medical Office Building
11 Nevins Street, #308
Brighton, MA 02135
P: 617-254-4200
F: 617-254-4242

(A) Rectenwald, John Edward
University of Michigan
CVC-5463
1500 E. Medical Center Drive
Ann Arbor, MI 48109-5867
P: 734-6476651
F: 734-6479867

(C) Reddy, Madan
Beth Israel Deaconess Medical
Center
330 Brookline Avenue
Department of Radiology
Boston, MA 02215

(A) Reed, Amy
Penn State University
500 University Drive
Hershey, PA 17033
P: 717-531-8866

(A) Rego, Alfred
South Florida Heart and Lung
Institute
21097 NE 27th Court, Suite 370
Aventura, FL 33180
P: 305-935-9883
F: 305-935-9711

(A) Rhodes, Jeffrey
Vascular Surgery Associates
1445 Portland Avenue, Suite 108
Rochester, NY 14621
P: 585-922-5550
F: 585-922-5950

(A) Ricci, Michael
Fletcher Allen Health Care
111 Colchester Ave, M/S 349MP5
Vascular Surgery
Burlington, VT 05401
P: 802-847-5155
F: 802-847-5907

(S) Rich, Norman
USUHS/Department of Surgery
4301 Jones Bridge Road
Bethesda, MD 20814
P: 301-295-3155
F: 301-295-3627

(I) Richardson, Graeme
Rural Clinical School, UNSW
PO Box 5695
Wagga Wagga, 2650
Australia

(A) Ricotta, John
Washington Hospital Center
Department of Surgery
110 Irving Street NW, Room G253
Washington, DC 20010
P: 202-877-5133
F: 202-877-3699

(A) Ricotta, II, Joseph
Mayo Clinic
200 First Street SW
Rochester, MN 55905
P: 507-266-0937

(A) Risley, Geoffrey
Cardiothoracic & Vascular
Surgical Associates, PA
1824 King Street, Suite 200
Jacksonville, FL 32204
P: 904-421-5586
F: 904-384-3838

(S) Robicsek, Francis
Carolinas Heart Institute
PO Box 32861
Charlotte, NC 28232-2861
P: 704-355-4005
F: 704-355-6227

(A) Rockman, Caron
NYU Medical Center
530 1st Avenue, 6F
New York, NY 10016
P: 212-263-7311
F: 212-263-7722

(A) Roddy, Sean
The Vascular Group, PLLC
43 New Scotland Ave, MC157
Albany, NY 12208
P: 518-262-8720
F: 518-262-6720

(A) Rodman, Charles John
Charles J. Rodman MD, PA
1756 Santa Fe Street
Corpus Christi, TX 78404
P: 361-888-4435
F: 361-888-4378

(A) Rodriguez, Agustin
University of Puerto Rico School
of Medicine
PO Box 364683
San Juan, PR 00936-4683
P: 787-763-2440
F: 787-763-3898

(A) Rogers, D. Michael
Harbin Clinic
1825 Martha Berry Blvd
Rome, GA 30165
P: 706-233-8508

(A) Rohrer, Michael
University of TN Medical School
1325 Eastmoreland Avenue,
Suite 310
Memphis, TN 38104
P: 901-448-4100
F: 901-448-4110

(S) Rolley, Ronald
610 Ridgewood Drive
West Lafayette, IN 47906
F: 317-477-9668

(A) Rollins, David
3660 Euclid Ave, #107
Willoughby, OH 44094
P: 440-269-8346
F: 440-975-5763

(A) Rooke, Thom
Mayo Clinic
200 First St, SW
Rochester, MN 55905
P: 507-266-7457
F: 507-266-1617

(A) Rosenfeld, Joel
St Luke's Hospital
801 Ostrum Street
Bethlehem, PA 18015
P: 610-954-2255
F: 610-954-6450

(A) Roth, Steven
Vein Care Pavilion of the South
447 North Belair Road, Suite 103
Evans, GA 30809
P: 706-854-3333

(A) Roupenian, Armen
Vein & Laser Center NE
45 Resnik Road, Suite 305
Plymouth, MA 02360
P: 508-747-1333
F: 508-747-2850

(A) Rubin, Brian
Washington University in St. Louis
660 S. Euclid Ave, Campus
Box 8109
St. Louis, MO 63110-1094
P: 314-362-7331
F: 314-362-7363

(A) Rubin, Jeffrey
Detroit Medical Center/Harper
University Hospital
Vascular Surgery
3990 John R
Detroit, MI 48201
P: 313-745-8637
F: 313-993-0244

(A) Ruby, Steven
St. Francis Hospital and Medical
Center
1000 Asylum Avenue, 2120
Hartford, CT 06105
P: 860-246-4000
F: 860-527-6985

(H) Ruckley, C. Vaughan
University of Edinburgh
1 Mayfield Terrace
Edinburgh, EH9 1 RU
UK
P: +13.166.78678

(C) Rupani, Bobby
UMDNJ – University Hospital
90 Bergen Street
Newark, NJ 07101
P: 973-972-6295
F: 973-972-0092

(S) Rutherford, Robert
14337 Dorsal Street
Corpus Christie, TX 78418
P: 361-949-0327
F: 361-949-8381

(A) Ryan, John
VA Medical Center
2501 East 22nd Street
Sioux Falls, SD 57105
P: 605-997-6277

(S) Sabety, Adrian

(As) Sadick, Neil
Sadick Aesthetic Surgery &
Dermatology
911 Park Avenue
New York, NY 10021-0337
P: 212-772-7242
F: 212-517-9566

(I) Sakuda, Hitoshi
Tomishiro Central Hosptial,
Vascular Surgery
25 Ueta
Tomigusuku Okinawa, 901-0243
Japan
P: +81.98.8951168
F: +81.98.8951422

(A) Salles-Cunha, Sergio

(A) Salvian, Anthony
#1214-750 West Broadway
Vancouver, BC V5ZJ2
Canada
P: 604-874-0532
F: 604-874-7806

(A) Samhouri, Farouq
Northeast Philadelphia Vascular
Surgeons, PC
2137 Welsh Road, Suite 1C
Philadelphia, PA 19115
P: 215-969-3944
F: 215-969-3886

(A) Samson, Russell
Mote Vascular Foundation
600 N. Cattlemen Road, Suite 220
Sarasota, FL 34232-6422
P: 941-371-6565
F: 941-377-7731

(S) Samuels, Peter

(A) Santilli, Steven
University of Minnesota
420 Delaware St SE, MMC 195
Department of Surgery, Division
of Vascular
Minneapolis, MN 55455
P: 612-625-1485

(A) Schadeck, Michel
Medical Center
5, Rue Michel Chasles
Paris, F-75012
France
P: +33.14.3892220
F: +33.01.43896627

(A) Schanzer, Harry
Mount Sinai Medical Center
993 Park Avenue
New York, NY 10028
P: 212-396-1254
F: 212-396-1338

(I) Schapira, Armando
Clinica de Flebolinfologia
Buenos Aires 1013
Rosario, 2000
Argentina

(A) Schellack, Jon
Vascular Clinic
5425 Brittany Drive, Suite B
Baton Rouge, LA 70808-4306
P: 225-767-5479
F: 225-445-7202

(As) Schepers, Helmut
Ganzoni Management AG
St. Georgen Str 70
Winterthur
Zuerich, 8401
Switzerland
P: +41.52.2650035
F: +41.52.2650001

(H) Schmid-Schonbein, GW
University of CA, San Diego
9500 Gilman Dr, Bioengr 0412
La Jolla, CA 92093-0412
P: 619-534-4272
F: 619-534-5722

(S) Schmidt, Frank
1137 Jefferson Avenue
New Orleans, LA 70115-3011
P: 504-568-4576
F: 504-568-4633

(A) Schneider, Joseph
Vascular & Interventional
Program of Central DuPage Hospital
Ambulatory Serv Pav Suite 201
25 North Winfield Road
Winfield, IL 60190
P: 630-933-4487
F: 630-933-2009

(A) Schul, Marlin
Lafayette Regional Vein Center
985 S. Creasy Lane
Lafayette, IN 47905
P: 765-807-2770
F: 765-807-0348

(S) Schuler, James
University of Illinois Vascular Surgery
1740 W. Taylor, #2200
Chicago, IL 60612
P: 312-996-7595
F: 312-996-2704

(S) Schultz-Ehrenburg, Ulrich
Wandlitz
Germany
P: +49.33.05682211
F: +49.33.05622654

(A) Scovell, Sherry
Massachusetts General Hospital
Division of Vascular Surgery
15 Parkman Street WAC 440
Boston, MA 02114
P: 671-543-9955

(I) Scurr, John
Lister Hospital, Lister House
Chelsea Bridge Rd
London, SW1W 8RH
UK
P: +44.027.07309563
F: +44.027.02599938

(A) Seabrook, Gary
Medical College of Wisconsin
9200 West Wisconsin Avenue
Vascular Surgery
Milwaukee, WI 53226
P: 414-805-9160
F: 414-805-9170

(S) Segal Halperin, Boris
Av Luis Maria Campos 1575, PB "C
Buenos Aires, 1426
Argentina
P: +54.17.849111
F: +54.17.849111

(As) Semrow, Carolyn
College Station Venous Diagnostic Center
1208 S. Magnolia Street
Hearne, TX 77859-3717
P: 979-279-4146
F: 979-764-5757

(A) Shafique, Shoaib
Indiana University School of Medicine
1001 W. 10th Street
OPE 303
Indianapolis, IN 46202
P: 317-630-7879
F: 317-639-0271

(C) Shah, Amit
Montefiore Medical Center
3400 Bainbridge – MAP 4
Department Vascular Surgery
Bronx, NY 10467

(C) Shah, Hemal
Englewood Hospital and Medical Center
350 Engle Street
Department of Surgery
Englewood, NJ0 7631

(I) Shaidakov, Evgeny
Military Medical Acadamy
Fontanka 106
St. Petersburg, 198013
Russia
P: +7.812.7468902

(A) Shamma, Asad
Artery & Vein Institute
PO Box 11-1666
Sodeco Sq; 8th Floor, Block B
Beirut, 111666
Lebanon

(A) Shanley, Charles
Beaumont Hospitals
3601 West 13 Mile Road
Hospital Administration
Royal Oak, MI 48073
P: 248-898-3321
F: 248-898-5418

(A) Shellito, John
Wichita Clinic
3311 E. Murdock
Wichita, KS 67208
P: 316-689-9453
F: 316-689-9710

(A) Shields, Raymond
Mayo Clinic
200 1st Street SW
Rochester, MN 55905
P: 507-266-9737
F: 507-266-1617

(A) Shin, David
Houston Vein Specialists
7501 Fannin Street, Suite 703
Houston, TX 77054
P: 713-790-0000
F: 713-790-1212

(A) Shortell, Cynthia
Duke University Medical Center
Box 3538
Durham, NC 27710
P: 919-681-2915
F: 919-681-3563

(A) Sidawy, Anton
7320 Yates Court
McLean, VA 22101
P: 202-745-8295
F: 202-745-8293

(A) Silva, Michael
UTMB
301 University Blvd
Galveston, TX 77555-0735
P: 409-772-6366

(I) Simkin, Roberto
University of Buenos Aires
Argentina
Talcahuano 1155, P. Baja Dto.5
Buenos Aires, 1013
Argentina
P: +54.11.48126098
F: +54.11.48054774

(A) Simkin, Carlos
Clinica Simkin Varicocenter
Talcauhano 1155 PB Dto:5
Buenos Aires, 1013
Argentina
P: +54.11.48124826

(S) Simonian, Simon
7616 Laurel Leaf Drive
Potomac, MD 20854
P: 301-983-8856

(As) Simons, Glen
Kentucky Vein Care
3229 Summit Square Place,
Suite 150
Lexington, KY 40509

(C) Singh, Kuldeep
Staten Island University Hospital
256 Mason Avenue
Staten Island, NY 10305

(As) Size, Gail
Inside Ultrasound, Inc.
13303 S. Desert Dawn Drive
Pearce, AZ 85625
P: 520-642-1303
F: 520-642-1304

(S) Sladen, Joseph
Canada

(C) Smolock, Christopher
The Methodist Hospital
6550 Fannin Street
Smith Tower 1401
Houston, TX 77030

(A) Sobel, Michael
VA Puget Sound Healthcare
System
1660 S. Columbian Way,
SS (112)
Seattle, WA 98108-1597
P: 206-764-2255
F: 206-764-2529

(A) Solit, Robert
Albert Einstein Medical Center
5401 Old York Road, Suite 203
Philadelphia, PA 19141
P: 215-456-6178
F: 215-456-6204

(A) Somaya, Anand
Bombay Vein Clinic
73 Lady Ratan Tata Medical &
Res Center
Maharshi Karve Road, Cooperage
Mumbai, 400 020
India
P: +91.22.040164

(A) Spence, Richard

(C) St. Louis, Myron
University of Connecticut/
Hartford Hospital
Department of Surgery
80 Seymour Street
Hartford, CT 6102

(As) Stagl, John
Sonosoft
1990 Main Street, Suite 750
Sarasota, FL 34236
P: 941-306-5800

(A) Stanley, Andrew
MCHV Campus Smith
111 Colchester Avenue
Burlington, VT 5401
P: 802-656-8474
F: 802-656-0680

(A) Steed, David
UPMC Shadyside
5200 Centre Avenue, Suite 307
Pittsburgh, PA 15232
P: 412-623-8437
F: 412-623-8440

(A) Stephanian, Edic
Baylor Medical Center
700 Walter Reed Blvd, Suite 311
Garland, TX 75042
P: 972-487-6400
F: 972-487-1686

(A) Stoughton, Julianne
Vein Solutions
92 Montvale Ave, Suite 3200
Stoneham, MA 02180
P: 781-438-8117
F: 781-438-8116

(A) Suh, Bo Yang
Yeungnam Medical Center
Department of Surgery
317-1 Daemyung-Dong, Nam-Gu
Daegu, 703-035
Korea
P: +10.3.5038011

(A) Sulkin, Michael
Horizon Surgical Group
9210 Corporate Blvd, Suite 100
Rockville, MD 20850
P: 301-330-1000

(C) Sullivan, Cornelius
Brigham & Women's Hospital
75 Francis Street
Department of Anesthesiology
Boston, MA 02115

(S) Sumner, David
2324 W. Lakeshore Drive
Springfield, IL 62707
P: 217-529-2910

(S) Taheri, Syde
268 Dan Troy
Williamsville, NY 14221
P: 716-633-1838
F: 716-634-4164

(A) Tapper, S. Scott
Symmetry Laser Cein Center
2169 SE Ocean Blvd
Stuart, FL 34996
P: 772-286-5501

(C) Thai, Janice
University of Arizona
1501 N. Campbell Avenue,
Rm 4404
Tucson, AZ 85724

(A) Thorpe, Patricia
Arizona Heart Institute & Hospital
7181 E. Camelback Road,
Suite 505
Scottsdale, AZ 85251
P: 602-206-7193
F: 480-584-6717

(H) Thulesius, Olav
University Hospital
Fac of Health Sciences
Linkoping, S-581 85
Sweden
F: +46.13.145949

(A) Towne, Jonathan
Medical College of Wisconsin
9200 West Wisconsin Avenue
Milwaukee, WI 53226
P: 414-456-6966
F: 414-456-6216

(S) Tretbar, Lawrence

(A) Tuerff, Sonya
Christiana Care Vascular
Specialists
4765 Ogletown Stanton Road
1E20
Newark, DE 19713
P: 302-733-5700

(A) Tzilinis, Argyrios
Physicians Regional Medical
Group
6101 Pine Ridge Road
Naples, FL 34119
P: 239-348-4093

(I) Uhl, Jean-Francois
Vanuse Veins Surgical Center
113 Av ch de Gaulle
Neuilly-sur-seine, 92200
France
P: +33.14.7472211
F: +33.14.7472060

(C) Vallabhaneni, Raghu
Washington University School of Medicine
660 S. Euclid Avenue
Campus Box 8109
St. Louis, MO 63110

(A) Van Bemmelen, Paul
Temple University
3401 No Broad St, Parkinson 4th Flr
Philadelphia, PA 19140
P: 215-707-3622
F: 215-707-5901

(I) Vandendriessche-Hobbs, Marianne
Vein Clinic
288 Maaltebrugge St
Gent, B9000
Belgium
P: +32.9.2454306
F: +32.9.2200781

(A) Varnagy, David
Vascular Surgery and Endovascular Therapeutics Surgery Spec of S. Florida
10000 West Colonial Drive, Off 495
Orlando, FL 34761
P: 305-904-8149

(A) Vasquez, Michael
The Venous Institute of Buffalo
The Wellness Center
415 Tremont Street
North Tonawanda, NY 14120
P: 716-690-2691
F: 716-690-2695

(A) Vazquez, Richard
Northwestern Memorial Hospital
201 E. Huron St, Galter,
Suite 11-250
Chicago, IL 60611
P: 312-649-6562
F: 312-649-9027

(A) Vedantham, Suresh
Mallinckrodt Institute of Radiology
510 S. Kings Highway Blvd
Box 8131
St. Louis, MO 63110
P: 314-719-3431
F: 314-362-2276

(C) Vegas, Dave
OHSU
3181 SW Sam Jackson Park Road
Portland, OR 97239

(C) Verma, Anil
Ochsner Medical Center
1514 Jefferson Highway
New Orleans, LA 70121

(A) Vermilion, Blair
Ohio State University
1654 Upham Drive
Means Hall N325
Columbus, OH 43210
P: 614-293-8536

(S) Villavicencio, J. Leonel
USUHS, Professor Surgery
4301 Jones Bridge Rd
Bethesda, MD 20814
P: 202-782-6592
F: 202-782-3371

(A) = Active (As) = Associate (H) = Honorary (S) = Senior (C) = Candidate (I) = International

(A) Vivekanandan, Uthan
North Atlanta Vascular Clinic
3400-A Old Milton Pkwy,
Ste 300
Alpharetta, GA 30005
P: 770-771-5260
F: 770-771-5269

(A) Vo, Danny
University of Florida
655 West 8th St
Jacksonville, FL 32209
P: 904-244-3925

(A) Vogt, Philip
Fox Valley Surgical Associates
1818 N. Meade Street
Appleton, WI 54911
P: 920-731-8131
F: 920-738-7850

(A) Wakefield, Thomas
University of Michigan Medical Center
1500 E. Medical Center Dr,
THCC 2210
Ann Arbor, MI 48109-0329
P: 734-936-5820
F: 734-647-9867

(A) Wasserman, Dean
Vein Treatment Center of NJ
1 West Ridgewood Avenue,
Suite 306
Paramus, NJ 07652
P: 201-612-1750
F: 201-612-1760

(A) Weingarten, Michael
Drexel University College of Medicine/Hahnemann Hospital
245 N. 15th Street #7150
Mailstop 413
Philadelphia, PA 19102
P: 215-762-4005
F: 215-762-8699

(A) Welch, Harold
Lahey Clinic
41 Mall Rd, Peripheral Vascular Surgery
Burlington, MA 01805
P: 781-744-8193
F: 781-744-5744

(A) Wennberg, Paul
Mayo Clinic
200 First Street SW
Rochester, MN 55905
P: 507-266-7231
F: 507-266-1617

(S) Wheeler, H. Brownell
University of Massachusetts Medical School
55 Lake Ave North, #S3-810,
Surgery
Worcester, MA 01655
P: 508-856-2201
F: 508-856-6941

(A) Whiting, John
Idaho Vein Center
444 Hospital Way, Suite 777
Pocatello, ID 83201
P: 208-233-1451

(A) Williams, David
University of Michigan B1-D530
1500 E. Medical Center Drive
Ann Arbor, MI 48109-0030
P: 734-662-2717

(S) Williams, G. Melville
Johns Hopkins Hospital
600 No Wolfe St, Harvey 611
Baltimore, MD 21287-8611
P: 410-955-5165
F: 410-614-2079

(I) Wittens, Cees HA
University Hospital Maastricht
P. Debeyelaan 25
Maastricht, 3056 LE
Netherlands
P: +31.64.3440660

(A) Wladis, Alan
Vascular Institute of Central Florida
2501 N. Orange Avenue, Suite 402
Orlando, FL 32804
P: 407-303-7250
F: 407-303-7255

(A) Wolk, Seth
Restoration Vein Care
5333 McAuley Drive, Suite 4016
Ann Arbor, MI 48106
P: 734-712-4310

(C) Wright, Mark
University of Arkansas for Medical Science
4301 W. Markham Street
Slot 520-2
Little Rock, AR 72205

(A) Xenos, Eleftherios
University of Kentucky
Division of General Surgery
800 Rose Street
Lexington, KY 40536
P: 859-323-6346
F: 859-323-6840

(A) Yamaki, Takashi
Tokyo Women's Medical University
8-1, Kawada-cho, Shinjuku-ku
Tokyo, 162-8666
Japan
P: +81.33.3538111
F: +81.33.2250940

(S) Yao, James ST
Northwestern University Medical School
675 N. Saint Clair, Suite 650
Chicago, IL 60611
P: 312-695-2716
F: 312-695-4955

(S) Yellin, Albert
59-415 Kawowo Road
Haleiwa, HI 96712
P: 808-638-0510

(C) Yunus, Tahir
William Beaumont Hospital
3601 W. 13 Mile Road
Royal Oak, MI 48073
P: 248-854-7972

(As) Zakaria, Aamir

(I) Zamboni, Paolo
University Degli Studi Di Ferrara
203 Corso Giovecca, Surgery
Ferrara, 44100
Italy
P: +39.053.2236524
F: +39.053.2237443

(A) Zatina, Michael
Maryland Vascular Associates, LLC
3350 Wilkens Ave, Ste 201
Baltimore, MD 21229-4615
P: 410-646-4888
F: 410-646-2828

(A) Zelenock, Gerald
University of Toledo Medical Center
3000 Arlington Avenue
Mailstop 1095
Toledo, OH 43614
P: 419-383-6298
F: 419-383-6636

(A) Zierler, Brenda
University of Washington
1959 NE Pacific Street,
Box 357266
Seattle, WA 98195-7266
P: 206-616-1910
F: 206-616-7495

(A) Zierler, R. Eugene
University of Washington
1959 NE Pacific Street
Box 356410
Seattle, WA 98195
P: 206-598-9851
F: 206-616-7495

(A) Zimmet, Steven
Zimmet Vein & Dermatology
1500 West 34th Street
Austin, TX 78703
P: 512-485-7700

(A) Zubicoa, Santiago Ezpeleta
Hospital Ruber Internacional
c/ la Maso N. 38
Madrid, 28034
Spain
P: +34.91.3875157
F: +34.91.3875158

(A) Zwolak, Robert
Dartmouth Hitchcock Medical Center
1 Medical Center Drive
Lebanon, NH 03756
P: 603-650-4973

AMERICAN VENOUS FORUM

Geographical Roster

ALABAMA

Birmingham
Isobe, James Hajime
Lochridge, Stanley K
Passman, Marc A

ARKANSAS

Little Rock
Ferris, Ernest J
Wright, Mark

ARIZONA

Chandler
Opie, John C

Pearce
Size, Gail P

Prescott
Fleck, Robin M

Scottsdale
Morrison, Nick
Puggioni, Alessandra
Thorpe, Patricia E

Tucson
Hunter, Glenn C
Ihnat, Daniel Michael
Thai, Janice

CALIFORNIA

Agoura
Barker, Wiley F
Baldwin Park
Murray, James D

Beverly Hills
Gradman, Wayne S

Burbank
Conrad, John Kenneth

Corona
Gorski, Yara C
Costa Mesa
Arata, Michael

Dana Point
Cannon, Jack A

Encinitas
Cheng, Van Le

Encino
Najibi, Sasan

Escondido
Bulkin, Anatoly

Glendale
Mihranian, Mardiros Haig

Irvine
Kanter, Alan

La Jolla
Bunke, Nisha J
Delaria, Giacomo A
Schmid-Schonbein, GW

Laguna Hills
Duensing, Robert A
Loma Linda
Hasaniya, Nahidh W

Orange
Flanigan, D. Preston

Portola Valley
Fogarty, Thomas J

Rancho Palos Verdes
Donayre, Carlos E

Roseville
Monahan, Daniel L

San Diego
Angle, Niren
Housman, Leland B
OByrne, Margaret G

San Francisco
Denbo, Howard E
Kwan, Sharon
Long, John B

San Jose
Kaplan, Jeff H

San Mateo
Harris, Edmund J
Santa Barbara
Mckittrick, James E

Santa Cruz
Jurnecka, Jan S

Santa Monica
Hoffman, Cheryl H

Seal Beach
Gaspar, Max R

Stanford
Harris, E. John

Thousand Oaks
Ortega, Raul E

Torrance
Duffy, David M

Walnut Creek
Isaacs, Mark

COLORADO

Aurora
Hammond, Sharon L

Fort Collins
Kaufman, Steven L

Palisade
Bernhard, Victor M

CONNECTICUT

Farmington
Menzoian, James O

Greenwich
Febles, Anthony
Mulcare, Robert

Hartford
Ruby, Steven T
St. Louis, Myron

Trumbull
Gagne, Paul

DISTRICT OF COLUMBIA

Washington
Beavers, Frederick P
DePalma, Ralph G
Laredo, James
Ricotta, John J

DELEWARE

Newark
Garcia, Mark J
Tuerff, Sonya N

FLORIDA

Aventura
Rego, Alfred

Boca Raton
Meretei, Attila

Jacksonville
Risley, Geoffrey L
Vo, Danny H

Miami
Almeida, Jose Ignacio
Ginzburg, Enrique

Naples
Tzilinis, Argyrios

Orlando
Varnagy, David
Wladis, Alan R

Palm Beach Gardens
Miskin, Barry M

Port Charlotte
Gruneiro, Laura A

Sarasota
Samson, Russell H
Stagl, John F

South Miami
Kang, Steven S

St. Petersburg
Collins, Paul S

Stuart
Tapper, S. Scott

Tampa
Kerr, Thomas M

West Palm Beach
Lynn, Richard A

Weston
Fernandez, Bernardo B

GEORGIA

Albany
Corr, John Price

Alpharetta
Vivekanandan, Uthan

Atlanta
Chaikof, Elliot L
Ferrier, Frank
Kasirajan, Karthikeshwar

Evans
Roth, Steven M

Gainesville
Procter, Charles D

Rome
Kirkland, John Smith
Rogers, D. Michael

Savannah
Alpert, Joseph

HAWAII

Haleiwa
Yellin, Albert E

Honolulu
Kistner, Robert L
Lurie, Fedor
Masuda, Elna M

IOWA

West Des Moines
Anderson, Robert

IDAHO

Pocatello
Whiting, John H

ILLINOIS

Arlington Heights
Forrestal, Mark

Chicago
Bassiouny, Hisham S
Durham, Joseph R
Ennis, William J
Matsumura, Jon S
McCarthy, Walter J
Morasch, Mark D
Naughton, Peter
Pearce, William H
Schuler, James J
Vazquez, Richard M
Yao, James ST

LaGrange
Gocke, John

Oak Brook
King, Ted

Park Ridge
Buckman, Jeffrey

Skokie
Caprini, Joseph A

Springfield
McLafferty, Robert B
Moore, Colleen M
Sumner, David S

Urbana
Hong, Steve C

Winfield
Schneider, Joseph R

INDIANA

Carmel
Finkelmeier, William R

Indianapolis
Crisostomo, Paul
Dalsing, Michael C
Goodson, Spencer F
Lemmon, Gary W
Shafique, Shoaib

Lafayette
Schul, Marlin W

Mishawaka
Kiser, Robert Cameron

West Lafayette
Rolley, Ronald T

KANSAS

Wichita
Shellito, John L

KENTUCKY

Lexington
Simons, Glen W
Xenos, Eleftherios S

Pikeville
Collins, David E

LOUISIANA
Baton Rouge
Frusha, John D
Schellack, Jon V

New Orleans
Amin, Rohit
Hollier, Larry H
Schmidt, Frank E
Verma, Anil

Shreveport
Knight, Jr., Charles D

MASSACHUSETTS

Arlington
Flynn, William F

Boston
Cantelmo, Nancy L
Iafrati, Mark D
Joglar, Fernando Luis
Nguyen, Tony
O'Donnell, Thomas F
Polak, Joseph F
Reddy, Madan
Scovell, Sherry D
Sullivan, Cornelius A

Brighton
Razvi, Syed A

Burlington
Welch, Harold J

Framingham
Donaldson, Magruder C

Milton
Kechejian, Gregory J

Plymouth
Roupenian, Armen L

Stoneham
Chang, Jeanette K
Nath, Ronald L
Stoughton, Julianne

Wellesley
Persson, Alfred V

West Roxbury
Raffetto, Joseph D

West Springfield
Goodman, Robert L

Worcester
Wheeler, H. Brownell

MARYLAND

Baltimore
Buchbinder, Dale
Flinn, William R
Heller, Jennifer A
Lal, Brajesh K
Williams, G. Melville
Zatina, Michael A

Bethesda
Chang, Richard
Rich, Norman M
Villavicencio, J. Leonel

Owings
Pietropaoli, John Anthony

Potomac
Simonian, Simon J

Rockville
Sulkin, Michael D

MAINE

Bangor
Cambria, Robert A

Lewiston
Blondeau, Benoit

Portland
Eldrup-Jorgensen, Jens

MICHIGAN

Ann Arbor
Criado, Enrique
Greenfield, Lazar J
Henke, Peter K
Myers, Jr., Daniel
Rectenwald, John Edward
Wakefield, Thomas W
Williams, David
Wolk, Seth W

Bingham Farms
Brown, O. William

Detroit
Lin, Judith C
Mattos, Mark A
Moreira, Barbara D'Agnoluzzo
Nypaver, Timothy J
Rubin, Jeffrey R

East Lansing
Garcia, Manuel E

Flint
Garner, Scott A

Grand Rapids
Mansour, M. Ashraf

Kalamazoo
Jain, Krishna M

Livonia
Cummings, Emily W
Pavone, Lisa E

Petoskey
Kazmers, Andris

Royal Oak
Shanley, Charles J
Yunus, Tahir

Troy
Dobzyniak, Christopher
Engle, Jennifer S

Warren
Hans, Sachinder S

West Bloomfield
Elliott, Joseph P
Granke, Kenneth

MINNESOTA

Edina
Nicholson, Phifer C

Minneapolis
Santilli, Steven M

Rochester
Bjarnason, Haraldur
Duncan, Audra A

Felty, Cindy
Gloviczki, Peter
Gloviczki, Monika L
Kalra, Manju
Lall, Purandath
Oderich, Gustavo
Pannu, Rajmony
Ricotta, II, Joseph J
Rooke, Thom W
Shields, Raymond C
Wennberg, Paul W

MISSOURI

Columbia
Gardner, Glenn P
Janzen, Mark
Creve Coeur
Bein, Norman N

St. Louis
Rubin, Brian G
Geraghty, Patrick J
Pennell, Richard C
Vallabhaneni, Raghu
Vedantham, Suresh

MISSISSIPPI

Flowood
Neglen, Peter
Raju, Seshadri

NORTH CAROLINA

Chapel Hill
Marston, William A

Charlotte
Robicsek, Francis

Concord
Cicci, Christopher K

Durham
Ahluwalia, Hardeep S
Shortell, Cynthia K

Winston-Salem
Davis, Ross
Fleming, Shawn

NEBRASKA

Omaha
Lynch, Thomas G

NEW HAMPSHIRE

Lebanon
Goodney, Philip P

Lebanon
Zwolak, Robert M

Manchester
Baribeau, Yvon R
Furey, Patricia C

NEW JERSEY

Denville
Mintz, Bruce

Englewood
Elias, Steven
Shah, Hemal
Highland Park
Konigsberg, Stephen F

Morristown
Moritz, Mark W
Oliver, Mark A
New Brunswick
Haser, Paul B

Newark
Huang, Joe
Jamil, Zafar
Padberg, Frank T
Pennycooke, Owano
Rupani, Bobby J

Paramus
Chubak, John A
Wasserman, Dean H

Somers Point
Gosin, Jeffrey S

Somerset
Deak, Steven T

Teaneck
Friedman, Joseph

Toms River
Ramnauth, Subhash C

NEW MEXICO

Albuquerque
Corson, John D
Peloso, Ole A

Santa Fe
Hertzman, Phillip
Martin, Alfred J

NEVADA

Las Vegas
Bernstein, Rick V

Reno
Daake, John W
Merchant, Robert F

NEW YORK

Albany
Chang, Benjamin B
Darling, R. Clement
Roddy, Sean P

Bronx
Shah, Amit

Brooklyn
Ascher, Enrico
Hingorani, Anil P
Jung, Daniel
Mutyala, Manikyam
Pappas, Peter J
Rai, Dinker B

Buffalo
Harris, Linda M

Hartsdale
Fleisher, Arlen G

Middletown
Fiorianti, John A

New York
Adelman, Mark A
Adler, Grit
Baron, Howard C
Fischman, Aaron
Green, Richard M
Honig, Shaun
Jacobowitz, Glenn R
Kabnick, Lowell S
Lantis, John Carlos
Min, Robert J
Nassiri, Naiem
Pamoukian, Vicken N
Rockman, Caron
Sadick, Neil S
Schanzer, Harry R

North Tonawanda
Vasquez, Michael A

Rochester
DeWeese, James A
Fanicullo, Dustin
Gillespie, David L
Glass, Carolyn
Illig, Karl A
Rhodes, Jeffrey

Roslyn
Chang, John B

Schenectady
Blumenberg, Robert M

Staten Island
Fodera, Maria Elena
Singh, Kuldeep

Stony Brook
Dias, Celso
Gasparis, Antonios P
Labropoulos, Nicos
Malgor, Rafael
Maru, Sandip T
Williamsville
Taheri, Syde A

OHIO

Cincinnati
Cranley, Robert D
Kempczinski, Richard
Lohr, Joann M
Muck, Patrick E

Cleveland
Blebea, John
Carman, Teresa L
Clair, Daniel G

Cleveland Heights
Perez, Alejandro

Columbus
Franz, Randall
Vermilion, Blair D

Lima
Malhotra, Praveen K

Portsmouth
Khoury, Thomas L

Toledo
Beebe, Hugh G
Balkany, Louis
Comerota, Anthony J
Gale, Steven S
Nazzal, Munier MS
Zelenock, Gerald B

Willoughby
Rollins, David L

OREGON

Bend
Jones, Andrew D

Grants Pass
Deatherage, Mark Frederick

Portland
Danczyk, Rachel
Edwards, James M
Landry, Gregory James
Liem, Timothy K
Moneta, Gregory L
Pavcnik, Dusan
Vegas, Dave

PENNSYLVANIA

Bethlehem
Rosenfeld, Joel C

Easton
Balshi, James D
Fisher, Jay B

Hershey
Reed, Amy B

Mechanicsburg
Calcagno, David

Monroeville
Plaza-Ponte, Mario T

Philadelphia
Merli, Geno J
Samhouri, Farouq A
Solit, Robert W
Van Bemmelen, Paul S
Weingarten, Michael S

Pittsburgh
Chaer, Rabih A
Cho, Jae-Sung
Jarrett, Fredric
Steed, David L

Reading
Impellizzeri, Paul

Sewickley
Collier, Paul E

Villanove
Kerstein, Morris D

Wayne
Ernst, Calvin B

York
Castronuovo, John J

RHODE ISLAND

Providence
Carney, Wilfred I
Garcia-Toca, Manuel
Patterson, Robert B

SOUTH CAROLINA

Charleston
Hallett, John W

SOUTH DAKOTA

Sioux Falls
Ryan, John J

TENNESSEE

Clarksville
Daugherty, Stephen Franklin

Knoxville
Goldman, Mitchell H

Memphis
Rohrer, Michael J

TEXAS

Austin
Dilling, Emery
Zimmet, Steven

College Station
Hansen, Henry Andrew

Corpus Christi
Rodman, Charles John
Rutherford, Robert B

Dallas
Clagett, G. Patrick

El Paso
Pester, Thomas L

Fort Worth
Paladugu, Ramesh

Galveston
Killewich, Lois A
Silva, Michael B

Garland
Stephanian, Edic

Hearne
Semrow, Carolyn M

Houston
Hallman, Grady L
Lin, Peter
Peden, Eric
Shin, David D
Smolock, Christopher

Lubbock
Baldwin, John C
Dickerson, Sandra Dee

McAllen
Hovorka, John W

McKinney
Proctor, Mary C

San Antonio
Martinez, Jeffrey M
Pounds, Lori C

Temple
Bohannon, W. Todd
Bush, Ruth L

Victoria
Johnston, Robert H

Wichita Falls
Brazil, Clark W

UTAH

West Jordan
Lazarus, Harrison M

VIRGINIA

Alexandria
Cordts, Paul R

Arlington
Bergan, John J

Charlottesville
Cherry, Kenneth J
Owens, Lewis

McLean
Sidawy, Anton N

Norfolk
Bonawitz, Cara A

Portsmouth
Arbid, Elias J

Reston
Lee, Byung-Boong

Roanoke
Drougas, James A

Williamsburg
Delaurentis, Dominic A

VERMONT

Burlington
Ricci, Michael A
Stanley, Andrew C

WASHINGTON

Bellevue
Gibson, Kathleen D

Seattle
Jayaraj, Arjun
Lundgren, Rachel
Meissner, Mark H
Sobel, Michael
Zierler, Brenda K
Zierler, R. Eugene

Vancouver
Nicholls, Stephen

WISCONSIN

Appleton
Vogt, Philip A

Green Bay
Hutto, John D

Madison
Carr, Sandra C
Kent, K. Craig

Manitowoc
Gueldner, Terry L

Milwaukee
Brown, Kellie
Seabrook, Gary R
Towne, Jonathan B

WEST VIRGINIA

Charleston
AbuRahma, Ali F
Boland, James P

International Members

ARGENTINA

Buenos Aires
Cigorraga, Jorge Raul
Enrici, Ermenegildo A
Papendieck, CM
Pietravallo, Antonio FR
Segal Halperin, Boris M
Simkin, Carlos G
Simkin, Roberto

Mendoza
Farmache, Alejandro H

Roasrio
Schapira, Armando E

AUSTRALIA

Wagga Wagga
Richardson, Graeme D

AUSTRIA

Vienna
Partsch, Hugo

BELGIUM

Ghent
Vandendriessche-Hobbs, Marianne

BRAZIL

Porto Alegre- RS
Komlos, Pedro P

Sao Paulo
Kikuchi, Rodrigo
Osse, Francisco

CANADA

Calgary
Hill, Douglas

Hamilton
Hirsh, Jack

Laval
Danylewick, Richard W

Quebec
Dion, Yves M

Vancouver
Hsiang, York N
Salvian, Anthony J
Sladen, Joseph G

CHILE

Viña del Mar
Orrego, Alvaro Esteban

CYPRUS

Ayios Dhometios
Nicolaides, Andrew N

DENMARK

Hellerup
Foegh, Pia

Naestved
Rasmussen, Lars H

FRANCE

Chassieu
Perrin, Michel R

Grenoble
Carpentier, Patrick H

Marseille
Hartung, Olivier

Montpelier
Milleret, Rene

Neuilly-sur-Seine
Cornu-Thenard, Andre M
Uhl, Jean-Francois

Nice
Guex, Jean-Jerome
Pittaluga, Paul

Paris
Cazaubon, Michele
Natali, Jean P
Schadeck, Michel P

GERMANY

Bonn
Rabe, Eberhard

Hirschberg
Proebstle, Thomas

Nuremberg
Noppeney, Thomas

Wandlitz
Schultz-Ehrenburg, Ulrich

GREECE

Athens
Balas, Panayiotis E
Liasis, Nikolaos E

GUATEMALA

Guatemala City
Corrales, Noel Ernesto

INDIA

Hyderabad
Gupta, Prem C

Mumbai
Somaya, Anand C

ISRAEL

Afula
Markel, Arie

Zerifin
Bass, Arie

ITALY

Ferrara
Zamboni, Paolo

Rome
Allegra, Claudio
Caggiati, Alberto
di Marzo, Luca

JAPAN

Fukushima
Hoshino, Shunichi
Ogawa, Tomohiro

Izumisano
Hirano, Tetsuya

Moriya City
Iwai, Takehisa

Okinawa
Sakuda, Hitoshi

Tokyo
Ishimaru, Shin
Yamaki, Takashi

KOREA

Daegu
Suh, Bo Yang

Seoul
Kim, Young-Wook

LEBANON

Beirut
Shamma, Asad R

LUXEMBOURG

Goetzingen
Lamesch, Alfred J

MALAYSIA

Kuala Lumpor
Liew, Ngoh C

MEXICO

Huixquilucan
Aguila Marquez, Roberto

Mexico City
Paramo, Marcelo

NETHERLANDS

Maastricht
Wittens, Cees HA

Rotterdam
Klem, Taco M

Utrecht
Disselhoff, Ben

POLAND

ul. Reduty Ordona
Kompf, Boguslaw

PUERTO RICO

Coto Laurel
Martinez Trabal, Jorge L

San Juan
Rodriguez, Agustin A

RUSSIA

Moscow
Bogachev, Vadim Y

St. Petersburg
Shaidakov, Evgeny V

Yekaterinburg
Belentsov, Sergey M

SERBIA

Nis
Milic, Dragan J

SOUTH KOREA

Seoul
Joh, Jin-Hyan

SPAIN

Madrid
Monedero, Javier Leal
Zubicoa, Santiago Ezpeleta

SWEDEN

Helsingborg
Eklof, Bo G

Linkoping
Thulesius, Olav

Orebro
Arfvidsson, Berndt

Uppsala
Bergqvist, David

SWITZERLAND

Geneva
Christenson, Jan T

Lucerne
Lauber, Andre F

Strafa
Bollinger, Alfred

Zurich
Schepers, Helmut

TURKEY

Istanbul
Kurtoglu, Mehmet H

UNITED KINGDOM

Alderney
Browse, Norman L

Edinburgh
Ruckley, C. Vaughan

London
Burnand, Kevin G
Davies, Alun Huw
Hobbs, John T
Scurr, John H

Solihull
Bradbury, Andrew W

Wexham
Coleridge Smith, Philip D

WEST INDIES

Trinidad
Maharaj, Dale A

AMERICAN VENOUS FORUM

BY LAWS

Article I – Name

The name of this organization shall be THE AMERICAN VENOUS FORUM.

Article II – Objectives

The objectives of this organization shall be (1) to promote venous and lymphatic health through innovative research, education, and technology; (2) to contribute to the active continuing education of its membership; (3) to hold annual meetings; and (4) to encourage the development and dissemination of knowledge regarding venous disease.

MISSION STATEMENT

The mission statement of this organization shall be to promote venous and lymphatic health through innovative research, education and technology.

Notwithstanding the foregoing, (a) no part of the organization's net earnings or assets shall inure to the benefit of any member, officer, or other person, except that the organization shall be authorized and empowered to pay reasonable compensation for services rendered and to make other payments and distributions in furtherance of the purposes set forth above, and (b) the organization shall not carry on any activity not permitted to an organization exempt from Federal income tax under Section 501 (c) (6) of the Internal Revenue Code of 1954, as amended (the "Code") or the corresponding provision of any future United States revenue statute.

Article III – Membership

Membership in the Venous Forum may include any physicians certified by their respective specialty Certifying Boards in the applicant's Country of practice who have demonstrated an interest in and contribution to the management of venous problems and who are in good standing in their State or Provincial Medical Societies. From time to time, the Membership Committee may recommend membership to scientists who are not M.D.'s and/or do not possess a doctoral degree but have demonstrated a major commitment to issues of venous disease.

1. Active Members: as identified above. Active members shall pay dues and have full voting privileges. Attendance at the Annual Scientific Program shall be expected of all Active members.

2. Senior Members included will be active members who have reached the age of 65 years; or members for whom, for reasons of health or other just cause, the Executive Committee recommends this category. They shall not be bound by meeting attendance and dues may be waived upon written request by Senior Member to waive dues. The Executive Committee may approve or disapprove the request at an executive meeting.

3. Honorary Members: individuals who have made outstanding contributions in the field of venous science. They shall not pay dues nor shall they have voting privileges.

4. Associate Members: Individuals who have an interest in the management of venous disorders, but do not necessarily hold a doctoral degree, such as nurses, registered vascular technologists, etc. Associate members will pay membership dues determined by the Executive Committee. Associate members are not eligible to vote or hold elective office.

5. Candidate Members: Physicians who are currently serving in a capacity of a resident or fellow in post-doctoral training programs and have demonstrated interest in and have made a contribution to the management of venous disease. Candidate members are not eligible to vote or hold elective office and are required to pay membership dues as set by the Executive Committee. Membership in this category shall not exceed 3 years. At the conclusion of post-doctoral training, Candidates may opt to become Active Members, by notifying the Forum in writing. In this instance, the application process will be waived, and the name shall automatically be placed on the Ballot.

Article IV – Election of Members

1. The process of election of Active members of the Society shall be as follows:

   a. Applications must be accompanied by a letter of interest, documenting the applicants experience in venous and lymphatic disease.

   b. Application forms must be accompanied by the curricula vitae of the candidates and shall be in the hands of the Secretary before the executive session at which it is desired that the candidate be considered for election.

   c. The Secretary shall send to the Chair of the Membership Committee these applications with all pertinent data before the annual meeting. The Membership Committee shall review the professional qualifications of the candidates.

   d. The Chair of the Membership Committee shall meet with the Executive Committee for the purpose of presenting the recommendations of the Membership Committee.

   e. The names of the candidates recommended by the Executive Committee for election shall be submitted by the Secretary to the membership in his or her annual report.

   f. Election to membership shall be by secret ballot, by a three fourths affirmative vote of those members present and voting at the annual business meeting.

   g. A candidate who fails to be elected at one meeting, may be presented to the membership at the next two (2) annual meeting of the Forum. The name of a candidate who fails of election a third time shall be dropped from the list of applications for membership. Such candidate's application may be resubmitted after an interval of two (2) years.

   h. New Member Attendance: Candidates, following their election to membership at the Annual Business Meeting of the organization, will be required to attend the next Annual Meeting of the Forum to be formally introduced to the membership.

2. The process of election for Associate and Candidate Members shall be as follows:

    a. Application forms presenting the curricula vitae of the candidates and signed by them shall be in the hands of the Secretary before the executive session at which it is desired that the candidate be considered for election.

    b. The Secretary shall send to the Chair of the Membership Committee these applications with all pertinent data before the annual meeting. The Membership Committee shall review the professional qualifications of the candidates.

    c. The Chair of the Membership Committee shall meet with the Executive Committee for the purpose of presenting the recommendations of the Membership Committee.

    d. The names of the candidates recommended by the Executive Committee for election shall be submitted by the Secretary to the membership in his or her annual report.

    e. Election to membership shall be by secret ballot, by a three fourths affirmative vote of those members present and voting at the annual business meeting

    f. A candidate who fails to be elected at one meeting may be presented to the membership at the next two (2) annual meeting of the Forum. The name of a candidate who fails of election a third time shall be dropped from the list of applications for membership. Such candidate's application may be resubmitted after an interval of two (2) years.

    g. New Member Attendance: Candidates, following their election to membership at the Annual Business Meeting of the organization, will be required to attend the next Annual Meeting of the Forum to be formally introduced to the membership.

3. The process of election of Honorary members of the Forum shall be as follows:

    a. Any Active or Senior member may nominate an individual for Honorary membership. The name and a brief description of the accomplishments of the nominee must be submitted to the Secretary before the Executive Session at which it is desired the nominee be considered for honorary membership. The Secretary shall distribute this information to the Honorary Membership Committee consisting of three (3) immediate past Presidents of the Executive Committee before the annual meeting.

    b. The Honorary Membership Committee shall make its recommendations to the Executive Committee.

    c. Following its deliberation, the Executive Committee may recommend that the candidate's name be submitted by the Secretary to the membership in the annual report at the Annual Business Meeting of the Forum.

    d. Election to Honorary Membership shall be by secret ballot by three fourths affirmative vote of the membership present and voting at the Annual Business Meeting.

Article V – Executive Committee

1. The Executive Committee of the Forum shall direct the activities of the Forum.

2. The Executive Committee shall be composed of the President, the President Elect, the Secretary, the Treasurer, the Recorder, at least three Councilors the Chairs of the Education and Research Councils, the immediate three Past Presidents, and the Archivist.

3. The Executive Committee shall be the governing body of the Forum and shall have full power to manage and act on all affairs on the Forum except as follows:

   a. It may not, without the approval of the Forum membership at an annual executive session, alter the initiation fees or levy any assessment against the membership, except that it may, set the annual dues rates and, in individual cases, waive annual dues or assessments.

   b. It may not amend the By Laws.

   c. It may neither elect new members nor alter the status of existing members, other than to apply the provisions of Article XI.

4. The President of the Forum shall serve as Chairman of the Executive Committee and the Secretary of the Forum as its Secretary.

5. Meeting of the Executive Committee shall be held at the call of the President of the Forum and each member of the Executive Committee must be notified in writing of the time and place of each such meeting no less than ten (10) days prior to the meeting.

6. The annual meeting of the Executive Committee shall precede the annual business meeting of the Forum membership.

7. A majority of the voting members of the Executive Committee shall constitute a quorum for the transaction of business.

8. The act of a majority of members of the Executive Committee present at a duly called meeting at which a quorum is present shall be the act of the Executive Committee unless the act of a greater number is required by applicable statute or these By Laws.

9. Any action which is required by law of the Articles of Incorporation or these By laws to be taken at a meeting of the Executive Committee, or any other action which may be taken without a meeting if a consent in writing, setting forth the action taken shall be signed by all of the members of the Executive Committee entitled to vote with respect to the subject matter thereof. Any such consent signed by all of the members of the Executive Committee shall have the same force and effect as a unanimous vote at a duly called and constituted meeting of the Executive Committee.

10. American Venous Forum Foundation: At its Annual Meeting, the Executive Committee shall elect up to eight (8) individuals to serve as members of the Board of Directors of the American Venous Forum Foundation. These eight individuals shall include the Secretary, Treasurer, and Immediate Past President of the American Venous Forum. Each elected Director, other than the Secretary and Treasurer, shall serve a staggered term of up to three (3) years and shall be eligible for an additional

reappointment of one (1) three-year term for a maximum of six (6) years of service to the Board.

Article VI – Councilors and Officers

1. The officers of the Forum shall be a President, a President elect, Secretary, Treasurer, and Recorder, all to be elected as provided in the By Laws. Said officers shall serve ex officio as voting members of the Executive Committee.

2. All officers of the Forum, except the Secretary, the Recorder, the Archivist, and the Treasurer, shall be elected for terms of one (1) year each and until their successors are elected and qualified. The President may not serve more than one (1) consecutive term. The Secretary, Recorder and Treasurer will serve three (3) years each and until their successors are elected and qualified. Councilors shall be elected serving overlapping terms of three (3) years each.

3. A Councilor, Archivist, and the officers of the Forum shall be nominated by the Nominating Committee, which shall present the slate to the Executive Committee at its annual meeting and to the members at the annual business meeting. Additional nominations may be made from the floor at the annual business meeting each year. The election shall take place at the executive session.

   Election of officers shall be by a majority of the votes cast. The three candidates for Councilor who receive the most votes shall be elected, provided that each member may vote for three candidates for Councilor and may not cumulate his or her votes.

4. The President shall preside at the meetings of the Forum membership Executive Committee, and Officers, and preserve order, regulate debates, announce results of elections, appoint committees not otherwise provided for in the Bylaws, sign certificates of membership, and perform all other duties normally appertaining to his office.

5. The President elect in the absence or incapacity of the President shall perform the duties of the President's office.

6. In the absence of both the President and the President elect, the position shall be taken by a chairman pro tem, nominated and elected by such members of the Executive Committee as are present.

7. The Secretary shall keep the minutes of the meetings of the Forum, the Executive Committee, and the Officers; attest all official acts requiring certification; notify councilors, officers and members of their election and take charge of all papers not otherwise provided for. The Secretary will be the Chair of the Administrative Council and make appointments as delineated in Article VII. At least ten (10) days but not more than thirty (30) days prior to each annual or special meeting, the Secretary shall issue to all members of the Society a program of the forthcoming meeting. The Secretary shall compile a written report to be read at the annual business meeting of the Forum in which shall be included the list of candidates proposed for membership, as approved by the Executive Committee.

8. The Treasurer shall receive all monies and funds belonging to the Forum to pay all bills; render bills for dues and assessments as soon as possible after the annual meeting; and report to the Executive Committee at each annual meeting the names of all members in arrears as to dues.

9. The Recorder shall receive all papers and reports of discussions on paper presented before the Forum or read by title.

10. The Archivist shall serve for three years and until a successor is elected and qualified. The Archivist shall be nominated by the Nominating Committee.

Article VII – Committees and Councils

1. The activities of the American Venous Forum will be conducted by designated committees under the oversight of four (4) councils, designated the Administrative, Research, Education, and Development Councils.

2. Each council will have a council chair or co-chair determined as follows.

    a. The President of the American Venous Forum will appoint the chair of the Research and Education councils at the time of the annual business meeting. The chair of the Research Council will serve a three (3) year term, and the chair of the Education council will serve a two (2) year term.

    b. The secretary of the Forum will serve as chair of the Administrative Council.

    c. The president and immediate past president of the American Venous Forum Foundation will serve as co-chairs of the Development Council.

3. The Administrative Council will consist of the chairmen of the Bylaws, Membership, Nominating, Program, Issues, and Honorary Membership committees (the Administrative committees), with the secretary of the Forum serving as chairman. The secretary of the forum will serve as an ex-officio member of all committees of the Administrative Council.

    a. The By-Laws Committee shall consist of three members to serve overlapping terms of three (3) years each with the secretary of the Forum serving as Chair. A new member shall be appointed annually by the Administrative Council Chair (secretary of the Forum). They will review the By-Laws from time to time as directed by the Executive Committee.

    b. The Membership Committee shall consist of three (3) members who shall be appointed, one in each year, by the Administrative Council Chair (secretary of the Forum) to serve overlapping terms of three (3) years each, plus the Secretary as an ex officio member. The senior member in terms of service on this committee shall be the chair. The functions of the Committee shall be to pass upon the professional and ethical qualifications of the applicants and to advise the Executive Committee of the recommendations of the Committee.

    c. The Nominating Committee shall consist of the three (3) most recent available Past Presidents and shall be appointed by the President one (1) month before the annual meeting. Its function shall be to comprise a slate of officers, and a member or members of the Membership Committee, to be presented at the annual meeting to the members at the Executive Session. The Senior Member in terms of service on this Committee shall be the Chairman.

d. The Program Committee shall consist of four (4) members who shall be appointed, one in each year, by the Administrative Council Chair (secretary of the Forum) to serve overlapping terms of four (4) years each. The senior member in terms of service on this committee shall be the chairman. The Secretary and Recorder shall be ex officio members of the Program Committee. The function of the Program Committee shall be to solicit papers and other presentations from members and other individuals and to make up the program for the annual meeting.

e. The Issues Committee shall consist of four (4) members who shall be appointed, one in each year, by the Administrative Council Chair (Secretary of the Forum) to serve overlapping terms of four (4) years each. The senior member in terms of service on this committee shall be the chairman. The Secretary shall serve as an Ex-Officio member of this Committee. The primary responsibility of the Committee on Issues will be the monitoring and interpretation of health care related issues. This will include responding in a timely manner to legislative and other issues of importance to the Forum, as well as investigation charges of unethical or unprofessional conduct, including erroneous medico legal testimony, by Forum members. The Committee shall present its observations and recommendations for action to the Executive Committee.

f. The Honorary Membership Committee shall consist of the three (3) most immediate past Presidents on the Executive Committee of the Forum. The most senior member shall serve as Chair. The Committee shall be responsible for reviewing candidates for Honorary Membership status and recommending actions to the Executive Committee.

4. The Research Council will consist of the chairs of the Research, Outcomes, Guidelines, and Grants and Awards committees (the Research committees) under the direction of the Research Council chair. The chair of the Research Council will serve as an ex-officio member of all committees of the Council.

a. The Research Committee will oversee all research activities sanctioned by the American Venous Forum. The responsibilities of this Council shall also include promotion of research in venous diseases; definition of areas of requiring multi-center clinical efforts; and promotion of research investment in venous disease by national granting agencies. The chair of the Research Committee will be appointed by the Research Council Chair of the Forum to serve a two (2) year term. Members of the Research Committee will be appointed by the chair of the Research Committee, and serve a two (2) year term.

b. The Outcomes Committee will be responsible for the creation and maintenance of all outcome measures and reporting standards produced under the auspices of the Forum. The chair of the Outcomes committee will be appointed by the Research Council Chair of the Forum to serve a two (2) year term. The chair of the Outcomes Committee will appoint members of the Outcomes Committee to two (2) year terms.

c. The Practice Guidelines Committee will be responsible for the creation and maintenance of all evidence-based practice guidelines produced under the auspices of the Forum. The chair of the Practice Guidelines committee will be appointed by the Research Council Chair of the Forum to serve a two (2) year term. The chairman of the Outcomes Committee will appoint members of the Practice Guidelines Committee to two (2) year terms.

d. The Grants & Awards Committee will be responsible for the selection of the recipients of all recurring grants and awards administered by the Forum. The Grants & Awards Committee shall consist of three (3) members who shall be appointed, one in each year, by the Research Council Chair to serve overlapping terms of three (3) years each. The senior member in terms of service on this committee shall be the chair.

5. The Education Council will consist of the chairs of the Fellow's Education, Patient Education, Physician/Allied Health Education, Website, and National Venous Screening Program committees (the Education committees) under the direction of the Education Council chair. The chair of the Education Council will serve as an ex-officio member of all committees of the Council.

a. The Fellow's Education Committee will be responsible for all components of resident and fellow's education in venous and lymphatic disease. Responsibilities will include development and maintenance of the fellow's venous curriculum as well as development and oversight of all fellow's courses held under the auspices of the Forum. The Committee shall consist of four (4) members who shall be appointed, one in each year, by the Education Council Chair to serve overlapping terms of four (4) years each. The senior member in terms of service on this committee shall be the chair.

b. The Patient Education Committee will be responsible for the creation, maintenance, and distribution of all laymen's educational materials produced by or under the auspices of the Forum. The chair of the Patient Education committee will be appointed by the Education Council Chair of the Forum to serve a two year term. The chair of the Committee will appoint members of the Patient Education Committee to serve two (2) year terms.

c. The Physician and Allied Health Education Committee will be responsible for the creation, maintenance, and distribution of all professional educational materials produced by or under the auspices of the Forum. The chair of the Physician and Allied Health Education committee will be appointed by the Education Council Chair of the Forum to serve a two (2) year term. The chair of the Committee will appoint members of the Physician and Allied Health Education Committee to serve (2) year terms.

d. The Website Committee will be responsible for maintenance of the Forum's website. The chair of the Website committee functions as webmaster and will be appointed by the Education Council Chair of the Forum to serve a two (2) year term. The chair of the Committee will appoint members of the Website Committee to two (2) year terms.

e. The National Venous Screening Program Committee all activities associated with the screening program. The chair of the Screening Committee will be appointed by the Education Council Chair of the Forum to serve a three (3) year term. The chair of the Committee will appoint members of the Physician and Allied Health Education Committee to three (3) year terms.

6. The Development Council will consist of the chairs of the Fundraising/Strategic Planning, Public and Industrial Relations, and Intersocietal Relations committees (the Development committees) under the direction of the Development Council co-chairs. The chair of the Industrial Advisory Committee will also serve as a council member. The co-chairs of the Development Council will serve as an ex-officio member of all committees of the Council.

   a. The Fundraising/Strategic Planning committee will oversee all long-term fundraising activities of the Forum in conjunction with administrative staff and any outside consultants. The Committee shall consist of the co-chairs of the Development council and their designated appointees.

   b. The Public and Industrial Relations Committee shall consist of three (3) members who shall be appointed, one in each year, by the Co-Chairs of the Development Council to serve overlapping terms of three (3) years each. The senior member in terms of service on this committee shall be the chairs.

   c. The Intersocietal Relations Committee shall consist of three (3) members who shall be appointed, one in each year, by the Co-chairs of the Development Council to serve overlapping terms of three (3) years each. The senior member in terms of service on this committee shall be the chair.

7. The Executive Committee may from time to time establish such other committees as it deems advisable, including committees established to augment and assist the Research, Education and Development Councils Each such committee shall consist of such persons and shall have such duties and powers as may be designated by the Executive Committee upon establishment of the committee or from time to time thereafter. Unless otherwise provided by the Executive Committee, the President shall appoint the members of each such committee or council.

8. Any vacancy occurring among the members of any elected committee of the Forum shall be filled by appointment by the President, the appointee to serve until the next annual meeting of the Forum membership.

9. Members of the Executive Committee, Officers or a Committee may participate in any meeting thereof with a conference telephone or similar communications equipment by means of which all persons participating in the meeting can hear each other, and such participation in a Committee meeting shall constitute presence in person at the meeting.

Article VIII – Meetings

1. The annual business meeting of the Forum shall be held at a time and place to be determined by the Executive Committee.

2. The Executive Committee shall meet in the week prior to the annual meeting, at a time and place designated by the President. The Chair of the Membership Committee, and the Nominating Committee shall meet with the Executive Committee in an advisory capacity.

3. Twenty five (25) voting members present in person shall constitute a quorum at a meeting of the membership.

4. The vote of a majority of members present and voting at a duly called meeting at which a quorum is present shall be necessary for the adoption of any matter voted upon by the members, unless a greater proportion is required by the applicable statute, the Articles of Incorporation, or these Bylaws.

5. Members may not cast their votes by proxy.

6. The executive session of the Forum shall be held at a time and place to be set by the President. The business of the Forum shall be conducted at this time.

7. The scientific sessions at the annual meeting shall consist of presentations of posters and papers and the discussion of these papers.

8. From time to time when deemed advisable by the Executive Committee, eminent investigators in the field of venous disease or allied sciences may be invited to present a special lecture during the annual meeting. This lecture shall be know as the " D. Eugene Strandness, Jr., M.D. Memorial Lecture. Each speaker who presents such a lecture shall receive an appropriate honorarium and a certificate of appreciation from the Forum.

Article IX – Invited Guests

1. Any member of the Forum may invite one or more guests to attend the annual meeting of the Forum.

2. The names of all guests attending the annual meeting shall be entered under a separate heading in the attendance list.

3. All invited guests shall be given the privilege of the floor by the President, but shall not be present at the executive session.

Article X – Fees and Dues

1. Initiation fees and assessments shall be proposed by the Executive Committee and approved by the membership at an annual executive session. The Executive Committee shall set dues for membership in all categories from time to time and publish same to the membership at the annual business meeting.

2. Any member of the Forum in arrears as to dues for one (1) year shall be notified of that fact by the Treasurer, by registered letter, which shall contain a copy of this Section 2. If the dues are not paid before the next annual business meeting or if some reasonable explanation of the delinquency is not forthcoming, the name of the delinquent member shall be presented at that Executive Committee meeting and, on a majority vote of the Executive Committee, the name may be stricken from the membership list. The Executive Committee may reinstate the delinquent member upon his payment of the dues in arrears.

Article XI – Resignations and Discipline

1. Resignations of members not in arrears as to dues may be accepted at any annual executive committee meeting by a majority vote of the members present.

2. Charges of unprofessional or unethical conduct may be brought against any member of the Forum by written complaint signed a member of the Forum and delivered to the Secretary. The Issues Committee will investigate said complaints and present them to the Executive Committee. The rules governing disciplinary proceedings based upon such charges shall be as established from time to time by the Executive Committee.

Article XII – Papers and Reports

1. All papers and reports read before the Forum shall be delivered to the Recorder at the time of their presentations and submitted online as directed by the Recorder.

2. No paper shall be published as having been read before the Forum unless it has been read by title or otherwise before the Forum.

Article XIII – Procedure

The proceedings of the Forum shall be conducted under Robert's Rules of Order Newly Revised and as amended from time to time.

Article XIV – Certificate of Membership

Every elected member of the Forum shall be entitled to a certificate of membership signed by the President and Secretary.

Article XV – Fiscal Year

The fiscal year of this corporation shall begin on the first of January in each year and shall run through the 31st day of December in that year.

Article XVI – Notice and Waiver of Notice

1. Whenever under applicable law, these By laws, or a resolution of the Executive Committee, notice is required to be given to any member, Executive Committee member or officer, such notice may be given in writing, by mail, addressed to such member, Executive Committee member or officer at his or her address as it appears on the records of the Forum. Such mailed notice shall be deemed to have been given when deposited in the United States mail in a sealed envelope so addressed, with postage thereon prepaid.

2. Whenever, under applicable law, these By laws or a resolution of the Executive Committee, any notice is required to be given, a waiver thereof in writing, signed by the person or persons entitled to such notice, whether before or after the time stated therein, shall be deemed equivalent to the giving of such notice. In addition, the attendance of a member or Executive Committee member at any meeting shall constitute a waiver of notice of such meeting, except where an individual attends the meeting for the express purpose of objecting to the transaction of any business because the meeting is not lawfully called or convened.

Article XVII – Indemnification

1. To the full extent specifically authorized by, and in accordance with the procedures prescribed in Section 108.75 of the Illinois General Not for Profit Corporation Act of 1986 (or the corresponding provisions of any future statute applicable to corporations organized under the Act), the Forum shall indemnify any and all members of the Executive Committee (which members shall hereinafter in this

Article be referred to as "Directors") and any and all of its officers, committee members, employees, agents and other authorized representatives for expenses and other amounts paid in connection with legal proceedings (whether threatened, pending or completed) in which any such person became involved by reason of serving in any such capacity for the Forum.

2. Upon specific authorization by the Executive Committee, the Forum may purchase and maintain insurance on behalf of any or all directors, officers, employees, agents or representatives of the Forum against any liability asserted against any such person and incurred in any such capacity, or arising out of the status of serving in any such capacity, whether or not the Forum would have the power to indemnify them against such liability under the provisions of Section I of this Article.

Article XVIII – Amendment

These By laws may be amended by a three fourths vote of the members present and voting at a properly called and convened of an annual business meeting or special meeting of the Forum provided that the proposed amendment has been submitted to the Secretary by at least three (3) voting members of the Forum at least three (3) months prior to the executive session of the Forum. The Secretary shall mail the proposed amendment to all voting members at least thirty (30) days prior to the executive session, accompanied by notice that such amendment will be acted upon at that business meeting

PROVISO TO THE BY LAWS

Article I

Effect of Proviso

This Proviso to the By laws (the "By laws") of the American Venous Forum, an Illinois not for profit corporation (the "Forum"), shall control and supersede the rules and regulations for the governance of the Forum contained in the By laws as of the date on which they are adopted. Except as specifically modified by this Proviso, all other provisions of the By laws shall remain in full force and effect.

Article II

Officers

The initial members of the Executive Committee of the Forum, which members are named in the Articles of Incorporation of the Forum as filed with the Illinois Secretary of State on February 7, 1989 shall elect the initial officers of the Forum from among the members of the Executive Committee. The officers so elected shall serve until the next annual executive session of the members of the Forum and until their successors shall have been elected and qualified.

DRAFTED: October 23, 1988
ADOPTED: February 22, 1989
AMENDED: February 19, 1999
AMENDED: February 16, 2007
AMENDED: February 22, 2008

AMERICAN VENOUS FORUM

Authors Index

IS YOUR AVF MEMBERSHIP INFORMATION CURRENT?

For Example:

- Do you have a new email address?
- Do you have a new address or phone number?

Please let us know so that your AVF records stay current and that all important updates and news reach you!

PLEASE PRINT

First M Last Suffix

Email Address

Daytime Phone Fax

MAILING ADDRESS

Institution

Street

City State Zip Country

Please return your completed form to the AVF Registration Desk, or fax your form to 978-927-7872.