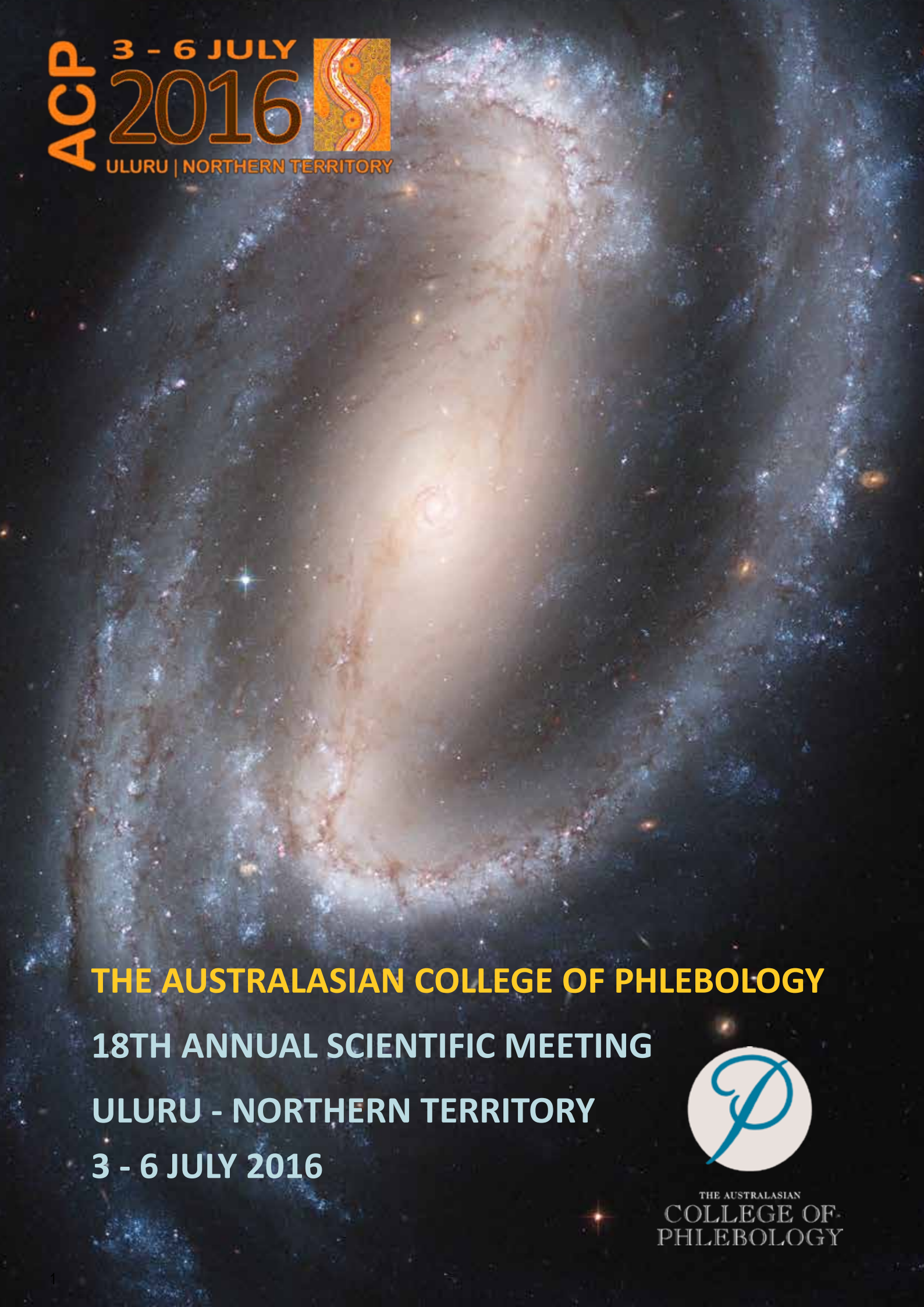


# THE AUSTRALASIAN COLLEGE OF PHLEBOLOGY

## 18TH ANNUAL SCIENTIFIC MEETING

## ULURU - NORTHERN TERRITORY

## 3 - 6 JULY 2016

THE AUSTRALASIAN
COLLEGE OF
PHLEBOLOGY

## VENUE AND CONTACTS

### Venue

**Voyages Sails in the Desert**
170 Yulara Dr, Yulara NT 0872
Phone: +61 2 8296 8010
Fax: +61 2 9299 2103
Web: www.ayersrockresort.com.au

**Organiser**

**Australasian College of Phlebology**
Level 5, 7 Help Street
Chatswood, NSW 2067
PO Box 705, Chatswood, NSW 2057
Phone: +61 2 9386 1811
Email: acpasm@phlebology.com.au
Web address: www.phlebology.com.au

Zivka Nicholls
Executive Manager
acp@phlebology.com.au

Sepand Asadolahi
Events & Marketing Coordinator
events@phlebology.com.au

**Registration and Hotel Accommodation**
www.phlebology.com.au/acp2016
Phone: +61 2 9386 1811
Email: acpasm@phlebology.com.au

**Exhibition and Sponsorship**
Request from sails opportunities or question by industry partners should be directed to:

Sepand Asadolahi
Phone: +61 2 9386 1811
events@phlebology.com.au

Zivka Nicholls
Phone: +61 2 9386 1811
acp@phlebology.com.au

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## WELCOME

Dear Colleagues and friends,

It is my great privilege and pleasure to invite you to Uluru in the Northern Territory, for the 18th Annual Scientific Meeting (ASM) of the Australasian College of Phlebology (ACP).

Uluru is the spiritual capital of Australia and we are quite grateful to be hosted at the beautiful Sails in the Desert resort. We are especially excited as this year we will hold our Welcome Party under the stars to listen to the Sounds of Silence and watch the Milky Way.

We will be thrilled to host our Emeritus fellow and friend, Dr Louis Grondin from Canada. The Ayers Rock, the Milky Way, the Orion and other stars will provide the perfect backdrop for Dr Grondin to take us through an inter-stellar journey through the space-time continuum.

The scientific meeting will be exciting and different with emphasis on new technologies, vascular dermatology and vascular medicine where speakers will talk freely on topics of interest to the audience.

We chose the meeting dates to coincide with NSW school holidays and overlapping with Victorian school holidays hoping that families will take this opportunity to show their children the heart of Australia and use this meeting as an opportunity to have some adventure.

Hope you will join us in Uluru.

A/Prof. Kurosh Parsi

President

Australasian College of Phlebology

ACP 3 - 6 JULY 2016 ULURU | NORTHERN TERRITORY

## ABOUT THE ACP

The Australasian College of Phlebology (ACP) is a multi-specialty organisation dedicated to promotion of phlebology research, teaching and training in Australasia.

Our membership includes medical practitioners and other health professionals such as scientists and sonographers dedicated to education and research in the field of phlebology. Our members have a shared interest in phlebology, but represent a variety of medical specialties, including vascular surgery and medicine, dermatology, haematology, interventional radiology, general surgery, and family medicine. Since its inception in 1993, the ACP has been active in promoting education and research in phlebology and serves the general public as a resource regarding vein disorders. Public educational initiatives such as patient education seminars, GP education workshops and media interviews are undertaken by College Fellows on a regular basis. Our mission is to improve the standards of practice and patient care as it relates to venous disorders.

The ACP is a member of the Union International de Phlébologie (UIP), a multi-national organisation that has phlebology society members from 35 countries from Europe, North America, Latin America, and Asia.

Annual Scientific Meetings and Workshops of ACP were initiated in 1994. These meetings have been instrumental in disseminating knowledge and experience among specialists from many medical disciplines.

THE AUSTRALASIAN COLLEGE OF PHLEBOLOGY

## Academic Board

**Chancellor**
Prof. Andre van Rij

**Emeritus Chancellor**
Prof. Ken Myers

**Deputy Chancellor**
Prof. Lourens Bester

## Executive Board

**President**
A/Prof. Kurosh Parsi

**Vice President**
A/Prof. David McClure

**Honorary Secretary**
Dr Louis Loizou

**Treasurer**
Dr Paul Thibault

## Board Members

Dr Stephen Benson
Dr Ivor Berman
Dr Elisabeth De Felice
Dr David Huber
Dr Chris Lekich
Dr Adrian Lim
Dr Peter Paraskevas
Dr Stefania Roberts

## ACP Faculties

**NSW**
Dr David Jenkins (Chair & DOT)
Dr Simon Thibault (Secretary)

**QLD**
Dr Paul Dinnen (Chair & DOT)
Dr Stuart McMaster (Secretary)

**SA**
Dr Anne Padbury (Chair & DOT)

**VIC**
Dr Stefania Roberts (Chair & DOT)
Dr Ivor Berman (Secretary)

**NZ**
Dr Stephen Benson (Chair & DOT)
Dr Elisabeth De Felice (Secretary)

**TAS**
Dr Asha Ram (Chair & DOT)
Dr Andrew Stirling (Secretary)

## ACP Committees

**AMC Accreditation Taskforce**
A/Prof. Kurosh Parsi (Chair)
Prof. Alun Davies
Dr Chris Lekich
Dr Adrian Lim
Dr Louis Loizou
A/Prof. David McClure
Prof. Ken Myers
Dr Peter Paraskevas
Dr Hugo Partsch
Prof. Ron Penny
Dr AA Ramelet
Prof. Andre van Rij
A/Prof. Stephen Shumack
Dr Paul Thibault
Dr Steven Zimmet

**Board of Censors**
Dr Simon Thibault (Chair)
Dr Ivor Berman
Dr Paul Hannah
Dr David Jenkins
A/Prof . David McClure
Dr Peter Paraskevas

**Board of Training**
Dr Adrian Lim (NDOT)
Dr David Jenkins (NSW)
Dr Stephen Benson (NZ)
Dr Anne Padbury (SA)
Dr Asha Ram (TAS)

**Code of Conduct and Ethics**
Dr Louis Loizou (Chair)
Dr Chris Lekich

**Continuing Professional Development**
Dr Peter Paraskevas (Chair)

**Finance and Fundraising**
Dr Paul Thibault (Chair)

**MBS and Private Health Insurance**
A/Prof. David McClure (Chair)
Dr John Barrett
Dr Paul Dinnen
Dr David Huber
Dr Chris Lekich
Dr Louis Loizou
Dr Peter Paraskevas
Prof. Ken Myers

**Scientific Meetings**
A/Prof Kurosh Parsi (Chair)
Dr David Connor

**Social Media**
Dr Asha Ram (Chair)

**Workshops**
Dr Chris Lekich
Ms Jenny Lekich
Dr Stefania Roberts

**Administration**
Zivka Nicholls
Executive Manager
acp@phlebology.com.au

Sepand Asadolahi
Events & Marketing Coordinator
events@phlebology.com.au

## ORGANISING COMMITTEE

**Convenor:**
A/Prof. Kurosh Parsi (Chair)

**Scientific Program:**
A/Prof. Kurosh Parsi, Dr David Connor, Dr Stefania Roberts

**Compression Workshop:**
Prof. Neil Piller

**Promotion and Sponsorship:**
Mr. Sepand Asadolahi, Dr. Stefania Roberts, Mrs. Yana Parsi, Ms. Stephanie Anderson

### Keynote Speakers - International

Dr. Afsaneh Alavi, Dermatologist, Canada
Prof. Pier Luigi Antignani, Phlebologist and Vascular Physician, Italy
Dr. Louis Grondin, Phlebologist, Canada
Prof. Mark Meissner, Phlebologist and Vascular Surgeon, USA
Dr. Angelo Scuderi, Phlebologist and Vascular Surgeon, Brazil

### Keynote Speakers - Australia & New Zealand

Prof. Andre van Rij, Phlebologist and Vascular Surgeon, New Zealand
Prof. Steven Krilis, Immunologist, Australia
Dr. John Vrazas, Interventional Radiologist, Australia
Prof. Harshal Nandurkar, Haematologist, Australia
Prof. Neil Piller, Lymphologist, Australia

### Local Faculty

Dr. David Conor (NSW)
Dr. Paul Dinnen (QLD)
Dr. Mark Elvy (NSW)
Dr. Richard Harris (NSW)
Dr. Keturah Hoffman (WA)
Dr. David Jenkins (NSW)
Ms. Jenny Lekich (QLD)
Dr. Adrian Lim (NSW)
Dr. Ewan Macaulay (SA)
Dr. Sanjay Nadkarni (WA)
Dr. Anne Padbury (SA)
A/Prof. Kurosh Parsi (NSW)
Dr. Stefania Roberts (VIC)
Dr. Paul Thibault (NSW)
Dr. Simon Thibault (NSW)
Dr. Ramon Varcoe (NSW)

## PROGRAM AT A GLANCE

| | SUNDAY 3 JULY | MONDAY 4 JULY | TUESDAY 5 JULY | WEDNESDAY 6 JULY |
|---|---|---|---|---|
| 08:00 | | Superficial Venous Disease: New Technologies | Debates and Controversies | Global Sclerotherapy |
| 08:30 | Practice Matters in Phlebology I | | | |
| 09:00 | | | Vascular Malformations | |
| 09:30 | | | | |
| 10:00 | **Morning Tea** | | | |
| 10:30 | Practice Matters in Phlebology II | Antiphospholipid Syndrome | Pelvic and Vulvar Veins | Chronic Venous Disease |
| 11:00 | | | | |
| 11:30 | | | | |
| 12:00 | | | | |
| 12:30 | **Lunch** | | | |
| 13:00 | | | | |
| 13:30 | Vascular Lasers | Venous Thromboembolism | Guided Poster Presentations | Venous Syndromes: Update in Diagnosis and Management |
| 14:00 | | | | |
| 14:30 | | | | |
| 15:00 | | | **Afternoon Tea** | |
| 15:30 | **Afternoon Tea** | **Afternoon Tea** | | **Afternoon Tea** |
| 16:00 | | | | Compression, Oedema and Lymphoedema |
| 16:30 | | | | |
| 17:00 | Optional Tour: Field of Light or Kata Tjuta | Welcome Reception Sounds of Silence | | |
| 17:30 | | | | |
| 18:00 | | | Conferring Ceremony & Gala Dinner | Pressure and Compression Workshop - EXHIBITION SPACE |
| 18:30 | | | | Closing Ceremony and Presentation of Awards |
| 19:00 | | | | |
| 19:30 | | | | Invited Speakers Dinner |
| 20:00 | | | | |
| 20:30 | | | | |
| 21:00 | | | | |
| 23:00 | | | | |

| | | | | |
|---|---|---|---|---|
| **Registration Hours** | 7.30am - 4.00pm | 7.00am - 4.00pm | 7.00am - 4.00pm | 7.00am - 4.00pm |
| **Exhibition Hours** | | 7.00am - 4.00pm | 7.00am - 4.00pm | 7.00am - 4.00pm |
| **Poster Viewing** | | 8.00am - 4.00pm | 8.00am - 4.00pm | 8.00am - 4.00pm |

## KEYNOTE SPEAKERS - INTERNATIONAL

**Dr. Afsaneh Alavi (Canada)**

**Dermatologist**

**President - elect, Canadian Association Wound Care**

Dr. Alavi is currently the director of the wound healing fellowship at University of Toronto-Women's College Hospital. She serves on the editorial boards for a number of journals in wound healing and dermatology. She is a board member and president elect of the Canadian Association of Wound Care.

**Prof. Pier Luigi Antignani (Italy)**

**Phlebologist and Vascular Physician**

**General Secretary, Union Internationale de Phlébologie (UIP)**

Professor of Angiology, as well as Director of Vascular Centre in Nuova Villa Claudia, Rome, Italy. He is President of Italian Society for Vascular Investigation, Vice-President of IUA and General Secretary of UIP.

**Dr. Louis Grondin (Canada)**

**Phlebologist**

**Past President, Canadian Society of Phlebology**

Echosclerotherapy and Ultrasound Cartography was performed for the first time in Canada at his clinic. He went on to pioneer the ultrasound guided catheter injection for difficult to reach varicose veins. Today, his technique is practiced worldwide.

**Prof. Mark Meissner (USA)**
**Phlebologist and Vascular Surgeon**
**Vice President, Union Internationale de Phlébologie (UIP)**
Prof. Meissner is a UW professor of surgery specializing in vascular surgery, with particular clinical and research interests in deep venous thrombosis, chronic venous disease, vascular trauma and abdominal aortic aneurysms.

**Dr. Angelo Scuderi (Brazil)**
**Phlebologist and Vascular Surgeon**
**Immediate Past President, Union Internationale de Phlébologie (UIP)**

Dr. Scuderi trained in general surgery and specialised in vascular surgery. He is the author of many publications in Portuguese, Spanish, Italian and English about Phlebology. He is the immediate past president of the International Union of Phlebologie (UIP).

## KEYNOTE SPEAKERS - AUSTRALIA & NEW ZEALAND

**Prof. Andre van Rij (NZ)**
**Phlebologist and Vascular Surgeon, New Zealand**
**Chancellor, Australasian College of Phlebology (ACP)**

Prof. Andre van Rij is Professor of Surgery at the Dunedin School of Medicine, University of Otago where he directs the Vascular Research Unit. His research has focused on venous disease and the biology of varicose vein recurrence and venous thrombosis. His translational research bridges new basic research into the venous clinic. Professor van Rij is a vascular surgeon and President of the NZ Association of General Surgeons. Professor van Rij is the Chancellor of the Australasian College of Phlebology.

## KEYNOTE SPEAKERS - AUSTRALIA & NEW ZEALAND

**Prof. Steven Krilis (NSW)**
**Director, Department of Infectious Disease, Immunology and Sexual Health at St. George Hospital, University of New South Wales , Australia**

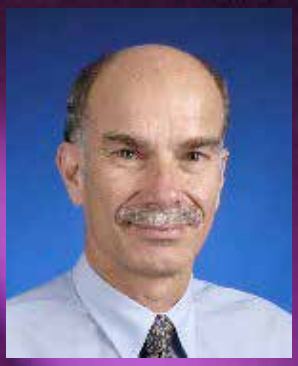
Prof. Krilis graduated MBBS (Hons) UNSW and received his PhD from the University of Sydney in 1984. He did postdoctoral work at the Harvard Medical School 1981 to 1984 funded by an Applied Science Fellowship from NH&MRC and a National Institute of Health Fogarty International postdoctoral research fellowship to study with Professor K. Frank Austen. He has received a number of international awards including a UICC Senior Fellowship, a Bob Pitney Award, AAAAI Training Program Directors Retreat award, Distinguished Alumni Award in Science and Engineering University of New South Wales, Honoris Causa Doctorate University of Athens, Greece and Distinguished Scientist Fellowship Award from the Japanese Society for Promotion of Science. He has had numerous visiting professorships in major universities including the University of Hokkaido, Sapporo, Japan, Harvard Medical School, Boston, USA, University of Athens, Athens, Greece, University of Tianjin, Tianjin, China, University of Istanbul, Turkey, Stanford University, California, USA and was a recipient of the Israel Academy of Science's Batsheva de Rothschild Fellowship.

**Dr. John Vrazas (VIC)**
**Interventional Radiologist**
**President, Interventional Radiology Society of Australasia (IRSA)**

Dr. Vrazas became head of the Department of Radiology and Director of Cardiovascular and Interventional Radiology at Western Hospital in 1999. In 2000 he moved to St Vincents Hospital. In 2002, he started the first vascular neuro-interventional procedures at St vincents Hospital, and following this, completed a sabbatical/mini fellowship ininterventional neuroradiology in Canada, and Switzerland. In 2006, he founded the first dedicated private interventional radiology practice in Australia, Melbourne Institute of Vascular and Interventional Radiology, and in 2014, he was instrumental in forming the first group IR practice, servicing one of the largest private hospital networks in Australia. In 2007, he founded the Paediatric Interventional Radiology section at the Royal Childrens Hospital, Melbourne and is the Senior IR, and holds co-appointments with the Departments of Transplantation and Hepatology, and the Department of Vascular Biology.

**Prof. Harshal Nandurkar (VIC)**
**President, Australasian Society of Thrombosis and Haemostasis (ASTH)**
**Vice President, International Union of Angiology (Australian Chapter)**
**Head, Australian Centre for Blood Diseases, Monash University**
**Director, Clinical Haematology, Alfred Health**

Prof. Harshal Nandurkar graduated in medicine from the University of Bombay, India and specialized in clinical and laboratory haematology at Westmead Hospital (Sydney). His research training included a PhD at the Walter and Eliza Hall Institute in the area of haemopoietic growth factors and postdoctoral research at Monash University, Department of Biochemistry was in the area of phosphoinositide signalling. His research interests include the development of new anticoagulants targeted to platelets or endothelium, identification of pathways that regulate haemostasis and understanding molecular mechanisms in antiphospholipid syndrome. Harshal's clinical service cover all areas of haematology, including blood malignancies, haemostasis-thrombosis and haemophilia.



**Prof. Neil Piller (SA)**
**Lymphologist**
**Director, Lymphoedema Clinical Research Unit in the Department of Surgery, School of Medicine at Flinders University, South Australia**
**Coordinator, Advanced Studies in the MD program**

Prof. Neil is Co-Director of the International Lymphoedema Framework based in the UK a member of the International Society for Lymphology, Clinical Sciences Editor for the "Journal of Lymphoedema" (UK), Australasian Editor of "Lymphatic Research and Biology" (USA). and a member of the Editorial Boards of "Phlebology" (USA) and "Lymphology" (USA)

Neil is a member International Advisory Board Union Internationale Phlebologie and a member of its consensus groups on Lymphoedema and phlebo-lymphoedema, a member of the steering committees for the Australian and American Lymphoedema Frameworks and the International Compression Club.

THE AUSTRALASIAN
COLLEGE OF
PHLEBOLOGY

# SCIENTIFIC PROGRAM

# SCIENTIFIC PROGRAM

## 18TH ANNUAL SCIENTIFIC MEETING OF THE AUSTRALASIAN COLLEGE OF PHLEBOLOGY

### Ayers Rock Resort, Uluru, NT, Australia

| | |
|---|---|
| **SUNDAY 3RD JULY, 2016**<br>**Pre-Conference Course** (included in registration)<br>**Lungkata Room** | |
| **Practice Matters in Phlebology I**<br>Chair: A/Prof. Kurosh Parsi (NSW)<br>Moderator: Dr Paul Hannah (QLD) | |
| **8:30** | **Employee Claims and Contracts: How to Avoid Appearing at the Fair Work Commission (80/20 rule)**<br>Stephanie Anderson (NSW) |
| **9:00** | **Employee Fraud: How to Detect and How to Prevent It**<br>Rebecca Fleming, Gow Gates (NSW) |
| **9:30** | **Panel Discussion** |
| **10:00** | **Morning Tea** |
| **Practice Matters in Phlebology II**<br>Chair: Jenny Lekich (QLD)<br>Moderator: Stephanie Anderson (NSW) | |
| **10:30** | **Accounting 101: How to be more Profitable by Understanding your Business Finances**<br>Andrew Ramsay (NSW) |
| **11:00** | **Refunds / Reimbursements and Discounts: Should we pay back?**<br>Jenny Lekich (QLD) |
| **11:30** | **Financing Your Practice**<br>Tim Wilson, ANZ Mobile (NSW) |
| **12:00** | **Panel Discussion** |
| **12:30** | **Lunch** |
| **Vascular Lasers**<br>Chair: Dr Stefania Roberts (VIC)<br>Moderator: Dr Anne Padbury (SA) | |
| **13:30** | **REVIEW LECTURE**<br>**Overview of Vascular Lasers**<br>Dr Stefania Roberts (VIC) |
| **14:00** | **Long Pulse Nd-YAG and Pulsed-Dye Lasers**<br>Dr Anne Padbury (SA) |
| **14:15** | **Q-switched Nd-YAG Laser to Treat Pigmentation Post-Sclerotherapy**<br>Dr Sanjay Nadkarni (WA) |
| **14:30** | **The new Yellow Laser**<br>A/Prof. Kurosh Parsi (NSW) |
| **14:45** | **Fractional and New Dermatological Lasers**<br>Dr Adrian Lim (NSW) |
| **15:00** | **Panel Discussion** |
| **15:30** | **Afternoon Tea** |
| **17:00** | **Optional Tour** - Visit the Rock! (Registration Required)<br>Choose from: Field of Light or Kata Tjuta<br>Coaches Leave the Resort at 17:00 |

## MONDAY 4TH JULY, 2016
## Uluru Meeting Place, Tjungu Room

| | |
|---|---|
| 7:55 | **Welcome to the Northern Territory**<br>A/Prof. Kurosh Parsi (President, Australasian College of Phlebology) |
| **Superficial Venous Disease: New Technologies**<br>Chair: Dr Paul Thibault (NSW)<br>Moderator: Dr David Jenkins (NSW) | |
| | REVIEW LECTURE |
| 8:00 | **Endovenous Modalities in 2016: Chemical Ablation, Endothermal Destruction and Adhesive Closure - What is the Evidence, How to Choose.**<br>Dr Stefania Roberts (VIC) |
| 8:30 | **Laser Beam Travelling Through Foam: What happens next?**<br>Dr Louis Grondin (Canada) |
| 8:45 | **A Large, Single-Centre Experience with Cyanoacrylate Adhesive Glue Embolisation for Saphenous Vein Insufficiency**<br>Dr Ramon Varcoe (NSW) |
| 9:00 | **Preliminary Experience with Adhesive Closure**<br>Dr Stefania Roberts (VIC) |
| 9:15 | **Trivex in Post-embolisation Management of Vascular Anomalies**<br>A/Prof. Kurosh Parsi (NSW) |
| 9:30 | **Panel Discussion** |
| 10:00 | **Morning Tea** |
| **Antiphospholipid Syndrome**<br>Chair: Prof. Steven Krilis (NSW)<br>Moderator: A/Prof. Kurosh Parsi (NSW) | |
| | KEYNOTE LECTURE |
| 10:30 | **Antiphospholipid Sydnrome: State-of-the-Art**<br>Prof. Steven Krilis (NSW) |
| 11:15 | **Antiphospholipid Sydnrome: Advances in Pathophysiology**<br>Prof. Harshal Nandurkar (VIC) |
| 11:30 | **Antiphospholipid Syndrome: Cutaneous Manifestations**<br>A/Prof. Kurosh Parsi (NSW) |
| 11:45 | **Livedo Vasculopathy**<br>Dr Afsaneh Alavi (Canada) |
| 12:00 | **Antiphospholipid Sydnrome: Looking beyond conventional tests**<br>Prof. Harshal Nandurkar (VIC) |
| 12:15 | **Panel Discussion** |
| 12:30 | **Lunch** |

## MONDAY 4TH JULY, 2016
## Uluru Meeting Place, Tjungu Room

**Venous Thromboembolism**
Chair: Dr Paul Dinnen (QLD)
Co-Chair: Prof. Harshal Nandurkar (VIC)
Moderator: Dr David Connor (NSW)

| | |
|---|---|
| 13:30 | **Cases in Venous Thrombosis: How would you manage them?**<br>Dr Paul Dinnen (QLD)<br>**Survey of the Audience - Pre**<br>Dr David Conor (NSW) |
| 13:45 | **Balancing the Risks and Benefits of Anticoagulation**<br>Prof. Mark Meissner (USA) |
| 14:00 | **DOACS: Simplifying VTE Management**<br>Prof. Harshal Nandurkar (VIC) |
| 14:15 | **Advanced Techniques in Iliac Venous Recanalisation**<br>Prof. Mark Meissner (USA) |
| 14:30 | **Interventional Techniques for Severe Pulmonary Embolism**<br>Dr Ramon Varcoe (NSW) |
| 14:45 | **Atypical Superficial Venous Thrombosis**<br>Prof. Pier Luigi Antignani (Italy) |
| 15:00 | **Survey of the Audience-Post**<br>Dr David Connor (NSW) |
| 15:15 | **Panel Discussion** |
| 15:30 | **Afternoon Tea** |

**Free Afternoon**

**17:00**

### Welcome Reception

**Phlebology Under the Stars**
**Buffet Dinner - Coaches leave the Resort at 17:15**
**Dress Code: Smart Casual**

## TUESDAY 5TH JULY, 2016
## Uluru Meeting Place, Tjungu Room

| | |
|---|---|
| **Debates and Controversies**<br>Chair: Prof. Mark Meissner (USA)<br>Co-Chair: Dr Ewan Macaulay (SA)<br>Moderator: Dr David Connor | |
| **8:00** | **Debate: Use of Ionising Radiation with Radiofrequency and Endovenous Laser Ablation**<br>**Brief Introduction and Survey of the Audience- Pre**<br>Dr David Connor (NSW) |
| **8:10** | **IRSA Position on the use of Ionising Radiation in Routine EVLA and RF: NO**<br>Dr John Vrazas (President, Interventional Radiology Society of Australia) |
| **8:20** | **Use of Ionising Radiation during endovenous ablation: YES**<br>Dr Ramon Varcoe (NSW) |
| **8:30** | **Panel Discussion** |
| **8:40** | **Survey of the Audience- Post**<br>**Dr David Connor (NSW)** |
| **Vascular Malformations**<br>Chair: Prof. Pier Luigi Antignani (Italy)<br>Moderator: Dr Afsaneh Alavi (Canada) | |
| **8:45** | **REVIEW LECTURE** |
| | **Overview of Vascular Anomalies: Clinical and Ultrasound Diagnosis**<br>A/Prof. Kurosh Parsi (NSW) |
| **9:15** | **ISVI-IUA Consensus. Diagnostic Guidelines of Vascular Anomalies: Vascular Malformations and Haemangiomas**<br>Prof. Pier Luigi Antignani (Italy) |
| **9:30** | **Embolisation of Arteriovenous Malformations**<br>Dr John Vrazas (VIC) |
| **9:45** | **Panel Discussion** |
| **10:00** | **Morning Tea** |
| **Pelvic and Vulvar Veins**<br>Chair: Dr Louis Grondin (Canada)<br>Moderator: Dr Richard Harris (NSW) | |
| **10:30** | **REVIEW LECTURE** |
| | **Pelvic and Vulvar Veins - Anatomy and an Approach to Treatment**<br>Dr Louis Grondin (Canada) |
| **11:00** | **KEYNOTE LECTURE** |
| | **Management of Pelvic Congestion Syndrome – Sense and Nonsense**<br>Prof. Mark Meissner (USA) |
| **11:30** | **Embolisation of Ovarian Veins**<br>Dr John Vrazas (VIC) |
| **11:45** | **Embolic Coils for Ovarian Veins: Which ones to Choose?**<br>Dr Sanjay Nadkarni (WA) |
| **12:00** | **Panel Discussion** |
| **12:30** | **Lunch** |

## TUESDAY 5TH JULY, 2016
## Uluru Meeting Place, Tjungu Room

**Guided Poster Presentations - EXHIBITION SPACE**
5 Minute Presentation, 5 Minute Questions
Judges: Prof. Andre van Rij (NZ), Dr Louis Grondin (Canada), Dr David Connor (NSW)

| | |
|---|---|
| **13:30** | **Apixaban versus enoxaparin in the prevention of venous thromboembolism following total knee arthroplasty: A single centre, single surgeon, retrospective analysis**<br>Dr Danika King (NSW) |
| **13:40** | **Onyx Embolisation of a Digital AVM**<br>Dr. Anthony Trimboli (NSW) |
| **13:50** | **Aggressive percutaneous pharmacomechanical thrombolysis for extensive proximal lower and upper extremity deep vein thrombosis with Angiojet; safety and feasibility – a case series.**<br>Dr Farshid Niknam (NSW) |
| **14:00** | **Retrieval of IVC and mechanical thrombectomy with catheter directed thrombolysis and repair of rupture IVC**<br>Dr Gagandeep Kaur (NSW) |
| **14:10** | **Livedo Racemosa secondary to cutaneous microcalcifications: A diagnostic challenge**<br>Dr Pooja Kadam (NSW) |
| **14:20** | **Detergent Sclerosants Induce Cellular Apoptosis**<br>Dr Osvaldo Cooley-Andrade (NSW) |
| **14:30** | **Venous Thromboembolism**<br>Dr George Nicola (NSW) |
| **15:00** | **Afternoon Tea** |

**Free Afternoon**

**18:00**

## Conferring Ceremony and Gala Dinner

**Welcome Speech** – Chief Petty Officer Neil Anderson
**Presentation of Awards and Certificates** – Prof. Andre van Rij
**Humanitarian Missions in Phlebology** – Dr Stefania Roberts
**Ken Myers Oration: Creativity in research-inspiration or perspiration** – Prof. Steven Krilis

THE AUSTRALASIAN COLLEGE OF PHLEBOLOGY

## WEDNESDAY 6TH JULY, 2016
## Uluru Meeting Place, Tjungu Room

**Global Sclerotherapy**
Chair: Dr Angelo Scuderi (Brasil)
Moderator: Dr Simon Thibault (NSW)

| | |
|---|---|
| 8:00 | **Sclerotherapy in Australia and New Zealand- what has been our contribution?**<br>Dr Paul Thibault (NSW) |
| 8:15 | **Glycerin and Foam Stabilisers: How should we use them?**<br>Dr Louis Grondin (Canada) |
| 8:30 | **Brazil- 75% Dextrose: Is it a good sclerosant?**<br>Dr Angelo Scuderi (Brazil) |
| 8:45 | **Sclerotherapy in Argentina**<br>Dr Miguel Huaman (Argentina) |
| 9:00 | **Impact of foam sclerotherapy upon respiratory system and central hemodynamics in an animal model**<br>Prof. Zbigniew Rybak (Poland) |
| 9:15 | **Microscopic Examination of Sclerocoagulum: What is Trapped Blood**<br>Dr Osvaldo Cooley-Andrade (NSW) |
| 9:30 | **Panel Discussion** |
| 10:00 | **Morning Tea** |

**Chronic Venous Disease**
Chair: Dr Adrian Lim (NSW)
Moderator: Dr Stefania Roberts (VIC)

| | |
|---|---|
| 10:30 | **KEYNOTE LECTURE**<br>**Update on Genetic Markers of Varicose Veins and Venous Insufficiency**<br>Prof. Andre van Rij (NZ) |
| 11:00 | **An Approach to Diagnosis and Management of Panniculitis**<br>Dr Afsaneh Alavi (Ontario, Canada) |
| 11:15 | **Non-invasive Evaluation of Vascular Leg Ulcers**<br>Prof. Pier Luigi Antignani (Italy) |
| 11:30 | **Approach to Atypical Wounds**<br>Dr Afsaneh Alavi (Canada) |
| 11:45 | **Foam Sclerotherapy in Elderly Patients with Severe CVD**<br>Prof. Pier Luigi Antignani (Italy) |
| 12:00 | **Panel Discussion** |
| 12:30 | **Lunch** |

## WEDNESDAY 6TH JULY, 2016
## Uluru Meeting Place, Tjungu Room

**Venous Syndromes: Update in Diagnosis and Management**
Chair: Prof. Andre van Rij (NZ)
Moderator: Dr Afsaneh Alavi (Canada)

| | |
|---|---|
| 13:30 | **The Nutcracker Syndrome – Laparotomy or Stent**<br>Prof. Mark Meissner (USA) |
| 13:45 | **Restless Leg Syndrome - Does it Really Exist?**<br>Prof. Pier Luigi Antignani (Italy) |
| 14:00 | **Popliteal Compression Syndrome**<br>Dr Richard Harris (NSW) |
| 14:15 | **May-Thurner Syndrome**<br>Prof. Mark Meissner (USA) |
| 14:30 | **Stewart-Blufarb Syndrome**<br>A/Prof. Kurosh Parsi (NSW) |
| 14:45 | **Chronic Cerebrospinal Venous Insufficiency (CCSVI)**<br>Dr Paul Thibault (NSW) |
| 15:00 | **Raynaud's Syndrome: Instrumental Approach**<br>Prof. Pier Luigi Antignani (Italy) |
| 15:15 | **Questions** |
| 15:30 | **Afternoon Tea** |

**Compression, Oedema and Lymphoedema**
Chair: Prof. Neil Piller (SA)
Moderator: Dr Mark Elvy (NSW)

| | |
|---|---|
| 16:00 | **An Approach to Patient with Oedema: Drugs, Foods and Reversible Causes**<br>Dr Keturah Hoffman (WA) |
| 16:15 | **A Review of Medical and Commercial Compression Stockings: A Female Perspective**<br>Dr Stefania Roberts (VIC) |
| 16:30 | **Why we may not always get good outcomes: The importance of pressure in chronic oedema/ lymphoedema**<br>Prof. Neil Piller (SA) |
| 16:45 | **The Risk for Lymphoedema in Lower Limb Trauma with Extensive Soft Tissue Loss**<br>Ms Malou van Zanten (SA) |
| 17:00 | **Do hydrocephalus shunts have a place in managing lymphoedema?**<br>Miss Jemima Bell (SA) |
| 17:15 | **What about compliance? Its impact on intermittent pneumatic compression outcomes**<br>Prof. Neil Piller (SA) |
| 17:30 | **Panel Discussion** |

**Pressure and Compression Workshop**
Prof. Neil Piller (SA)

| | |
|---|---|
| 18:00 | **Test to see if you are getting the pressures right and the garments you order are applying the right pressures!** |

**Closing Ceremony and Presentation of Best Abstract Awards**

| | |
|---|---|
| 19:30 | **Invited Speakers Dinner** |

For A Night At The Opera:

INTRODUCING

Yvette Masters

# WELCOME RECEPTION

# MONDAY 4 JULY 2016

# SOUNDS OF SILENCE

MEET AT THE SAILS IN THE DESERT LOBBY AT 5PM

DRESS: SMART CASUAL

LADIES ADVISED TO NOT TO WEAR HEELS

BE MINDFULL THAT DESERT TEMPERATURE DO DROP DURING THE NIGHT

WEAR WARM CLOTHING

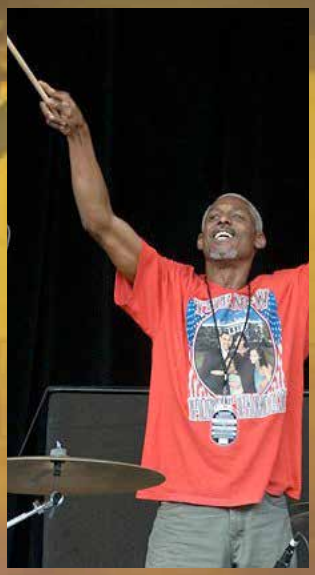
AND LOOK OUT FOR ALIENS

## GUEST SPEAKER DR. LOUIS GRONDIN

## The Fundamental Foamy Structure of the Universe

Enjoy an evening of catching up with College members and delegates at the Welcome Reception. Your Sounds of Silence experience begins with canapés and chilled sparkling wine served on a viewing platform overlooking the Uluru-Kata Tjuta National Park. As the sun sets and darkness falls, listen to the sound of a didgeridoo and join your table for an unforgettable dining experience. Cocktail, canapé and buffet dinner served.

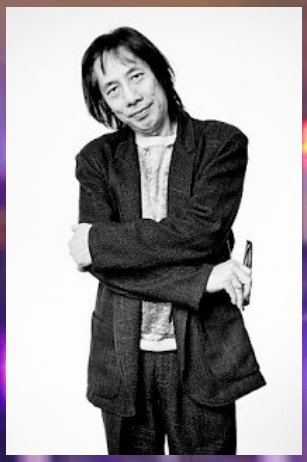
Settle back and listen to our resident star talker decode the southern night sky. Locate the Southern Cross, the signs of the zodiac, the Milky Way, as well as planets and galaxies that are visible due to the exceptional clarity of the atmosphere.



Drums / Percussion
**Calvin Welch**

Bass Guitar / Double Bass
**James Haselwood**

Vocals
**Berni Love**



Guitar/Vocals
**Rex Goh**

Keyboard/Violin/Vocals
**Gerard Masters**

# *Conferring Ceremony & Gala Dinner*

Tuesday 5 July 2016, 6:00 pm

Sails in the Desert

Tjungu Ballroom, Uluru Meeting Place

Dress: formal

## Exhibitors

### AMSL & NZMS

Phone: + 612 9882 3666 (AMSL)
Phone: + 649 259 4062 (NZMS)
Email: amsl@amsl.com.au
Email: nzms@nzms.co.nz
Contact: Suzi Trajkovski (AMSL)
Contact: Amber Johnston (NZMS)
Website: www.amsl.com.au
Website: www.nzms.co.nz

AMSL & NZMS have been looking after the needs of Vascular Surgeons, Phlebologists and Cosmetic Physicians for over 25 years. We are exclusive distributors in Australia and NZ of FibroVein by STD, UK. In NZ we distribute the Angio Dynamics range of EVLT 1470 Lasers with the Never Touch and Never Touch Direct fibers.

### ANZ Mobile Lending

Phone: +614 17 693 423
Contact: Tim Wilson
Email: wilsont@anzmortgagesolutions.com
Website: www.anzmobilelending.com.au/stives

ANZ Health provides medical practitioners, businesses and professionals with single industry expertise allowing them to focus on their patient care. ANZ is Australia's most awarded home lender. Our specialists can meet wherever to deliver optimal financing solutions.

### CRYOMED

Phone: 1300 346 448
Email: info@cryomed.com.au
Contact: JOHN FILLER
Website: www.cryomed.com.au

Cryomed Australia: Bringing premium aesthetic medical technology to plastic surgeons, dermatologists, cosmetic physicians, and aesthetic clinics in Australasia. A successful medical aesthetic practice is built on excellent patient outcomes. Cryomed's Research and Development team rigorously researches and tests the latest technology from the world's leading manufacturers. We are committed to providing you with the highest quality technology in a business model that allows you to provide excellent treatment to your patients, while ensuring a successful return on your investment.

### Device Consulting PTY LTD

Phone: +614 16 199 141
Contact: Milivoj Boltuzic
Website: www.deviceconsulting.com.au
Email: sales@deviceconsulting.com.au

With over 17 years' experience in distributing laser and energy based equipment, Device Consulting is once again very proud to be introducing cutting edge equipment which is clinically backed with an eye on a fantastic return on investment.

Please visit our booth to meet visiting international doctors and speakers to discuss your needs in more details.

### Endotherapeutics Pty Ltd

Phone: +612 9869 2868
Email: lturkrivero@endotherapeutics .com.au
Contact: Lian Turk Rivero
Website: www.Endotherapeutics .com.au

Endotherapeutics is an Australian medical devices company founded in 1999. Since its inception, Endotherapeutics has gained a reputation for being a leading specialist medical device distributor. Endotherapeutics prides itself in providing leading medical technologies to the healthcare specialist. Endotherapeutics is also incorporated in NZ, trading as Endoventure.

### GE Healthcare

Phone: +612 9846 4705
Email: steven.e.mclean@ge.com
Contact: Steven McLean
Website: www.gehealthcare.com.au

### Getz Healthcare

Phone: +614 3497 0780
Contact: Daniel Walsh
Email: danielwalsh@getzhealthcare.com.au
Website: www.basepharma.com

In March 2016 BASE Pharma became part of Getz Healthcare. We are committed to supporting Phlebology in ANZ and to offering Phlebologists a broad range of leading products for vein treatment as well as excellent service and support.

## Exhibitors

### LeMaitre Vascular

Phone: +614 08 025 045
Email: dluckin@lemaitre.com
Contact: David Luckin & Michael Whitley
Website: www.lemaitre.com

Lemaitre Vascular is a company dedicated to Vascular and Venous surgeons, providing ethical and innovative devices and product to improve patients outcomes. Based in the USA, we manufacture our biosynthetic grafts in Melbourne and the remainder of our extensive ranges, including the TRIVEX vein machine come from the USA.

### Life Healthcare

Phone: +612 8114 1549
Email: jessica.lee@lifehealthcare.com.au
Contact: Jessica Lee
Website: www.lifehealthcare.com.au

At LifeHealthcare we bring healthcare professionals innovative solutions by partnering with world class companies who share our vision for innovation and making a real difference to people's lives. LifeHealthcare understands the importance of providing our customers access to the most up-to-date technology enabling them to improve the clinical outcome for their patients. With its recent acquisition of M4 Healthcare, LifeHealthcare can offer a complete ultrasound solution across cardiac, vascular and Point-of-Care ultrasound markets. LifeHealthcare's portfolio of ultrasound products includes Philips, Terason, Mindray and Supersonic.

### Medtronic Australasia Pty Ltd

Phone: +612 9857 9235
Contact: Miranda Okazima
Email: miranda.okazima@medtronic.com
Website: www.medtronic.com.au

As a global leader in medical technology, services and solutions, Medtronic improves the lives and health of millions of people each year. We use our deep clinical, therapeutic and economic expertise to address the complex challenges faced by healthcare systems today. Let's take healthcare Further, Together. Learn more at www.Medtronic.com.au

### SIGVARIS

Phone: +613 9329 2772
Email: info.au@sigvaris.com
Contact: Alison Pringle
Website: www.sigvaris.com/au/en-au

More than 150 years ago the foundations were laid for the family business, SigVARIS. A perpetual endeavour to set standards in the field of compression textile is very much a part of our creed, and we aim to manufacture the best compression stockings.

### VENOSAN

Phone: 1300 527 127
Contact: Clare Anstiss
Email: clare@venosan.com.au
Website: www.venosan.com.au

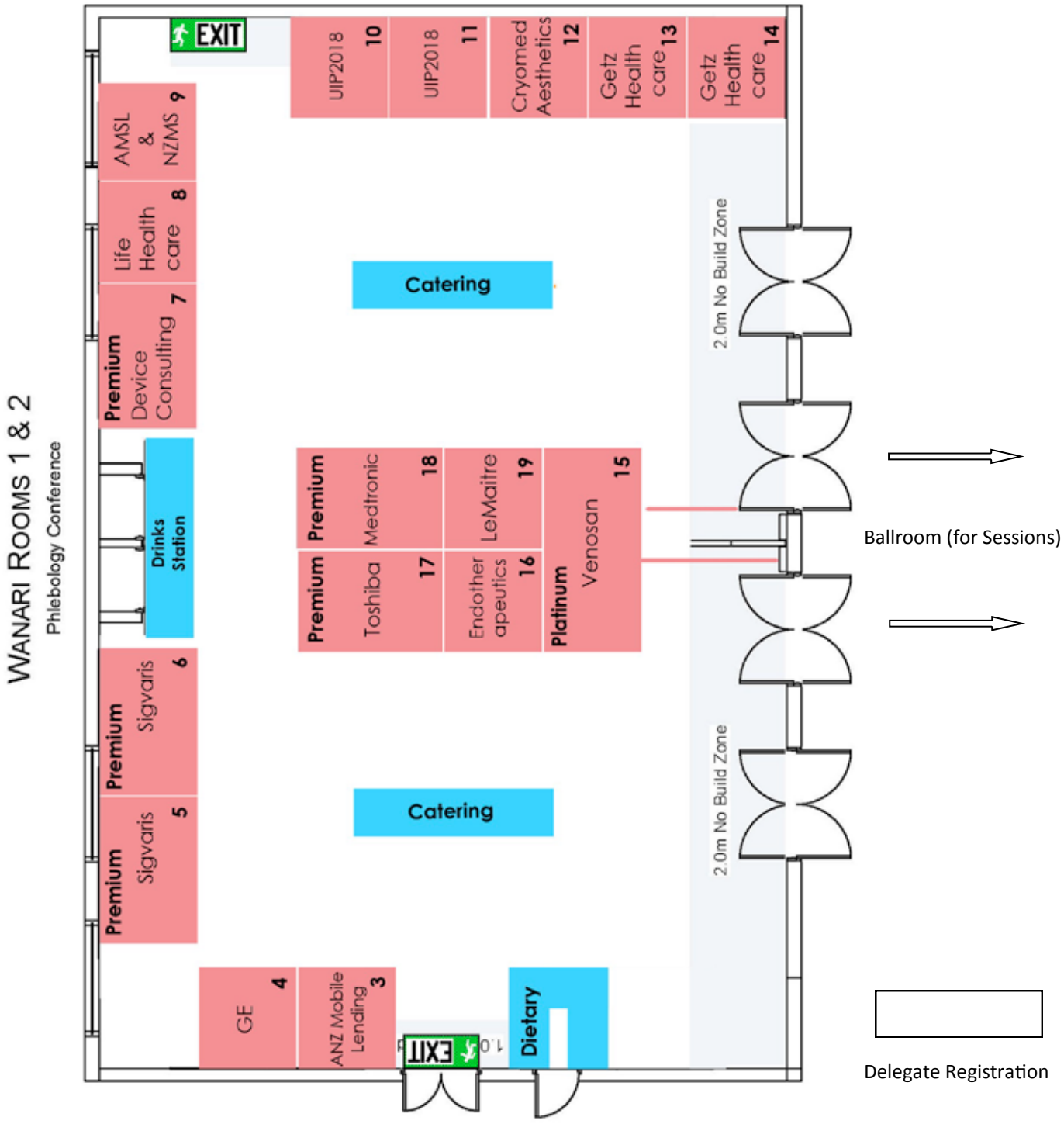
Venosan® Compression Stockings are manufactured in Switzerland by Swisslastic AG and has fulfilled a vision to supply the perfect compression and appropriate support stocking for all relevant medical indications and life situations. Medical Accessories of Australia with over 25 years of experience in healthcare are proud to be Australia Distributors.

### Toshiba

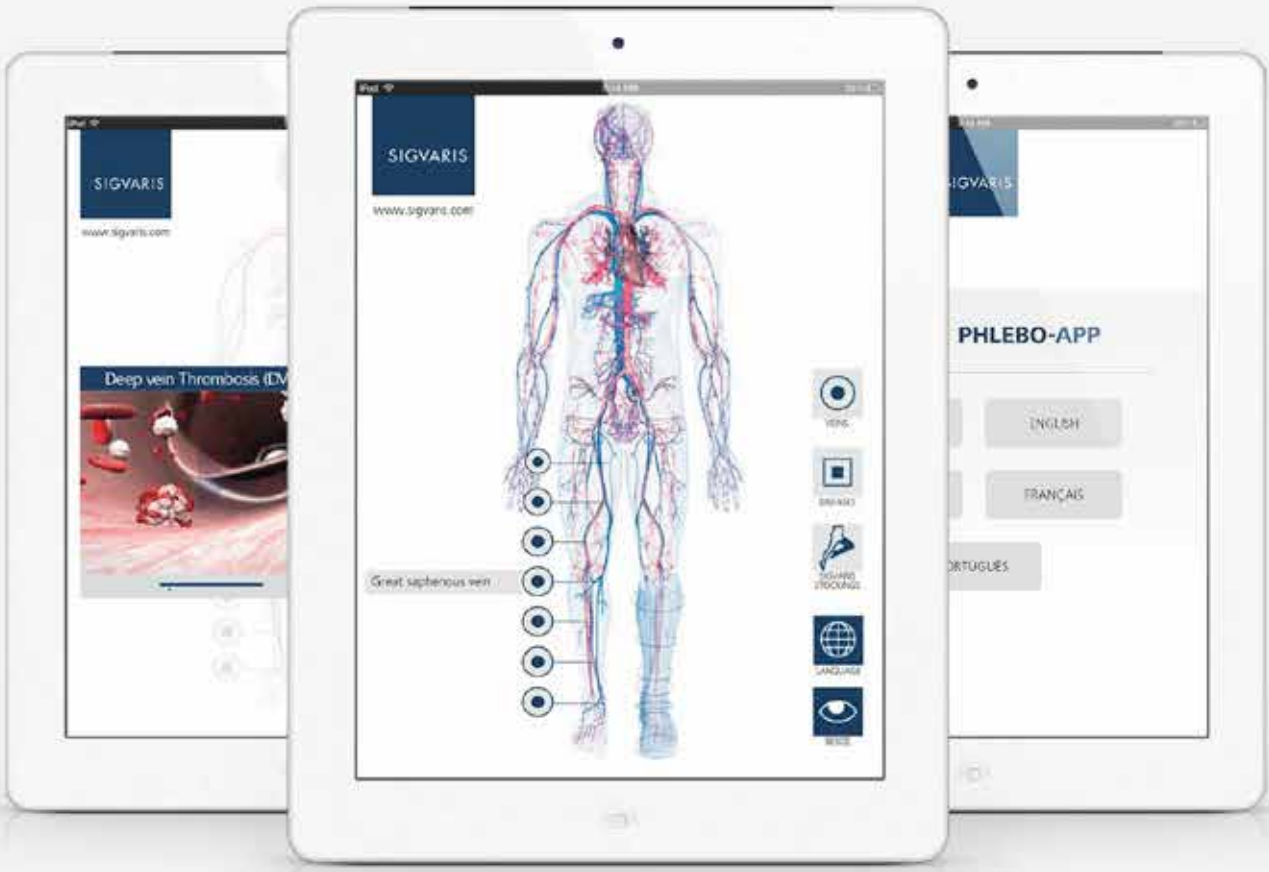
Phone: 1300 655 155
Email: intouch@toshiba-tap.com
Website: Medical.toshiba.com.au

For over 100 years Toshiba has been at the forefront of the medical device industry with leading innovations that challenge the status quo and change clinical pathways. Our award winning ultrasound systems and technologies are setting new industry standards. Made for Patients, Made for you, Made for life.

## Exhibitors Floor Plan



# The SIGVARIS Phlebo-App!




**The SIGVARIS Phlebo-App for your tablet:**

- See the most important venous diseases with location, video clip of how they develop, and explanatory text box
- Find out the anatomical location of the most important veins in phlebology
- Be fascinated by the pumping heart and blood flow in arteries and veins
- Show your patients the effectiveness of SIGVARIS medical compression stockings in a video clip

Visit our homepage and find out more:

**www.sigvaris.com/en/app/phlebo-app**

Available on App Store

Available on Google Play

Available on Windows Store

**www.sigvaris.com**



07999

# PRO Yellow

## The Ultimate Vascular Laser

### Applications

- Spider Veins/Telangiectasia
- Rosacea
- Vascular Lesions

### Improved Patient Outcomes

- The optimal wavelength in table top design for the treatment of vascular lesions
- Clinically effective
- The latest technological advancement
- Integrated skin cooling

### Greater Productivity

- No running costs
- Can be used on a broad range of skin types

PRO Yellow is the world's first tabletop vascular laser, utilising 577 nm tabletop technology to achieve the best clinical result. The 577 nm wavelength delivers 39% more blood absorption than 532 nm, and 75% more than 1064 nm, but with significantly lower melanin and $H_2O$ absorbtion.

Weighing just 13kg, PRO Yellow is both compact and portable. Its advanced German "solid state" semiconductor diode technology ensures fewer moving parts, making it among the most reliable of today's laser technologies, with no ongoing consumables costs.

DEVICE CONSULTING

16/24 Brand Drive, Thomastown
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f +61 3 9998 2019
e sales@deviceconsulting.com.au

**www.deviceconsulting.com.au**

# Simple. Fast. Precise.

## The new LOGIQ™ *e* Ultrasound System
## Ideal for Vascular Specialists

For more information, visit booth 4 or contact
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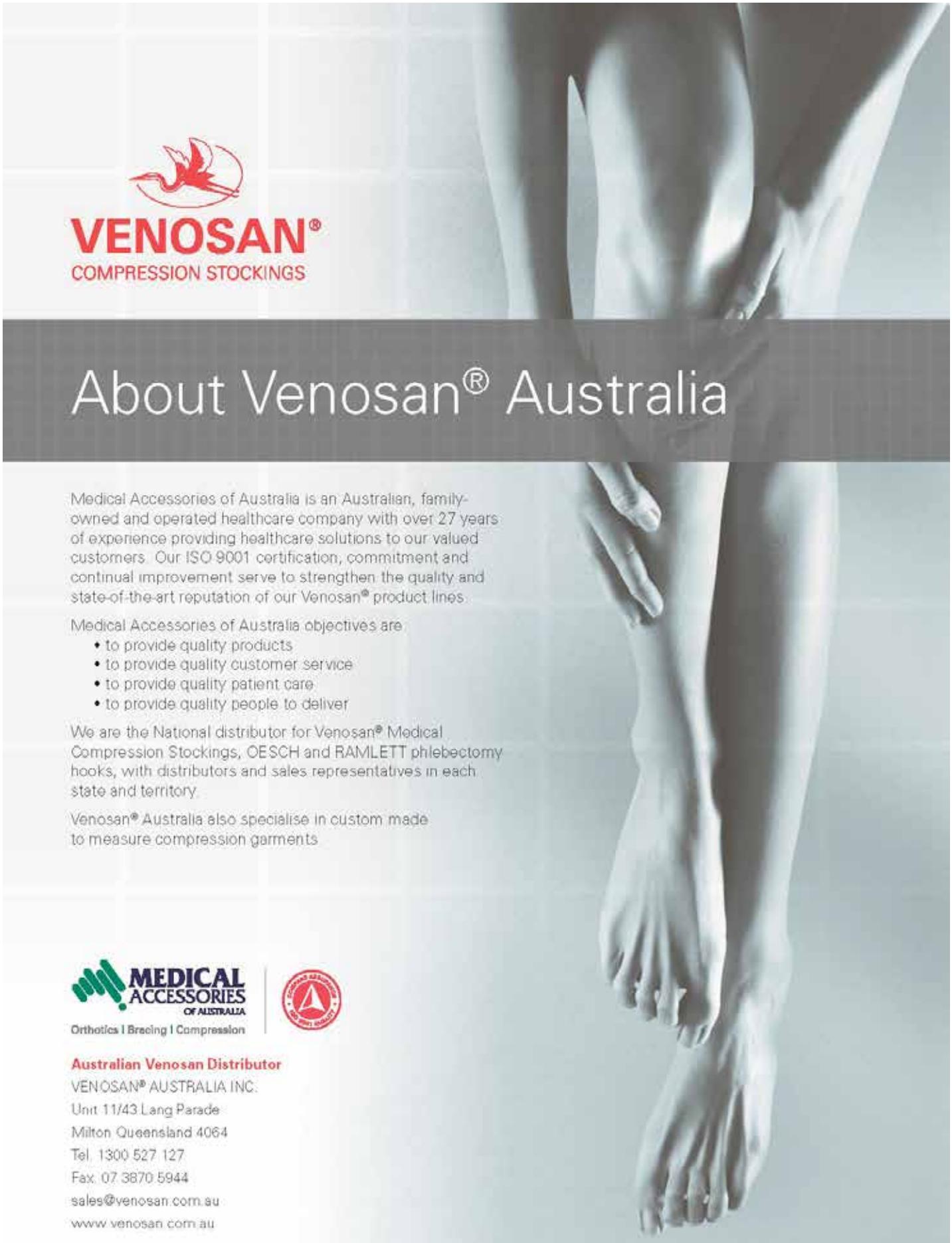


VENOSAN®
COMPRESSION STOCKINGS

# About Venosan® Australia

Medical Accessories of Australia is an Australian, family-owned and operated healthcare company with over 27 years of experience providing healthcare solutions to our valued customers. Our ISO 9001 certification, commitment and continual improvement serve to strengthen the quality and state-of-the-art reputation of our Venosan® product lines.

Medical Accessories of Australia objectives are:

- to provide quality products
- to provide quality customer service
- to provide quality patient care
- to provide quality people to deliver

We are the National distributor for Venosan® Medical Compression Stockings, OESCH and RAMLETT phlebectomy hooks, with distributors and sales representatives in each state and territory.

Venosan® Australia also specialise in custom made to measure compression garments.

MEDICAL ACCESSORIES OF AUSTRALIA
Orthotics | Bracing | Compression

**Australian Venosan Distributor**
VENOSAN® AUSTRALIA INC.
Unit 11/43 Lang Parade
Milton Queensland 4064
Tel. 1300 527 127
Fax. 07 3870 5944
sales@venosan.com.au
www.venosan.com.au

Call **1300 527 127** or email **sales@venosan.com.au**

The industry leader in compression stockings

# Venosan® one step ahead

Venosan® garments are made in Switzerland by Salzmann AG, a leading developer and manufacturer of specialised elastic yarns since 1883.

Venosan® compression garments have been manufactured since 1942 and are distributed in over 70 countries.

The on-going transfer of knowledge and technology between manufacturing and development teams, make for constant improvement in production techniques and the development of new ideas and yarns, which make Venosan® medical stockings an industry leader.

Knitted to German standards, graduated compression, correct pressure ratio and excellent fit are guaranteed.

**Your Guarantee**
Our medical compression garments are independently tested to the RAL standard by the Hohenstein Institute in Germany.

finest comfort

www.venosan.com.au

# LATEST NEWS:

BASE Pharma is now...

Getz Healthcare

**ABPI MD**
*Ankle Brachial Index*


**Aethoxysklerol**
*Sclerotherapy*

**Sclero-X**
*Compression Stockings*

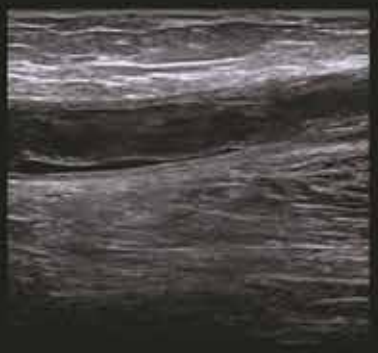
**VeinGogh**
*Spider Vein Thermolysis*

**VeinLite LEDX**
*Vein Illumination*

• Comprehensive Range • Service • Commitment to sup

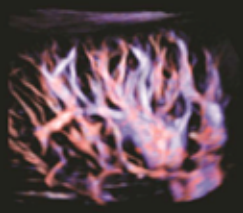
TOSHIBA

# ADVANCED APPROACH TO VEIN VISUALISATION



*Toshiba's Ultrasound product range covers a wide range of phlebology applications from advanced volume imaging through to high resolution B mode scanning. All of Toshiba Ultrasounds systems utilise unique technologies to help demonstrate venous anatomy and to guide interventions. The systems are designed to be easy to use, ergonomic and are supported by a team of Applications Specialist and service engineers.*

Made For life™

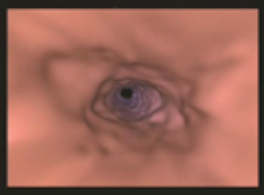


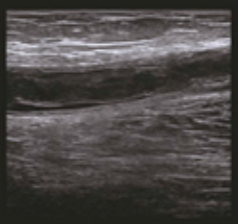
### 3D Smart Fusion

3D Smart fusion provides a 3 dimensional view of arterial venous malformations providing more structural information when compared to 2D imaging alone. 3D Smart Fusion provides an overall perspective of vascular lesions to help planning the treatment of these complex malformations.

### Fly Thru

Fly Thru provides a unique insight inside the blood vessels to demonstrate pathology from within. Fly Thru allows the ultrasound navigation through the lumen giving unprecedented views of the patients vascular anatomy from inside the vessels Fly Thru provides a endoscopic view from inside blood vessels using non-invasive ultrasound.





### High Frequency Transducers for the highest resolution where you need to be confident

Toshiba's high frequency transducers provide the highest resolution for detection of small clots and other abnormalities in superficial veins. This shows a thrombus in the GSV and the borders of the thrombus can be clearly resolved. Unique Toshiba technology such as Differential Tissue Harmonic imaging gives greater confidence in detecting and defining pathology in small vessels.

Come and see the **TOSHIBA** team at the ACP conference for **your conference special**

**Toshiba are proud sponsors of The Australasian College Of Phlebology**

# ZUMBA
## Class

**Fitness has never been so much fun until now**

**Join us for free**

**Monday 4th - Wednesday 6th July**

**7:00am - 7:30am**

**Instructor: Susan Hannah**

**Sails in the Desert**

**Lungkata Room**

## Stay Fit While in Uluru

FREE ADMISSION

**Train with our local trainer**

**Free for ACP Delegates**

**Monday 4th - Wednesday 6th July**

**6:15am - 7:00am**

**Sails in the Desert**

**Lungkata Room**

# THURSDAY 7 JULY 2016

# OPTIONAL TOURS

Uluru Motorcycle Tours offer a fun-filled adventure on the back of a Harley Davidson Motorcycle or a 3 Wheeler Trike. Offering anything from a quick 30-min spin to the ultimate sunset tour. The 3 Wheeler Trike's are great for families too as this can carry three passengers in total (max of two adults). Be adventurous and experience the beauty of Uluru and Kata Tjuta with the wind in your face. From $119. Tour duration approx. 30 minutes

Time: Morning, Afternoon CALL 1300 134 044

Drive through the sand hill country towards Kings Canyon, stopping for breakfast at Kings Creek Station (own expense). Continue on to Watarrka National Park where you can make the rocky climb with your Guide to the rim of Kings Canyon to be rewarded with marvellous views. The climb may take up to 3 hours and is for those with a good level of fitness. Or you may wish to explore the boulder-strewn canyon floor, an easier walk. From $225. Tour duration approx. 12 hours

Time: 04:45 AM CALL 1300 134 044

Catch the camel train and ride our good natured camels for 45 minutes through desert landscape with Uluru and Kata Tjuta as a stunning backdrop. Back at the farm, learn about the camel history of the Red Centre or browse our gift shop for that special souvenir. This tour always finishes in time for the Sounds of Silence dinner. Tour duration approx. 1.5 hours, and runs from April to October only. Suitable for children 5 years and over. From $80

Time: 14:00 PM CALL 1300 134 044

Launch off on a Rock Blasting adventure, taking in the sights of Ayers Rock Resort and surrounding deserts on the way to Uluru a panorama of breathtaking scenery unique to the Red Centre.

Tour bookings: Require a minimum 2 passengers per booking. If you're travelling with children under 2 years of age bookings must be made through the Voyages Travel Centre, call +61 (02) 8296 8010 or email travel@voyages.com.au. From $150

Time: Morning, Afternoon

## ULURU SUNRISE VIEWING

90 MINS PRIOR TO SUNRISE
From $69 Per adult
From $33 Per child

Travel to the sunrise viewing area, Talinguru Nyakunytjaku, near Uluru and watch the first sunlight of the day creep across the desert plains.

Enjoy a cup of tea or coffee and biscuits as the morning sun slowly changes the colour of Uluru

In summer, watching sunrise at Uluru in the cool morning air is the perfect start to the day.

Tour duration approx. 3 hours.

## FIELD OF LIGHT STAR PASS

1 HOUR 15 MINS AFTER SUNS
From $35 Per adult
From $25 Per child

Your Field of Light experienc
hotel pick-up and short tra
location.

As darkness has fallen, the 5
with radiant frosted-glass spl
rhythms of coloured light. Ter
monumental light installation

Tour duration: 2hrs 15mins in

These activities are not included in conference registration,
www.ayersrockre

ET

ce begins with a convenient nsfer to the remote desert

0,000 slender stems crowned neres will gently bloom with mporarily lose yourself in this its pathways draw you in.

cluding transfers.

to book please contact resort on 1300 134 044 or visit esort.com.au

**KATA TJUTA SUNSET**

1 HOUR 30 MINS AFTER SUNSET
From $95 Per adult
From $48 Per child

Enjoy a leisurely drive out to Kata Tjuta (the Olgas) for one of our favourite sunset experiences in Central Australia. Along the way your Driver Guide will explain the geological and cultural history of the area.

Marvel at the unique flora and admire the view of the central valley of the domes of Kata Tjuta while you take in a magnificent outback sunset with gourmet canapés and Australian wine.

Tour duration approx. 2.5 hours.

## LOCATION

### ULURU & KATA TJUTA

Ancient rock formations soar hundreds of metres into the desert sky, surrounded by the Red Centre's unique wildlife and spirit of the Anangu people's Tjukurpa. Sunset and sunrise over Uluru and Kata Tjuta are spectacular, with the colours at both sites becoming more vibrant and ever changing. Uluru and Kata Tjuta have significant meaning to Aboriginal people. They both form an important focus of their spiritual life.

**By Air**

Both Jetstar and Virgin Australia fly daily directly into Ayers Rock Airport from Sydney. Jetstar has a 4 weekly return service from Melbourne Tullamarine to Ayers Rock Airport (Tuesday, Wednesday, Friday and Sunday)

Qantas operates daily flights to Ayers Rock Airport via Alice Springs from Sydney. Qantas also offers direct flights from Cairns and Alice Springs.

All three airlines offer connecting flights from most capital cities to Ayers Rock Airport.

International guests may like to consider a Qantas Aussie AirPass for a more convenient way to include Ayers Rock (Uluru) in their itinerary.

Alice Springs to Ayers Rock Resort is a 1 hour flight or a 4.5 hour drive (450kms).

Contact the Voyages Travel Centre for accommodation, flight, touring, car hire and travel insurance quotes on 1300 134 044 (+612 8296 8010) or email travel@voyages.com.au.

**Airport Transfers**

Complimentary return coach transfers from Ayers Rock Airport to Voyages Ayers Rock Resort meet every scheduled flight. The return transfer to Ayers Rock Resort Airport collects you from your hotel approximately 2 hours prior to flight departure please check with reception for exact departure times.

### WEATHER

Uluru is situated near the centre of a semi-arid desert, which most people would associate with a hot and dry climate. However, it surprises - in that the temperature can vary so dramatically. Temperature in July can on average range from 5°C to 22°C.

For overseas visitors, Australian winter months are June, July, August and summer months are December, January and February.

# VENUE

## ULURU MEETING PLACE

The Uluru-Kata Tjuta National Park is an awe-inspiring living cultural landscape. The grandeur and unrefined charm of this iconic Australian destination is celebrated in Ayers Rock Resort's advanced, purposed built conference centre. At Uluru Meeting Place event practicalities are delivered in modern luxury from a location of extraordinary wonder.

## AYERS ROCK RESORT

Regardless of where you stay while you're at Voyages Ayers Rock Resort, you can experience the beauty of the living cultural landscape of Uluru-Kata Tjuta National Park, in Australia's Northern Territory. With over 65 tours, local activities and attractions within the Resort and the Uluru (Ayers Rock) - Kata Tjuta (The Olgas) National Park, your days will be action-packed. Ride a camel across the desert dunes. Hop on a Harley, or embark on a base walk of Uluru (Ayers Rock).

If you're looking for relaxation and pampering after a day of discovering the outback, make sure you visit the beautiful Red Ochre Spa. The Red Ochre Spa has been designed with total indulgence in mind - a sanctuary where guests exchange stressed states for tranquillity. By night, dine under a canopy of stars at the award-winning Sounds of Silence buffet barbeque dining experience. See the sun set behind Uluru, and after dinner, tour the southern night sky with a resident star talker.

Ayers Rock Resort provides a variety of accommodation options for every possible taste and budget - from the award winning 5-star Sails in the Desert, and modern Desert Gardens Hotel, to the self contained Emu Walk Apartments, the authentic Outback Pioneer Hotel and Lodge, and the Ayers Rock Campground, offering powered campsites and air conditioned cabins.

## AYERS ROCK RESORT MAP

## ACCOMMODATION

Four hotels unite to form Ayers Rock Resort. Each is distinct in design and budget. All share a common passion for complimenting unforgettable events with our own style. Our service is professional and refined and made unique by a sense of relaxed outback charm.

All properties are within close walking distance of each other. This practical layout makes it possible for large groups to use multiple hotels for the same event group.

Ayers Rock Resort is complimented by the exclusive and award-winning Longitude 131°. Located a short 10 minute drive from the Resort, Longitude 131° is an experience of luxury rather an accommodation choice.

Special rates have been negotiated with the Ayers Rock Resort for delegates attending the Annual Scientific Meeting.

### SAILS IN THE DESERT - 5 STAR

In the heart of Australia's Red Centre, Sails in the Desert hotel beautifully contrasts Uluru's raw natural beauty with a decidedly luxurious outback holiday experience.

### DESERT GARDENS HOTEL - 4.5 STAR

The giant Australian ghost gums dwarf flowering native shrubs in the gardens surrounding this hotel. Sip sparkling wine on your balcony while gazing out over uninterrupted views of Uluru from the hotel's Deluxe Rock View Rooms.

### EMU WALK APARTMENTS – 4 STAR

Strolling along the shady avenues that meander through the heart of the Resort you will find a collection of welcoming terrace style apartments.

### Outback Pioneer Hotel - 3.5 Star

Experience traditional Aussie hospitality at the Outback Pioneer Hotel - with a choice of 3 ½-star hotel or 2-star budget accommodation.

Rooms are either configured with one queen and one single bed or one queen bed and two single beds, both with a private ensuite, television and movies on demand. Interconnecting rooms and wheelchair accessible rooms are available in some standard rooms. Cots and roll-away beds, and wheelchair accessible rooms are subject to availability. Your stay includes a FREE Indigenous Activities Program, return Ayers Rock Airport transfers, free use of Ayers Rock Resort shuttle bus service, Virgin Velocity Points and children 15 years and under stay free using existing bedding.

## Notes

# ABSTRACTS

| Practice Matters in Phlebology I | |
|---|---|
| Sunday, 3rd July, 2016 | 8:30 - 9:00 |

## Employee Fraud: How to Detect and How to Prevent it

Rebecca Fleming

Gow Gates, Sydney NSW, Australia

Businesses of all kinds and sizes are increasingly threatened by fraud. One of the most difficult situation to come to terms with is when that fraud is committed by your own employees, many who you have trusted and been with you for years. Employee dishonesty can be damaging not only to your bottom line, but also to your reputation. After an event, it is common to feel a sense of betrayal and shock.

In these high pressure times, you have missed the subtle warning signs. The aim of this presentation is to provide you with practical tools that may prevent you becoming a victim, because while you can insure for the financial loss, the time involved and disruption to your business is sometimes impossible to quantify. In the case of Employee Fraud, prevention is always the better option.

This presentation will address the following points:

- Who commits fraud
- Why employees commit fraud
- How to detect the red flags of common scenarios
- How to implement anti-fraud controls
- How to ensure you Insurance Programme includes protection from this exposure

NOTES

| Practice Matters in Phlebology I | |
|---|---|
| Sunday, 3rd July, 2016 | 9:00 - 9:30 |

## Employee Claims and Contracts: How to Avoid Appearing at the Fair Work Commission (80/20 rule)

Stephanie Anderson

Sydney Skin and Vein Clinic, Chatswood, NSW Australia

Avoiding the Fair Work Commission will save your business time and effort. Learn what the most crucial steps to creating a workplace environment that protects and minimises your risk of having to spend time in the Commission.

Employee workplace contracts are just one way to ensure safe, fair, productive and successful Australian workplaces for both the employee and employer. Contracts are the backbones of the modern day workplace with performance management a crucial process.

Negotiating a good contract that is compliant with the Act and that will stand up to any action in the Commission can entail is the first step. Claims can be costly to business, in lost time, time which is better spent on core business activities.

Planning the role thoroughly, along with the development of competency based job descriptions are all crucial steps to preparing up front, and planning to not appear in the Fair Work Commission! These steps will ensure that the employer has a solid start to the employment relationship with the employee.

Excellent performance management and record keeping are an employer's best friend when it comes to responding to employee claims. Your insurer, legal, or IR specialist will call upon your HR records as their first point of investigation! Keep your records relevant, up to date and record them as you go!

Employee claims are more common than employers think. Plan to stay out of the Commission by understanding your obligations.

It is not enough to have read the Act, or to have a copy of the National Employment Standards that can be referred to, ensure that contracts that are offered are compliant with the Act and NES. Ensure your managers understand the core of the NES and the Fair Work Act!

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| Practice Matters in Phlebology II | |
|---|---|
| Sunday, 3rd July, 2016 | 10:30 - 11:00 |

## Accounting 101: How to be more Profitable by Understanding your Business Finances

Andrew Ramsay

Ramsay Financial, NSW, Australia

Have you ever wondered what the difference is between a practice that consistently and predictably realizes extraordinary growth and another practice that struggles just to make ends meet?
Ever wondered what a balance sheet is as opposed to a profit and loss statement, and how they can help you understand where your business is heading? Does your practice have a management information system in place that you get meaningful reporting out of? Learn what these reports are, and how they can allow you to deeply understand your business. What costs you money, and where to reign in the budget is another key area. Budgeting and how it can manage your business is an area worth focusing on.
Financial management and reporting are key to excellent operations, a good understanding of the reports you actually need and how they can increase your profitability will allow you to fully understand your business. Slim down your reports to what gives you a good understanding of your practice, rather than a fat load of reports which you need to sift through to get the real answers.
Having good reporting in place will reveal where you should devote time and resources and let you address any real problems you are facing before they get out of control. You can only really succeed with managing your business through understanding your financial management reporting by planning and implementing a good system that's tailor fit to your requirements.
You will be provided with revealing insights into financial management and reporting to get you geared up to implement some of the ideas and concepts you will learn.

NOTES

| Practice Matters in Phlebology II | |
|---|---|
| Sunday, 3rd July, 2016 | 11:00 - 11:30 |

## Refunds / Reimbursements and Discounts: Should we pay back?

Jennie Lekich

Vein Doctors Group / Miami Day Hospital, Gold Coast QLD, Australia

NOTES

| Practice Matters in Phlebology II | |
|---|---|
| Sunday, 3rd July, 2016 | 11:30 - 12:00 |

## Financing your Practice

Tim Wilson

ANZ Mobile, NSW, Australia

ANZ Health provides medical practitioners, businesses and professionals with single industry expertise allowing them to focus on their patient care. Our team of specialists can help with all financial services from buying a home or commercial premises to all business needs for practitioners who want to own or grow their existing business. We understand practitioners can be time poor, so our team is happy to come to you at a time that suits you.

We offer tailored financial solutions for:

- Business Property (up to 100%)
- Goodwill / Business Purchase (up to 100%)
- Fit out
- Working Capital
- Medical Equipment
- Cars
- Merchant services
- Transactional & internet banking

We also offer up to 90% LVR with no LMI for Owner Occupied and Investor Properties

All wealth needs such as Risk protection, Needle Stick Insurance and Superannuation are also catered for by our health specialised financial planners.

NOTES

| Vascular Lasers | REVIEW LECTURE |
| --- | --- |
| Sunday, 3rd July, 2016 | 13:30 - 14:00 |

## Overview of Vascular Lasers

Stefania Roberts

Victoria Vein Clinic, East Melbourne, Australia

NOTES

| Vascular Lasers | |
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| Sunday, 3rd July, 2016 | 14:00 - 14:15 |

## Long Pulse Nd-YAG and Pulsed-Dye Lasers

Anne Padbury

Lasers In Medicine, Adelaide SA, Australia

The Cutera Excel V offers a best-in-class technology for the treatment of vascular and benign pigmented lesions. The Cutera Excel V's unique design allows combinations of pulse widths, spot sizes and fluences at both 532 nm and 1064 nm exclusively.

The Syneron Candela V Beam Pulsed Dye Laser at 595 nm has been the gold standard for treatment of vascular lesions. Both lasers provide a wide range of parameters to enable the clinician to treat effectively.

NOTES

| Vascular Lasers | |
|---|---|
| Sunday, 3rd July, 2016 | 14:15 - 14:30 |

## Q-switched Nd-YAG Laser to Treat Pigmentation Post-Sclerotherapy

Sanjay Nadkarni

Endovascular WA, Claremont WA, Australia

NOTES

| Vascular Lasers | |
|---|---|
| Sunday, 3rd July, 2016 | 14:30 - 14:45 |

## The new Yellow Laser

Kurosh Parsi

Department of Dermatology, St Vincent's Hospital, Darlinghurst NSW, Australia.
Dermatology, Phlebology and Fluid Mechanics Research Laboratory, St Vincent's Centre for Applied Medical Research, Darlinghurst NSW, Australia.
The University of New South Wales, NSW, Australia.

The QuadroStar PROYELLOW is the first yellow table-top laser in dermatology. Thanks to its special wavelength of 577 nm, the QuadroStarPROYELLOW can treat telangiectasia, spider and cherry angiomas, venous lakes and capillary malformations. This laser is suitable for the treatment of darker skin types due to the lower melanin absorption of the 577 nm wavelength compared to 532nm.

The QuadroStarPROYELLOW features the HOPSL (high-power optically pumped semiconductor laser), the state of the art in solid-state laser technology. This technology makes the yellow wavelength available in a very small and light device (less than 12 kg). The device has no ongoing running costs.

In addition to the standard handpiece, the QuadroStarPROYELLOW also offers a scanner with integrated skin cooling, for the treatment of larger areas. This feature is especially suitable to treat diffuse erythema and capillary malformations.

NOTES

| Vascular Lasers | |
|---|---|
| Sunday, 3rd July, 2016 | 14:45 - 15:00 |

## Fractional and New Dermatological Lasers

Adrian Lim

Department of Dermatology, Royal North Shore Hospital, St Leonards, Australia.
uRepublic Cosmetic Dermatology and Veins, Sydney, Australia

Lasers and laser-like devices have become an accepted part of clinical practice ever since the first laser was introduced 50 years ago. Over the last decade, the demand for these devices has grown considerably. Whilst technology improves, the cost is also starting to fall, making ownership of one or more devices a reality for many.

Phlebologists are familiar with endovenous and external beam vascular lasers. This talk will provide an overview of the some of the latest dermatology devices some of which are directly relevant to phlebology while others will be of interest to 'vein and cosmetic/laser' practitioners.

The talk will include discussion of:

- Lasers for staining and matting
- Lasers for treating periorbital veins
- Fractional lasers
- Colour-blind lasers for darker skin types
- Skin tightening devices
- Fat-freezing devices

The talk will be framed by my personal experience - what works in my practice and what doesn't – and the pearls and pitfalls of running a 'vein and cosmetic/laser' practice.

NOTES

| Superficial Venous Disease: New Technologies | REVIEW LECTURE |
|---|---|
| Monday, 4th July, 2016 | 8:00 - 8:30 |

## Endovenous Modalities in 2016: Chemical Ablation, Endothermal Destruction and Adhesive Closure - What is the Evidence, How to Choose.

Stefania Roberts

Victoria Vein Clinic, East Melbourne, Australia

NOTES

| Superficial Venous Disease: New Technologies | |
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| Monday, 4th July, 2016 | 8:30 - 8:45 |

## Laser Beam Travelling Through Foam - What happens next?

Louis Grondin

In the last 10-15 years, endovenous thermal ablation, has nearly replace surgery as the treatment of choice of incompetent Saphenous Veins. In the case of endovenous laser ablation, the ambulatory nature of the procedure, the reduced incidence of complications (especially when higher laser wave lengths are used), and the predictably good and lasting outcomes, likely played a major role in this transition.

The procedure in not free of complications however, thrombus extension into the deep venous system, and adventitial arterio-venous fistulas have been reported. In order to reduce these complication 2 technique have been developed: one is the use of radial tip fiber, and the other is to laser through foam. The later offers the added the advantage of overcoming one of endovenous laser's innate weakness: which is reaching and eliminating the often complex and multiple saphenous termination into the deep venous system.

But what exactly does happen when a laser beam interacts with foam? Two main phenomena have been observed:

1. A resonant effect, which results in profound laser beam refraction and directional change, which effectively transforms a bare tip fiber emission into a radial tip emission, distributing laser light energy uniformly to the adjacent vein wall.
2. And a dominant un-resonant effect, which results, within 100 micro-seconds after laser impact, in nano-water droplet deformity, and hydro-jets propulsions at hypersonic range, which should have a destabilizing effect on the foam structure. This un-resonant effect is proportional the the energy delivered and inversely proportional to the wave length emitted.

Of late Laser irradiation of antimicrobials prior to drug delivery has induce increased biological activity in the irradiated chemical. No such enhanced endothelial cell wall activity have yet been demonstrated despite evidence of enhanced Raman spectroscopy enhancement which is likely produced by the intense laser light scattering on Polidocanol *in vitro*, which paradoxically may indicate suspended foam decay. This has found some interesting application in the management of reticular varicosities.

NOTES

| Superficial Venous Disease: New Technologies | |
|---|---|
| Monday, 4th July, 2016 | 8:45 - 9:00 |

## A Large, Single-Centre Experience with Cyanoacrylate Adhesive Glue Embolisation for Saphenous Vein Insufficiency

Ramon Varcoe[1,2], Shannon Thomas[1,2], Mark Yang[2], Nicole Rubesamen[2], Andrew Lennox[2]

[1] University of New South Wales, Randwick, Australia,
[2] Prince of Wales Hospital, Randwick, Australia

Objectives: To report the results of the use of cyanoacrylate adhesive embolisation for the treatment of superficial venous reflux in an Australian patient cohort.

Methods: A prospective, single-centre analysis of patients with chronic venous disease treated with saphenous vein ablation with cyanoacrylate between March 2015 and May 2016.

Results: 69 venous trunks in 64 limbs of 46 patients (76% female) with a mean age of 51.6 years (range 25-79 years) were treated. CEAP categories included C2 (21; 46%), C3 (17; 37%), C4A (4; 9%), C5 (1; 2%) and C6 (1; 2%). 60 great-saphenous and 9 small-saphenous veins were ablated with 100% technical success; 82.6% underwent concomitant phlebectomy, 6.5% sclerotherapy and 4.3% had no additional treatment for their varicosities. There were no adverse events prior to discharge. Of the 38 & 33 patients eligible for 1- & 3-month follow-up respectively, 34(89%) and 21(64%) attended. Target vein closure rates were 100% and 90.6% at 1 & 3 months. Complications included phlebitis (15%), neuropraxia (9%) and skin matting (3%).

Conclusion: The use of cyanoacrylate adhesive embolization for saphenous vein insufficiency is safe and has excellent short-term efficacy. Longer follow-up is necessary to determine treatment durability.

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| Superficial Venous Disease: New Technologies | |
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| Monday, 4th July, 2016 | 9:00 - 9:15 |

## Preliminary experience with Adhesive Closure

Stefania Roberts, Kenneth Myers

Victoria Vein Clinic, East Melbourne, Australia

Adhesive closure is an innovative modality in the treatment of varicose veins. It is as effective as the current endothermal technologies with a safer side effect profile. Preliminary results from a single centre observational study with technical success from life table analysis, QOL and clinical outcome will be presented.

Adhesive closure has a number of advantages. From the patient's perspective there is no pain associated with tumescent anaesthesia, no compression stockings, no downtime and good results. From the doctor's perspective it is a quick, easy to perform procedure with minimal complications.

NOTES

| Superficial Venous Disease: New Technologies | |
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| Monday, 4th July, 2016 | 9:15 - 9:30 |

## Trivex in Post-embolisation Management of Vascular Anomalies

Kurosh Parsi[1,2,3] and Anthony Trimboli[1,2]

[1] Department of Dermatology, St Vincent's Hospital, Darlinghurst NSW, Australia.
[2] Dermatology, Phlebology and Fluid Mechanics Research Laboratory, St Vincent's Centre for Applied Medical Research, Darlinghurst NSW, Australia.
[3] The University of New South Wales, NSW, Australia.

Large vascular anomalies on the trunk or limbs are uncommon and often prove challenging to treat. Currently, treatment options include conservative management, embolization, sclerotherapy or surgical excision. Once the lesion has been embolised, or if it undergoes spontaneous evolution, the residual fibrofatty tissue may need to be surgically debulked. Surgical debulking may be associated with complicated wound healing, infection and scarring. Here we present our technique of using TrivexTM powered phlebectomy to debulk the residual vascular anomaly.

The first case was a 62 year old female with a symptomatic involuted congenital haemangionma on the lower back. She complained of not being able to sleep on her back for 40 years due to pain. The second case was a 29 year old female with a combined venolymphatic malformation (VLM) on the medial aspect of her leg. Both lesions were removed without adverse sequelae and with excellent cosmetic and symptomatic results.

NOTES

| Antiphospholipid Syndrome | KEYNOTE LECTURE |
|---|---|
| Monday, 4th July, 2016 | 10:30 - 11:00 |

## Antiphospholipid Syndrome: State-of-the-Art

Fatima El-Assaad[1,2], Steven A. Krilis[1,2,3] and Bill Giannakopoulos[1,2]

[1] Department of Medicine, St George Hospital, University of New South Wales, Australia
[2] Department of Infectious Disease, Immunology and Sexual Health, St George Hospital, University of New South Wales, Australia
[3] School of Biomedical Sciences and Pharmacy, University of Newcastle, Australia

Clinical manifestations of the antiphospholipid syndrome (APS) include both venous and arterial thrombosis that can affect a variety of vascular beds. Laboratory investigations are crucial in establishing a diagnosis of APS as there is a lack of specificity of the clinical manifestations of the syndrome. The laboratory investigations detect autoantibodies to specific autoantigens using ELISA assays and the lupus anticoagulant (LA) the *in vitro* coagulation based assay used to detect antiphospholipid (aPL) antibodies. These tests measure the time to clot *in vitro* of the patients' plasma, relative to the time taken by healthy control samples. Lupus anticoagulant correlates best with clinical features of APS more than positive result on ELISA testing. A significant proportion of patients with APS that are LA positive also have anti-$\beta_2$GPI autoantibodies detected in the $\beta_2$GPI ELISA. There are patients that are negative on both the anti-$\beta_2$GPI and aCL ELISAs but test LA positive. The term aPL antibodies is a misnomer as the autoantibodies bind protein autoantigens, the major APS autoantigen being $\beta_2$GPI an abundant plasma glycoprotein that binds negatively charged phospholipids. It is suggested based on extensive laboratory and clinical studies that the anti-$\beta_2$GPI antibodies may be directly pathogenic.

$\beta_2$GPI is a protein that consists of 5 modules designated domain I-V. Domain V of $\beta_2$GPI contains a surface exposed patch that is highly positively charged and an area which inserts into lipid membranes interacting with negatively charged phospholipids. In plasma, the $\beta_2$GPI molecule can potentially be in equilibrium with 2 types of configurations, a closed loop and open conformation. In fluid phase the anti-$\beta_2$GPI autoantibodies do not bind $\beta_2$GPI as it circulates in plasma predominantly in a closed configuration such that the antibody binding site on domain I is hidden.

NOTES

| Antiphospholipid Syndrome | |
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| Monday, 4th July, 2016 | 11:00 - 11:15 |

## The Structure and Post-Translational Modification of Beta-2-Glycoprotein-I

Fatima El-Assaad[1,2], Steven A. Krilis[1,2,3] and Bill Giannakopoulos[1,2]

[1] Department of Medicine, St George Hospital, University of New South Wales, Australia
[2] Department of Infectious Disease, Immunology and Sexual Health, St George Hospital, University of New South Wales, Australia
[3] School of Biomedical Sciences and Pharmacy, University of Newcastle, Australia

The antiphospholipid syndrome (APS) is an autoimmune condition with its major manifestations characterized by thrombophilia and/or recurrent miscarriage. The major autoantigen in this condition is $\beta_2$GPI. This is a plasma glycoprotein which circulates in plasma at a high concentration. It is composed of 5 domains. The first 4 domains consist of 60 amino acid residues linked by 2 disulfide bonds. The 5th domain has an extra disulfide bond with the 3rd disulfide bond ending in a cysteine at 326 covalently linked to cysteine 288. The molecule can exist in different redox forms where the disulfide bridge in the 5th domain at the C-terminus between Cys288 and Cys326 can be either in an oxidised or in a free thiol form. The 5th domain of $\beta_2$GPI binds and inhibits a number of coagulation proteins such as thrombin, Factor XI and von Willebrand Factor (vWF). Human endothelial cells modulate the redox status of $\beta_2$GPI amplifying the free thiol form and nitrosylation of $\beta_2$GPI. The oxidised form of $\beta_2$GPI is significantly more immunogenic than the free thiol form.

Posttranslational oxidative modification of $\beta_2$GPI occurs during oxidative and nitrosative stress. Cys326 can be nitrosylated in-vitro. The free thiol form of $\beta_2$GPI has been shown to protect endothelial cells from oxidative stress induced cell death. Patients with APS have significantly lower levels of the endothelium protective free thiol form of $\beta_2$GPI. *In vivo* it is thought that posttranslational modification of $\beta_2$GPI induces a conformational change such that the major B cell epitope on Domain I of the molecule and the major T cell binding site on Domain V of the molecule induce autoantibodies to $\beta_2$GPI. The proportion of the free thiol $\beta_2$GPI and oxidised $\beta_2$GPI is important in the development of autoantibodies in patients with APS. These findings provide a new approach to risk stratification and therapy in APS.

NOTES

| Antiphospholipid Syndrome | |
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| Monday, 4th July, 2016 | 11:15 - 11:30 |

## Antiphospholipid Syndrome: Advances in Pathophysiology

Harshal Nandurkar

Australian Centre for Blood Diseases, Monash University, Melbourne, Australia

Antiphospholipid syndrome (APS) is an autoimmune disease characterized by the presence of antiphospholipid antibodies (aPL) and can share overlap with lupus. The clinical syndrome includes vascular thrombosis and/or pregnancy morbidity. Thrombotic manifestations include arterial and venous events. aPL's activate procoagulant pathways by several mechanisms and evidence is supported by *in vitro*, animal and human studies. A 'two hit hypothesis' is suggested as an explanation for the clinical observation that thrombotic events occur only occasionally even though aPLs are persistently present. In this model, the autoantibody creates a thrombophilic state as the first hit. However, a second hit is necessary to progress to thrombosis. Implicated mechanisms include increased generation of reactive oxygen species, disruption of annexin A5 protective shield, increased expression of tissue factor and signalling via Toll-like receptors. Fetal complications of APS could be mediated by placental thrombosis, inflammatory signalling and toxicity to trophoblasts. Activation of complement by aPL has also been shown to play a prominent role by the confirmation that mice deficient in complement components or treated with inhibitors of complement activation, show reduced fetal loss. There is evidence that aPLs activate the mammalian target of rapamycin complex (mTORC) pathway in patients with primary or secondary antiphospholipid syndrome nephropathy and treatment with sirolimus led to improvement in renal function.

NOTES

| Antiphospholipid Syndrome | |
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| Monday, 4th July, 2016 | 11:30 - 11:45 |

## Antiphospholipid Syndrome: Cutaneous Manifestations

Kurosh Parsi

Department of Dermatology, St Vincent's Hospital, Darlinghurst NSW, Australia.
Dermatology, Phlebology and Fluid Mechanics Research Laboratory, St Vincent's Centre for Applied Medical Research, Darlinghurst NSW, Australia.
The University of New South Wales, NSW, Australia.

Antiphospholipid syndrome (APS) can present with a range of cutaneous manifestations. These can be divided into three broad categories of cutaneous manifestations secondary to 1) hyper-viscosity and 2) vessel inflammation and occlusion 3) the underlying disease.

Blood hyperviscosity is an important contributor to the cutaneous eruptions observed in APS. High serum levels of circulating AP antibodes result in stagnant blood flow in the sub-dermal venous plexus. This results in the veno-congestive sub-type of livedo reticularis. This sub-type presents with large cyanotic venous rings that correlate with the sub-dermal reticular veins. This eruption is completely blanchable, may be symmetrical and often diffuse. While the veno-congestive sub-type of livedo reticularis always represents an underlying pathology, the vaso-spastic sub-type is more commonly due to a physiological response to cold in young females.

Inflammation and partial obstuction will result in the partly blanchable eruption of livedo racemosa. Livedo racemosa is a branched, partially blanchable, non-symmetrical eruption that always signifies an underlying pathology. APS can result in a medium-size vasculitis that will clinically present with livedo racemosa. Livedo racemosa and livedo reticularis maybe concurrently present. Small vessel vasculitis can also occur presenting with erythematous papules of palpable purpura.

APS can also result in thrombotic occlusion of small, medium or large size vessels. Small vessel thrombosis is found in livedo vasculopathy (LV) with APS as the underlying disease. LV will present with reticulate pigmentation, atrophie blanche and seasonal stellate ulceration. APS can also result in deep vein thrombosis (DVT) or superficial thrombophlebitis (STP). Arterial thrombosis will result in ischaemic signs and tissue necrosis.

The underling disease, for example systemic lupus, will present with its own cutaneous signs such as peri-ungal telangiectasias and malar erythema.

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| Antiphospholipid Syndrome | |
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| Monday, 4th July, 2016 | 11:45 - 12:00 |

## Livedoid Vasculopathy

Afsaneh Alavi

University of Toronto, Toronto, Canada

Livedoid vasculopathy (LV) is a non-inflammatory thrombotic condition presenting in a primary idiopathic or secondary subtype associated with abnormal coagulation factors. Clinically this condition is associated with recurrent livedo reticularis with chronic and painful skin ulcers at the distal extremities, particularly around the ankle region, and at the dorsum of the feet. The histology is characterized by segmental hyalinizing changes at the subintimal region of small dermal vessels with thrombotic occlusions. LV skin ulcers resolve with stellate, porcelain-white scars that need to be distinguished from similar changes with venous stasis. The term atrophie blanche (AB) has employed to describe spontaneously occurring porcelain-white skin areas with red dots that typically occur in the context of skin changes attributed to chronic venous insufficiency (CVI). The two forms of AB – (1) the LV-AB-complex and (2) AB in the context of CVI – are unrelated and require separate diagnostic and therapeutic approaches.

Objectives:

- Review the clinical presentation and approach to livedoid vasculopathy
- Discuss the current management of livedoid vasculopathy

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| Antiphospholipid Syndrome | |
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| Monday, 4th July, 2016 | 12:00 - 12:15 |

## Antiphospholipid Syndrome: Looking beyond conventional tests

Harshal Nandurkar

Australian Centre for Blood Diseases, Monash University, Melbourne, Australia

The common autoantibodies tested in the diagnosis of antiphospholipid syndrome (APS) include anti-β2-glycoprotein I or anticardiolipin antibodies (IgG and IgM) as quantified by ELISA and lupus anticoagulant as detected by a clotting based assay. Correlation of assays with clinical phenotype has shown that the strongest association is with lupus anticoagulant (LA). Also, LA that is due to anti-β2-glycoprotein I autoantibodies correlates with thrombosis better than LA due to antiprothrombin antibodies. A number of other coagulant pathway-related proteins are also known as autoantigens in APS; for eg, anti-protein C antibodies are associated with a severe thrombotic phenotype. Annexin A5 is found on trophoblasts and endothelial cells and has anticoagulant activity. Anti-2-glycoprotein I antibodies disrupt annexin A5 function to create a procoagulant environment. A novel annexin A5 'resistance' assay has been developed to better understand the pathogenetic relationships with antibody subsets. Other autoantibodies that have been discovered to play a role include anti-annexin A2 antibodies, anti-phosphatidylethanolamine antibodies, and anti-lyso-bis-phosphatidic acid antibodies. A number of new diagnostic techniques such as chemiluminescence, dot blot and thin layer chromatography immunostaining are being developed to gain a better understanding of APS pathophysiology. 'Seronegative APS' is proposed to explain patients that have strong clinical manifestation of APS, with no alternative explanation and are persistently negative for the routine APS tests and this is an area of active research to identify new autoantigens.

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| Venous Thromboembolism | |
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| Monday, 4th July, 2016 | 13:30 - 13:45 |

## Cases in Venous Thromboembolism – How would you manage them?

Paul Dinnen

Gold Coast Vascular, Benowa, Gold Coast QLD, Australia

NOTES

| Venous Thromboembolism | |
|---|---|
| Monday, 4th July, 2016 | 13:45 - 14:00 |

## Balancing the Risks and Benefits of Anticoagulation

Mark Meissner

University of Washington, Washington, USA

More so than in many areas of medicine, the treatment of acute deep venous thrombosis is based upon the results of rigorously conducted randomized clinical trials in which the standard endpoints have been balancing the risks of recurrent VTE and bleeding. Based upon the results of these trials, current guidelines for the treatment of unprovoked VTE include anticoagulation for 3 months with extended anticoagulant therapy for those at low or moderate bleeding risk. The estimated risk of recurrence in such patients is 30% at 5 years, increasing 1.5 fold after a second unprovoked VTE. Despite these risks, the benefits of indefinite anticoagulation must be balanced against the risk of bleeding. Bleeding risk can be stratified as low (0 risk factors), moderate (1 risk factor), or high (≥ 2 risk factors), corresponding to an annual bleeding risk of 0.8%, 1.6% and > 6.5% during extended therapy with a vitamin K antagonist. However, recent meta-analysis suggests that the direct factor Xa inhibitors rivaroxaban and apixaban may be associated with a lower bleeding risk than the vitamin K antagonists. For those patients in whom the risks versus benefits of extended anticoagulation remain unclear, consideration of patient gender (1.75 X increased risk of recurrence in males) and D-Dimer levels after completing a 3 month course of anticoagulation may assist decision making.

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| Venous Thromboembolism | |
|---|---|
| Monday, 4th July, 2016 | 14:00 - 14:15 |

## DOACs: Simplifying VTE Management

Harshal Nandurkar

Australian Centre for Blood Diseases, Monash University, Melbourne, Australia

The availability of direct oral anticoagulants has introduced more ease and safety in VTE management. There are two DOACS currently available on PBS (apixaban and rivaxoxaban, both Xa antagonists) and dabigatran (thrombin antagonist) is registered with TGA. Inclusion of over 25,000 patients in phase 3 randomized trials has confirmed that DOACs are non-inferior compared with vitamin K antagonists (VKA) for efficacy. Moreover, DOACs have demonstrated a substantial reduction in major bleeding, intracranial bleeding and clinically-relevant non-major bleeding (CRNM). DOACs have the advantage of initiating therapy without a low molecular weight heparin bridge (apixaban and rivaroxaban). Tests such as INR for dose adjustments are not necessary. Tests for quantifying drug levels are available in major hospital laboratories but are not required for routine management. 'Antidotes' are being developed and a monoclonal antibody for very efficient dabigatran reversal was recently approved by TGA. There is ongoing clinical trial research for the use of DOACs in subgroups including cancer-associated thrombosis and antiphospholipid syndrome.

NOTES

| Venous Thromboembolism | |
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| Monday, 4th July, 2016 | 14:15 - 14:30 |

## Advanced Techniques in Iliac Venous Recanalisation

Mark Meissner

University of Washington, Washington, USA

Percutaneous angioplasty and stenting has become the standard of care for the management of chronic iliac venous obstruction. Unfortunately, the long term results for treatment of post-thrombotic obstructions are inferior to those obtained for non-thrombotic lesions. Primary and secondary patency rates of 57% and 86% have been reported for such lesions, in comparison to 79% and 100% for non-thrombotic lesions. Iliocaval venous occlusions present particular challenges including difficult crossing secondary to multiple collaterals and dense scar, long segment occlusions and pre-existing intravenous devices such as IVC filters and failed stents. Although such lesions can be treated in over 90% of cases, advanced techniques including multiple access points, specialized crossing catheters, and sharp recanalization are often required. Reconstruction of the iliac venous confluences in cases of bilateral occlusion presents particular difficulties, with double barreled stent configurations having the best primary patency (73%) and lowest incidence or re-intervention (8%). Despite the challenges, good symptom relief and acceptable mid-term patency can be achieved with these techniques.

NOTES

| Venous Thromboembolism | |
|---|---|
| Monday, 4th July, 2016 | 14:30 - 14:45 |

## Interventional Techniques for Severe Pulmonary Embolism

Ramon Varcoe

The University of New South Wales, Randwick, Australia

An overview of the current interventional strategies for the treatment of massive and sub-massive pulmonary embolus.

NOTES

| Venous Thromboembolism | |
|---|---|
| Monday, 4th July, 2016 | 14:45 - 15:00 |

## Atypical Superficial Venous Thrombosis

Pier Luigi Antignani

Vascular Center, Nuova Villa Claudia, Rome, Italy

Although superficial venous thrombosis usually occurs in the lower extremities, it also has been described in the penis and the breast (Mondor disease). Superficial venous thrombosis can also develop anywhere that medical interventions occur, such as in the arm or neck (external jugular vein) when intravenous (IV) catheters are used or in the cerebral venous system.

Superficial venous thrombosis is most often associated with one of the components of the Virchow triad; i.e., intimal damage (which can result from trauma, infection, or inflammation), stasis or turbulent flow, or changes in blood constituents (presumably causing increased coagulability).

Superficial venous thrombosis is a clinical diagnosis in which the clinician identifies tender and inflamed superficial veins. However, further testing is often required to evaluate the etiology of this condition.

Phlebitis also occurs in diseases associated with vasculitis, such as polyarteritis nodosa (periarteritis nodosa) and Buerger disease (thromboangiitis obliterans).

Superficial venous thrombosis with infection, such as phlebitis originating at an IV catheter site, is referred to as septic thrombophlebitis, a clinical entity requiring diagnostic and therapeutic approaches that are different from those applied to sterile phlebitis.

Superficial venous thrombosis following an injury usually occurs in an extremity, manifesting as a tender cord along the course of a vein juxtaposing the area of trauma. Ecchymosis may be present early in the disease, indicating extravasation of blood associated with injury to the vein; this may turn to brownish pigmentation over the vein as the inflammation resolves.

Migratory superficial venous thrombosis is defined as an entity characterized by repeated thromboses developing in superficial veins at varying sites but occurring most commonly in the lower extremity. Although numerous etiologic factors have been proposed for this condition, none have been confirmed. It has a strong association with adenocarcinoma of the pancreas and lung.

NOTES

| DEBATE: The Use of Ionising Radiation with Radiofrequency and Endovenous Laser Ablation | |
|---|---|
| Tuesday, 5th July, 2016 | 8:00 - 8:45 |

8:00 – 8:10

## Brief Introduction and Survey of the Audience

David Connor

8:10 – 8:20

## IRSA Position on the use of Ionising Radiation in Routine EVLA and RF: NO

John Vrazas

President, Interventional Radiology Society of Australia

8:20-8:30

## Use of Ionising Radiation during endovenous ablation: YES

Ramon Varcoe

The University of New South Wales, NSW Australia

08:30-08:40

## Panel Discussion

8:40 - 8:45

## Survey of the Audience

David Connor

NOTES

| Vascular Malformations | REVIEW LECTURE |
|---|---|
| Tuesday, 5th July, 2016 | 8:45 – 9:15 |

# Overview of Vascular Anomalies: Clinical and Ultrasound Diagnosis

Kurosh Parsi

Department of Dermatology, St Vincent's Hospital, Darlinghurst NSW, Australia.
Dermatology, Phlebology and Fluid Mechanics Research Laboratory, St Vincent's Centre for Applied Medical Research, Darlinghurst NSW, Australia.
The University of New South Wales, NSW, Australia.

Vascular malformations are congenital anomalies of the vascular system. These anomalies are present at birth but may not be apparent until later in life. Venous malformations (VMs) are the most common vascular malformations followed by lymphatic malformations (LMs) and capillary malformations (CMs). Arterio-venous malformations (AVMs) are fortunately the least common but the most aggressive and the most difficult to treat.

Vascular malformations may be classified into truncular and extra-truncular sub-categories. Truncular malformations arise at later stages (>3 weeks) of embryogenesis and hence involve mature vessels. Truncular LMs present with primary lymphoedema. Examples of truncular VMs include embryonic persistent Marginal vein, sciatic vein, agenesis of individual veins, valvular agenesis or hypoplasia and venous aneurysms. Extra-truncular malformations arise earlier (<3 weeks) than truncular malformations during the reticular phase of vasculogenesis and may result in anomalous arteriovenous communications (AVMs), venous spaces within other tiussues (VMs) and cystic deformities (LMs). Extra-truncular LMs are further characterized into macrocystic (cysts >1cm), micro-cystic (cysts <1cm) or mixed cystic lesions.

Duplex Ultrasound (DUS)

Every patient with a vascular anomaly should have a duplex ultrasound (DUS) study performed. Such studies should be undertaken by dedicated phlebology/vascular laboratories familiar with vascular tumours and malformations. This will avoid the need for repeat studies, un-necessary expenses and run around for patients and parents. DUS is the first line of investigation to confirm the diagnosis of a congenital vascular anomaly given its non-invasive nature, low cost, availability and ability to exclude a wide range of differential diagnoses.The aim of the DUS study is to confirm the clinical diagnosis of a vascular malformation, exclude other causes and detect co-existing anomalies.

*1. Vascular Malformation vs. Vascular Tumour*

The first question to address is whether the presenting mass is a soft tissue mass, a vascular tumour or a malformation. The majority of soft tissue masses have a non-specific ultrasound appearance. Vascular tumours are primarily a soft tissue mass whereas malformations are primarily formed of vascular spaces. B-mode can readily make the distinction based on the echogenic morphology of a tumour as against the anechoic vascular spaces of a malformation. Doppler studies will demonstrate the vascularity of a proliferating tumour such as a haemangioma but will be negative if the heamangioma has already involted. Hence, Doppler cannot be completely relied upon and B-mode characteristics are very important in making the distinction between tumours and malformations.

2. *Non-compressible Vascular Spaces: LMs vs. AVMs*

On B-mode ultrasound, macrocystic LMs appear as non-compressible anechoic cystic spaces containing and separated by thin echogenic septae. AVMs are also non-compressible and may have a 'honeycomb' appearance that can be confused with the cystic structure of LMs. AVMs however have thicker walls compared to the thin septae of LMs and the surrounding tissue may show echogenic fibrosis due to chronic trauma. Doppler studies will further characterize an AVM by demonstrating a low resistance high velocity arterial flow pattern. By contrast, LMs will show no spontaneous flow. Importantly, vascularity (pulsatile arterioles) may be evident in the septae of a LM and in the surrounding tissues and this should not be interpreted as high flow within the lesion. This is why it is important to obtain adequate B-mode information before proceeding to, and be confused by a Doppler study.

3. *Low or No Flow on Doppler: LMs vs. VMs*

VMs can be differentiated from macrocystic LMs by their relatively thicker walls and compressibility. Wall thickening may be even more prominent secondary to recurrent thrombophlebitis and localised intravascular coagulopathy (LIC), a common finding in larger VMs. Venous wall thickness in contrast to thin lymphatic septae is an important distinguishing feature on B-mode ultrasound. Compressibility is another important useful B-mode feature. Patent VMs are compressible whereas LMs are relatively non-compressible. Doppler examination should only be used to confirm the B-mode findings. LMs and small VMs may show no detectable flow on Doppler studies. Larger VMs would demonstrate low flow induced by compression. By comparison, no flow would be detected within the cystic spaces of a LM.

4. *Previously-treated or Thrombosed Lesions*

Thrombosed or sclerosed VMs will be non- or partially-compressible depending on the extent of the intra-luminal occlusion. With high resolution B-mode imaging, thrombus within a VM would appear slightly hypoechoic. Sclerosed VMs should appear echogenic unless they contain intraluminal haemolysed blood which would appear hypoechoic. Treated LMs with no discernable cyst will also be echogenic on DUS.

Magnetic Resonance Imaging (MRI)

MRI is considered mandatory in all patients with vascular malformations. Macrocystic LMs show hyperintense signal in T2 images and low intensity signal in T1 images, with post-contrast enhancement of the septa. Microcystic lesions generally appear as T2 images with homogeneous hyperintense signal. Non-enhancement and fluid-fluid levels from intracystic haemorrhages are characteristic features of LMs whereas enhancement of the vascular space, presence of phleboliths and accompanying venous channels would define VMs. MR and CT Angiography (MRA and CTA) are used to confirm the duplex diagnosis of AVMs. MRA is a better modality to diagnose AVMs of the soft tissue in comparison to CTA which is better in diagnosing lesions involving bone, bowel or lung. CT is also an alternative to MR in patients with cardiac or respiratory failure due to faster image acquisition and in patients who have contraindications to sedation or MR. Time-resolved MRA is more likely to identify an AV communication compared with CTA which is not time-resolved. The hallmark of AVMs on MRA is early venous filling proportional to the severity of the anomalous AV connections.

| Vascular Malformations | |
|---|---|
| Tuesday, 5th July, 2016 | 9:15 – 9:30 |

## ISVI-IUA Consensus. Diagnostic Guidelines of Vascular Anomalies: Vascular Malformations and Haemangiomas

Pier Luigi Antignani

Vascular Center, Nuova Villa Claudia, Rome, Italy

With the rapid proliferation of descriptive terms and eponyms, the need to develop a meaningful classification of vascular anomalies became evident. The milestone was the development of the Hamburg classification. This classification was soon modified to incorporate the newly discovered embryological findings.

The management of these lesions is often complex. The relatively low incidence of vascular anomalies among general population combined with the fact that their management often falls within the purview of several different medical and surgical specialties has traditionally resulted in insufficient expertise in the management of these conditions. For this reason it is necessary to provide guidelines for the diagnosis of vascular anomalies.

The diagnostic algorithm used in the evaluation of vascular anomalies should be based on an accurate clinical assessment, which includes a thorough history and a detailed physical examination. The diagnostic approach to vascular anomalies should include the distinction between vascular tumors (i.e. hemangiomas) and congenital vascular malformations (CVMs). This step is based more on history and clinical examination rather than on instrumental evaluation. In children Duplex ultrasound and histology can be helpful to separate hypervascularized tumors from CVMs.

Appropriate record of objective measures as size or flow volume is required in order to evaluate the progress of the pathology and/or to assess the results of adopted therapeutic interventions. The anatomic, pathological and hemodynamic characteristics, the secondary effects on the surrounding tissues and the systemic manifestations should be defined. Basic diagnostic tools are Duplex sonography followed by MRI or CT scanning.

Diagnostic investigations are best undertaken at centers where subsequent therapeutic interventions will be performed. The treatment is based on a multidisciplinary approach including plastic surgery, vascular surgery and sclerotherapy. Actually, the foam sclerotherapy is the best method to treat the low flow malformations as venous and lymphatic ones.

NOTES

| Vascular Malformations | |
|---|---|
| Tuesday, 5th July, 2016 | 9:30 – 9:45 |

## Embolisation of Arteriovenous Malformations

John Vrazas

Melbourne Institute of Vascular and Interventional Radiology

NOTES

| Pelvic and Vulvar Veins | REVIEW LECTURE |
|---|---|
| Tuesday, 5th July, 2016 | 10:30 – 11:00 |

## Pelvic and Vulvar Veins - Anatomy and an Approach to Treatment

Louis Grondin

Since Hoeltgen and later Hobbs initially described the Pelvic congestion syndrome (PCS), many diagnostic and treatment approaches have been proposed for this condition. Nevertheless, the condition remains ill-defined and is still a relative new comer to medical disease states. PCS found a welcomed yet marginal home in Phlebology as a vascular condition, and found no-home whatsoever in gynecology where it is paradoxically quite common. Described as a cause of recurrent leg varices following surgery, It is still a source of confusion in the medical literature.

Today the PCS is described as chronic pelvic pain (CPP) arising from dilated and refluxing pelvic veins, although the causal relationship between pelvic vein incompetence (PVI) and CPP is still poorly established. Non-invasive screening methods such as Doppler ultrasound and magnetic resonance venography are used before confirmation by venography. Percutaneous embolization has become the principal treatment for PCS, with high success. Although there has been variation in approaches between investigators, the goal is the elimination of ovarian vein reflux with or without direct sclerosis of enlarged pelvic varicosities. Symptom reduction is seen in 70 to 90% of the treated females despite technical variation. Associated conditions may present with the PCS, such as the Nutcracker syndrome, Iliac vein occlusion (May-Thurner Syndrome) or insufficiency, and pelvic tumors. Therefore MR Venography is perhaps the best initial test to diagnose the PCS, as it can effectively uncover these occult conditions.

In the absence of PCS, literature review shows that embolization is not essential in the treatment of leg varices of pelvic origin. Foam sclerotherapy or phlebectomy offer good results in patients with vulvar or pudendal varicose veins.

NOTES

| Pelvic and Vulvar Veins | KEYNOTE LECTURE |
|---|---|
| Tuesday, 5th July, 2016 | 11:00 – 11:30 |

## Management of Pelvic Congestion Syndrome – Sense and Nonsense

Mark Meissner

University of Washington, Washington, USA

Chronic pelvic pain accounts for approximately 10% of outpatient gynecologic visits and among the varied causes, pelvic congestion syndrome is second only to endometriosis in frequency. Manifestations may include pelvic pain, dyspareunia, dysuria, and dysmenorrhea as well as external varices and a number of psychosocial symptoms. Although a variety of treatments have been proposed - including pharmacologic ovarian suppression; hysterectomy with or without oophorectomy; and ovarian vein resection - transcatheter embolization is the least invasive and most efficacious management option. Complete or partial symptom improvement has been reported in 68.2 – 100% of patients and there has been a consistent reduction in visual analog pain scores after treatment. Based upon these results, recommendation of either pharmacotherapy or other surgical procedures is difficult to justify. However, it is also clear that 6% - 31.8% of patients do not get substantial relief from pelvic venous embolization. Potential explanations for an inadequate response to treatment include patient variability, procedural variability, and inadequate outcome measures. The latter are particularly important and future investigation should focus on the development of disease-specific quality of life measures as well as identifying those aspects of the procedure, such as choice of embolic agents and extent of embolization, associated with the best clinical outcomes.

NOTES

| Pelvic and Vulvar Veins | |
|---|---|
| Tuesday, 5th July, 2016 | 11:30 – 11:45 |

## Embolisation of Ovarian Veins

John Vrazas

Melbourne Institute of Vascular and Interventional Radiology

NOTES

| Pelvic and Vulvar Veins | |
|---|---|
| Tuesday, 5th July, 2016 | 11:45 – 12:00 |

## Embolic Coils for Ovarian Veins: Which ones to choose?

Sanjay Nadkarni

Endovascular WA, Claremont WA, Australia

NOTES

| Guided Poster Presentations | |
|---|---|
| Tuesday, 5th July, 2016 | 13:30 – 13:40 |

# Apixaban versus enoxaparin in the prevention of venous thromboembolism following total knee arthroplasty: A single centre, single surgeon, retrospective analysis

**Danika King**[1], Richard Pow[1], David Dickison[2], Peter Vale[1,3]

[1] School of Medicine, The University of Notre Dame, Sydney, Australia,
[2] Department of Orthopaedic Surgery, Mater Hospital, North Sydney, Australia,
[3] Department of Cardiovascular Medicine, Mater Hospital, North Sydney, Australia

Background: There is a high risk of developing venous thromboembolism (VTE), following total knee arthroplasty (TKA). Conventional thromboprophylactic agents have limitations, such as route of administration, the need for monitoring, narrow therapeutic windows, and interactions. Apixaban is a new oral anticoagulant with the potential to overcome these limitations.

Objectives: To report the efficacy and safety of apixaban and a low molecular weight heparin, enoxaparin, in VTE prophylaxis following TKA.

Methods: This single centre, single surgeon, retrospective analysis included 506 consecutive patients who underwent TKA between 2009 and 2015 and received enoxaparin or apixaban as thromboprophylaxis. Baseline characteristics of patients, in-hospital rates of VTE, total DVT, proximal or distal DVT, PE, bleeding outcomes and mortality were compared between the two groups.

Results: In-hospital VTE occurred in 22 (8.9%) patients in the enoxaparin group and 11 (4.5%) patients in the apixaban group (p=0.049). Nine (3.6%) patients in the in the enoxaparin group and 1 (0.4%) in the apixaban group experienced a post-operative drop in haemoglobin ≥ 20 g/L-1 that either necessitated transfusion of ≥2 units of blood, caused haemodynamic instability, or both (p=0.020). Thirty-five patients experienced other bleeding events, with 25 (9.9%) in the enoxaparin group and 10 (4.0%) in the apixaban group (p=0.009). There were no statistically significant differences in rates of total DVT, proximal or distal DVT, PE, or mortality between the groups.

Conclusions: Compared with enoxaparin, thromboprophylaxis with apixaban resulted in a lower VTE incidence and fewer haemorrhagic complications.

NOTES

| Guided Poster Presentations | |
|---|---|
| Tuesday, 5th July, 2016 | 13:40 – 13:50 |

## Onyx Embolisation of a Digital AVM

Anthony Trimboli, Lourens Bester, Kurosh Parsi

St Vincent's Hospital, Darlinghurst NSW Australia

Objectives: To demonstrate a case of treating a peripheral arteriovenous malformation (AVM) with Onyx, and the challenges involved in the treatment of these lesions.

Methods: We present a case of 66 year old female with a peripheral AVM of her third digit for which management had been previously attempted with the insertion of a vascular coil. Following characterization of the lesion with spectral Doppler, and confirmation of details of the lesion via angiogram, Onyx was infused into the nidus of the lesion until occluded. Onyx was chosen as the embolization agent as it allowed infusion of the agent at different stages through the microcatheter without losing access.

Results: The lesion was effectively embolised and the AVM substantially reduced in size both clinically and sonographically. In addition to occluding the nidus of the AVM, onyx also occluded the arterial supply to part of the digit, resulting in intractable finger pain and dry gangrene and necrosis of the tip of the finger. After unsuccessful attempts to manage the pain, the distal digit was amputated surgically. On follow up, the patient reported no pain of the digit, and an improved level of function compared with before the procedure.

Conclusions: This case highlights the challenges, potential complications and treatment options that can be involved with the management of peripheral AVM's.

NOTES

| Guided Poster Presentations | |
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| Tuesday, 5th July, 2016 | 13:50 – 14:00 |

## Aggressive Percutaneous Pharmacomechanical Thrombolysis for Extensive Proximal Lower and Upper Extremity Deep Vein Thrombosis with Angiojet; Safety and Feasibility – A Case Series.

Farshid Niknam, Laurencia Villalba, Tam Nguyen, David Huber

Wollongong Hospital, Wollongong, Australia

Objectives: Venous lysis is usually reserved for symptomatic patients with acute deep vein thrombosis (DVT) and a low risk of bleeding. The aim of this study is to report cases involving the successful use of pharmaco-mechanical thrombectomy (PMT) for DVT. Endovascular removal of intra-vascular thrombus using the AngioJet rheolytic thrombectomy (RT) system has been shown to be clinically effective 1. The RT system also permits the concomitant infusion of thrombolytic agents in conjunction with mechanical thrombectomy, thus creating a strategy known as pharmaco-mechanical thrombectomy (PMT).

Methodology: In this study we reviewed 10 cases where an extensive DVT (6 ilio-popliteal or ilio-femoral DVT and 4 subclavian and axillary vein DVT) was treated using PMT at the Wollongong Hospital in NSW, Australia. The average time of DVT symptoms till intervention was 15.4 days. For all cases we utilized urokinase as our chemical thrombolytic, with an average dose of 565,000 units (minimum 250,000 units to a maximum of 1,000,000 units) with 30 minutes of dwell time before mechanical thrombectomy. Venography post-mechanical thrombectomy revealed severe luminal stenosis in all cases. Five out of six patients with ilio-femoral-popliteal DVT proceeded to venous stenting. All four subclavian DVT were because of thoracic outlet syndrome (TOS), and these patients underwent first rib resection on the next available list.

Results: All of the proximal extensive thrombosis were successfully managed with PMT and their follow up DUS) showed venous patency with no significant residual stenosis or thrombosis. Clinical symptoms significantly improved within 24 hours post-intervention. One patient developed an acute renal failure which required hemodialysis. None of the patients experienced bleeding or clinical PE.

Conclusion: Our experience shows that percutaneous pharmaco-mechanical thrombectomy with the Angiojet device is a safe and feasible option for operative management of extensive proximal DVT.

NOTES

| Guided Poster Presentations | |
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| Tuesday, 5th July, 2016 | 14:00 – 14:10 |

## Retrieval of IVC and Mechanical Thrombectomy with Pharmoco-kinetics Directed Thrombolysis and Repair of Rupture IVC

Gagandeep Kaur, Laurencia Villalba

Wollongong Hospital, Wollongong, Australia

A 64 yrs old lady presented to hospital two days post-septoplasty with PE secondary to DVT. Admitted to ICU had severe epistaxis and hypotension. Anticoagulation was stopped and IVC filter inserted.

Nine months later patient presented to Vascular surgeon with significant PTS and bilateral venous claudication.

Patient underwent Pharmaco-Mechanical Thrombectomy (Angiojet) in mid January 2016 of IVC Thrombus and attempted removal of IVC filter. Retrieval failed due to high burden of thrombus.

Patient was re booked to have the IVC filter removed with different techniques and once again had pharmaco- mechanical thrombectomy with Angio jet of IVC and right ilio-femoral system.

In the attempt to remove the embedded IVC filter the IVC ruptured and was repaired with a covered stent. The right iliac system was stented with Veniti (venous stents).

She was discharged 5 days later with no complications.

In follow –up the patient has had a full recovery, can walk 1 km with no pain and the edema in the right leg is resolved, the left leg has minor residual edema.

NOTES

| Guided Poster Presentations | |
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| Tuesday, 5th July, 2016 | 14:10 – 14:20 |

## Livedo Racemosa Secondary to Cutaneous Microcalcifications: A diagnostic challenge

Pooja Kadam[1,2], Steven Kossard[3], Kurosh Parsi[1,2]

[1] Department of Dermatology, St Vincent's Hospital, Darlinghurst NSW, Australia.
[2] Dermatology, Phlebology and Fluid Mechanics Research Laboratory, St Vincent's Centre for Applied Medical Research, Darlinghurst NSW, Australia
[3] Sydney Skin Hospital, Sydney NSW, Australia

A 44-year-old beautician presented with an eighteen-month history of a reticular eruption affecting her trunk and four limbs. Cold temperature resulted in increasing erythema. Medical history was significant for a patent foramen ovale, depression, recurrent sinusitis with nasal cautery, breast augmentation (ruptured once) and miscarriages. Regular medications included escitalopram and loratidine. She had a sister with dermatitis and brother and daughter with asthma.

Examination of the skin revealed a partially blanchable, violaceous, branched reticulate eruption on the trunk and four limbs consistent with livedo racemosa. Blood tests revealed MTHFR polymorphism with a normal homocysteine, positive cold agglutin screen, ANA 1:640, iron 8µmol/L, transferrin 2g/L and positive EBV IgG serology. Right posterior thigh punch biopsy revealed minimal perivascular infiltrate in the superficial and mid dermal region. Eosinophils were scant with mild dermal sclerosis.

High frequency probe ultrasound (Toshiba Aplio V, 18-7 MHz linear transducer) of the livedo racemosa affected regions was performed. It demonstrated cutaneous microcalcifications within the dermis producing linear shadows extending down to the subcutaneous layer. Von Kossa staining of previous biopsy specimens revealed positive fine granular staining for calcium.

Livedo racemosa is a rare disease characterised by persistent violaceous netlike patterning, mottled discoloration of the skin largely affecting the arms and legs with occasional buttocks and trunk involvement. It is always pathological and permanent. No underlying cause was established in our patient despite extensive investigations. The role of ultrasound in the diagnosis of livedo racemosa is a novel concept. The cutaneous microcalcifications appear to play an important role in the pathogenesis of livedo racemosa. Given that no underlying cause has yet been established, it is judicious to postulate that the livedo racemosa is secondary to cutaneous microcalcifications.

NOTES

| Guided Poster Presentations | |
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| Tuesday, 5th July, 2016 | 14:20 – 14:30 |

## Venous Thromboembolism

George Nicola

Ashton Medical Practice, Rockdale, Australia

Introduction: Venous thromboembolism (VTE) is considered the 3rd most common cardiovascular cause of death in Australia. This case highlights the importance of primary and secondary prevention when contemplating both surgical and phlebological procedures.

Case: 28 year old female patient who presented for assessment and management of her bilateral legs varicose veins. She gives a 4 years history of bilateral symptomatic varicose veins of the great saphenous system starting to be noticed during her first pregnancy in 2012. She had one leg treated surgically in 2014.

The case is complicated by a recent pulmonary embolism (PE) 11 days post abdominoplasty in November 2015. She had then just started oral contraception (OC) for 1 month and stopped it 1 week prior to the operation. She is overweight, non-smoker and has a history of typical migraine attacks. She has a strong family history of varicose veins but no prior history or family history of VTE.

This case shows the importance of a systematic thorough history and examination prior to any varicose vein procedure. Preventative VTE management plan should be implemented as part of the treatment plan according to each individual's risk stratification.

NOTES

| Guided Poster Presentations | |
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| Tuesday, 5th July, 2016 | 14:30 – 14:40 |

## Detergent Sclerosants Induce Cellular Apoptosis

**Osvaldo Cooley**, David Connor, Kurosh Parsi

Dermatology, Phlebology and Fluid Mechanics Research Laboratory, St Vincent's Centre for Applied Medical Research, Darlinghurst NSW, Australia
The University of New South Wales, NSW, Australia

Objectives: To investigate the effects of detergent sclerosants sodium tetradecyl sulphate (STS) and polidocanol (POL) on human leukocytes and endothelial cells at sub-lytic concentrations.

Materials and methods: Human Umbilical Vein Endothelial Cells and human leukocytes were labelled with antibodies to assess for apoptosis and oncosis by fluorescence microscopy and flow cytometry. Cell viability and membrane integrity were assessed using trypan blue, fluo-3 and propidium iodide (PI) staining. Phosphatidylserine (PS) exposure (apoptosis) was identified by flow cytometry using lactadherin. Caspase 8 expression was used as a marker of the extrinsic pathway of apoptosis and Bax for the intrinsic pathway. Porimin expression was used to assess oncosis.

Results: Up to 40% of leukocytes and endothelial cells maintained membrane integrity at sub-lytic concentrations (≤0.15%) of sclerosants. The remaining 60% did not maintain membrane integrity but were not completely lysed. PS exposure was increased with both STS and POL exhibiting a dose- and time-dependent trend. Expression of both Caspase 8 and Bax was increased in both leukocytes and endothelial cells treated with STS while those exposed to POL expressed increased Bax only. Both agents increased the leukocyte expression of porimin at 0.075%. On fluorescence microscopy, stains for Caspase 8 and Bax were slightly increased for STS and only Bax was increased for POL. Porimin stain was markedly positive for both STS and POL.

Conclusions: Both sclerosants induced leukocyte and endothelial cell apoptosis and oncosis at sub-lytic concentrations. STS activated both extrinsic and intrinsic pathways of apoptosis while POL stimulated the intrinsic pathway of apoptosis only. Both agents stimulated the porimin pathway of oncosis.

NOTES

| Global Sclerotherapy | |
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| Wednesday, 6th July, 2016 | 8:00 – 8:15 |

## Sclerotherapy in Australia and New Zealand: What has been our contribution?

Paul Thibault

Central Vein and Cosmetic Centre, Broadmeadow, Australia

Over the past 30 years Australian and New Zealand sclerotherapists have pioneered, published, established and formalised techniques and methods in sclerotherapy that have become mainstream throughout the world. These techniques have included pre-treatment ultrasound mapping, ultrasound-guided sclerotherapy, ultrasound monitoring post-treatment, methods to reduce adverse effects and refinements such as peri-venous compression to improve results.

At a fundamental scientific level, Australian phlebology researchers have explored the physiological qualities of sclerosant solutions to an advanced level possibly never seen before in the field of Phlebology.

These advances have been able to be established as mainstream theory and methods through formalised education and training programs developed by the Australiasian College of Phlebology, that are now being adopted by the International Union of Phlebology and other Phlebology Societies and Colleges.

These achievements will be detailed in this concise lecture.

NOTES

| Global Sclerotherapy | |
|---|---|
| Wednesday, 6th July, 2016 | 8:15 – 8:30 |

## Glycerin and other foam stabilisers - how should we use them?

Louis Grondin

Aqueous foams are by definition a dispersions of gas in liquid, stabilized by surfactant at the air–liquid interface. In the short-run foam-sclerotherapy has an impressive sclerotic effect on saphenous and non-saphenous varices with minimal effect on peripheral blood. A renewed interest in Ultrasound guided Foam sclerotherapy is resurging in view of the favorably low (0.8%) risk of post procedure DVT compared to radiofrequency ablation (4.4%), multiple same-day-therapies (3.4%); laser ablation (3.1%), and surgery (2.4%).

Aqueous foam sclerosants are however, inherently unstable, and evolve over time by gravitational drainage of liquid, to coarsening, cavitation, and film-rupture. Foam decay is felt to be responsible for gas embolism and the neurologic complications such as transient visual disturbances and transient confusional states that have been widely reported.

The efficacy and quite possibly the safety of foam sclerotherapy ultimately depends on the quality and stability of foam. Thus the never-ending and maddening quest for methods of creating a stable and durable foam. Foam sclerosants are more stable when produced at lower temperatures. Foam stability if further influenced by the choice of the sclerosants, it's concentration and the air-liquid fraction.

The addition of small concentrations of nonionic surfactants such as of glycerin, Hyaluronic acid, contrast agent (iopromide), and, when inducing fallopian tube sclerosis Benzalkonium chloride, have proven successful in increasing foam stability, postponing cavitation and decay *in vitro*. It's ultimate effects on treatment outcome, and improved safety, are still far from evident.

NOTES

| Global Sclerotherapy | |
|---|---|
| Wednesday, 6th July, 2016 | 8:30 – 8:45 |

## Brazil - 75% Dextrose - Is it a good sclerosant

Angelo Scuderi

Flebologia Brasil, Sorocaba, Sao Paulo, Brazil.

Many drugs have been used to perform sclerotherapy. Among the osmotic stands out hypertonic dextrose at 50 or 75%. The use of this drug is very common in Brazil. It may be useful in the treatment of small reticular veins or telangiectasias. It can be classified as a weak sclerosant. Among its main advantages we can point out that it hardly cause scar or pigmentation and there is no risk of allergic shock. However, often have sub results, forcing the reinjection in the same vein. The concentration of 75% provides increased power, though the expanded viscosity makes it difficult to use a thin needle. In addition, injections are painful often requiring the addition of local anesthetics. In larger veins it is completely inefficient. Since there are practically no studies published about the efficiency or inefficiency of dextrose, my conclusions are based solely on empirical in my personal experience of forty years. I've used Dextrose and already abandoned for nearly 20 years. I do not consider Dextrose a good sclerosant.

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| Global Sclerotherapy | |
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| Wednesday, 6th July, 2016 | 8:45 – 9:00 |

## Sclerotherapy in Argentina

Miguel Huaman

NOTES

| Global Sclerotherapy | |
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| Wednesday, 6th July, 2016 | 9:00 – 9:15 |

## Impact of Foam Sclerotherapy Upon Respiratory System and Central Hemodynamics in an Animal Model

Zbigniew Rybak, M Janeczek, A Noszczyk-Nowak, M Dobrzyński, M Szymonowicz

Wroclaw Medical University, Wroclaw, Poland

Objective: The aim of the study was to assess the impact of foam sclerotherapy upon respiratory system and central hemodynamics in sheep.

Material and methods: In nine sheep underwent procedure of injecting 5 ML of polidocanol foam (PF) into external jugular vein. ECG was performed with SCHILLER AT-1 Echocardiography was performed with TERASON. Left atrium volume, flow velocity in pulmonary artery, volume of right and left ventricle, thickness of ventricle wall and septum in systole and diastole were assessed. All these parameters as well as breathe action of animals were calculated before injection of PF –T0 after 3-5 min following injection – T1, after 10-15 min – T2, and 20-25 min. – T3.

Results: Disturbances in breathe action were observed in 6 out of 9 sheep as an apnoea. After 30-180 sec. breathing return spontaneously. Tachycardia 240/min. was in 2 animals. Important lowering of ST segment in 3, elevation of ST segment in 1, inverting T wave in 1. In two sheep ECG trace was normal. There were no statistical changes in heart rate (102+/-22.68 vs. 138+/-73,18) and volume of left ventricle during systole (p=n.s.). It has been pointed out increase in volume of right ventricle T1 compare to T0 and return to initial value in T3 (p=0,00057). It has been found negative correlation between flow increase in pulmonary artery and volume of right ventricle (r= -0.31, $p<0.05$). There was statistical significant decrease in shortening fraction of left ventricle in T1, T2 compare to T0 and return to initial value in T3 (p=0,0052).

Conclusion: The study have pointed out that FS has impact upon breathe action and circulation hemodynamics in sheep. These results should be taken into consideration in all cases of human sclerotherapy when adverse effects have taken place.

NOTES

## Microscopic Examination of Sclerocoagulum: What is Trapped Blood?

**Osvaldo Cooley**, David Connor, Kurosh Parsi

Dermatology, Phlebology and Fluid Mechanics Research Laboratory, St Vincent's Centre for Applied Medical Research, Darlinghurst NSW, Australia
The University of New South Wales, NSW, Australia

Objectives: The aim of this study was to determine the microscopic characteristics and structural composition of *ex-vivo* coagulum/trapped blood post-sclerotherapy.

Methods: Coagulum/trapped blood was identified and extracted with a 20mL syringe Samples were stained for fibrinogen and analysed with fluorescence microscopy or dehydrated and coated in gold palladium and analysed by scanning electron microscopy.

Results: On fluorescence microscopy fibrin strands in trapped blood appeared to be thinner than the strands found in spontaneous thrombus samples. Trapped blood displayed a disorganised mesh-like pattern. On scanning electron microscopy, a disorganised pattern was evident. There was a small number of clusters of platelets and multiple polyhedrocytes generated during the platelet contraction stage of the clot. There were also multiple debris and structures resembling casts of cells.

Conclusions: In conclusion, coagulum/trapped blood seen after sclerotherapy shares similarities with spontaneous thrombus formed in superficial veins. Trapped blood contains a vast number of polyhedrocytes confined into the fibrin strands. They also present a reduced number of clusters of platelets. However, the distribution of the fibrin strands is different showing a disorganized, mesh-like pattern and the strands seem to be thinner. There were also an increased number of cast structures that have not been described previously.

NOTES

| Chronic Venous Disease | KEYNOTE LECTURE |
|---|---|
| Wednesday, 6th July, 2016 | 10:30 – 11:00 |

## Update on Genetic Markers of Varicose Veins and Venous Insufficiency

Andre Van Rij, Greg Jones, Gerry Hill, Jo Krysa, Victoria Phillips

University of Otago, New Zealand

Objective: Describe new development in genetics of varicose veins

Method: There has been little progress in identifying genetic markers of varicose vein and venous insufficiency which have been validated by other research groups. We have previously recommended that an international collaborative approach be taken with workers in venous genetics to carryout genome wide association studies (GWAS) to make some headway. These require very large numbers of patients to identify genes that are relevant to venous disease. Attempts to do this are being made.

The commercial genetics company '23andMe' Inc. have an extremely large population of subjects using their service to provide their individual whole genome analyses. The subjects (92,666) were asked whether they had varicose veins or not. From this analysis of 13.7M imputed SNPs, '23andMe' have posted twelve possible SNP locations which are associated with this self declared status of varicose veins.

Results: We have tested those SNPs that had the highest probability, in 1697 patients with varicose veins and a similar sized cohort of controls. One of these was validated to be associated with varicose veins ($p< 5x10^{-7}$) and with an odds ratio that is related to the severity of disease. The other SNPs did not.

Conclusion: This SNP is the first one validated to be associated with varicose veins and appears to have a relationship to severity of venous insufficiency. The role of the SNP in venous development is to be determined.

NOTES

| Chronic Venous Disease | |
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| Wednesday, 6th July, 2016 | 11:00 – 11:15 |

## An Approach to Diagnosis and Management of Panniculitis

Afsaneh Alavi

University of Toronto, Toronto, Canada

Panniculitis or inflammation of the subcutis encompasses a wide range of disease processes. Patients with panniculitis commonly develop deep ulcers requiring wound care expertise. The diagnosis of panniculitides is difficult because the clinical presentation is often nonspecific and the histopathological changes vary. The proper biopsy is required to render a diagnosis with certainty.

The histopathological diagnosis is usually based on determination of the predominant location of the cellular infiltrate and the presence of a coexisting vasculitis. The aim of this presentation is to provide a review of cases seen in wound clinics.

Objectives:

- Review different types of panniculitis
- Discuss the common presentations of cases with panniculitis in wound clinic

NOTES

| Chronic Venous Disease | |
|---|---|
| Wednesday, 6th July, 2016 | 11:15 – 11:30 |

## Non-invasive Evaluation of Vascular Leg Ulcers

Pier Luigi Antignani

Vascular Center, Nuova Villa Claudia, Rome, Italy

Chronic vascular leg ulcers typically manifest as arterial, neurotrophic, or venous ulcers. They are distinct with regard to their location, appearance, bleeding, and associated pain and findings. it is mandatory to evaluate the patient to define correctly the etiology of lesion.

Ankle-brachial indices (ABIs) and toe digital pressures with pulse volume recordings can provide good clues to the perfusion of the foot.

Transcutaneous oxygen tension ($TcPO_2$) may be measured; however, a wide discrepancy exists with the minimal level below which wound healing does not occur. Most agree that a pressure of 30-35 mm Hg is sufficient for healing of more than 90% of wounds. Regarding the arterial ulcers, when noninvasive tests as reveal unacceptable pedal perfusion, perform imaging studies of the lower extremity to identify the level of obstruction and to evaluate the distal runoff.

Perform angiography when visualization of the vessels of the lower extremities is desired. Actually, Magnetic resonance angiography (MRA) is performed when evaluating lower extremity disease.

Imaging tests for venous disease can also reveal important preoperative issues.

Doppler duplex scanning can detect venous reflux with a very high sensitivity. Some authors suggest that combining duplex scanning with air plethysmography helps differentiate severe venous disease from mild venous disease.

Ascending venography also may be considered to obtain detailed anatomic information. This study can reveal axial channel patency, perforator incompetence, obstruction, and the presence of deep venous thrombosis.

If an ulcer is recurring, etiology is unclear, and all invasive and noninvasive studies have been performed, a biopsy is essential to establish a diagnosis and further understand the etiology of the disease. As always, management of chronic wounds can be improved by understanding the true etiology and therefore treating the underlying problem.

NOTES

| Chronic Venous Disease | |
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| Wednesday, 6th July, 2016 | 11:30 – 11:45 |

## Approach to Atypical Wounds

Afsaneh Alavi

University of Toronto, Toronto, Canada

Atypical ulcers have a wide differential diagnosis that often creates difficult diagnostic and treatment challenges. These ulcers may present with features that clinicians not previously encountered or uncommon ulcers may appear in typical places and cause phenotypic mimicry. The underlying disease in atypical ulcers heightened urgency to achieving an accurate diagnosis and initiating appropriate therapy.

Atypical ulcers have different aetiologies including vasculitis, vasculopathy, infections and malignant ulcers. The prevalence of malignancy in patients with lower extremity ulcers continues to rise, representing a significant health concern for patients and healthcare systems globally. This presentation highlights some clinical scenarios of atypical ulcers and approach to these conditions

Objectives:

- Formulate an approach to management of atypical wounds
- Discuss some scenarios presenting to wound clinic

NOTES

| Chronic Venous Disease | |
|---|---|
| Wednesday, 6th July, 2016 | 11:45 – 12:00 |

## Foam Sclerotherapy in the Elderly Patients with Severe CVD

Pier Luigi Antignani

Vascular Center, Nuova Villa Claudia, Rome, Italy

The increase in the average age of the general population has caused a continuous increase in the occurrence of severe Chronic Venous Disease among the elderly with serious effects on the quality of life. Ultrasound guided foam sclerotherapy appears to be the most promising alternative to surgery as it is minimally invasive and because of its reduced cost and favourable safety profile.

Between December 2005 and December 2015 we performed ultrasound-guided foam sclerotherapy in 104 patients with C4-C6 (CEAP classification) CVD, with a mean age of 73.8 years (range 68-85). All patients were evaluated before and after treatment and every year for 8 years follow up through the Venous Severity Score System (VSSS) and quality of life questionnaire (SF12). 46 had been suffering from one or more leg ulcers (C5 - CEAP). At the end of treatment, all patients were followed up with objective clinical exams, CDU, VCSS, VDS and SF12 questionnaire at 6-12- months and each year thereafter.

During the 6-84 months follow-up period (mean/average, 31.9 months) symptoms improved or disappeared in all patients. Ulcer healing was observed in 38 out of 46 patients with an average treatment time of 3.7 months. VCSS improved from a baseline value of 13.6 to an after-treatment value of 4.1, $p<0.001$. The statistical evaluation of the SF12 has showed the improvement of the quality of life for both the physical and mental component.

All patients expressed their gratitude and a high level of satisfaction for the functional, clinical improvement after the treatment, especially patients with a more severe CVD (C4-C6) presented significant improvement in their quality of life (SF12).

NOTES

| Venous Syndromes: Update in Diagnosis and Management | |
|---|---|
| Wednesday, 6th July, 2016 | 13:30 – 13:45 |

## The Nutcracker Syndrome – Laparotomy or Stent

Mark Meissner

University of Washington, Washington, USA.

As asymptomatic compression of the left renal vein is common, treatment should not be based on radiographic findings alone. Many patients with minor symptoms, microscopic hematuria, or intermittent gross hematuria without anemia will stabilize or improve without specific treatment. Conservative management may be particularly important in children, in whom renal vein hemodynamics may change with growth and the development of collaterals and visceral fat. However, intervention may be warranted in patients with severe or persistent hematuria and in those with lifestyle limiting symptoms. A variety of surgical and endovascular approaches to treatment have been described, although both have limitations. Left renal vein transposition has been the most common surgical approach, and although small series have reported relief of flank pain and hematuria in 90 to100% of patients, relief of associated pelvic symptoms has often been less complete and anatomic challenges such as persistent compression may be encountered. Not surprisingly, there has been recent enthusiasm for endovascular treatment of the nutcracker syndrome with self-expanding stents. Although relief of flank pain and hematuria has been reported to be equivalent to renal vein transposition, there are similarly challenges related to stent migration and incomplete relief of compression. It is likely that treatment approaches will continue to evolve with advances in endovenous techniques and improved stent technology.

NOTES

## Restless Leg Syndrome - Does It Really Exist?

Pier Luigi Antignani

Vascular Center, Nuova Villa Claudia, Rome, Italy

Restless legs syndrome (RLS) is a neurologic movement disorder of the limbs that is often associated with a sleep complaint. RLS can lead to significant physical and emotional disability. Patients with RLS may report sensations, such as an almost irresistible urge to move the legs, that are not painful but are distinctly bothersome.

Symptoms occur at least 3 times per week and have persisted for at least 3 months. They cause significant distress or impairment in social, occupational, educational, academic, behavioral or other areas of functioning.

The symptoms cannot be attributed to another mental disorder or medical condition (e.g., Chronic venous disease, leg edema, arthritis, leg cramps) or behavioral condition (e.g. positional discomfort, habitual foot tapping).

The disturbance cannot be explained by the effects of a drug of abuse or medication.The RLS is not a symptom of chronic venous disease but it appears in patients with CVD more frequently than other.

All patients with symptoms of RLS should be tested for iron deficiency. At a minimum, a ferritin level should be obtained, although a complete iron panel is preferable (iron levels, ferritin, transferrin saturation, total iron binding capacity).

Other studies can be useful as Needle electromyography and nerve conduction studies (should be considered if polyneuropathy or radiculopathy is suspected on clinical grounds, even if the results of the neurologic examination are apparently normal) and polysomnography.

Drug therapy for RLS is largely symptomatic. Medications used in the treatment of RLS include: Dopaminergic agents, Benzodiazepines, Opioids, Anticonvulsants,Presynaptic alpha2-adrenergic agonists, Iron salt.

In general, physical measures are only partially or temporarily helpful and should be avoided before bedtime. Some patients benefit from different physical modalities before bedtime, such as a hot or cold bath, a whirlpool bath, limb massage, or vibratory or electrical stimulation of the feet and toes.

NOTES

| Venous Syndromes: Update in Diagnosis and Management | |
|---|---|
| Wednesday, 6th July, 2016 | 14:00 – 14:15 |

## Popliteal Vein Compression

Richard Harris

Kuring-gai Vascular Ultrasound, NSW, Australia

Popliteal vein compression syndrome is a poorly understood and managed but very common condition that will be encountered by vascular specialists who are aware of the condition every week that they consult. There is a clear clinical syndrome that is backed up by straightforward and reproducible ultrasound findings. The patient is usually obese and often has venous hypertensive changes of considerable significance without demonstrable deep vein reflux or even superficial venous reflux. The patient avoids having the leg in the straight locked position for any period of time and acceleration of venous hypertensive changes may be seen when superficial reflux is dealt with without decompression of the affected popliteal segment. Significant symptoms of fullness, throbbing and pain are often encountered. Frank ulceration is also seen.

A new procedure will be described which has been shown to be effective for the condition and has replaced a much more invasive but equally technically effective procedure which was a standard approach for decompression.

NOTES

# May-Thurner Syndrome

Mark Meissner

University of Washington, Washington, USA

May and Thurner were the first to systematically evaluate lesions at the crossing of the right common iliac artery and the left common iliac, describing "spurs" at the arterial crossing in 24% of cadavers. A remarkably similar 24% of patients undergoing CT scanning for abdominal pain have been found to have > 50% compression of the L common iliac vein. The incidence is even higher among patients with advanced venous disease, cross-sectional imaging demonstrating > 50% iliocaval venous obstruction in 37% of limbs with healed or active ulcers. Although lesions at the crossing of the right common iliac artery and left common iliac vein are most common, compressive lesions may also occur at the left internal iliac artery crossing and inguinal ligament as well on the right side.

Anatomic compression of the iliac veins is best characterized by intravascular ultrasound, although definition of a hemodynamically significant lesion remains elusive. Arterial concepts of "critical" stenosis do not apply in the venous circulation. In the arterial circulation, critical stenoses are determined by sharp declines in pressure and flow, while in the venous circulation, the critical parameter is upstream pressure rather than downstream perfusion. The determinants of a "critical" stenosis are far more complex in the venous circulation, with some of the components including the degree of outflow stenosis, the inflow volume, the Starling (tissue or intra-abdominal) pressure, and left atrial pressure.

Endovascular approaches to non-thrombotic lesions of the iliac veins are now standard and are associated with excellent midterm patency, good relief of symptoms and improved quality of life. Although most reports have utilized self expanding Wallstents, the use of stents designed for other purposes does have some limitations. Although long-term outcomes are pending, the recent availability of stents specifically designed for the venous circulation should overcome many of these limitation.

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| Venous Syndromes: Update in Diagnosis and Management | |
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| Wednesday, 6th July, 2016 | 14:30 – 14:45 |

## Stewart-Bluefarb Syndrome

Kurosh Parsi

Department of Dermatology, St Vincent's Hospital, Darlinghurst NSW, Australia.
Dermatology, Phlebology and Fluid Mechanics Research Laboratory, St Vincent's Centre for Applied Medical Research, Darlinghurst NSW, Australia.
The University of New South Wales, NSW, Australia.

Objectives: This presentation will discuss Stewart-Bluefarb syndrome (SBS), a rare angioproliferative disorder characterised by acroangiodermatitis (AAD) associated with an underlying arteriovenous shunt.

Methods: Five patients with SBS will be discussed and literature pertaining to this condition reviewed.

Results: In this case series, all underlying AV communications were initially diagnosed on duplex ultrasound and confirmed with magnetic resonance angiography (MRA). Four patients were found to have a congenital AV malformation while one patient was diagnosed with a post-thrombotic AV fistula. In one female and two male patients the diagnosis was delayed as the AAD closely resembled other conditions. Management included observation and intervention using a variety of techniques including percutaneous or trans-catheter embolization, endovenous laser, radiofrequency ablation and foam ultrasound guided sclerotherapy.

Conclusion: This case series highlights the challenges involved in the diagnosis and management of SBS. Given the local and systemic sequelae of high flow shunts, correct diagnosis and early detection of the underlying AV abnormality is crucial in the long-term management of these patients and in preventing the associated complications.

NOTES

| Venous Syndromes: Update in Diagnosis and Management | |
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| Wednesday, 6th July, 2016 | 14:45 – 15:00 |

## Chronic Cerebrospinal Venous Insufficiency (CCSVI)

Paul Thibault

Central Vein and Cosmetic Centre, Broadmeadow, Australia

Paolo Zamboni suggests that Multiple Sclerosis is associated with stenoses and obstructions of the internal jugular and azygos veins. However there is controversy as to the nature of these extracranial venous anomalies, their association with multiple sclerosis and whether they represent pathological findings. Zamboni postulates that these venous obstructions are congenital venous malformations and may contribute to the development and progression of MS. In contrast Thibault has proposed that the venous obstructions are a result of a chronic infective cerebrospinal venulitis that occurs early in the disease process and progress with the disease. Others have refuted that there is any relationship between these venous anomalies and the aetiology or pathophysiology of MS.

The term chronic cerebrospinal venous insufficiency (CCSVI) has been used to refer to the venous anomaly associated with MS. Zamboni has used five parameters of duplex ultrasound to indicate the likely presence of CCSVI in MS patients:

1. Reflux in the Internal Jugular Veins (IJV) and/or Vertebral Veins (VV) in sitting and/or supine posture, assessed using Extracranial Doppler (ECD) methodology;
2. Reflux in the deep cerebral veins in sitting and/or supine position, assessed using trans-cranial Doppler;
3. High resolution B-mode evidence of IJV stenoses;
4. Flow not Doppler detectable in the IJVs and/or VVs;
5. Reverted postural control of the main cerebral venous outflow pathways, as determined by cross-sectional area changes.

Thibault also uses blood volume flow (BVF) measurements, calculated as a product of blood velocity and cross-sectional area, to provide objective quantification of flow disturbances and indicate the likely presence of stenoses.

There is now evidence that CCSVI is also associated with other chronic diseases of the head, neck and chest and that the venous obstructions are associated with chronic persistent chlamydophila pneumoniae infection. These concepts will be elucidated in this lecture.

NOTES

| Venous Syndromes: Update in Diagnosis and Management | |
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| Wednesday, 6th July, 2016 | 15:00 – 15:15 |

## Raynaud's Syndrome: Instrumental approach

Pier Luigi Antignani

Vascular Center, Nuova Villa Claudia, Rome, Italy

In primary and secondary Raynaud's phenomenon, measurement of activity or severity of the digital vascular disease is a major challenge. We need to identify objective measures of digital vascular disease that are helpful in predicting those patients with Raynaud's who have underlying connective tissue disease, and to measure digital vascular disease progression, and responses to treatment.

Diagnostic criteria for primary Raynaud phenomenon include the following: attacks triggered by exposure to cold and/or stress, symmetric bilateral involvement, absence of necrosis, absence of a detectable underlying cause, normal capillaroscopy findings, normal laboratory findings for inflammation, absence of antinuclear factors.

Different physiological measurement techniques may be used in combination, especially if investigators wish to examine both digital artery and microvascular flow. The diagnostic panel includes: Physical examination, Diagnosis of etiology (laboratory studies as immunological findings), Instrumental evaluation (Plethysmography, Doppler ultrasound, finger systolic blood pressure measurements, Nailfold (video and dynamic) capillaroscopy, Laser - Doppler, $TcPO_2$ - TcPCO2, Thermography, Radiography, angioRM, angio TC (eg. thoracic outlet syndrome).

Traditional capillaroscopy proved very useful for an early diagnosis of Raynaud's phenomenon with connective tissue involvement. Dynamic capillaroscopy revealed velocities below normal but somewhat overlapping among different connective tissue disorders, thus it did not provide diagnostic value. Video capillaroscopy may allow quantitation of microvascular disease progression over time and therefore be an important research tool. While current techniques of quantifying capillary abnormalities are not ideal, in that they are time-consuming or incorporate a degree of subjectivity, or both, this is an area being actively researched. Fluorescence video microscopy can be used to measure capillary permeability, which is increased in SSc. This technique could be used to evaluate drug induced changes in capillary permeability.

Laser Doppler flowmetry is well established in the measurement of cutaneous microcirculatory flow.

NOTES

| Compression, Oedema and Lymphoedema | |
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| Wednesday, 6th July, 2016 | 16:00 – 16:15 |

## An Approach to Patients with Oedema: Drugs, Foods and Reversible Causes

Keturah Hoffman

Restoration Clinic of WA, WA Australia.

Oedema is a common yet poorly treated problem. It is usually multifactorial and often contributed to by factors within the patient's control. In order to best help patients with oedema we need to accurately diagnose all the contributing factors and address each one.

Contributing factors include systemic disease such as cardiac, respiratory, renal or hepatic concerns, medication including over the counter tablets, dietary issues and lifestyle factors. Treatment includes withdrawing medications where possible, improving sleep and diet, using proteases and compression and other novel treatments.

More research is needed into cellular mechanisms of oedema & lymphoedema in order to determine more specific treatments.

NOTES

| Compression, Oedema and Lymphoedema | |
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| Wednesday, 6th July, 2016 | 16:15 – 16:30 |

## A Review of Medical and Commercial Compression Stockings: A Female Perspective

Stefania Roberts

Victoria Vein Clinic, East Melbourne, Australia

NOTES

| Compression, Oedema and Lymphoedema | |
|---|---|
| Wednesday, 6th July, 2016 | 16:30 – 16:45 |

## Why we may not always get good outcomes: The Importance of Pressure in Chronic Oedema/Lymphoedema

Neil Piller, John Arkwright, Luke Parkinson, Malou van Zanten

Flinders University, Bedford Park, Australia

Background: Venous obstruction and lymphedema can both lead to chronic oedema and are exacerbated by obesity and immobility. The failure of one system may precipitate failure in the other leading to Phlebo-lymphoedema. A mainstay of treatment is compression and research indicates that efficacy is strongly linked to achieving the optimal recommended pressures and pressure gradients however measuring these parameters remains a challenge.

Objectives: To demonstrate that achieving the correct sub-bandage pressure profiles is challenging, even for experienced medical professionals. To show how feedback from a sub-bandage pressure sensor array can lead to improved accuracy and compliance to compression orders.

Methods: A fibre-optic pressure sensoring array comprising 33 independent pressure sensors at 10 mm spacing was fixed along healthy lower leg (antero-lateral surface). A trained nurse with 10 years' experience in chronic wound care was then asked to apply a 4 layer compression bandage system to the lower leg with 40 mmHg compression at the ankle and 20 mmHg compression below the knee. Pressures were recorded continuously during bandaging and the $r^2$ value was used to quantify "closeness" to the target gradient. Initially bandaging was conducted without reference to the measured pressure profile. The nurse then re-applied the bandage using the measured pressure profile as a reference.

Results: For the blinded trial, the pressure profile achieved ranged from 45 mm Hg at the ankle to 38 mm Hg at the knee, with $R^2 = 0.085$. By using sensor feedback during bandaging, a linear gradient from 50 mmHg to 18 mmHg ($R^2=0.9021$) was achieved.

Conclusions: Distributed fibre-optic pressure sensing is a useful training and learning aid even for experienced bandaging therapists, enabling them to achieve improved accuracy in the application of bandage pressures and gradients. We hypothesize that its use will dramatically improve outcomes for patients with oedema who are prescribed bandaging.

NOTES

| Compression, Oedema and Lymphoedema | |
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| Wednesday, 6th July, 2016 | 16:45 – 17:00 |

## The Risk for Lymphoedema in Lower Limb Trauma with Extensive Soft Tissue Loss

Malou van Zanten[1], Yugesh Caplash[2], Neil Piller[1]

[1] Department of Surgery, Lymphoedema Clinical Research Unit, Flinders University, Adelaide, Australia,
[2] Department of Plastic and Reconstructive Surgery, Royal Adelaide Hospital, Adelaide, Australia

Background: Lymphoedema is a chronic progressive condition often producing significant morbidity. An in-depth understanding of an individual's lymphatic architecture is valuable both in the understanding of underlying pathology and for targeting and tailoring treatment. Severe lower limb injuries resulting in extensive loss of soft tissue requires transposition of a flap consisting of muscle and/or soft tissue to close the defect. There is limited data on lymphatic response following lower limb trauma

Methods: Twenty-three patients have been recruited from the lower limb trauma database (2009-2015) kept at the Department of Plastic and Reconstructive Surgery.

Basic socio-demographic data was collected next to General Short Health questionnaire (SF12), and the Lower Limb Functioning Score (LEFS). Objectively for segmental oedema measurement a Bio-Impedance Spectroscopy SFB7 unit was used (Impedimed, Queensland) and for measurement of local fluids at 2.5mm depth a Di-electric constant Moisturemeter (Delphin Technologies, Finland). Eighteen participants were imaged with a novel lymphatic imaging technique Indocyanine Green, Near Infra-Red Lymphography. With all measurements the non-affected leg was used as a control and all measurements were repeated at 12 months.

Results: The majority of patients were male (94%). The average age at presentation was 47 years (range 26-73 years) with a follow up of 38 months (range 2-62). Measurements with Di-electric constant showed a significant higher fluid content in the reconstructed area compared to the control leg. This is consistent with a significantly high extracellular fluid measured with Bio-Impedance in the affected leg. Muscle free flaps demonstrated no functional lymphatic vessel regrowth; fasciocutaneous flaps demonstrated impaired lymphatic vessel function and dermal backflow pattern. Local flaps demonstrated lymphatic block at the scar edge.

Conclusion: Patients with severe soft tissue trauma can be at risk for lymphoedema and thus at risk for reduced quality of life and potential recurrent infections.

NOTES

| Compression, Oedema and Lymphoedema | |
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| Wednesday, 6th July, 2016 | 17:00 – 17:15 |

## Do hydrocephalus shunts have a place in managing lymphoedema?

Jemima Bell, Neil Piller

Flinders University, Bedford Park, Australia

Background: Lymphoedema is a consequence of impaired lymphatic drainage. Various treatment options are available with varying degrees of efficacy and impact on the individual. These include manual lymphatic drainage, physical therapy, compression, massage, exercise, anastomoses, nodal transplants, liposuction, microsurgery/super microsurgery and preventative chemotherapy for lymphatic filariasis (Champaneria 2015). Whilst all have been shown to be useful in reducing the extent and impact of lymphoedema, there are confounding factors such as patient compliance, the financial and physical costs and of course the often unpredictable variable outcomes. There seems to be no single treatment that is affordable, effective and sustainable for patients with or at risk of lymphoedema.

Aims: This review examines lymphoedema, its pathogenesis, treatment options available and introduces the novel idea of a modified hydrocephalus shunt as a surgical alternative to treat (and perhaps prevent) lymphoedema.

Description: Hydrocephalus shunts allow cerebrospinal fluid (CSF) to circumvent an obstruction during periods of impaired absorption, removing the build-up of fluid that causes hydrocephalus. Shunts work on a low pressure system, and generally consist of a ventricular catheter, a one-way valve, and a distal catheter.

Normal human limb pumping pressures have been recorded to be between 10 and 60mmHg, whilst maximum pressures recorded in failing lymphatics is approximately 50-60mmHg. As hydrocephalus shunts work on pressures as low as 15-25mmHg at flow rates as low as 5ml/hr it is plausible that they could be used to prevent retrograde flow of lymph in failed lymphatics, thereby reducing lymphoedema. Their functioning and role will be described in the talk

Clinical Implications: The use of hydrocephalus shunts in the treatment and prevention of lymphoedema would greatly reduce the need for other interventions that require higher patient compliance and ongoing management.

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| Compression, Oedema and Lymphoedema | |
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| Wednesday, 6th July, 2016 | 17:15 – 17:30 |

# What about compliance? Its impact on Intermittent pneumatic compression outcomes.

Neil Piller, Beth Kean, Edward Mitchell, Malou van Zanten, Marielle Esplin, Janet Douglass, Shahid Ullah

Flinders University, Bedford Park, Australia

Background: This report is part of a larger study investigating the objective/subjective benefits of multi-chambered intermittent pneumatic compression treatment on leg lymphoedemas. For some, the demands of compliance are difficult due to treatment fatigue, physical challenges, time, and travel issues. However non-compliance can affect outcomes.

Aims: To determine the benefits of patient compliance with pneumatic compression treatment on a range of objective and subjective parameters over 12 weeks of IPC treatment.

Methods Baseline objective measurements utilizing whole limb bio-impedance spectroscopy, perometry, tape measurement and volume calculation, tonometry/indurometry, moisture content and the subjective completion of the LYMQOL instrument were conducted. Patients were then allocated a compression unit with a 27-32 chambered compression garment (Tactile Medical), which provided wave-like compression to the trunk and affected limb. Patients were instructed to use the system daily for one hour. Objective measures were repeated at 3 days, 1,2,4,6, 8 and 12 weeks. The LYMQOL was completed again at 12 weeks. Patients were instructed to undertake normal (best practice) limb care during the study period. The untreated limb was used as a control.

Results: An interim analysis of the first 40 patients (of whom 11 were totally compliant) was undertaken. A whole group analysis indicated large variability in outcomes but when totally compliant patients (determined by log book) were compared to partially compliant patients, some large practically and statistically significant differences were found, indicating clear benefits of compliance. Major differences occurred in lower limb circumference and volume and in limb total fluids and local site fluids. These are important for tissue and cellular health. Some differences in LYMQOL were also observed with heaviness, tension and pins and needles sensations showing the greatest difference between the two groups

Conclusion: Fully compliant patients gain improved subjective and objective outcomes with intermittent pneumatic compression treatment compared to those who are not.

NOTES

| Pressure and Compression Workshop | |
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| Tuesday, 5th July, 2016 | 14:50 – 15:30 |

## Can you Handle the Pressure? A test to see if you are getting the pressures right and the garments you order are applying the right pressures!

Neil Piller

Flinders University, Bedford Park, Australia

The workshop will begin with a short presentation of the critical factors and laws which indicate and affect pressure and the pressure gradient around and along a limb. Workshop attendees will have an opportunity to apply a garment which should provide an indicated pressure. Using a range of pressure sensors, we will determine if the pressure attained was anywhere near the pressure expected. Following this, attendees will be invited to apply a specific bandage to a simulated limb and achieve an indicated pressure and gradient. The truth is you "Don't always get what you want". No wonder why some outcomes from compression are so poor! Over the remainder of the Conference there will be a competition to see who can achieve an optimal pressure/pressure gradient for an oedematous lower limb with and without sensor based input. Winner takes all!

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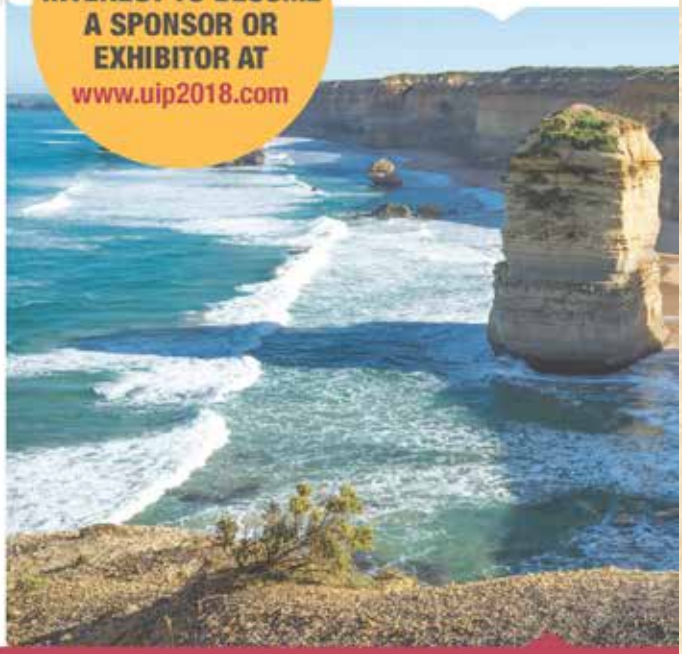

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