Report from a Phlebology "Task Force" in Ecuador

In January, 2007 and again in January, 2008 a group of Italian and American medical volunteers traveled to Guayaquil, Ecuador on the west coast of South America to deliver much needed medical care to indigent patients with severe chronic venous disease, mostly CEAP classification 4-6. Attilio Cavezzi, Valerio Carigi, and Allesandro Frullini led the Italian delegation and Nick Morrison, Terri Morrison, Diana Neuhardt, Joseph Zygmunt and Sergio Salles-Cunha led the American group.

Working 10-12 hour days in the Military Hospital for one week, the volunteers managed to exam and perform duplex scans on some 600 patients, and treated nearly that many with application of compression bandages or garments, duplex-guided foam sclerotherapy, endovenous laser ablation, ambulatory phlebectomy, and sclerotherapy. Patients with venous malformations, superficial venous insufficiency, deep venous obstruction or insufficiency, and combinations of all were seen in abundance. Supplies and equipment were largely borrowed or donated, although there was also need to purchase some supplies not readily available, or in response to equipment malfunction while in Ecuador.

These trips were taken in conjunction with Amigos de Salud, a volunteer medical organization founded some 20 years ago by Nick and Terri Morrison of the U.S., in order to bring medical care of all disciplines to Ecuador on an annual or semi-annual basis. A typical Amigos trip might include specialists in General Surgery, Pediatrics, Colorectal Surgery, Audiology, Orthopedics, Plastic Surgery, Gynecology, Internal Medicine, Family Medicine, Gastroenterology, and Pulmonary Medicine.

For twenty years, Amigos de Salud has worked with the director of an Ecuadorian foundation, Dra. Zorayda Figueroa, who manages the logistical support for this large endeavor in Ecuador. Preliminary discussions have also commenced with the International Union of Phlebology regarding a joint humanitarian project.

With reference to the details of our Phlebology experience in Guayaquil (2007 and 2008), you may read below some details concerning the patients, methods and the short-term outcomes.

The flow chart of our activity was the following:

a) through newspapers, radios, TVs and other forms of information diffusion, the foundation collected the patients in the Military Hospital of Guayaquil.

b) all the patients underwent a preliminary history and directed physical examination, and subsequently a colour-duplex ultrasound investigation (CDUI) intended to highlight the main venous and arterial abnormalities

c) if the patient was eligible for treatment, a drawing/paper map of the main morphologic and haemodynamic pathologic findings was created and a scheduled date for the subsequent treatment was provided.
d) the treatment consisted mainly in ultrasound-guided FS of the saphenous trunk, perforators and varicose tributaries, as well of the groin/popliteal recurrence venous networks when necessary (one-step procedure)

e) 810nm (Diomed) endovenous laser treatment (EVLT) under tumescent local anaesthesia was provided, this treatment modality especially indicated in large saphenous trunks (>8mm); EVLT was always combined with FS of all the varicose tributaries when needed

f) venous malformations or peri-ulcer varices were treated with FS as well

g) compression by 20-30 or 30-40 mmHg elastic stocking + pads were provided in all cases, with the help of some personnel of the Hospital and/or of the foundation

h) all patients signed an informed consent and received a leaflet containing a detailed list of suggestions, precautions, explanations about the treatment as a whole and its potential complications and the relative therapy; on-call availability of a medical doctor was provided in all cases

Together with the treatment of patients, the “phlebology task force” in Ecuador provided some education to local medical doctors and nurses who freely accessed our location and who helped us or simply attended our daily sessions. The educational purpose of this charity initiative has been occasionally slowed because of inherent difficulties (e.g. discontinuity of the presence of the local doctors/nurses), but the overall acceptance of our work within the local vascular surgeons/medical doctors has been good.

From the diagnostic point of view a significant rate of pelvic venous insufficiency and related non-saphenous varices, including a remarkable number of sciatic nerve varices, was noticed in female patients (presumably due to the high number of pregnancies, more than four in the majority of cases). The most relevant finding which was encountered was the extremely advanced stages of CEAP together with the relevant size of the saphenous trunks (mid thigh diameter exceeded 8-10 mm in many patients with some 40-50 mm saphenous trunks and varicose tributaries.

FS was the elected treatment for the vast majority of the patients in 2007, while 6 VV surgical procedures were performed for patients with larger varices. In 2008 only FS was provided to all the treated patients, due to the difficulties (and the resulting low number of the treated patients per day) encountered the previous year with the surgical approach.

With reference to the material we used in FS, only CO2 was used to form the sclerosant foam, together with sodium tetradecylsulfate or polidocanol (1-3%).

The average amount of sclerosant foam which was injected per patient varied in a significant manner due to the relevant differences in the varicose state of the patients. A minimum of 12 ml of sclerosant foam per session(patient) was injected and the maximum dose was 51 ml.

In cases of ulcers or vascular malformation a second look and a possible re-treatment were performed, together with the application of specific compression bandages and dressings.

Finally in 2008 there was the possibility to review clinically and with CDUI a small percentage of the patients who were treated the previous year.

The immediate outcomes of ultrasound-guided FS were good in the vast majority of the cases: obliteration of the saphenous trunk and/or disappearance of the varicose veins were achieved in the vast majority of the cases, though a few cases which were available at mid-term (one year) follow-up revealed an overall better result in case of smaller diameter and in case of usage of larger volumes and higher concentrations (no statistical data available).

The side effects and complication rates were monitored through the immediate control of all the treated patients (up to 60 minutes after the treatment), as well as through the collection of all the patients who contacted the Foundation Centre for any possible atypical consequence of the treatment. The 2007 and
2008 treatments were characterised by one probable case of (late) TIA (without sequelae), one case of gastroenteric malaise and one case of short-lasting (1 hour) sensorial neural lesion. Because of the large size of the varices, and our inability to perform ambulatory phlebectomy efficiently, a significant number of varicophlebites in the treated areas was highlighted after laser treatment or after FS. No specific symptoms/signs of pulmonary embolism or of immediate cerebral ischemia were spontaneously reported by the patients or noticed by the medical staff.

In general, the advanced stages of venous disease seen on these trips are a result of long-suffering patients who have not had the means to obtain any type of medical care for their venous disease, not even graduated compression hose. Some of the worst complications of chronic venous disease are seen, including circumferential leg ulcers, phlebolymphedema, and huge varicosities. These patients often travel great distances and undergo considerable hardship to see the volunteer medical team, and they are among the most grateful patients one will encounter in a medical career. Nearly as gratifying as the patient’s kind words is the opportunity to work side by side with colleagues from around the world. Spending a week working hard with a dedicated team is tremendously rewarding in itself, let alone the good one does for the hundreds of patients impacted by the effort.

These volunteer medical trips are self-funded by the physicians and nurses who attend, while some funding is occasionally available to help assistants and technologists, without whose help these trips are simply not possible. Industry partners are also very supportive of these efforts with equipment and supplies. A tax-exempt foundation has been created by Amigos de Salud to facilitate donations. For more information, please feel free to contact us at nickmorrison2002@yahoo.com

A.Cavezzi, N.Morrison
**INTRODUCTION**

The Australasian College of Phlebology, in late January 2008, appointed an Executive Director in Noel Hadjimichael a 45 year old executive with an impressive background in management, policy development, law and industry association interchange with Government. Noel came to the College following recent roles as a University Lecturer in Management and Law and the very dynamic role as the Director of the Governor General’s Award Program in Civics Education. Noel spent time with me (online) to allow me to interview him on the direction the College is taking, the upcoming ASM and what attributes and skills he brings to this critical role for Phlebology.

**EDITOR:** Firstly Noel, why join us when most people are still somewhat ignorant about Phlebology as a discipline or field of medicine?

**NOEL:** Well Jacqui, I was tempted by the twin features that I see as the College’s strength – a specialty field that encompasses such a diverse set of professionals and an organization that is still charting its way to establishment – both very attractive if you are an ideas person and value the public interest impact of the work you do.

**EDITOR:** But you had a cozy job with teaching how many hours a week, was it 26?

**NOEL:** Yes, it was 26 hours face to face, but as some of your readers will know you have the absolute joy of dealing with the demands of more than 500 students across four subjects and leaving the office exhausted after a 12 hour day with limited direct evidence that you have made a difference.

**EDITOR:** Where did the role relating to the Governor General take you?

**NOEL:** In my 18 months, I oversaw a number of key programs aimed at improving and enhancing the quality of civics education for young Australians aged 8 to 25. Whilst the prestigious and very lucrative essay competition for undergraduates exposes you to some of the best young minds in Australia (with a judging panel including people like Justice Michael Kirby or former Tasmanian Governor Sir Guy Greene) the real excitement was bringing schools in isolated regions into pilot programs with their peers in Sydney and Perth.

**EDITOR:** BEc, MPP, LLB (Hons) ? you must like study?

**NOEL:** Yes and no. Like most kids of my generation I loved sports (Cricket, Golf and Volleyball were my interests) but reading got me excited about history, economics, politics and social issues. Following an early placement with the Royal Australian Naval College, I saw my way through an economics degree in Government/Industrial Relations, a masters degree in Public Policy and a law degree.

**EDITOR:** What do you bring to the College at this time?

**NOEL:** More than any technical skills – drawing on work experience in a parliamentary, banking, industry association and legal capacity – I believe that I bring the capacity to identify and assess issues and risks well. I may not have the answer to hand, but I am very determined to find the answer best tailored to our needs.

**EDITOR:** Where is the College now placed?

**NOEL:** Well I started at the end of January 2008 and want to place on record a thank you to Charles Baker the former Executive Officer. I have the pleasure of working with Camilla Millazzo - the Executive Assistant - who is a University graduate in Sociology and a person with direct experience in Phlebology at a clinical level. We have established a stand alone office located at level 16 of Tower One at Westfield Bondi Junction just a few minutes train ride from the Sydney CBD. We have also set in train a major overhaul of the internal workings of the College with three key outcomes: efficiency, effectiveness and community education. But to do this I rely on the hard work of many Fellows and Members from the Board down!

**EDITOR:** What are the current projects that dominate your thinking?

**NOEL:** Accreditation is a big task – one that might take some time. However I am confident that we can make a strong case for recognition in the light of our excellent international reputation in training and continuing professional development. The second big task is the roll out of the regional Faculties. NSW has its election on 5 April 2008 – having set up both the
Vein School and Journal Club activities successfully. Victoria is hoping to launch in July 2008 and Queensland is on track to launch during the ASM. I am still looking around for the right mix of leaders to take the Faculties forward – in particular New Zealand, South Australia and Western Australia. All volunteers please step forward!

EDITOR: Wow, you have your hands busy. What else is in the wings?

NOEL: I play a supportive role towards the ASM and have engaged with leaders such as Professor Rabe of Germany to encourage a very international spirit with the week long conference. The College is optimistic that the delegation from the German society will make a significant impact and I look forward to the conjoint session. We also have plans to bring the Vein Schools and Journal Clubs to other venues beyond Sydney – in particular Brisbane, Melbourne and somewhere in New Zealand.

EDITOR: How can we contact you? Do you have time to speak with Members and Fellows?

NOEL: Absolutely – it is affirmative. Without communication, the College will not prosper and I will not able to do my job. My mobile 0409 151521 is always an option – yes I do check the messages – and my email at noel.hadjimichael@phlebology.com.au is a very good start. Although parented by a couple who spoke nine languages, I have only schoolboy German (which is getting better over time) and I am always available to discuss issues or deal with matters of interest to the College or the profession.

EDITOR: What do you in your spare time?

NOEL: Well, a balance is very important in life. I have started my PhD (looking at the relationship between the medical profession and governments – what else) and still find time for my partner Carol, her daughters Sarah and Nina, and my sons James and David. With a hectic family life and an interesting job, I am very much a 7 day active person. Very similar to the many Members and Fellows I have met, so far, via the College. Whether attending a school Seder, a Highland Games (my sons are very keen on bagpipes) or catching up with old friends from Navy or Parliament House days, I think I have found a very sound balance.

EDITOR: Thank you Noel for allowing me to interview you and give readers a glimpse into your situation.

NOEL: Thank Jacqui, I know that I will need the patience and assistance of the Board to achieve our strategic vision. Thank you for asking me to tell all!

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The CoolLipo/CTEV Combo unit combines lipolysis, skin tightening and treatment of varicose veins in one easily affordable box.

Before
After
Unretouched photos courtesy of Robert Weiss, MD

Before
After
Photos courtesy of Charles Mok, D.O.
Catheter-directed thrombolytic techniques can be invaluable in managing patients with the most severe forms of venous thrombosis. They have also shown promise to improve clinical outcomes in the larger population of patients with venous thromboembolic disease. However, the proper indications for thrombolytic therapy have not been conclusively established and the wide range of thrombolytic techniques offered can be confusing. The purpose of this document is to provide a straightforward approach to patient selection, procedure performance, and post-procedure monitoring that can be easily adopted by IRs starting to perform venous thrombolysis.

**Patient Selection**

At present, the proper indications for catheter-directed thrombolysis (CDT) in the treatment of lower extremity DVT are controversial.

1. Patient selection should take into account the anatomic extent of thrombosis, symptom duration and severity.
2. Patient’s likelihood of having a bleeding complication.

**Absolute contraindications to CDT include:**
- Active internal bleeding
- Disseminated intravascular coagulation
- Recent (< 3 months) cerebrovascular event, neurosurgery, or intracranial trauma
- Presence of an absolute contraindication to anticoagulation.

**Strong relative contraindications include:**
- Recent CPR
- Recent major surgery, delivery, biopsy, trauma
- Bleeding event
- Intracranial lesion or seizure
- Uncontrolled hypertension
- Thrombocytopenia
- Known right-to-left cardiopulmonary shunt
- Renal failure
- Severe hepatic dysfunction
- Septic thrombophlebitis
- Diabetic hemorrhagic retinopathy.

**A. Lower Extremity and Inferior Vena Cava DVT:**

A multicentre CDT registry clearly demonstrated that CDT is more effective in removing acute thrombus compared with chronic organized thrombus (3). There are two primary reasons to perform CDT in patients with acute DVT:

1. To prevent major immediate adverse clinical sequelae such as death, limb loss, pulmonary embolism, and renal failure in patients with phlegmasia or acute IVC thrombosis
2. To prevent post-thrombotic syndrome in patients with acute proximal DVT of lesser severity (iliofemoral and femoropopliteal).

It is, however, important to recognize that the ability of CDT to prevent PTS has not been conclusively established in randomized trials. Therefore, patients evaluated for CDT with the primary goal of post-thrombotic syndrome (PTS) prevention should be informed of the long-term risks of PTS; the risks, benefits, and alternatives to CDT; and the lack of conclusive evidence in favor of (or against) CDT’s ability to prevent PTS.

**B. Upper Extremity DVT:**

Post-thrombotic syndrome of the upper extremity, particularly when within the dominant arm, can also significantly impair quality of life (4). For this reason, patients with symptomatic, acute axillosubclavian DVT may also be candidates for CDT. In general, the etiology of thrombosis plays a major role in determining the optimal therapeutic approach. Primary axillosubclavian vein thrombosis is typically caused by compression of the subclavian vein from surrounding ligamentous and muscular structures in the thoracic outlet. These patients are often young and otherwise healthy and need more drastic measures.

Modern treatment features a combined interventional-surgical approach: CDT to eliminate the acute thrombus, followed by surgical thoracic outlet decompression to prevent recurrence. Angioplasty and stent placement are not performed in order to avoid further traumatizing the subclavian vein, and because stents
tend to fracture in this location. Treatment of patients with secondary axillousubclavian venous thrombosis is largely dependent upon the degree of symptoms and overall patient condition. In general, symptomatic younger patients without major comorbidities that would elevate bleeding risk are candidates for CDT. Because many such cases are related to stenosis caused by prior central venous catheters and other devices, balloon angioplasty may play a role in treatment as well.

PROCEDURAL TECHNIQUE

The following is a brief description of the basic CDT technique for acute DVT (5):

A. Venous Access: When thrombolytic therapy is planned, venous access should be obtained using ultrasound guidance in order to avoid inadvertent arterial punctures. When possible, a lower extremity vein should be selected at a site lower than the most distal extent of thrombosis. For many patients with iliofemoral DVT, the popliteal vein provides a suitable access site; however, patients with thrombus involving the popliteal vein and upper calf veins may be better treated with access into the small saphenous vein or posterior tibial vein. The internal jugular vein is another option.

B. Catheter Venography: Once venous access is obtained, diagnostic venography is performed to accurately define the extent of thrombosis.

C. Retrievable IVC Filter is placed

D. Initiation of Thrombolysis: A multisidehole catheter is embedded within the thrombus and the thrombolytic drug is infused in drip fashion. The patient is simultaneously anticoagulated using heparin infusion. The patient is monitored in an ICU or stepdown unit, and serial laboratory values are obtained every 6 hours. Specifically, the hematocrit, partial thromboplastin time, and fibrinogen are monitored for change and dose adjustments made accordingly.

F. Follow-Up Checks: At 6-18 hour intervals, the patient is brought back to the interventional suite and repeat venography performed to define the extent of thrombolysis. At each follow-up check, one of several findings is seen:

a) Complete thrombolysis with no venous stenosis – therapy is deemed successful, the thrombolytic infusion is terminated, and the patient is anticoagulated.

b) Complete thrombolysis with a venous stenosis identified – iliac vein stenoses are typically treated with endovascular stent placement and femoral vein stenoses are typically treated with balloon angioplasty

c) Incomplete thrombolysis – an angioplasty balloon is used to macerate the thrombus, the infusion catheter is repositioned within the residual thrombus, and the infusion is continued.

If no thrombolysis is seen within 24-48 hours, therapy is discontinued.

CHOICE OF THROMBOLYTIC DRUG:

Although UK is the drug most commonly used in published CDT studies, its intermittent unavailability has prompted the use of alternative drugs including tissue plasminogen activator (TPA), reteplase (RPA), and tenecteplase (TNK). To date, no studies have demonstrated differences in safety or efficacy of these agents using current dosing regimens (Appendix).

Concomitant Anticoagulation: Although there is no direct evidence available to support use of any anticoagulation regimen over another, the current consensus is for full-dose therapeutic-level heparin (PTT 1.5 – 2.5x control) when using UK. On the other hand, subtherapeutic heparin (300-500 units/hr, PTT < 1.5x control) is suggested when using TPA, RPA, and TNK.

Percutaneous Mechanical Thrombectomy (PMT): To date, no stand-alone PMT method has proven effective in DVT treatment without associated pharmacologic thrombolytic therapy. However, the combination of pharmacologic CDT with PMT, known as pharmacomechanical thrombolysis, has shown potential to speed thrombolysis, reduce the required drug dose, and reduce complication rates.

Several different pharmacomechanical methods have been used:

a) Thrombolytic drug infusion followed by PMT to macerate or remove thrombus.

b) Initial PMT to debulk the thrombus and create a flow channel, followed by thrombolytic drug infusion.

c) Recently, several pharmacomechanical techniques have been introduced to enable on-table, single session DVT treatment.
These techniques involve vigorous pulse-spray injection of a bolus dose of thrombolytic drug into the thrombus, with concomitant/subsequent PMT device use to macerate and/or aspirate residual thrombus. To date, mid-term results of the single-session treatment methods have not been published.

**APPENDIX: CDT DOSING**

The following represent acceptable dosing regiments for CDT of DVT.

1. Urokinase: 120,000 – 200,000 units/hr – Dissolve 1 million units UK in 500 ml normal saline (= 2000 units/ml). Infuse at 60-100 ml/hr.

2. Tissue Plasminogen Activator: 0.5 – 1.0 mg/hr – Dissolve 10 units TPA in 1000 ml normal saline (= 0.01 mg/ml). Infuse at 50-100 ml/hr.

3. Reteplase: 0.25 – 0.75 units/hr – Dissolve 10 units reteplase in 1000 ml normal saline (= 0.01 units/ml). Infuse at 25-75 ml/hr.

4. Tenecteplase: 0.25 – 0.5 mg/hr – Dissolve 5 mg TNK in 500 ml normal saline (= 0.01 mg/ml). Infuse at 25-50 ml/hr.

**POST-PROCEDURE CARE**

Patients must transition to long-term anticoagulant therapy. He/she may ambulate soon after sheath removal and can usually be discharged from the hospital within 1-2 days afterwards. Typically patients are placed on oral warfarin and are given low-molecular weight heparin during the transition period – this is discontinued when the patient reaches the desired therapeutic range (INR 2.0 – 3.0 for most first-episode DVT patients, 2.5 – 3.5 for selected subgroups of patients).

Patients receiving stents are often also placed on anti-platelet therapy for several months. Patients also undergo risk factor evaluation to determine the appropriate duration of anticoagulant therapy, per American College of Chest Physicians guidelines – this may be done with hematology consultation in many instances. Patients with lower extremity DVT should be asked to wear a Class II (30-40 mmHg) graduated compression stocking to the affected limb for prevention of PTS – two randomized trials have shown that this intervention may decrease PTS rates by 50%. Patients and their physicians should be educated about the need to inform the interventionalist should symptoms recur, since re-stenosis can sometimes be treated with repeat balloon angioplasty or stent placement before re-thrombosis occurs.

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Australasian College of Phlebology
2008 Annual Scientific Meeting and Workshops

The Australasian College of Phlebology will be holding its Annual Scientific Meeting at the Crowne Plaza Surfers Paradise on the Gold Coast from Saturday 6th September to Thursday 11th September.

The programme for the Annual Scientific Meeting has changed this year, and is set to provide a comprehensive and enlightening look at the widening field of Phlebology. Members of the German Society of Phlebology will be attending, along with noted speakers from other parts of the world, including America and Italy.

The first two days of the conference will be devoted to a two-day Basic Phlebology course, running concurrently with a two-day Advanced Phlebology course. These will have a much more hands-on approach this year, with planned workshops on Doppler, Coagulation, Embolic agents & Filters, Sclerotherapy, Compression, UGS and EVLA.

The Scientific Programme will follow over the next three days, and will include symposiums on Foam, EVLA, Lymphoedema, the Basic Sciences as well as a symposium from the German Society of Phlebology. It is expected these will address recent controversies in both Foam and EVLA and provide a forum to hear the latest information on these issues.

So far, the list of invited speakers from overseas is impressive, and includes:
- Professor Philip Coleridge Smith, Phlebologist, Vascular Surgeon, UK
- Dr Stephan Guggenbichler, Phlebologist, Germany
- Professor BB Lee, Phlebologist, Vascular Surgeon, USA and Korea
- Dr Nick Morrison, Phlebologist, Vascular Surgeon, USA
- Professor Eberhard Rabe, Phlebologist, Dermatologist, President Union International Phlebologie, Germany

Following the scientific programme, there will be a one-day Vascular Anomalies Symposium, running concurrently with the traditional one-day Diagnostic Imaging sonography workshop.

The structure of the programme will provide ample opportunity to attend a wide variety of practical workshops and sessions. Live workshop sessions will alternate with lecture and tutorial sessions featuring presentations involving audience participation. The Basic and Advanced Phlebology Courses will allow for examination question time.

A partner programme is planned and will offer a diverse range of activities to suit all tastes. The Gold Coast is renowned for its leisure and entertainment activities and delegates, sponsors and exhibitors are encouraged to bring their families to make the most of this wonderful holiday venue.
**Location and Venue**

Set along a sweeping 70 kilometre pristine coastline, the Gold Coast is world famous for its sun, surf and sand. From ancient rainforests and boutique wineries to thrilling theme parks and wildlife reserves, the surrounding areas are full of options to explore. The Gold Coast is Australia’s largest regional city and renowned as the country’s leisure and entertainment capital. Gold Coast beaches are safe for swimming throughout the year.

Delegates, sponsors and exhibitors are encouraged to bring their families and make the most of this stunning location.

Positioned between Surfers Paradise and Broadbeach, Crowne Plaza Surfers Paradise is perfectly located for the ideal getaway and successful conference with a tropical resort atmosphere. The 26 floor luxury hotel has two distinctive towers with all rooms offering ocean views and private balconies. Crowne Plaza Surfers Paradise facilities include 2 restaurants, 3 bars, entertainment centre, 2 swimming pools and spas, gym, sauna & tennis court. The hotel is walking distance to major shopping centres, a variety of restaurants, nightlife and beaches.

It is a 20 minutes’ drive from the Coolangatta Airport or an hour from Brisbane domestic and international airports.

**Keynote Speakers**

- Professor Philip Coleridge Smith, Vascular Surgeon, UK
- Professor BB Lee, Phlebologist, Vascular Surgeon, USA and Korea
- Dr Nick Morrison, Phlebologist, Vascular Surgeon, USA
- Professor Ken Myers, Phlebologist, Vascular Surgeon, Victoria
- Professor Eberhard Rabe, Dermatologist, Phlebologist, President Union International Phlebologie, Germany
- Dr George Somjen, Phlebologist, Vascular Surgeon, Melbourne
- Professor Andre Van Rij, Phlebologist, Vascular Surgeon, Dunedin

**Invited International Speakers**

- Professor Philip Coleridge Smith, Vascular Surgeon, UK
- Dr Stephan Guggenbichler, Phlebologist, Germany
- Professor BB Lee, Phlebologist, Vascular Surgeon, USA and Korea
- Dr Uldis Maurins, Phlebologist, Latvia
- Dr Nick Morrison, Phlebologist, Vascular Surgeon, USA
- Professor Eberhard Rabe, Phlebologist, Dermatologist, President Union International Phlebologie, Germany

**Preliminary Conference Program**

**Saturday 06 September**
- Basic Phlebology Certificate Course
- Advanced Phlebology & Refresher Course

**Sunday 07 September**
- Basic Phlebology Certificate Course
- Advanced Phlebology & Refresher Course
- Welcome Cocktail Function

**Monday 08 September**
- Scientific Program

**Tuesday 09 September**
- Scientific Program
- Ken Myers: Oration & Launch of Queensland Facility
- Invited Speaker: Her Excellency Quentin Bryce AC, Governor of Queensland
- Graduation Ceremony - University of Qld Conference Dinner - Customs House Brisbane

**Wednesday 10 September**
- Scientific Program

**Thursday 11 September**
- Practical Workshops & Scientific Program

**Partner Program Activities**

A partner program is planned offering a diverse range of activities to suit all tastes.

Options include:
- Theme Park Day (three park pass available)
- Gold Coast Half Day Sight Seeing
- Mt Tamborine Half Day Tour
- Holden Performance Driving Centre Hot Laps
- Whale Watching
- Harbour Town Outlet Shopping
- Surfing Lesson
- Palazzo Versace High-Tea & Harbour Town
- Outlet Shopping
- Currimbin Wildlife Sanctuary
- Mt Tamborine Wineries & Gallery Walk
- Byron Bay Full Day Tour
- Hot Air Ballooning

We look forward to welcoming you to the Gold Coast in September 2008

For further details visit www.phlebology.com.au or to register your expression of interest email (providing full contact details): info@conferencematters.co.nz
9TH INTERNATIONAL CONFERENCE OF PHLEBOLOGY - BOLOGNE

My wife and I have just returned from the 9th International Conference of Phlebology organised by Attilio Cavezzi. I had the good fortune to meet up there with George Somjen. Experienced phlebologists will appreciate the importance of becoming acclimatised to the country well before the meeting. We commenced this arduous task by spending a few days in the delightful seaside city of Taormina in Sicily, overlooked by the 300 BC Teatro Greco and Mount Etna. We continued our preparation with a similar time in Venice and then Florence. Now thoroughly prepared, we continued on to Bologna for the conference. The faculty dinner on the night before was highlighted for me by meeting Stefano Ricci - anyone visiting Rome would be well advised to spend some time in his delightful company. The first day of the meeting was somewhat confusing - I was taken by bus to a venue where I was a effusively greeted by an Italian doctor as Professor Malkowski, the noted expert on sleep apnoea. It took a little time to appreciate that I was at a meeting on Rheumatology and that my venue was some 20 minutes taxi ride away on the other side of town.

The first two sessions were workshops conducted by Drs Rabe and Ricci on techniques for ultrasound scanning, and Drs Hugo Partsch, Coleridge Smith and others on methods for compression after treatment. These were of interest though rather basic, and little new was learned except that there was agreement that class 2 stockings alone provide insufficient compression after endovenous treatment and that adequate compression requires elastic bandaging with further eccentric compression over the treated veins. The first morning was completed by my personal endovenous treatment and that adequate compression requires external pressure of 40 mm Hg is required to measured by ultrasound showing that an venous diameter reduction under compression result without further treatment of the varices.

Dr Bernie Partsch presented a study on venous diameter reduction under compression measured by ultrasound showing that an external pressure of 40 mm Hg is required to compress thigh veins although this is reduced to half by using external pads. A highlight from me was a presentation by Drs Somjen of a detailed ultrasound study of the saphenofemoral junction after EVL in 204 legs. In 80%, the saphenofemoral junction remained open though competent and draining pelvic veins with the GSV obliterated 1-3 cm below the junction. If the vein was occluded to the junction, a trend was noted for thrombus retraction, occasionally to beyond thigh tributaries. Dr King from Chicago emphasised the need to identify the saphenous and sural nerves during EVL for their protection.

The Scientific Sessions continued on the Saturday morning with Dr Cavezzi presenting his experience preferring phlebectomy to sclerotherapy after treating saphenous reflux, and Dr Morrison sharing his experience of reduced systemic symptoms with sclerotherapy using carbon dioxide and oxygen rather than air. Dr Tessari then made an important contribution relating to the passage of bubbles and sclerosant into the right heart and pulmonary circulation. Echocardiography showed that bubbles appear whatever technique is used, but that they arrive later if the leg is elevated. Labeling the sclerosant with an isotope then showed that the sclerosant did not accumulate in the lung so that it is presumably dissociated from the bubbles. I then gave an address on evidence-based analysis of the factors which influence the efficacy and safety in foam sclerotherapy in which I summarised our observational studies on success rates in superficial veins and factors affecting the incidence of deep vein occlusion, and Kurosh Parsi's studies of in vitro effects of sclerosants on coagulation and cell destruction. Dr Frullini then gave an overview of the preparation of foam sclerosant pointing out that we do not know enough about the influence of concentration and dosage as well as site of injection. Dr Winczakiewicz presented a study on histologic changes of rat lung tissue post foam
sclerotherapy with aethoxysclerol. Initially, there was erythrocyte extravasation, perivascular, intra-alveolar and interstitial oedema, hemosiderin deposits, presence of siderophages and atelectasis but these all disappeared within 12 weeks.

The meeting then continued with selected submitted abstracts for which there was a generous prize of a trip to a meeting of the speaker’s choice with all expenses paid. The prize was won by Dr Kaspar from the Czech Republic for a study that used correct statistical techniques of survival analysis to follow late results after EVL. There were significantly worse late results in patients with a high BMI or where low power settings had been used. The results supported a concept of “slow and gentle heating” during EVL with lower or medium power settings (8 to 13 W) and slower pull-back speed of the laser fibre (0.2 to 2 mm/sec). Dr Brachmann presented a histological study of samples taken from the great saphenous vein after treatment by radio-frequency using the Closure-Fast system. All veins showed complete destruction of the intima (0.1-0.2 mm deep), denaturation of collagen and subintimal oedema, with complete obliteration of the vein and organised thrombus at four weeks. Dr Vandekerckhove from Belgium presented early results using a new 1500 nm laser system with continuous pullback at 1 mm/sec and 6 Watt energy with good early occlusion rates. Dr Vuylsteke from the same unit presented histological studies from goat veins using the 980 nm and 1500 nm systems. The perivenous temperature in the tumescent liquid could rise to 40-50°C. There was uneven destruction of the vein wall with ulcerations and perforations followed by cell destruction of the wall with surrounding inflammation. The energy penetration and perivenous cell destruction was deeper with the 980 nm laser and a more even destruction of the vein wall was found with the 1500 nm diode laser.

The meeting then concluded with round table discussions of foam sclerotherapy and endovenous laser therapy with an interchange of many ideas but few new conclusions. The long trip home from this very successful meeting then commenced leaving us with the usual desire to get home and back to normality. Italy is fun though.

Prof Ken Myers
GENETICS AND LYMPHOEDEMA:
FROM DIAGNOSIS TO TREATMENT

SUMMARY

A patient presents. There is no real cause for their lymphoedema. We call it “primary”. We superficially examine the patient, see if there are any clinically manifest external signs of lymph vascular malformation, ask them about the nature of their swollen limb, when it started, how it progressed, if there is a family history, make some measurements and if they are lucky, they may be able to be referred to a genetic specialist. In the examination we may find (or be told of) other associated angiodyplasias or associated syndromes such as disachiasis or a webbed neck or others. We classify and then begin treatment of the outwardly appearing symptoms of a dysfunctional lymphatic system. Phlebologists especially will focus on the venous system to elucidate if that is the cause and as to how that aspect could be re-mediated.

In reality, we have little idea of what has happened or is happening at the genetic, molecular and biochemical levels. We often cannot be sure of whether what we suggest will work. There is little or no pro-activity in this. We react and then treat in a “shotgun” approach with little knowledge or ability to target the real issues – that is the structure and functional aberrations of the lympho-vascular system. The time is right for change. Our better understanding of genetics and molecular biology will soon hopefully mean a change to this. We will act on sound knowledge, look at the structure and function of the lymphatic system and change and improve it.

We are not there yet but in the meantime the best we can do is early detection, recognition and acknowledgement of a dysfunctional lymphatic system, hopefully some prophylactic intervention (for those who have been lucky enough to gain an early referral) and an offering of the best treatment targeting the range of problems using a team familiar with the dynamics of the lymph, blood and tissue systems and of their dynamics. What we do know now though is if there are underlying issues of primary lymphoedema (lymphatic dysplasia of some type), then there are also likely to be similar issues with the vascular system, although their effects may not be as strongly manifest at times.

ARTICLE

Our genes are the blueprint for the bodies development, growth and functioning but sometimes there is an error in the code we get from each of our parents, sometimes the code is read wrongly and sometimes the code changes as we move through life.

The effect of this are changes to our biochemical and physiological events which lead to the manifestation of lymphoedema.

In the world of lymphoedemas and associated syndromes these code and misreading errors are what can form the basis of a range of dysplasias of the lympho-vascular systems, the end point of which, for some, is the development of one of a range of what we currently call primary lymphoedemas. Lately it seems that the proportion of primary lymphoedemas compared to secondary ones seems to be declining. Perhaps this is a consequence of our ability now to more accurately determine the causes and paths of lymphoedemas than previously. Not withstanding this when a primary lymphoedema (and its associated co-morbidities) manifest it is a situation which needs to be reacted to, and just as importantly, to look at the family history or the disorder and glance into the future for those who are part of the family future who may be at risk of it or already have a sub-clinical or latent form of it.

Sometimes these errors are severe and the embryo fails to thrive, while on other occasions the errors are small. Depending on the nature of these errors and on the impact of this misreading or wrong reading of coded information there can be aberrations to the structure and thus function of the lympho-vascular system and associated structures. The more wide spread these are across the lympho-vascular structures of the body the earlier they present.

For instance for those embryos which survive the earliest form of lymphoedema which forms is the congenital (hereditary/type 1/Nonne-Milroy) form. It seems to affect boys and girls equally, is more common in the legs, but certainly occurs in the genitalia, arms trunk and face at times. Its incidence is about 1 in 6000 births so in real terms it’s a fairly common genetic disorder. In this form of lymphoedema the fault seems to be on chromosome 5 and with VEGFR-3 (the FLT4 gene) for a majority of the families.

For the later types of primary lymphoedema (juvenile and later onset/Meige) it’s different in that mostly girls are affected and then usually in one limb (or more so in one limb than the other). While the exact gene here is not certain, many of those who have it do not express it (develop lymphoedema), but there is still the risk that it will be passed to their children.

Often the genetic coding or transcription error which leads to lymphoedema (or to an increased risk of it), are associated with other disorders (commonly also with a genetic basis). Under these conditions the term “syndromic lymphoedema” is sometimes used. Examples of when lymphoedema occurs with other syndromes include, yellow nail syndrome (adults), distichiasis (teenagers mostly), choleostasis, intestinal lymphangiectasia, hypoparathyroidism, Noonan’s syndrome, microcephaly and a range of other infrequent but often significant conditions. As an example of what is happening if we take the case of distichiasis there is a haplo-insufficiency due to a mutation in one allele of the gene which encodes the FOXC2 transcription factor. (Ericson, 2001). An excellent paper to read about the syndromic classification of heritable lymphoedema is by Northup et al (2003)

What is clear is that a dysplasia of the lymphatic system often is associated with a range of other malformations in other structures and systems of the body. The severity of these malformations will often determine the timing of their appearance. (Chaft et al 2003).
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One gets the impression that a really pure primary lymphoedema is rare given the strong association of lymphangiopathies with carriers of chromosomal aberrations. Because of this if one comes across a person with just a lymphangiopathic aberration (no matter what the age) then there should perhaps be an attempt to screen for symptoms of chromosomal aberrations as described above.

So focussing in on the lymphangiopathies, what has gone wrong and what is the future for those with these conditions and what are we likely to be able to do to prevent or remediate them? Our best chance seems to be linked to our expansion of our knowledge of factors affecting the growth and development of the lympho-vascular system to an extent where we can use it to, suppress abnormal growth of the lymph vascular system, grow functional vessels (where they previously did not grow) and repair lymphatic damage caused by surgery, radiotherapy, tissue trauma and other diseases/disorders?

There are proteins circulating around our body called vascular endothelial growth factors (one is VEGF-A for short). When this protein meets a special receptor (in this case VEGFR 1 and 2) then blood vessels grow, but excessive overgrowth can be bad (as occurs in tumors). Of course the opposite can also be bad – undergrowth or poorly organised growth - when there are just not enough functional vessels to supply cell and tissue demands.

There are also growth factors (VEGF-C and D) and receptors (VEGFR-1 and 2) in the lymphatic vessels and like those of the blood vascular system, high growth levels may be associated with higher risk of tumor spread but that lower levels of growth may be associated with hypoplastic conditions such as primary lymphoedemas. The importance of VEGF-C and D in the sprouting and guidance in the migration of lymphatic vessels is crucial; if levels are low then growth is poor.

For the problems associated with high growth levels we now have some indications of an anti-VEGFR-3 neutralising anti-body which seems to be able to inhibit lymph vascular regeneration, which will be just fantastic in situations where we have pathological overgrowth such as Klippel-Trenauney syndrome and others. There are many actions and interactions between the range of growth factors and receptors and the endothelium of the lymph and vascular systems leading to significant debate about which was first. (Wilting et al 2004), this part of the argument is far from settled but its clear that when there are vascular issues there are often associated lymphatic ones as well (and vice versa!). So does it really matter which was first, as long as we have better identified the growth factors, their receptors and the switches?

Of course the whole issue is not as simple as this and looking at it simplistically is often dangerous but it can at least help us understand some of the inter-relationships between our genes on one extreme and the lymphatic system and cell and tissue health, and lymphoedema on the other. What will be important to help us make better clinical decisions however will be a close look at the relationships which exist between our clinical descriptions of the range of lymph vascular dysplasias which confront us (which most often are nothing more fancy than a description of what we see externally) and what’s actually going on at the genetic, molecular and biochemical levels.

Also as suggested earlier, tied in with lymphatic vessel development issues are other dysfunctionalities such those indicated in the paper of Harvey et al and Harvey (2005) who indicated that the inactivation of a single allele of the homeobox gene (Prox1) could lead to adult onset obesity seemingly due to abnormal leakage from poorly structured lymph vessels. Maybe this is in part the reason for the unique increase in epifascial adiposity in middle and latter stage lymphoedema! Or course it might not be just this but also the fact, as lymph flow is slow in lymphoedemas and as it seems that slow lymph flow (due to possible adiopigenic factors in it) this also might also help in the deposition of fatty tissue!

We also need to consider the other crucial role of the lymphatic system and associated structures and that is its role in tumor metastases and specific immunity. High levels of VGEF-C and D correlate with high numbers of intra tumoral vessels and this with the tendency to metastasize. (Well at least that is one view and others disagree!). Again a detailed genetic interrogation and the use of the tools of molecular biology have also a lot to offer our understanding in this area.

Even further along the pathway we have the genetic basis of how patients respond to pharmacological treatment. Some time ago coumarin (Lodema) was used as a common treatment for lymphoedemas, however the discovery of significant hepatotoxicity effects and possible linked death of some patients meant it was taken from the market. And yet now it seems that the problem of hepatotoxicity could have been related to reductions in CYP2A6 activity (the major enzyme involved in coumarin metabolism), with poor CYP2A6 metabolisers metabolised coumarin via a cytotoxic pathway. There are multiple CYP2A6 variants and some of these are alleles which express the enzyme with reduced or no activity. Those who are homozygous for these alleles are poor metabolizers and thus are the ones possibly more susceptible to coumarin toxicity. (Farinola and Piller, 2007)

So there are genetic factors which will affect the development of lymphoedema, its inter-relationship with vascular development, and a wide range of other syndromes, some of which remain to be clearly identified. There are genetic factors which may affect the response to pharmacological means to manage the lymphoedema. As yet we do not have an adequate understanding to intervene and achieve better outcomes for the individual. We do hope that soon we will be able to tailor medicines to a patient, based on what we know of their genetic presentation.

We certainly now can switch genes one and off and control growth of small capillary like lymphatics as the work of Stacker, Achen and others have shown experimentally. There are quite a few steps before we can apply this practically but we are well on the way.

On very interesting step will be the application of this knowledge to tissue engineering (Neumann et al 2003) where once we have control over lymph vascular proliferation we may be able to build up a three dimensional tissue with a functional lymph vascular system and have an array of not only lymphatic capillaries but also collectors and nodes.

Harvey (2005) has indicated our most important questions of the moment relate to the nature of the signals which separates the lymphatic and blood vasculature, why there are different responses
of the superficial and visceral lymphatic networks to growth factors and which signals determine endothelial cell growth and its fate.

In the meantime we must encourage and participate in further research into primary lymphoedemas and their associated syndromes and of how best to treat and manage them. But what we can all do today is to earlier and better recognise them and begin appropriate treatment and at least show we can have some control over them even if we have an incomplete knowledge of all the reasons for their presentation. Some of our strategies here are involved around measurement of the structural and functional changes of the lympho vascular systems both generally and locally and early detection of these symptoms (such as subtle fluid accumulation through bio-impedance spectroscopy) and the functional status of the lymphatic system through lymphoscintigraphy, since it is the blood-tissue lymph interface, the pre-lymphatic fluid and the lymph which carries the signals, mediates the response and the site of action of every thing we have been talking about. Optimal flow, physio-chemical properties and communication with other entities is crucial irrespective of all of the issues or transcription or coding errors. We must recognise and react to changes while we wait for our knowledge at the genomic and molecular level to reach a level where we can begin to seriously influence the development, structure and function of the lymphatic system.

One thing seems certain however is that if the phlebologist is confronted by a swelling and it’s of relatively long duration and if it’s mostly fluid (ie no significant increase in epi-fascial fat) then it’s likely that the lymphatic system (in terms of the speed of lymph flow and flow per se) is just fine! It’s just that there is a load on the lymphatic system which is greater than its maximum transport capacity. But if there are increased fatty deposits then its likely that there are some issues with the lymphatic system and lymph flow and then its time to talk to a lymphatic expert and perhaps consider sending the patient for a functional assessment of their lymphatic system and perhaps for some appropriate lympho-stimulatory and (in the future) some lymphogenic treatment!

**Key References:**

Farinola, N and Piller, N (2007) CYP2A6 polymorphisms: Is there a role for pharmacogenomics in preventing coumarin induced hepatotoxicity in lymphoedema patients? Pharmacogenomics 8(2) 151-158

Harvey, N (2005) Embryonic Lymphatic Development: Recent Advances and Unanswered Questions. Lymphatic research and Biology 3(3) 157-165


Stacker and Achen

Wiltig 2004

Northup 2003


**Professor Neil Piller**
**Director Lymphoedema Assessment Clinic**
**Department of Surgery, School of Medicine, Flinders University and Medical Centre Bedford Park, South Australia**

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11 FEBRUARY 2008 submission Australian Financial Review

Dear Editor,

Yesterday’s editorial [What’s needed now is courage] is relevant to the withholding tax debate. Kevin Rudd can show both policy courage and political acumen by aggressively growing the Australian-managed funds industry at a time of global economic uncertainty. Achieving growth in the services sectors of medicine, finance and science would be a welcome change from dependence on natural resources, agribusiness or tourism.

Noel Hadjimichael
Executive Director

26 FEBRUARY 2008 submission Australian Financial Review

Dear Editor,

The Australasian College of Phlebology welcomes the establishment of the NHHRC (AFR 26 February) with the proviso that its outcomes reflect scientific and medical consensus on preventative health strategies. The acknowledged world class expertise of Australian phlebologists, vascular surgeons and other specialists should not be overlooked in preference to economists or commercial interests.

Noel Hadjimichael
Executive Director

4 MARCH 2008 submission Australian Financial Review

Dear Editor,

Tony Harris (AFR Corruption A Matter of Time 4 March 2008) is right to point out the pitfalls of selling Ministers’ time. As a professional college representing medical practitioners, scientists and other health care specialists, we choose for ethical and economic reasons to conduct our stakeholder relationships outside the fund-raising loop. Money spent on such access would deny resources to our outreach activities, research scholarships and social justice initiatives.

Noel Hadjimichael
Executive Director

10 MARCH 2008 submission Australian Financial Review

Dear Editor,

Reports (Rudd’s $4 billion dilemma over budget handouts, AFR, 10 March) that vitally needed health funding of medical research facilities might be cut are worrying. The Australasian College of Phlebology, like many health stakeholders, does not benefit from any of these dollars. However, the quality and reputation of our medical community does. We are able to punch above our weight in international forums and bilateral partnerships due in the main to our researchers’ high reputation amongst medical leaders in North America, Europe and Asia.

Dr Kurosh Parsi
President

11 MARCH 2008 submission SMH, Wentworth Courier

Dear Editor,

A report of any death on a long haul flight (Backpacker dies in flight, SMH, breaking news 11 March 2008) is a timely reminder of the common sense risk management steps that all passengers should take. Phlebologists and other specialist doctors interested in the treatment of venous thrombosis are only too well aware of the potential dangers for many passengers. A study completed by our team at St Vincent’s Hospital, Sydney demonstrated the importance of individual risk factors such as inherited clotting tendencies. All passengers with a serious current illness or a history of deep vein thrombosis should see their general practitioners or an appropriate specialist before long distance travel. Graduated compression stockings and other simple measures such as regular walking and good hydration can help prevent such tragic events.

Dr Kurosh Parsi
President

18 MARCH 2008 submission Australian Financial Review

Dear Editor,

Reports (Study of Unis Likely, AFR, page 7, 13 March) that graduates going overseas for more than 6 months should pay HECS immediately are sending the wrong message about professional standards. Medical graduates, at various post admission stages, find work in Britain, Canada or the less developed world a valid and enhancing professional experience. We should entice them back to utilize their valuable experience. My own post graduate training in Canada, with an acknowledged world expert, has benefited Australian patients ever since. Time to think globally and act locally.

Dr Kurosh Parsi
President

25 MARCH 2008 submission SMH, Age, AFR

Dear Editor,

The COAG meeting should steer away from any knee jerk decision to register doctors nationally. If the AMA, the Council of Presidents of Medical Colleges, and specifically the Australian Commission on Safety and Quality on Health Care, are apparently opposed to a Canberra power grab, then the national register is a "bridge too far" for the new government.

Noel Hadjimichael
Executive Director
27 MARCH 2008

Dear Editor,

The decision by COAG to proceed with the proposed national register of doctors ($1 bn health injection restores balance, AFR page 4, 27 March 2008) awaits consultation with the most important key performance asset: doctors. In the face of strong opposition, on policy and safety grounds, from the AMA and the Presidents of the Medical Colleges, this decision has wishful thinking written all over it. Standards, ethics and professional development are what the profession does best; financing and resourcing health delivery systems is what governments are elected to do. For the sake of patients and taxpayers, doctors will no doubt co-operate to make a bad decision workable.

Dr Kurosh Parsi
President

15 APRIL 2008

Dear Editor,

Prime Minister Rudd (Classic Rudd promises to respond to all, AFR, Page 10, 15 April 2008) has rightfully identified the value of a longer term focus for the 2020 Summit. However to achieve a sustainable and proactive preventative health care strategy he needs to take some emergency medicine now: swallow hard and appoint the AMA Federal President as a co-chair of the Health stream. If we can have a co-chair for the Arts we can afford a co-chair for Health.

Dr Kurosh Parsi, FACP, FACD
President

8 APRIL 2008

Dear Editor,

With the 2020 Summit a few weeks away it is timely to note that there is no guaranteed, to date, radio or television coverage of proceedings. It would be helpful if at least one of the ten mini-summits was broadcast. Health would be the ideal choice: it affects all members of the community and its recommendations have a direct impact on our youngest, most vulnerable and oldest citizens. Transparency would make healthier the whole exercise, lest backroom conclusions have been made already.

Noel Hadjimichael
Executive Director

8 APRIL 2008

Dear Editor,

Reports (Migrants add to skills crisis Herald 29 April) that all is not well with skilled migration intakes require both caution and care. It is recognized that there is a skills deficient, in both numbers and distribution, in the area of health care. Some say at least 14,000 more skilled staff are needed to meet social, regional and economic needs. Supporting local Medical Colleges to train and prepare skilled health professionals is a necessary policy in tandem with importing motivated migrants from a Third World that has its own critical needs.

Noel Hadjimichael
Executive Director

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The Australasian College of Phlebology website (www.phlebology.com.au) is the key communication tool between the College and its members. Similarly the website is the key communication pathway between members of the public and Phlebology as it is practiced in Oceania. On any one day up to 1500 visitors might peruse our data, visit the search engine, send an email or just visit a page of handy public health information. The website is under constant review and significant funds have been expended to improve and enhance its effectiveness. When many people use search engines they will find that www.phlebology.com.au will appear at the very top of their searches. The College has been active in gaining a degree of prominence in this electronic marketplace.

The key features that are well known and well patronized are the:

• Search for a doctor function
• Notices
• Professional development and continuing medical training pages
• Information about preceptorships

Recent updates to the search engine on the home page mean that members of the public can now search for College members based on the particular services that the member provides. For example, a patient will now be able to search for a practitioner in their area who can provide services related to varicose veins, or perhaps venous malformations. Members are able to select and/or change the list of services they provide through their login. Such changes will be immediately reflected in the search engine. Previously, the search engine was largely aimed at providing information based only on a member’s geographical location rather than the particular area of interest of each member.

At Vein School we have covered popliteal vein aneurysm, mechanisms of action of sclerosing agents and sclerotherapy techniques. At the next vein school we are privileged to have Dr Joanne Joseph (Haematologist) and Dr Jennifer Dew (O & G) covering procoagulant conditions, and the risks of thrombosis associated with the pill, HRT and treatment. Dr Phil Artemi will be speaking at the August Vein School on Pharmacology in Phlebology – including the use of Anthogenol, Venotrex and Paroven. Subsequent topics are to be advised and speakers confirmed.

At our most recent Clinical Meeting we had a 16-year-old boy with a thrombosis of the SSV who is homozygous for Factor V Leiden, his mother has had a DVT presumed related to her SLE. We were also able to examine and discuss the management two patients with Klippel Trenaunay syndrome, bilateral popliteal vein aneurysm, and venous malformation.

We have had a high attendance level from the registrars, a commendable performance given the time and cost it takes to be involved and I am pleased to see a number of our sonographer members attending the monthly meetings too. The plan is to create a three-year cycle of topics so that registrars will be exposed to the whole program over their period of training.

David Jenkins
Director of Training, NSW
STOP THE PRESS

Sole Health Care’s application to the Medical Services Advisory Committee (MSAC) seeking public funding for Endovenous Laser Treatment (EVLT®) for Varicose Veins has been successful. The Federal Minister of Health & Ageing signed off on the MSAC recommendation on 20th May.

When we introduced EVLT® into Australia in 2001 and commenced the initial Clinical Trial of the Diomed Laser for EVLT® with Professor Ken Myers in Melbourne our intention was always to reach this objective. Whilst this has come at the cost of countless man hours in its preparation and tens of thousands of dollars in support infrastructure for its presentation and submission we have achieved what we set out to do.

Paul Nielsen
Managing Director,
Sole Health Care Products Pty Ltd.

CALENDAR OF EVENTS

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<td>Thursday 07 August</td>
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<td>Tuesday 09 September</td>
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<tr>
<td>Friday 05 December</td>
<td>Interviews Advanced Trainee Selection ESD</td>
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<td>13:00-15:00</td>
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<tr>
<td>Saturday 06 December</td>
<td>Board of Censors Meeting College</td>
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<td>9:00-11:30</td>
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<tr>
<td>Saturday 06 December</td>
<td>AGM College</td>
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<tr>
<td>11:30-12:00</td>
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<tr>
<td>Saturday 06 December</td>
<td>Trainee Orientation ESD</td>
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<td>13:00-17:00</td>
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21
recover faster

- World-first patented Bodyflow™ Technology clinically proven to flush fluid by specifically stimulating the smooth muscle within veins, arteries and lymphatics.
- Increases lymphatic drainage and blood flow, accelerating patient recovery.
- Independent clinical trials as presented at 2007 ACP Conference have shown Bodyflow™ effective for:
  - Lymphoedema Piller, N. et al. (Flinders Medical Centre, Adelaide)
  - Blood Flow, Fibrinolysis and Anti-Procoagulant Activity After Treatment with a Portable Electrostimulation Device (Bodyflow™) in Healthy Subjects Parsi, K. et al. (Haematology Research Lab, St. Vincent’s Hospital)

If you're interested in this world-first technology and how it may assist with the growth of your business, contact:
www.bodyflow.com.au or 1300 bodyflow
Dr. Lawrence L. Tretbar, MD FACS - innovative surgeon, medical inventor, cartoonist, humorist and photographer - passed away on October 14, 2007 in Kansas at the age of 74. Larry, as he was known to most Phlebologists around the world, lived and practiced in Shawnee Mission, Kansas, where he founded the Vein Clinic of Kansas City.

His medical insight, modesty, artistic ability and rare good humor endeared him to his many friends and medical colleagues around the world. His moustache was legendary. He educated peers and students in medicine and photography, pioneered surgical procedures for obesity, venous disease and modified mastectomies, and directed a series of educational films for the American Medical Association.

In recent years Larry was dedicated to establishing Lymphology as a medical specialty in the United States. He always sought to improve medical techniques, instruments, and therapies according to the working principle of his mentor, Dr. George Crile, Jr., of the Cleveland Clinic: “Question the time-honored procedures.”

Larry’s private practice was far from typical. In addition to caring for patients, he was a prolific research scientist, publishing papers and lecturing worldwide. Central in his research was the study of varicose vein treatment, for which he wrote a textbook and developed surgical and non-surgical techniques. He was widely-known by colleagues as “The Grandfather of American Phlebology”.

Larry was born at Stafford, Kansas, on Feb. 5, 1933, the youngest son of Dr. and Mrs. J.J. Tretbar. He began college at University of New Mexico, then went on to graduate from the University of Kansas in 1955 where he was also an outstanding photographer for the Daily Kansan. Following a graduate year at the University of Vienna, he returned to enter Kansas University School of Medicine where he graduated in 1960.

On June 23, 1957, Larry married Kathleen Paulsen, whom he always described as the love of his life. Their marriage was full of laughter, ideas, and adventure. Their home - a modern “tree house” at Black Swan Lake - was a warm, inviting haven for many. In June 2007, Larry and Kathleen celebrated their 50th wedding anniversary.

From 1960-67, Larry trained in General Surgery at the Cleveland Clinic at Cleveland, Ohio, where he also worked in the laboratory of Dr. Willem Kolff, developing a prototype of the artificial heart and creating an early procedure for human liver transplant. After completing his residency in Cleveland, he was Senior Surgical Registrar at the West Middlesex Hospital in London, England.

Larry began practicing in Kansas City in 1968, first in partnership with Dr. Earl C. Sifers, and later, in solo practice. In the mid-80s, he founded the Vein Clinic of Kansas City to devote his time to the treatment of venous disease.

He was a Fellow of the American College of Surgeons, serving as President of the Kansas Chapter, 1976-77. From 1988-91, he was President of the Phlebology Society of America. From 1996-2001, he served on the Board of Directors of the American College of Phlebology. He was a founding member of the American Venous Forum, and of the American Society of Lymphology. At the time of his death, he was serving as its President and had just completed a textbook on Lymphology, now at press, with his colleague and family friend, Cheryl Morgan, PhD. For three years in the mid-1980s, he was President of the Cleveland Clinic Alumna Association. He was also actively involved with Cancer Connection and Hospice of Kansas City. From 1970-73, he was Chairman, Department of BioMedical Communication at the New UMKC School of Medicine. He was Assistant Professor of Surgery from 1970-78 at Kansas University School of Medicine.

From an Australia and New Zealand perspective, we were first blessed with Larry’s enthusiasm and humour at the inaugural Annual Scientific Meeting of the Sclerotherapy Society of Australia held in Sydney in 1994. Larry attended without any formal invitation and his presence was greatly appreciated by our then fledgling organisation. As a result of his contribution on that occasion, Larry was invited back as an International Speaker on 2 subsequent occasions and his participation at these conferences, both formally and informally were special moments never to be forgotten.

Throughout his life, Larry was always innovative and he can be credited for pioneering and reporting on the method of diluting the sclerosant, sodium tetradecyl sulphate, to relatively low concentrations to treat smaller venules and telangiectasias. He clearly demonstrated that this technique greatly reduced adverse effects such as post-sclerotherapy pigmentation and cutaneous ulceration. Although he had a great respect for the use of sclerotherapy in treatment of varicose veins and telangiectasias, he also remained enthusiastic in developing better methods for surgical management of venous disease. As such, he was the complete phlebologist. If only we could all think like Larry.

Dr Paul Thibault

Registrar's Representative

At the February Board Meeting, the College agreed that a representative of the registrars under training be sought. Dr Peter Paraskevas, of Melbourne, was subsequently chosen by the registrars to promote this communication between trainees and the College.
College Calendar – Key Dates
(See website www.phlebology.com.au for updates)

SATURDAY 31 MAY 2008
Extraordinary General Meeting 1145-1200 College Boardroom
NSW Vein School/Journal Club 1400-1700 East Sub Div

SATURDAY 5 JULY 2008
NSW Clinical Meeting 1400-1700 West Sub Div

SATURDAY 2 AUGUST 2008
ACP Board 800-1130 College Boardroom
NSW Vein School/Journal Club 1300-1700 East Sub Div

THURSDAY 7 AUGUST 2008
Launch of Victorian Faculty 1730-1930 Como House, Melbourne

SATURDAY 6 – THURSDAY 11 SEPTEMBER 2008
Annual Conference/Workshops see website Crowne Plaza Gold Coast

TUESDAY 9 SEPTEMBER
Graduation Ceremony & Launch of Queensland Faculty 1800-1900 Customs House, Uni Qld

SATURDAY 4 OCTOBER
Part I Written exams 900-1100 Vibe Hotel

SATURDAY 1 NOVEMBER
NSW Clinical Meeting 1300-1700 West Sub Div

SATURDAY 15 NOVEMBER
Part I Clinical Exams 800-1200 East Sub Div

FRIDAY 5 DECEMBER
Interviews/Adv Trainee Selection 1300-1500 East Sub Div

S ATURDAY 6 DECEMBER
Board Censors Meeting 0900-1130 College Boardroom
AGM 1130-1200 College Boardroom
Trainee Orientation 1300-1700 East Sub Div

TUESDAY 17-FRIDAY 20 MARCH 2009
ACP Workshops Sydney

MONDAY 31 AUGUST – FRIDAY 4 SEPTEMBER
UIP World Congress Monaco

The launch of the Victorian Faculty of the College will be conducted by His Excellency Professor David de Kretser AC, Governor of Victoria, on Thursday 7 August 2008 at Como House. Please contact the College Executive Director on 0409 151521 to obtain information about entry tickets. This is a special occasion for all Victorian members and fellows.

CONTACT
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www.phlebology.com.au email: info@phlebology.com.au

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Dep. Chancellor     Professor Andre van Rij

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                     Professor Lourens Bester
                     Dr Jacqueline Chirgwin
                     Dr Mark Elvy
                     Dr Gabrielle McMullin

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QLD Faculty Interim Chairman Dr Paul Dinnen
VIC Faculty Interim Chairman Dr Stefania Roberts

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Executive Director
Noel Hadjimichael