# CLINICAL ASSESSMENT OF VENOUS DISEASE

**0800-0825** Dr Abdullah Omari  
An Overview of Venous Disease

**0825-0845** Dr Louis Loizou  
Patient Assessment

**0845-0905** Dr Joseph Graiche  
Doppler Principles and the Applications of CW-Doppler

**0905-0925** Dr Robert McDonald  
Thrombophilia and Hypercoagulable States

**0925-0945** Dr Louis Loizou  
Review of CEAP Classification

## ANATOMY AND PATHOPHYSIOLOGY

**1030-1050** Dr Peter Paraskevas  
Anatomy and Physiology of the Venous System

**1050-1110** Dr Mark Elvy  
Venous Hypertension and its Complications

**1110-1130** Dr Paul Thibault  
Patterns of Telangiectasias

**1130-1150** Dr Joseph Graiche  
Interpreting Duplex Reports

## SCLEROTHERAPY

**1300-1320** Dr Paul Thibault  
Treatment Overview

**1320-1340** Dr David Jenkins  
Techniques of Sclerotherapy

**1340-1400** Dr Paul Thibault  
Sclerosing Agents and their Mechanisms of Action

**1400-1420** Dr Phillip Artemi  
Pharmacokinetics and Pharmacodynamics of the Sclerosing Agents

**1420-1440** Dr David Jenkins  
Sclerotherapy of Non-Leg Veins

## COMPLICATIONS OF SCLEROTHERAPY

**1530-1545** Dr Adrian Lim  
Absolute and Relative Contraindications of Sclerotherapy

**1545-1600** Dr Joseph Graiche  
Overview of Complications of Sclerotherapy

**1600-1615** Dr Kurosh Parsi  
Post-Sclerotherapy Ulcers

**1615-1630** Dr Adrian Lim  
Anaphylaxis, Anaphylactoid and Allergies

**1630-1645** Dr Kurosh Parsi  
Post-Sclerotherapy DVT and DVS

**1645-1700** Dr Paul Thibault  
Post-Sclerotherapy Telangiectatic Matting and Pigmentation

## MEDICOLEGAL AND TRAINING

**1730-1745** Dr Chris Lekich  
Medicolegal Issues

**1745-1800** Dr Jillian Tatham  
How to Write a Scientific Paper

**1800-1810** Ms Lucy Taylor-Turner  
Certificate of Ultrasound in Phlebology

**1810-1820** Dr Joseph Graiche  
An Overview of Training Program

**1810-1820** Dr Joseph Graiche  
An Overview of the Examination Process

**1800-1810** Ms Lucy Taylor-Turner  
Certificate of Ultrasound in Phlebology

## WIDER ASPECTS OF PHLEBOPY

**0905-0925** Dr Robert McDonald  
Thrombophilia and Hypercoagulable States

**0925-0950** Dr Joseph Graiche  
Doppler Principles and the Applications of CW-Doppler

**0845-0905** Dr David Gibson  
Overview of Ultrasound and Laser Physics

**0800-0820** Dr Mark Malouf  
Anatomy and New Venous Terminology

**0740-0800** Dr Phillip Artemi  
Pharmacology of Phlebology

## BASIC SCIENCES

**0740-0800** Dr Phillip Artemi  
Pharmacology of Phlebology

**0800-0820** Dr Mark Malouf  
Anatomy and New Venous Terminology

**0820-0850** Dr David Gibson  
Overview of Ultrasound and Laser Physics

**0850-0920** Professor Andre van Rij  
Assessment of Venous Function and Physiology

**0920-0950** Professor Hugo Partsch  
Physics of Compression

## VENOUS INSUFFICIENCY

**1030-1050** Dr Kurosh Parsi  
Dermatologic Manifestations of Venous Diseases

**1050-1110** Professor Hugo Partsch  
Swollen Limb

**1110-1130** Dr Phillip Artemi  
Leg Ulcers

**1130-1150** Dr Abdullah Omari  
Medical Management of Leg Ulcers

## VENOUS THROMBOEMBOLISM

**1300-1320** Mr David Connor  
Haemostasis and Coagulation

**1320-1340** Professor Ken Myers  
Ultrasound Assessment of Venous Thrombosis

**1340-1400** Dr Abdullah Omari  
Management of Superficial Thrombophlebitis

**1400-1420** Dr Joanne Joseph  
Management of Acute DVT

**1420-1440** Professor Lourens Bester  
Thrombolysis and IVC Filters: Indications, Contraindications, Technical Aspects

## WIDER ASPECTS OF PHLEBOPY

**1530-1550** Dr Kurosh Parsi  
Vascular Anomalies

**1550-1610** Professor Lourens Bester  
Agents used in Treatment of Vascular Malformations and Pelvic Veins

**1610-1630** Dr John Pereira  
Treatment of Venous Malformations in Children

**1630-1650** Professor Neil Piller  
Lymphoedema

**1650-1710** Professor Hugo Partsch  
Lymphography and Lymphoscintigraphy: Practical Aspects

## MANAGEMENT OF VENOUS INCOMPETENCE

**1745-1800** Professor Lourens Bester  
Pelvic Congestion Syndrome

**1800-1815** Dr David Robinson  
Surgery for Varicose Veins

**1815-1830** Dr David Jenkins  
Ultrasound Guided Sclerotherapy

**1830-1845** Professor Lourens Bester  
Radiofrequency Ablation

**1845-1900** Professor Ken Myers  
Endovenous Laser Ablation

## Supplementary Notes

**0800-0815** Professor Ted King  
Endovenous Laser Ablation: Does Fluence Make a Difference? Notes

**1630-1640** Professor Ramesh Tripathi  
Deep Vein Valvular Reconstructions - How To Do It and Why?
PART I
Basic Phlebology Certificate Course

THE AUSTRALASIAN
COLLEGE OF PHLEBOLOGY

- CLINICAL ASSESSMENT OF VENOUS DISEASE
- ANATOMY AND PATHOPHYSIOLOGY
- SCLEROTHERAPY
- COMPLICATIONS OF SCLEROTHERAPY
- MEDICOLEGAL AND TRAINING
Not available at time of printing
Patient Assessment: Venous History, Examination and Introduction to Doppler and PPG
Dr Louis Loizou

THE CONSULTATION
This is the time we are evaluating the patient and the patient is evaluating us.

DURING THE CONSULTATION
- The patient concerns are being addressed.
- Your concerns about the patient condition are adequately conveyed to the patient.
- You convey an atmosphere of confidence, competence and thoroughness.
- The patient is motivated to partake in discussion.
- Taking a thorough history.
- Performing a methodical and thorough examination.
- Performing and/or requesting appropriate investigations.
- Providing informative discussion and literature for patient education.
- Why the questionnaire?-as there are approximately 120 questions that need to be asked. The questionnaire dramatically reduces the consultation time and yet is thorough and allows the practitioner to focus on the important issues.

Become familiar with at least the revised BASIC CEAP classification- it is the universal language of the Phlebologist.
What do we want to achieve by the end of the patient assessment?
- CEAP classify
- Determine the patients relative risk of thrombosis.
- Be confident that you have had a meaningful discussion with the patient.

Basic CEAP Classification
- Co-no visible or palpable signs of venous disease.
- C1-Telangiectasia or reticular veins
- C2-Varicose veins>3mm
- C3-Oedema
- C4a-Pigmentation or eczema
- C4b-Lipodermatosclerosis or atrophie blanche
- C5-healed venous ulcer
- C6-active ulcer
- In addition to these can add S-for symptomatic and A for asymptomatic

- Etiologic Classification
- Ec-congenital
- Ep-primary
- Es-secondary(Post-thrombosis)
- En-No venous cause identified
- Anatomical Classification
As-superficial veins
Ap-perforator veins
Ad-deep veins
An-No venous location identified
Pathophysiological Classification
Pr-reflux
Po-obstruction
Pr,o-reflux and obstruction
Pn-No venous pathophysiology identified

History
Seeks to determine and clarify the answers given by the patient in the questionnaire.

Questions of special interest.
1. Patients current concerns
Do they have symptoms?

Or is the problem “cosmetic”?
Symptoms attributable to venous disease
- Aching
- Heaviness
- Tiredness
- Pain
- Throbbing
- Burning
- Tingling
- Itching
- Cramping

2. Family History
Is there a family history of varicose veins?
Inquire about blood relatives such as uncles, aunties and grandparents.

3. History of Thromboembolic Disease
Is there any family or personal history of thromboembolic disease, in particular, a past history of DVT?
- Warfarin
- Heparin
- Plane Travel
- Pregnancy
- Past Leg fractures- these can localise venous abnormalities and can also be a site of undetected DVT.

5. Past Procedures to Varicose Veins
• When and what venous procedures have been performed?
  – Ligation and stripping
  – Ambulatory phlebectomy
  – Ultrasound guided sclerotherapy
  – EVLT
  – Normal sclerotherapy
  – Cutaneous laser therapy

6. Satisfaction
• Was the patient satisfied with the treatment they received?
• If not – why?
• Was it due to
  – Pain
  – Time involved
  – Cost
  – Poor results – below patient expectations (is the patient realistic)
  – Perceived complications

7. Future Plans
• Is the patient planning an overseas trip in the near future?
• Is the treatment appropriate before travel?
• If we proceed with treatment are we putting the patient at increased risk?
• Are we able to achieve the expected result before the trip?

8. Medication
• What medication are they taking?
  – HRT
  – OCP
  – NSAI
  – ASPIRIN
  – FE / VITAMINS

Examination
• Relate to the patient’s concerns
• Clarify the venous anatomy
• Locate sites of reflux
• Assess the severity of reflux

Stand

Inspection
• A quick look at the abdomen for any distended veins.

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• Examine the legs for MALIGNANT skin lesions.
• Look at foot posture.
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- Look at the foot posture
- Perineum and inner thigh area-Looking for vulval veins which implies a possible pelvic source of reflux.

**Inspection**
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- Examine the legs for MALIGNANT skin lesions.
- Look at the foot posture
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- Thighs.
- Legs.
- Non Venous clues to venous disease.

**Non Venous Clues to Venous Disease**
- Asymmetrical swelling or oedema – ? suggesting possible past DVT (C3)
- Scars – may have caused venous distortion
- Skin discolouration (C4a)

- Early Venous Eczema
  - Also known as erythematous dermatitis and can progress to blistering, weeping and scaly eruptions (C4a).
  - Suggestive of CVD

- **ATROPHIE BLANCHE**
  - Usually around the ankle and appears like white porcelain (C4b)
  - Represents skin atrophy
  - Sign of severe CVD
  - Lipodermatosclerosis-chronic inflammation with fibrosis of the skin and subcutaneous tissue (C4b)
  - Corona Phlebectasia-early sign of advancing venous disease
  - ULCERATION- full thickness skin defect that fails to heal and is sustained by CVD (C6)
- Venous Examination
- Large Varicose Veins

—Truncal
Branch Veins

- Related to Incompetent Perforators
  - Small Veins

Reticular Vein Patterns

- Telangiectasis

Most Common Sites of Reflux
- Sapheno-femoral junction
- Sapheno-popliteal junction
- Perforating Veins
- Superficial Veins
  - Reticular
  - Telangiectasias

Most Common Sites of Reflux
- **Sapheno-femoral junction**
  - Most Common Sites of Reflux
  - Sapheno-femoral Junction
- **Sapheno-popliteal junction**
  - Most Common Sites of Reflux
  - Sapheno-femoral junction
  - Sapheno-popliteal junction
- **Perforating Veins**

Perforator veins (Old Names)

Note new classification - depicting the designated location is now preferred (e.g. Cocketts 1st, 2nd and 3rd now known as upper, middle and lower posterior tibial perforators.

- Clinical assessment alone is an incomplete and often insufficient assessment of the venous system.
- Often the underlying etiology is rarely apparent particularly in a complex presentation.
- Diagnostic evaluation is necessary if one is to understand the particular venous hemodynamics of an individual patient.
- Most advanced phlebologists would not proceed without further investigation – the two simplest being continuous wave Doppler and photoplethysmography but of course the “gold standard” is a duplex scan.
- Comments
- Duplex ultrasound has become the “gold standard” in the diagnosis of both deep venous thrombosis and venous insufficiency. Most diagnostic centres have replaced CWD and PPG with duplex ultrasound.
- It is controversial whether HHD examination alone is sufficient before undertaking treatment.
- However educated phlebologists must have knowledge of these tests and their physiologic background.
- Is there a Role of CWD?
- ABI index in patients with suspected arterial insufficiency who also have features of CVD.
- May not be very specific but is very sensitive to weak signals such as small perforators in areas of poor responding telangiectasias. These small sites of reflux are often missed with duplex scanning.
“Stethoscope of the phlebologist should be present at every venous examination which in essence allows for coupling of the physical examination and physical investigation.”
This is considered by many the minimum level of investigation before any treatment is initiated.
Is an excellent screening tool.
CWD
Takes practice and is user dependant and can identify flow abnormalities such as reflux in the superficial veins and deep veins (Austrian Physicist-Johann Christian Doppler).

- Requires methodical and systematic approach

Who do we use CWD on?
- All patients during the initial examination
- And most patients at subsequent reviews

Where on the Lower Limb do we use CWD?
- I focus on the most common sites of superficial reflux
  - 1. Sapheno-femoral junction and great saphenous vein in the thigh
  - 2. Sapheno-popliteal junction and small saphenous vein in the calf
  - 4. Perforator sites
  - 5. Small incompetent veins.

Examination of Sapheno-Femoral Junction with CWD
Examination of Great Saphenous vein with CWD
Examination of Sapheno-popliteal junction with CWD
Examination of Perforator Veins with CWD
CWD and deep system.
- The CWD was first introduced by Strandness and Baker in 1960’s. It was applied to the diagnosis of DVT and later deep vein insufficiency.
- Examination is performed with patient supine and slightly leg down.
- Again the commonest sites of deep vein reflux are examined.
- Commonest sites of deep vein reflux
- Common femoral vein at the inguinal region.
- Popliteal vein in the popliteal fossa.
- Post tibial vein behind the medial malleolus.

Photoplethysmography (PPG)

CWD can assist with identifying sites of reflux. PPG assists by quantifying the reflux in a reproducible manner.

Photoplethysmography – Method of Use

Basic Fact regarding the use of PPG
- The fact that needs to be understood is that normally refilling of blood into the venous circulation occurs only through the arterial circuit and this normally takes at least 20 – 25 seconds
• A value of less than 20 seconds indicates the presence of an abnormal refilling channel – namely retrograde flow through the superficial or deep venous system.
• But to have retrograde flow – this means the presence of incompetent valves in the system. PPG measures the Degree of Venous Valvular Incompetence
Venous History, Examination and Introduction to Doppler and PPG
Topic: Doppler Principles and the Applications of CW-Doppler
Date: Tuesday 18th September, 2007
Time: 0845-0905

Speaker:
Dr Joseph Graiche
MB,BS(HONS),FACP
Phlebologist

Conference:
Australian College of Phlebology 2007 Scientific Meeting and Workshops
Basic Phlebology Certificate Course (Phlebology Part 1)
18-21 September, Stamford Plaza Double Bay, Sydney, Australia

Session Content
• Indications
• Examination Types
• Results/Reporting
• Sensitivity & Specificity
• Limitations

Audience Survey
• Use of CW doppler
**Indications**
- Screening for venous reflux
- Assessment prior to micro-sclerotherapy
- Exclude co-existent perforator vein, truncal vein &/or tributary vein reflux
- ABI

**CW Doppler Principles**

\[
\text{Doppler frequency } (f_d) = \frac{2 \cdot f \cdot V \cdot \cos \Theta}{c}
\]

- \(f_d\) - doppler shift
- \(c\) - is speed of sound in tissue
- \(f\) - transmitted beam
- \(V\) - velocity of the blood
- \(\Theta\) - angle of incidence between the ultrasound beam and the direction of the flow

Higher Doppler frequency obtained if:
- velocity is increased
- beam is more aligned to flow direction
- higher frequency is used
Fig 3. A, At the groin, the anterior accessory of the great saphenous vein (GSV) (arrow) courses deeply in the subcutaneous layer, and below a hyperechoic fascia that resembles the GSV covering. B, The small lumen of a hypoplastic GSV as seen by duplex scan. Note the compensatory enlargement of the overlying saphenous accessory. C, Real double GSV. The two veins course within the saphenous compartment and are connected by the saphenous ligament (arrow). D, Real double femoral vein. The two veins (in blue) course close to the femoral artery (in red).
Results/Reporting

• Venous Map
• Report

Results Examples

Limitations & Examples

— Anatomical
— ?SPJ or PV
— ?SFJ or FV
— ?Perforator Vein or Saphenous Trunk

• Operator
  — Training
  — Skill
  — Experience
Ankle Brachial Indices (ABI)

1. LEFT ARM PRESSURE - With patient lying down, take the left brachial pressure. Record the value on the ABI/Risk Assessment Form.
2. RIGHT ARM PRESSURE - Take the right brachial pressure and record the value on the report form.
3. RIGHT ANKLE WAVEFORM* - Acquire arterial pulse with the Doppler at the right foot and print the waveform prior to taking the ankle pressure. Attach the waveform to the report form and mark the appropriate boxes.
4. RIGHT ANKLE PRESSURE – Take the right ankle pressure and record the value on the report form.
5. LEFT ANKLE WAVEFORM * - Acquire arterial pulse with the Doppler at the left foot and print the waveform prior to taking the ankle pressure. Attach the waveform to the report form and mark the appropriate boxes.
6. LEFT ANKLE PRESSURE - Take the left ankle pressure and record the value on the report form.
7. CALCULATE ABI VALUES - Calculate the ABI for both sides.
   (For each side, divide the ankle pressure by the higher of the two arm pressures.)

<table>
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<th>Ankle/Brachial Index Chart</th>
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<td>Ankle Pressure (mmHg)</td>
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Index | Assessment/Condition
--- | ---
Above 1.30 | Incompressible Artery
0.90 to 1.30 | Normal
0.70 to 0.89 | Mild Disease
0.40 to 0.69 | Moderate Disease
0.35 and Below | Severe Disease

Resources

Books

- Gent, R (1997), Applied Physics and Technology of Diagnostic Ultrasound, Women's and Children's Hospital, South Australia.
Tues 18 Sept 0905-0925  Dr Robert McDonald  Thrombophilia and Hypercoagulable States

POWERPOINT (see next page)
Why do we want to classify venous disease?

To CEAP or not to CEAP that is the question?

Classification of diseases is a basic instrument for the UNIFORM diagnosis and for the meaningful COMMUNICATION about the disease no matter where a doctor has been trained.

Brief History of venous classification

• 1978- WIDMER-proposed a classification mainly related to clinical appearance.
• 1979-HACH-proposed a classification according to the degree of GSV reflux.
• 1980-PARTSCH-proposed a classification based on anatomical involvement of the superficial, perforating and deep veins BUT relating this to reproducible objective measurements to help select and distinguish the patients that can best be treated.
• 1985-SYTCHER-proposed a classification with considerations similar to the current CEAP.

With CVD the reliance for too long had been placed on clinical appearance only without relating this appearance to an accurate diagnosis of the underlying venous pathology and reflux.

**CEAP Classification**

• C- Clinical Manifestations
• E-Etiologic Factors
• A-Anatomical Consideration
• P-Pathophysiology

**TERMINOLOGY and DEFINITIONS**

• The committee quickly established that to develop a UNIVERSAL classification system for CVD also required a consensus on the definitions of the words used in the classification.

• **CVD** – including the full spectrum of morphological and functional abnormalities of the venous system from telangiectasias to venous ulcers.
• **Telangiectasias** – a confluence of dilated intradermal venules of<1mm in diameter. Synonyms include spider veins, hyphen webs and thread veins.
• **Reticular veins** – dilated bluish subdermal veins usually between 1mm -3mm in diameter. Synonyms include blue veins, subdermal varices and venulectasias.

• **Varicose veins** – subcutaneous dilated veins greater than or equal to 3mm in diameter as measured in the upright position. Synonyms include varix, varices and varicosities.
Corona Phlebectatica – a fan shaped pattern of numerous intradermal veins on the medial or lateral aspects of the ankle and foot. Synonyms include malleolar flare and ankle flare.

Oedema – a perceptible increase in volume of fluid in the skin and subcutaneous tissue characteristically indenting with pressure. Usually located in the ankle but also can extend to the leg and foot.

Pigmentation – a brownish darkening of the skin resulting from extravasated blood, which usually occurs in the ankle region but may extend to the leg and foot. A sign of increasing venous pressure due to CVD.

Eczema – an erythematous dermatitis which may progress to blistering, weeping, or scaly eruption of the skin of the leg. Commonly located near varicose veins but can be localised anywhere on the leg and reflects uncontrolled CVD.

Lipodermatosclerosis – localised chronic inflammation and fibrosis of the skin and subcutaneous tissue of the lower leg and suggests severe CVD.

Atrophie Blanche – localised whitish and atrophic skin areas surrounded by dilated capillaries and sometimes hyperpigmentation and is a sign of severe CVD.

Venous Ulcer – full thickness defect of the skin most frequently seen in the ankle region that fails to heal spontaneously and is sustained by CVD.

CEAP- Clinical Classification
* C0 – No visible or palpable signs of venous disease.
* C1 - Telangiectasia or reticular veins.
* C2 – Varicose veins.
* C3 – Oedema.
* C4a – Pigmentation and/or eczema.
* C4b – Lipodermatosclerosis and/or atrophie blanche.
* C5 – Healed venous ulcer.
* C6 - Active venous ulcer.
* S - Symptomatic-including ache, pain, tightness, skin irritation, heaviness, muscle cramps, burning as well as other complaints due to venous dysfunction.
* A – Asymptomatic.

CEAP – Etiologic Classification
* Ec – Congenital
* Ep – Primary
* Es – Secondary (post thrombotic)
* En – No venous etiology identified

CEAP – Anatomic Classification
* As – Superficial veins
* Ap – Perforator veins
Ad – Deep veins
An – No venous location identified

CEAP – Pathophysiologic Classification
Pr – Reflux
Po - Obstruction
Pr,o – Reflux and Obstruction
Pn – No venous pathophysiology identified

Basic CEAP classification - example
• Painful swelling of the leg and varicose veins plus lipodermatosclerosis and active ulceration where duplex scan on May 17, 2007 demonstrated axial reflux of the GSV above and below the knee, incompetent perforators and axial reflux in the femoral and popliteal veins.
• Basic CEAP- C6,S,Ep,As,p,d,Pr

Additions to Basic CEAP

Date of Classification
• As venous disease is not static it is recommended that any CEAP classification be followed by a date that that classification was made.

Level of Investigation
• As a precise diagnosis of the underlying venous pathology is the basis for a correct classification it is recommended that the level of investigation utilised be included in any CEAP classification.
• Level 1- office visit with history and examination and/ or use of HHD.
• Level 11- non invasive – Duplex scan and /or PPG.
• Level 111 – Invasive – varicography, ascending and descending venography, venous pressure measurements, spiral CT scan or MRI.

Anatomical locations
• Same as basic CEAP with the addition of 18 named and numbered venous segments used to localise the venous pathology.

•SUPERFICIAL VEINS
• 1) telangiectasias/ reticular veins.
• 2) great saphenous vein reflux above knee.
• 3) great saphenous vein below knee.
• 4) small saphenous vein.
• 5) nonsaphenous veins.

•DEEP VEINS
• 6) inferior vena cava.
• 7) common iliac vein.
• 8) internal iliac vein.
• 9) external iliac vein.
• 10) pelvic-gonadal, broad ligament veins, other.
• 11) common femoral vein.
• 12) deep femoral vein.
• 13) femoral vein.
• 14) popliteal vein.
• 15) crural-anterior tibial, posterior tibial, peroneal veins (all paired).
• 16) muscular-gastrocnemial, soleal veins, other.
• 17) perforating veins, thigh.
• 18) perforating veins, calf

**Advanced CEAP classification example.**

* Painful swelling of the leg and varicose veins plus lipodermatosclerosis and active ulceration where duplex scan on May 17, 2007 demonstrated axial reflux of the GSV above and below the knee, incompetent calf perforators and axial reflux in the femoral and popliteal veins. No obstruction.
  * Basic CEAP- C6,S,Ep,As,p,d,Pr
  * Advanced CEAP-C2,3,4b,6,S,Ep,As,p,d,Pr 2,3,18,13,14 (2007-05-17, L11)
Anatomy & Physiology of the Peripheral Venous System
By: Dr Peter Paraskevas

Main Physiological Functions
- Return of Venous Blood back to the Heart
- Thermoregulation
- Storage of blood
  - 70% of blood stored in the venous system
- Regulation of cardiac output

Thermoregulation
- Vasoconstrictor fibres supply the SVS
- Heat response:
  - Removal of vasoconstriction
  - Cooler blood diverted towards skin helping in heat dissipation
- Cold response:
  - Constriction of arterioles and superficial veins
  - This allows heat conservation by diverting venous blood through the perforators into the deep veins which lie closer to the arteries

The Physiology of Venous Return
- As arterial blood flows into the leg, distal superficial veins constantly fill
- Venous Blood is regularly emptied from the superficial system into the deep venous system via the SFJ, SPJ and perforators
- This blood is then returned to the right side of the heart through one-way valves by calf muscle contraction.
- Venous Return occurs uphill against gravity, against fluctuating thoraco-abdominal pressures and sometimes in the face of additional back-pressures such as CCF.
- The process of Venous Return depends on the patency of the flow circuit and on the normal functioning of the Calf Muscle Pump and Venous Valves

Valvular System
- ‘One-way’, Bi-cuspid
- These valves permit blood flow from superficial to deep (foot is only exception) and from distal to proximal
- Located every few centimeters in veins below the CFV
- Located in Perforators and at Major Junctions (SFJ, SPJ)
- Made up of thin sheets of collagen/smooth muscle, covered by endothelium

Calf Muscle Pump and Ambulatory Venous Pressure (AVP)
- When calf muscles are at rest, deep veins expand and blood is drawn in from the superficial veins.
- Venous Refilling occurs via arterial inflow (VRT 25-30 s).
- Normal Resting Supine Venous Pressure in the foot is approximately 80-100 mmHg.
- With calf-muscle contraction, blood is forced up the deep veins
- Foot Pump also contributes.
- The immediate post-AVP is about 20% of the resting supine venous pressure.
Venous Pathophysiology

**Chronic Venous Insufficiency** is caused by either impaired venous outflow or abnormal (retrograde) venous inflow.

- Chronic Venous Hypertension occurs when the AVP is persistently elevated
- Exercise can no longer empty the leg of blood and thereby decrease AVP.
- Failure of this normal process, leads to a persistently elevated venous pressure despite ambulation.
- This subsequently results in Chronic Venous Hypertension.
- Any condition that increases venous inflow or impedes venous outflow will result in a persistently elevated venous pressure during or immediately after ambulation.

**Calf Muscle Pump Failure**

- CMP failure leads to incomplete emptying of venous blood from the leg and hence an increase in post AVP
- Muscle Atrophy
  - bed rest and immobility (plaster casts)
  - muscle injury
  - deliberate dieting, malnutrition, malabsorptive states, eating disorders
  - Neuromuscular causes – neuropathies, MND, nerve injuries, neuralgias
  - Malignancy (pancreatic, stomach, lung, etc...)
  - Chronic Infection (HIV, TB)
  - CCF
  - Endocrine Causes (Hyperthyroidism, Addisons, Cushings)
  - Arthritic (OA, Rheumatoid)
  - Seven Masquerades (Depression, Diabetes, Drugs, Anaemia, Thyroid, etc...)
  - Prolonged Standing

**Deep Venous Obstruction**

- Thrombotic
  - Spontaneous
  - Induced
  - Hereditary
  - Acquired
- Non-thrombotic
  - Diaphragm-like membrane
    (Kilken et al, 2004)
  - Fibrous Tissue compressing DVS (KTS)
  - Tumour
1. Mesenchymal Chondrosarcoma
   (Kim et al., 2003)
2. Primary Venous Leiomyosarcoma
   (Zhang & Wang, 2006)
3. Primary Malignant Lymphoma
   (Rulli et al., 2002)
4. Popliteal Vein Compression

**Valvular Incompetence**

- Deep Incompetence
  - primary valve agenesis (?KTS)
  - Prior Valve damage
  - direct trauma
  - dilation with 2ndary valve failure
- Perforator Incompetence
- trauma
- secondary to deep vein obstruction

Superficial Incompetence Valve Failure
- Gravitational Hydrostatic Pressure (prolonged standing)
- Congenital Weakness of valves
- thrombophlebitis
- trauma
- hormonal influences

Superficial Venous System

Great Saphenous Vein
- Originates in Medial Foot as part of dorsal venous arch
- Continues proximally, along the medial aspect of the foot as the medial marginal vein of the foot
- GSV then passes anterior to the medial malleolus
- Ascends along the tibial edge of the medial calf to cross the knee.
- Lies within a fascial compartment (not as large and well defined as in the thigh)
- From the upper calf to the groin, the GSV lies within a very clearly defined fascial compartment (superficial and deep fascial fascia) known as the “saphenous eye”.
- Typical normal GSV is 3-4 mm in diameter
- Usually has 10-20 valves

Saphenous Nerve and its association with the GSV
- Very close association with the “Saphenous Nerve”, in the lower leg, which may be injured during surgical stripping/EVLT/UGS
- Saphenous Nerve is the largest branch of the Femoral Nerve and is purely sensory, supplying the anteromedial and posteromedial aspects of the lower leg.

Sapheno-Femoral Junction
- The GSV terminates into the SFJ (a short segment that receives multiple tributaries)
- There is a constant terminal valve 1-2 mm distal to the termination of the GSV
- There is a preterminal valve a further 2 cm distal which marks the distal area of the SFJ – this is the upper limit for EVLT.

GSV Tributaries
- Anterior Accessory Saphenous Vein
- Posterior Accessory Saphenous Vein (PASV)
- Anterior Thigh Circumflex Vein (ATCV)
- Posterior Accessory GSV of leg (aka Posterior Arch Vein)
- Anterior Accessory GSV of leg
- Communicating Branch with SSV, usually via another tributary.

Small Saphenous Vein
- Drains the Postero-Lateral Aspect of the Leg and lateral aspect of the foot.
- Originates in the lateral foot as part of the dorsal venous arch.
- Ascends proximally behind the lateral malleolus as continuation of lateral marginal vein of foot
- Frequently terminates at the popliteal vein, but this may vary.
- The SSV lies for its entire length in an inter-fascial compartment defined by the deep muscular fascia and superficial fascia.
The distal compartment appears on ultrasound as an “Egyptian eye”

The proximal compartment is defined by the medial and lateral heads of gastrocnemius and the superficial fascia

Sural Nerve intimately associated in distal 1/3
Medial Cutaneous Sural nerve in upper 2/3s
9-12 valves

**Sural Nerve and its association with the SSV**

- Sural Nerve is formed in the distal portion of the leg by the union of the Medial Sural Cutaneous Nerve (branch of tibial nerve) and a Peroneal Communicating Branch. In 20%, the peroneal communicating branch may be absent. The Lateral Sural Cutaneous Nerve may also contribute.
- Although the sural nerve is considered to be a sensory nerve, motor fibres have been found in 4.5% of cases. (Amoiridis G, Schols L, Ameridis N, Przuntek H. *Motor fibers in the sural nerve of humans*. Neurology 1997;49:1725-8)
- The Sural Nerve is intimately associated with the SSV in the distal calf. It lies lateral to the SSV in the distal leg.
- Injury to the sural nerve following surgery can cause permanent lateral leg/foot paraesthesia
- Care must also be taken with EVLT – in the lower third of the leg.

**Thigh Extension of the SSV**

- Confirmed by ultrasound
- Present in 95% of limbs
- Lies deep to the fascia of the posterior thigh
- 4 distinct patterns
  1. Continue as single vein to the Gluteal area
  2. Join FV via post. or post.lateral perforator
  3. Divide into many muscular or s/c branches
  4. Connect to the PTCV which then connects to the GSV. This complex of veins (TE + PTCV) is termed Giacomini Vein.
Termination of the SSV – 3 possible variations

1. SSV joins popliteal vein at the SPJ, joins deep veins at higher level via TE, or joins GSV via Giacomini
2. SSV continues as TE/Giacomini but communicates with popliteal vein via small anastamosis
3. There may be no connection to the popliteal or deep veins. Hence, SSV continues as TE or Giacomini Vein

Sapheno-Popliteal Junction
- Position of the SPJ is highly variable
- Most often situated within 2-4 cm above the knee crease, but above this level in 25%
- SSV joins popliteal vein from the posterior aspect in 15%, postero-medial in 30%, lateral in 42% and antero-lateral in 1%
- Terminal SSV has a terminal valve in close proximity to the popliteal vein and a pre-terminal valve just below the depart of the TE of the SSV

Tributaries of the SSV
- Subcutaneous tributaries pierce the superficial fascia
- Common tributary seen on regular U/S is the so-called “popliteal fossa perforating vein”. First described by Dodd
- Runs s/c along post.aspect of calf and popliteal fossa, sometimes parallel to SSV
- Typically forms a separate junction with the popliteal vein, usually lateral to the SPJ
- Communicating branch with GSV or its tributaries

Lateral Venous System
- aka Lateral Subdermic Venous System or Albanese system
- May represent the remnant of the embryonic lateral marginal vein
- Extremely common and accounts for large % of phlebologist’s practice
- Normal flow is paradoxically downwards from proximal thigh into lateral thigh and lateral knee perforators.
- Reflux commonly occurs via these perforators
- Small percentage occur via incompetent ATCV/GSV

Perforators
- Perforators act as alternative pathways from superficial to deep
- They pass through anatomical defects in the deep fascia and join directly with deep veins of the thigh or calf
- They usually contain one way bicuspid valves that allow blood flow from superficial to deep
- All perforators are accompanied by an artery

GSV System Perforators
- Perforators of the femoral canal (formally Dodd) connect the GSV to the Femoral Vein.
- Para-tibial Perforators (formally Sherman in the lower and mid leg and Boyd in the upper leg) connect the GSV or its tributaries to the Posterior Tibial Veins.
- Posterior Tibial Vein Perforators (formally Cockett’s) are divided into upper, middle and lower and connect the Posterior Arch Vein to the Posterior Tibial Veins.
- Anterior Leg Perforators (pierce the Anterior Tibial compartment to connect the ant. GSV tributaries to the anterior tibial veins.
SSV System Perforators
- Soleal Perforators – perf. of May
- Para-Achillean Perforators – perf. of Bassi

Deep Veins of the Calf
- Intra-muscular
  (venous sinusoids within the corresponding muscle, coalesce to form these veins. In most cases, these are paired and run with a corresponding artery)
  - soleal, gastrocnemius
- Inter-muscular veins
  (these veins are all paired and run with their accompanying artery)
  - peroneal, post. tibial, ant. tibial
- Outflow tract
  - popliteal vein

Deep Veins of the Thigh
- Popliteal vein
- Femoral Vein (not to be referred to as the Superficial Femoral Vein)
- Deep Femoral Vein (aka Profunda Femoris Vein)
- Common Femoral Vein
- External Iliac Vein

Important Nerves for Phlebologists to consider:
- Saphenous Nerve
- Sural Nerve
- Sciatic Nerve
- Common Peroneal Nerve

References:
3. Weiss RA, Weiss MA, Fied CF (2001), Vein Diagnosis & Treatment, Chapter 4, pp. 23-30, USA, McGraw-Hill Companies
Chronic venous insufficiency (CVI) is a common condition, affecting 2-5% of the population. It is related to primary or secondary dysfunction of the musculo-venous system resulting in chronic ambulatory venous hypertension and eventual microangiopathic changes of the skin and pannicular with associated complications.

Primary dysfunction result in reflux from
- Varicose vein disease with valvular and or venous wall abnormalities of the superficial and deep system

Secondary changes from
- Deep and Superficial Venous Thrombosis with subsequent recanalization resulting in obstruction and or reflux due to valvular damage

Chronic venous insufficiency is caused by malfunction of the venous system associated with chronic venous hypertension. When the venous system is normal, exercise such as walking activates the calf muscle pump, reducing the venous pressure in the foot from 90 mm Hg on standing to around 30 mm Hg during walking.

With every thigh and calf muscle contraction, blood is expelled out of the leg. During muscle relaxation, the competent valves prevent reflux. The venous pressure remains low because of slow filling from arterial inflow. Obstruction to venous outflow or reflux due to valve damage interferes with this normal mechanism and results in high ambulatory venous pressure during exercise.

Many previous pathophysiological explanations for the changes seen in CVI are now considered to be out of date.

Dysfunctional Venous systems are now believed to be related to injury to the vein walls and venous valves. This injury is largely due to a sterile inflammatory process. There are other contributing factors and these may include
- Genetics
- Obesity
- Female gender
- Pregnancy
- Standing occupations in females.

Vein wall injury results in the clinical presentation of tortuosity and dilatation seen in varicose veins.

Increasing venous diameter leads to functional valvular failure and reflux.

The effect of this reflux is chronic ambulatory venous hypertension leading to microangiopathic skin changes.

Clinically it manifests itself as lower extremity oedema, pain, itch, skin colour change and ulceration. The earliest sign of venous hypertension are elongated and dilated veins in the epidermis and dermis called telangiectasia. Deeper subcutaneous reticular veins may dilate and elongate. Above the superficial fascia are the varicose veins which become tortuous, dilated and incompetent.

The exact mechanism of vein wall and valvular dysfunction seen in CVI is still controversial.

However, we can consider this to be a chronic inflammatory process triggered by venous hypertension, in which there is a white cell/endothelial interaction with the markers of chronic sterile inflammation.
There is evidence that leukocyte migration occurs into the parenchyma of venous valves and vein walls, resulting in destruction of elastin and possibly collagen leading to perforated scarred, non-functioning valves and elongated, thinned wall varicose veins.

The other manifestation of venous hypertension is in the skin, where again leukocyte activation and inflammatory responses have been implicated as playing a major role in the pathophysiology of CVI.

Complications of CVI
- Varicose veins
  - Cosmetic problem
  - Symptoms
  - Complications SVT, Bleeding, Varicose ulcers
- Oedema
- Pain
  - Venous hypertension in muscles and fascial compartments of the lower leg from exercise and prolonged standing results in the characteristic ache of CVI. The discomfort is described as pain, pressure, burning, itching, dull ache, or heaviness in affected calves or leg
- Venous dermatitis,
- Lipodermatosclerosis:
  - These characteristic skin changes in the lower extremities include capillary proliferation, fat necrosis, and fibrosis of skin and subcutaneous tissues. Skin becomes reddish or brown because of the deposition of hemosiderin from red blood cells
- Ulceration

Current theory implicates inflammation as the cause for valve, vein wall and advanced skin changes in chronic venous dysfunction. This is triggered by chronic ambulatory venous hypertension.

Hence the main stay of treatment is correction of this venotensive state.
Lower limb telangiectasias are visible, ectatic dermal veins measuring 0.1 to 1mm in diameter. Initially telangiectasias appear as faint erythematous lines, but with time they become progressively more dilated, tortuous, and elevated above the skin surface and turn blue. The term “venulectasia” is used to describe larger blue telangiectasia measuring 1 to 2mm in diameter. Invariably telangiectasias are closely associated with either dilated subcutaneous reticular veins or varicose veins. Frequently a family history of similar abnormally dilated lower limb veins is noted, but this factor is not invariable. The disease appears to be related to western lifestyle (diet, occupation and physical inactivity) with hormonal influences (particularly oestrogen) being pivotal in the ultimate expression of the disorder. Hence telangiectasias and reticular veins are more common in women with their onset frequently precipitated by the hormonal surges of menarche and pregnancy. In certain patients telangiectasias may develop or be aggravated by treatment with oral contraceptives or menopausal hormone replacement therapy. The most common time of presentation for treatment is between the ages of 30 and 50 years, but with the modern trend of emphasizing physical attractiveness, presentation as early as 14 years and as late as 75 years is not unusual. It is less common for men to seek treatment for lower limb telangiectasias.

Unattractive or disfiguring visual appearance is the most common presenting symptom of patients with reticular veins and telangiectasias. However, mild to severe pain is also a well-recognized symptom. Isaacs has found that aching/pain, excessive tiredness/fatigue, and throbbing in the legs correlate well with patients presenting with non-bulging reticular veins and telangiectasias compared with a matching control group. Furthermore, these symptoms were independent of the size of the veins.

The patient who presents with reticular veins and telangiectasias first should have a directed history and examination. Important information to obtain in the history includes age of onset, possibility of aggravation by past pregnancies, and whether the condition is stable or deteriorating. It is important to note whether the patient’s chief complaint is predominantly cosmetic or pain-related because this factor may significantly influence the patient’s expectations regarding treatment outcomes and consequently affect treatment decisions. History taking should focus on whether the patient has had any bleeding disorders, episodes of superficial thrombophlebitis or deep venous thrombosis, and previous treatment, including surgery and sclerotherapy. Past and present use of hormonal contraceptives and hormonal replacement therapy, cigarette consumption, and allergy to any medications also should be noted. A thorough family history will provide valuable information as to the potential severity of the telangiectasia.

Examination of the lower limbs is best performed with the patient standing on a platform in front of the physician. Good lighting is essential for a systematic inspection of each aspect of the leg from the groin to the toes. The patterns of telangiectasias and their relationships to underlying reticular and varicose veins must be noted. It is important to remember that patients with cosmetic symptoms associated with lower limb telangiectatic veins may have incompetence of the major superficial veins (greater or short saphenous veins). In a study of patients presenting with cosmetic symptoms related to lower limb superficial veins, duplex evaluation of limbs with telangiectasias without clinical evidence of associated bulging reticular veins revealed a significant incidence of incompetence in the greater or short saphenous veins (Table-1). It is uncommon, however, for telangiectasias to be associated with deep venous incompetence. Therefore after physical examination, further diagnostic evaluation with Doppler ultrasound, or duplex imaging should be considered if there is clinical suspicion of incompetence of the greater or short saphenous systems. For example, telangiectasias occurring on the medial aspect of the leg are frequently associated with incompetence in the greater saphenous system. In particular, the appearance of telangiectasias on the proximal medial calf should arouse suspicion of incompetence in the greater saphenous vein and necessitate further
evaluation with Doppler or duplex. In the absence of obvious truncal varices, the incompetence is general segmental with a competent saphenofemoral junction.

Isolated telangiectasias located on the mid and distal calf should arouse the suspicion of short saphenous vein incompetence. If Doppler examination confirms reflux in the short saphenous system, duplex imaging will be required to determine the extent of incompetence and whether the short saphenous vein terminates in the popliteal vein, femoropopliteal vein or as a branch of the thigh segment of the long saphenous vein (thigh posterior circumflex vein).

REFERENCES
<table>
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<tr>
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<th>Telangiectasias only (N = 83)</th>
<th>Telangiectasias and varicose veins (N = 314)</th>
<th>Varicose Veins only (N = 84)</th>
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<td>Average age (yr)</td>
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<td>41.4</td>
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<td>Superficial incompetence %</td>
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<td>66.7</td>
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<td>Deep incompetence %</td>
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<tr>
<td>Perforator incompetence %</td>
<td>1.2</td>
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<td>13.1</td>
</tr>
</tbody>
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Topic: Interpreting Duplex Reports
Date: Tuesday 18th September, 2007
Time: 1130-1150
Speaker:
  Dr Joseph Graiche
  MB,BS(HONS),FACP
  Phlebologist
Conference:
  Australian College of Phlebology 2007 Scientific Meeting and Workshops
  Basic Phlebology Certificate Course (Phlebology Part 1)
  18-21 September, Stamford Plaza Double Bay, Sydney, Australia

Session Content
• Indications
• Nomenclature
• Examination Types
• Results/Reporting
• Limitations/Artifacts
• Resources
• Future Directions

Audience Survey
• Access to Diagnostic Ultrasound
• Visiting Medical Officers
• Ownership of Equipment
• Operation of Diagnostic Ultrasound
**Indications**
- Assessment
- Diagnosis
- Intervention
- Follow-up
- Predictive Tool

**Nomenclature**
- Consensus Statement
- Relationship to fascial planes

**Lower Limb Venous Framework**

**Saphenofemoral Junction**

Fig 2. A. Schematic representation of the hemodynamic role of the sapheno-femoral junction (SFJ) valves (modified from Pieri et al., 1995). B, The first exhaustive representation of the SFJ with its valves. Modified from the *De Venarum Osannis*, of Jeronimus Fabricius Ab Acquapendente, Venice, 1603. **TV**, Terminal valve; **PTV**, preterminal valve; **SSV**, suprasaphenic valve; **ISV**, infrasaphenic valve.
**GSV Anatomy**

*Fig 3.* A, At the groin, the anterior accessory of the great saphenous vein (GSV) (*arrow*) courses deeply in the subcutaneous layer, and below a hyperchoic fascia that resembles the GSV covering. B, The small lumen of a hypoplastic GSV as seen by duplex scan. Note the compensatory enlargement of the overlying saphenous accessory. C, Real double GSV. The two veins course within the saphenous compartment and are connected by the saphenous ligament (*arrow*). D, Real double femoral vein. The two veins (*in blue*) course close to the femoral artery (*in red*).

**Saphenous System Anatomy**

*Fig 1.* A, Axial computed tomography scan of the thigh. The greater saphenous vein (*) and the saphenous accessories (*arrow*) course in different planes, separated by the saphenous fascia (*arrowheads*). B, Axial section from a cadaveric limb showing the close relationships of the great saphenous vein (*) with the saphenous fascia (*arrowheads*) and the underlying muscular fascia (*MF*). *SL*, Saphenous ligament.
Examination Types
• Venous Mapping (SCM)
• Pre-Operative Markings (SCP)
• Cross Sectional Echography (SCE)
• Deep Venous Scan (SCD)

Venous Mapping (SCM)
• Deep Venous Map
• Truncal Venous Map
• Perforators map
• Tributaries map
• Critical Positive/Negative Findings
  — Anatomical Variations
  — Reflux
  — Abnormal Flow

Deep Venous Scan (SCD)
• Post Treatment
• Investigation of DVT

Results/Reporting
• Venous Map
• B Mode image
• B Mode-Colour Image (Duplex)
• Pulsed Doppler (Triplex)
• Report
• Data Entry
Limitations & Examples

• Technology
  — Artifacts
  — Physics

• Operator
  — Experience
  — Skill

• 2D slice of moving 3D object
  — Training

Artifacts

• Attenuation Artifacts
  — Acoustic Shadowing
  — Acoustic Enhancement
  — Edge Effect

• Beam Dimension Artifacts
  — Beam Width
  — Slice Thickness
  — Side Lobes
  — Grating Lobe

More Artifacts

• Depth of Origin Artifacts
  — Reverberation
  — Comet Tail
  — Ringdown
  — Velocity Artifacts
  — Range Ambiguity

• Beam Path Artifacts
  — Refraction Artifacts
  — Reflection Artifacts
  — Mirror Artifacts

Even More Artifacts

• Equipment Settings Artifacts
  — TGA Artifacts
  — Multiple Focal Zones Artifacts
  — Electronic Noise Artifacts

• Operator Dependant

Future Directions

• Image/Resolution Improvement

• Post-processing Power

• Smart Probes

Resources

Books
— Gent, R (1997), Applied Physics and Technology of Diagnostic Ultrasound, Women’s and Children’s Hospital, South Australia.
Web
• http://pear.co.nz/asum/docs.php?s=1&m=clinical_guides

Journals
References
Compression.

Compression is an important treatment modality in the management of:
1. Venous thrombosis
2. Varicose veins
3. Prevention of varicose veins
4. Functional venous problems
5. Varicose veins in pregnancy
6. Post-partum venous disease
7. Deep venous insufficiency
8. Venous ulcers
9. Lymphoedema

Compression is usually used in conjunction with mobilisation wherever possible.

Surgery

1. Surgery is the traditional treatment for major varicose veins. The stripping operation for truncal (GSV and SSV) incompetence has been shown to give superior long-term results compared with simple junctional ligation. Currently, due mainly to higher adverse effects of surgery compared to more recently developed less invasive treatment modalities, surgery is being used less often.

2. Endoscopic ligation of incompetent perforators is another surgical technique occasionally used.

3. Ambulatory phlebectomy is commonly used as a method of eliminating varicose tributaries of all sizes. This method has gained popularity over the past 15 years.

4. External valvular “cuffing”

Sclerotherapy

Treatment of choice for reticular veins and telangiectasias

Ultrasound guided foam sclerotherapy used for:
1. truncal incompetence with vein diameters < 6mm,
2. incompetent perforators,
3. recurrent post-surgical varicose veins
4. other atypical refluxes eg Giacomini and femoro-popliteal refluxes

Physical Ablation Techniques

1. Endovenous laser
2. Radio-frequency ablation

These 2 methods are used as alternative, less invasive approaches to the surgical stripping procedure. They are generally indicated for truncal incompetence (GSV or SSV) where the vein diameter in greater than 5mm.

Vascular Laser

Has limited applications in phlebology but has been used for:
1. Progressive ascending telangiectasia
2. Vascular malformations
3. Post-sclerotherapy pigmentation
Sclerotherapy developed in the mid 1800s following the invention of the syringe in 1851. By the end of the 19th century it was considered a dangerous procedure and not to be recommended. This was mainly due to the hazardous solutions being used. Following World War 1 safer solutions were used and a renaissance of ‘the injection technique’ during the 1920s saw the popularity of sclerotherapy dramatically increase. With the development of antibiotics and safer anaesthetics, surgical management flourished from the 1930s until recent years.

Superficial sclerotherapy – has been used for the treatment of varicose veins that didn’t look bad enough to the treating doctor to justify surgery, to tidy up persistent varicose veins that remained after surgery, or if the patient didn’t want surgery in the first place. The hit-and-miss nature of the procedure guaranteed variable and poor results. It was recognised from the 1920s that a good cosmetic result could be achieved, however, social attitudes towards seeking cosmetic improvement, frequent complications from injecting and the absence of ultrasound assessment precluded the refinement of sclerotherapy techniques until the 1990s!

The three major techniques of the 20th century (Tournay, Sigg and Fegan) will be presented, as well as that of Hobbs and the ‘combined technique’ which involves injecting from superficial to deep, proximal to distal and largest to smallest. The advantages and disadvantages of various techniques described in sclerotherapy texts will be discussed.
SCLEROSING AGENTS
Dr PK Thibault

The purpose of sclerotherapy is to produce endothelial damage that results in permanent endofibrosis and clinical obliteration of the vessel. The ideal sclerosant would have a highly specific mechanism of action, would be free of adverse effects when used for this purpose, and would not produce allergic reactions. Although many agents have been used in treating varicose veins and telangiectasias, thus far none have completely satisfied the criteria for the ideal sclerosant.

The sclerotherapist should have a sound knowledge of the mechanism of action and the adverse effects of all available solutions in order to select the sclerosant that will optimize results in each patient. The following agents are available for use in Australia and New Zealand.

Osmotic Agents

Osmotic agents exert their effects by dehydrating endothelial cells through osmosis, which results in endothelial destruction. They are hypertonic solutions, and their effect is dependent on the existence of an osmotic gradient. Because osmotic agents are rapidly diluted in the bloodstream, they lose their potency within a short distance of injection and are less effective in the treatment of veins larger than 3 to 4mm in diameter.

Hypertonic saline solution. Hypertonic saline is the most commonly used osmotic agent. The advantages of hypertonic saline are its low cost, ready availability, lack of allergenicity of unadulterated solutions, and rapid clinical effect. It also has been reported to cause less telangiectatic matting compared with polidocanol. The significant adverse effects of hypertonic saline relate to its non-specific action of destroying cells within its osmotic gradient. Therefore extravascular injection is liable to cause cutaneous ulceration, which can also occur with intravascular injection via diffusion through a damaged endothelium. Diffusion of hypertonic saline through the vein wall also results in irritation of adventitial nerves, causing postinjection pain and transient muscle cramping. Therefore injection technique, concentration, and volume are particularly important when this agent is being used.

Attempts have been made to reduce postinjection pain from hypertonic saline by the addition of local anesthetics such as lignocaine. However, this practice appears to be counterproductive because the local anesthetics are acidic (and therefore contribute to transient pain on injection) and have known allergenicity, the very properties that proponents of hypertonic saline wish to avoid. The addition of heparin to hypertonic saline in an attempt to prevent thrombosis in larger vessels and to reduce the incidence of thrombophlebitis and postsclerotherapy pigmentation has been found to be of no therapeutic benefit in treating telangiectasias. Concentrations of hypertonic saline used to treat telangiectasias range from 11.7% to 23.4%, the latter being the standard concentration available for use as an abortifacient. The incidence of postinjection pain, muscle cramping, cutaneous ulceration, and postsclerotherapy pigmentation is proportional to the concentration of solution used.

Detergent Solutions

Detergent solutions include sodium morrhuate, ethanolamine oleate, STS, and polidocanol. These agents act specifically on venous endothelium. They induce sclerosis by damaging the endothelium via interference with cell membrane lipids. They exert their effect along the vessel until either diluted or inactivated by serum surfactants. Only STS and polidocanol are widely used for the treatment of telangiectasias. Table 1 gives the approximate equivalent concentrations of STS and polidocanol required for the treatment of lower limb veins. Because of their different mechanisms of action, it is difficult to compare concentrations of detergents solutions with hypertonic saline. However, in the dorsal rabbit ear vein, polidocanol 1% is equivalent in potency to hypertonic saline 23.4%. Other factors apart from vein diameter need to be considered in selecting the concentration of solution.
Patients younger than 25 years of age generally require weaker solutions to achieve effective sclerosis. Also, care should be taken with elderly patients and cigarette smokers because they may have coexisting diminished cutaneous perfusion which increases the risk of post-sclerotherapy cutaneous necrosis. The area to be treated also influences the choice of concentration. Slightly lower concentrations are required on the medial distal thigh and around the ankle; slightly higher concentrations are needed for treating the lateral and posterior thighs and calves.

**Sodium tetradecyl sulphate.** STS is a versatile sclerosant that can be effectively used for the treatment of a wide range of varicose veins and telangiectasias. The essential concept in using STS successfully is to use the appropriate concentration for the given vein diameter. This will result in minimisation of adverse effects. Although STS has a predictable and constant effect within the same caliber and type of vein at the same level or area of the leg, there can be variation in sensitivity among patients. For example, when treating reticular veins and telangiectasias I have observed that cigarette smokers appear more sensitive to solution concentration, possibly as a result of chronic endothelial damage and increased endothelial permeability caused by carbon monoxide and nicotine. The main disadvantage of STS is the pain it causes for several minutes after injection, particularly when reticular and small varicose veins are being treated. However this disadvantage is outweighed by the relatively large total volumes that can be safely injected in a treatment session, especially with the diluted solutions.

STS is commonly used in concentrations of 0.50% to 3% to sclerose lower limb varicose veins. Concentrations of 0.25% to 0.50% are used to treat reticular veins, and concentrations of 0.10% to 0.20% are used for microinjection of telangiectasias. Recently the lower concentrations 0.2%, 0.5% and 1% have become available for use in Australia and New Zealand and are marketed under the brand-name “Fibrovein” (Australasian Medical and Scientific Limited). Other concentrations are achieved by diluting the available strengths of 0.2% 0.5%, 1% and 3% with normal saline. Injection of STS results in immediate vessel spasm, which aids hemostasis after needle withdrawal and can be used as a guide to the effectiveness of a particular injection. The most common serious adverse effect with STS is anaphylaxis which has an incidence of 1 in 700 when using the 3% solution. Typically, the reaction occurs from 10 –30 minutes from commencement of the injections and is manifested by facial flushing, generalised urticaria followed by the patient feeling unwell (a feeling often described as impending disaster), dizziness (hypotension) compensatory tachycardia, shortness of breath and wheeze and finally gastrointestinal symptoms of nausea, vomiting and abdominal pain. Patients with this reaction will generally respond well to immediate injection of adrenaline 0.5mL of 1:1000 aqueous solution subcutaneously or intramuscularly, followed by promethazine HCl 25mg or 50mg intramuscularly. Therefore it is advisable that any physician using STS should be well aware of the early signs of anaphylactoid reaction and have the appropriate emergency equipment immediately at hand, as prompt treatment prevents the patient’s condition becoming critical.

Systemic reactions to STS, including allergy and anaphylaxis, are extremely rare in the treatment of reticular veins and telangiectasias probably due to the lower concentrations used.

STS can cause a burning sensation pain for several minutes after injection, especially when reticular veins are being treated. The pain usually is relieved by applying pressure to the injection sites by using cotton balls held firmly in place with paper tape. Alternatively, an ice pack placed over the treated area for several minutes will reduce the pain significantly. The 0.10% to 0.20% solutions used for microinjection cause minor discomfort and pain is minimal when treating large varicose veins with the appropriate concentration.

Cutaneous ulceration has been commonly reported when STS is used for the treatment of telangiectasias. These high rates of cutaneous ulceration always have occurred when excessive concentrations were used for microinjection. Reiner showed that cutaneous necrosis after intradermal injection of STS in rabbits was concentration-dependent, with 0.313% solution producing...
no necrosis and 1.25% solution producing necrosis. In the microinjection of telangiectasias, STS rarely causes cutaneous necrosis when diluted to 0.1% - 0.15%.

**Polidocanol.** Polidocanol is used in concentrations of 0.25% to 3% to sclerose lower limb varicose veins and telangiectasias. Concentrations of 1% to 3% are used to treat reticular and varicose veins, and concentrations of 0.25% to 0.75% are used for microinjection of telangiectasias. Polidocanol is available in standard concentrations of 0.5%, 1%, 2%, and 3%. Other concentrations are achieved by diluting with normal saline. Experimental evidence in the dorsal rabbit ear vein indicates that polidocanol is a weaker agent than STS in producing effective endosclerosis of telangiectasias.

Compared with STS, polidocanol causes less vessel spasm and more erythema, resulting in increased bleeding after needle withdrawal. Like STS, polidocanol has a predictable effect within the same caliber of vein and area of the leg, but variation in effect between patients occurs. Systemic reactions to polidocanol when used in the treatment of telangiectasias have been reported and occur at a frequency of 0.2 - 0.3%. However, it is essential that the manufacturer’s maximum dose is not exceeded as bradycardia and hypotension will occur due to the intrinsic negative inotropic effect of polidocanol. This may limit the extent of treatment possible in one session when compared with STS.

Because of its inherent local anesthetic properties, polidocanol causes less pain after intravascular injection compared with STS however it tends to be slightly more painful at the moment of injection, especially if there is any extravasation. Cutaneous ulceration is uncommon when concentrations of 0.25% to 1% are used, but it does occur with solutions over 1%

A problem that can occur when using polidocanol is inadvertent sclerosis of normal veins far from the site of injection owing to spread of the solution further from the site of injection than anticipated due to its high solubility.

**Sclerosant Foam**

In the treatment of major varicose veins and incompetent axial trunks, the effectiveness of both STS and POL can be enhanced by converting the standard solution into a foam using a simple 3 way tap and pumping a mixture of sclerosant and air between 2 syringes. By using this method larger veins can be more effectively treated with smaller volumes of sclerosant. This also appears to reduce the incidence of local thrombophlebitis and may reduce the incidence of anaphylaxis with STS.

**Alcohol**

Alcohol has been used for over 100 years in treating varicose veins. It’s quickly diluted and inexpensive. Alcohol is the most potent sclerosant available. It’s typically used as either a 95% or a 100% solution and the mode of action is by chemically injuring the vascular endothelium and denaturing blood protein, which results in an intense thrombosis. Alcohol is typically used in the venous type of malformation. It has also been reported to successfully treat extremity and renal arteriovenous malformations and this has led to investigations of its use in the treatment of AVMs, AV fistulas and lymphatic malformations in the head and neck region as well.
While sclerotherapy is most commonly performed on leg veins, it can be useful in the treatment of prominent veins on the face, hands, back, breasts, shoulders and abdomen. While many of these veins are abnormal (such as spider angiomas and venous malformations), veins such as those on the dorsum of the hands are completely normal and are frequently prominent but considered unsightly.

Patients must be warned that there can be complications from sclerotherapy and this must be discussed fully with the patient prior to undertaking any form of treatment, more particularly if it is done for purely cosmetic reasons.

Facial capillaries can be quite prominent, particularly if they arise following previous skin cancer surgery. Actinic damage and rosacea are also common causes. While fine capillaries are better treated by laser or IPL, the larger ones respond well to sclerotherapy. Spider angiomas on the face of children are best left alone as they tend to disappear spontaneously. Periorbital veins respond well, however there is the theoretical risk of cavernous sinus thrombosis. Reticular veins on the hands respond to 3% L-9 whether used as foam or liquid. Compression garments are available but of questionable necessity. Back, breast, shoulder and abdominal veins are treated with similar strength solutions to leg veins.
Lists for sclerotherapy contraindications can be derived from sources such as sclerosant product information and empirical/theoretical extrapolation. Recommendations on this issue can also be found on physician websites, journal articles, text-books as well as from consensus documents relating to sclerotherapy.

No single list is entirely comprehensive. A typical recommendation is the Australasian College of Phlebology consensus document on sclerotherapy which reasonably separates absolute from relative contraindications. This presentation will address and discuss a typical contraindication list for sclerotherapy.

Absolute contraindications include:
- Previous anaphylaxis to proposed sclerosants
- Acute DVT

Relative contraindications include:
- Deep venous obstruction
- Peripheral vascular disease
- Documented thrombophilia (w increased risk of DVT)
- Inability to mobilize
- Acute SVT
- Pregnancy/Breastfeeding
- OCP/HRT
- Skin disease (systemic disease)
- Uncontrolled asthma
- Uncontrolled migraine
- Poor tolerance of compression

Other listed contraindications from published journals include:
- Obesity
- Deep vein incompetence
- Needle phobia
- Pts on Disulfiram (POL – contains ethyl alcohol)
- Advanced age
- Anticoagulation
- Pelvic tumours causing VV
- Acute infections/fever
- Tamoxifen (increased risk of superficial phlebitis)
- Foramen ovale/visual disturbance (foam)
- Recent long distance travel, lower extremity cast, enforced bed rest (one month)

All of the above listed contraindications are reasonable and acceptable to most patients and physicians. Few would argue against the absolute contraindication of anaphylaxis and acute DVT in sclerotherapy. However, for relative contraindications, this “relativity” is at times tested in clinical practice. The experienced Phlebologists may consider the benefits of treatment to outweigh potential risks associated with the mitigating factor and elect to treat.

Three clinical conundrums involving patients with relative contraindications to treatment are presented to illustrate how treatment decisions are made on a case-by-case basis in daily practice.
Case 1 – “The breast feeding mother”
A 36 yr old flight attendant, 12 weeks post-partum, presents with varicose veins for treatment. She has full length right long saphenous vein reflux (max diameter 5mm) with incompetent tributaries. She is still breast feeding and wants to have her veins treated before going back to work and while she is still able to benefit from the medicare safety net rebate which runs out in a month. What can you offer her?

Discussion
PI listing for both STS and POL state that: “it is not known whether (STS or POL) is excreted into human milk…. Use in lactations is not recommended (STS)…. Caution should be exercised when used in nursing mothers (POL).” Weiss recommends waiting for a period of 4-6hrs after treatment before breast feeding.* Breast pumps can be used pre-sclerotherapy to store breast milk for feeds immediately after sclerotherapy. The T1/2 for POL is 4h and that of STS is unknown. It is generally accepted that treatment with hypertonic saline is safe in nursing mothers.

This patient was scheduled for endovenous laser ablation (‘chemical-free’). Intra-operatively, the procedure was unsuccessful due to extreme venospasm of the target vein (prox GSV). The patient requested foam injections and promised to change to bottle feeds. Treatment was uneventful and she elected to continue breastfeeding the following day.

One other relevant issue is the timing of treatment postpartum. This is generally accepted as 3 months to accommodate spontaneous reversal of varicose veins post partum and regression of pregnancy associated thrombotic risk towards baseline.

Case 2 – “Mr Thrombophilia”
A 35 year old man with anti-cardiolipin antibody syndrome and heterozygous MTHFR re-presents with painful left medial ankle ulcer secondary to recurrent DVTs and post-thrombotic syndrome. He has been struggling with the ulcer for over 2 years and is desperate for intervention. His experienced his first DVT/PE at 26 and has been warfarinised since and is currently on daily clexane. He wears a class 2 stocking daily and has attempted profore on several occasions which helps but does not prevent recurrence of the ulcer. How can you help him?

Discussion
Many sclerotherapy contraindication lists include thrombophilia which is relative indication on the ACP consensus document. The concern is sclerotherapy causing a DVT in an individual already predisposed towards this event. Acute DVT is a clear absolute contraindication. Patients with a past hx of thrombophilia and DVT should best be left alone unless the potential gain from treatment outweighs the risk.

Thrombophilias with low to moderate clotting risks are relatively contra-indicated and should only be managed by experienced Phlebologists. Patients with anti-thrombin 3 deficiency should never be injected as they have a tendency to develop DVTs and, if they develop a clot, heparin is ineffective in the absence of AT3.

This man was treated with a couple of sessions of echosclerotherapy. He did not develop a DVT on serial duplex follow-up scans. Following treatment, the ulcer healed within 3 weeks to the relief and astonishment of the patient. He remains ulcer-free at 9 months follow up.

Although anticoagulation is often cited as a relative contraindication, there are no drug interaction issues per se and the real issue is the underlying primary prothrombotic disease. As such, should anticoagulation in non-thrombotic conditions such as AF be a barrier to sclerotherapy? Certainly extra-precaution is required (increased bleeding tendency) but this is the case with all skin related procedures such as excision of skin cancers where ongoing (intraoperative anticoagulation) is no
longer an issue. Is it time to reconsider our position on blood thinners and large vessel sclerotherapy?

**Case 3 - “Pregnant and willing”**

A 36 year old mother, G8P8, with painful venous ulcers capping off her severe bilateral varicose veins seeks treatment. She also has severe stasis dermatitis, hyperpigmentation, and atrophie blanche/ healed ulcer scars. Whilst waiting for her treatment session, she falls pregnant again. You cancel her treatment appointments and recommend compression and for a review 3 months after her delivery. You are surprised to get a call from her solicitor husband who has researched the matter and tells you that “thousands of women have been treated during pregnancy without harm”. He wants to know the risk and danger to mother and pregnancy by not treating - and specifically points out maternal distress, infection, clots etc – as detrimental factors to the pregnancy. He feels that the benefit of treatment may, in this case, outweigh the risks. What is your response to the husband’s proposal?

**Discussion**

The Phlebology literature supports treating painful vulval varicosities (W263) during pregnancy. It is true that that many thousands of pts have been safely treated with sclerotherapy (STS and POL) with no evidence of detriment to the pregnancy. The pregnancy rating for STS and POL is B2 and B3 respectively (animal studies only- refer to MIMS). Both PIs do not recommend treatment during pregnancy in the absence of well controlled studies.

Another issue is the hypercoagulable state of pregnancy (which persists for up to 3 months post partum) increasing this patient’s risk for DVT.

I elected not to treat this patient with sclerotherapy. She should persevere with compression and consider profore-type compression to accelerate ulcer healing. She should be seen at regular intervals and watched for complications such as clots and thrombophlebitis. Non urgent elective procedures should be deferred till after pregnancy. It is especially important to avoid the first trimester as this is the period of fetal organogenesis.

**Recommended reading**


The experienced Phlebologist should be able to promptly identify sclerotherapy complications and commence appropriate treatment immediately. Most sclerotherapy complications may be classed as minor and readily manageable without significant sequelae. Though a catastrophic event is unlikely, all treatments should be performed in a clinic properly equipped with acute resuscitative equipment. See the college for guidelines on clinic set-up.

Important references:
(Guex, Allaert, Gillet, & Chleir, 2005)
Transient Dermal Pigmentation

- Grade/Severity: minor but may persist for up to 12 months after a treatment session.
- Aetiology/Predisposing Factors:
  - Operator Dependent: more likely if high sclerosant concentrations in superficial and dermal veins.
  - Patient Dependant: more likely if patient has high FP classifications (FPC III to V)
  - Drug Dependant: HT Saline then STS more likely to produce pigmentation than Aethoxysclerol.
  - Unknown/Undiscovered: ???
- Prevention: eliminate predisposing factors and treat proximal disease first.
- Diagnosis:
  - History: gradual onset following treatment session. Peak usually at 4-6 weeks.
  - Physical Examination: non-blanching, non-tender, non-raised brown discolouration.
  - Further Investigations: CW doppler for inadequate treatment of incompetent reticular veins.
  - Exclude: Trapped blood, heamosiderrin staining, tetracycline induced.
- Mechanism:
  - Pathophysiology: inflammatory mediated, increased melanin production by melanocytes. Heamosiderrin and RBC degradation products produced by sclerosed vessels.
  - Anatomy:
    - Histology: Increased melanin granules within dermis and persistent heamosiderrin within the dermis and hypodermis.
- Hydrodynamics:
- Treatment:
  - Acute: NIL
  - Long Term Management: Avoid extreme UV exposure which may cause exacerbation or permanence.
- Prognosis: Good
Permanent Dermal Pigmentation

- Incidence: 1 in 80-120 (Georgiev, 1993; Tafazzoli, Rostan, & Goldman, 2000; Thibault & Wlodarczyk, 1992; Weiss & Weiss, 1990)
- Grade/Severity: moderate
- Aetiology/Predisposing Factors:
  - Operator Dependent:
  - Patient Dependent: Iron Supplementation, Oral Tetracycline
  - Unknown/Undiscovered: ???
- Prevention: Avoid predisposing factors.
- Diagnosis:
  - History: pigmentation >12 months duration.
  - Physical Examination: as with transient dermal pigmentation.
  - Further Investigations: Skin biopsy
- Mechanism:
  - Pathophysiology: inability of to resorb heamosiderrin and iron from dermis.
  - Anatomy
  - Histology: granules in dermis.
  - Hydrodynamics
- Treatment:
  - Acute:
  - Long Term Management: Q switched ruby laser
- Prognosis: Poor
Telangiectatic Matting

- Incidence: 1 in 10-30 (Davis & Duffy, 1990)
- Grade/Severity: mild-moderate
- Aetiology/Predisposing Factors:
  - Operator Dependent: high concentration, high volume, high hydrostatic injection pressure
  - Drug Dependant: related to drug sclerosing power.
  - Patient Dependent: Transparent skin, females and high oestrogen more likely, untreated proximal disease – persistent reflux.
  - Unknown/Undiscovered: ????
- Prevention: avoid predisposing factors.
- Diagnosis:
  - History: patchy pigmentation, onset 4-6 weeks post treatment.
  - Physical Examination: Blanching, visible telangiectatic matting.
  - Further Investigations: CW to identify or exclude underlying incompetent reticular veins.
- Mechanism:
  - Pathophysiology: Chemical induced angioneogenesis.
  - Anatomy
  - Histology: Diffuse thin walled vessels at the with in the superficial most layers of the dermis.
  - Hydrodynamics
- Treatment:
  - Acute: Nil
  - Long Term Management: identify and treat proximal disease, treat remaining telangiectatic matting with sclerotherapy.
- Prognosis: Good if retreated.
Trapped Blood

- Incidence: 1 in 3-5 (Kern, 2002)
- Grade/Severity: mild
- Aetiology/Predisposing Factors:
  - Operator Dependent: ?
  - Drug Dependant: ?
  - Patient Dependent: ?
  - Unknown/Undiscovered: ?
- Prevention: ?
- Diagnosis:
  - History: tender lump, onset 1 to 6 weeks post-sclerotherapy.
  - Physical Examination: tender, non-blanching, brown discoloration.
  - Further Investigations: CW Doppler used to confirm abolition of reflux, evacuation of liquefied thrombus.
- Mechanism:
  - Pathophysiology: liquefied thrombus trapped between two treated ends of vessel. Intraleisonal pressure increases due to increased volume of blood or reduced volume of untreated segment of vessel.
  - Anatomy: image of treated ends.
  - Histology:

- Treatment:
  - Acute: evacuation using 19G needle.
  - Long Term Management: repeat evacuation up to 6 months after treatment.
- Prognosis: Excellent.
Dermal Necrosis/Ulceration

- Grade/Severity: mild to catastrophic.
- Aetiology/Predisposing Factors:
  - Operator Dependent: inadvertent intra-arterial or arteriolar injection (microsclerotherapy). Excessive injection pressure into the superficial veins may cause retrograde flow of sclerosant into the arterial capillary vasculature
  - Drug Dependant:
  - Patient Dependent: more likely in smokers, vasculitis (Henoch-Schonlein, Erythema nodosum, Polyarteritis nodosa, Temporal (giant cell) arteritis, Takayasu's arteritis, Wegener's granulomatosis.)
  - Unknown/Undiscovered: ???
- Prevention: Exclude risk factors such as vasculitis and cease smoking.
- Diagnosis:
  - History: onset of intense pain within 6 to 24 hours after the ischaemic event. Skin is pale and discoloured.
  - Physical Examination: initially dermis is pale with blue-grey dusky appearance. Dermal sloughing occurs within 24 to 72 hours after the ischaemic event. Moderately tender ulcer crater develops quickly.
  - Further Investigations: Check for underlying vasculitis, duplex doppler to check for arterial wall thickness and abnormalities.
- Mechanism:
  - Pathophysiology: Ischaemic necrosis resulting from complete occlusion of arterial component of capillary vascular bed. Unrecognised arterio-venous shunts allow sclerosant to enter the arterial circulation. Compare this to occlusion of non end-artery or end-arteriole. Sclerosant may also enter the arterial capillary vasculature via retrograde flow due to excessive injection pressure.
  - Anatomy: diagram of anastomosis.
  - Histology:
  - Hydrodynamics:
- Treatment:
  - Acute: Supportive, compression, occlusive dressings
  - Long Term Management: dermal scars may need further treatment.
- Prognosis: Very good when extent of necrosis is minimal.
Superficial Thrombophlebitis

- Incidence: 1 in 30 to 90 (Coleridge Smith, 2005; Sadick, 1991)
- Grade/Severity: mild to moderate
- Aetiology/Predisposing Factors:
  - Operator Dependent: high sclerosant concentration, high injection pressure and increased contact time.
  - Drug Dependant: relates to sclerosant power.
  - Patient Dependent:
    - Unknown/Undiscovered: ???
- Prevention: Post-Sclerotherapy compression therapy.
- Diagnosis:
  - History: gradual onset of tender red track approximately 2-6 weeks after sclerotherapy.
  - Physical Examination: tender, indurated, erythematous longitudinal lesion which follows the course of the saphenous trunk.
  - Further Investigations: Duplex doppler examination is essential to establish the diagnosis and to exclude a concomitant or subsequent DVT.
- Mechanism:
  - Pathophysiology: Chemical injury causing an inflammatory reaction within the segment of saphenous vein that has been treated. Intraluminal thrombus and causes complete occlusion of the vessel but may also demonstrate antegrade progression. There is perivascular oedema and this can be readily identified on B-mode ultrasound.
  - Anatomy:
  - Histology: see diagram
  - Hydrodynamics
- Treatment:
  - Acute: compression, ambulation, anti-inflammatory medication
  - Long Term Management:
- Prognosis: Excellent
Deep Vein Thrombosis
- Incidence: 1 in 500-1000 treatments
- Grade/Severity: mild to severe
- Aetiology/Predisposing Factors:
  o Operator Dependent:
  o Procedure Dependent: prolonged procedure time, perforator vein treatment, high injection volumes, treatment of distal leg varices.
  o Drug Dependant: Aethoxysclerol may produce greater effect at distant sites, sinus thrombosis risk with periocular inections.
  o Patient Dependent: Gene mutations, thrombophilia, previous history of DVT, immobilisation, smoking, hyperhomosysteinaemia, long haul flights, dehydration.
  o Unknown/Undiscovered: ???
- Prevention: graded compression, exercise, anticoagulation.
- Diagnosis:
  o History: sudden onset of calf pain and/or ankle swelling, pain when walking.
  o Physical Examination: tenderness to palpation, positive Homan’s sign.
  o Further Investigations: Duplex ultrasound examination is essential to establish the diagnosis and assess progression of the disease.
- Mechanism:
  o Pathophysiology: Virchow’s triad
  o Anatomy: Axial veins and crural veins
  o Histology:
  o Hydrodynamics:
- Treatment:
  o Acute: Assessment, compression, anticoagulation
  o Long Term Management: CVI, CVH, recurrence, prophylaxis
- Prognosis: Good to very poor
Other Dermal Complications
  Hirsutism
  Contusion
Deep Venous Injury
  Deep Venous Thrombosis
Lymphatic Injury
  Lymphocele – Phlebectomy Only
Pulmonary Injury
  Embolism- Air/Thrombotic
  Sclerosis
Arterial Injury
  Ischaemia
Allergic (Anaphylaxis)
CNS
  Migraine (Hanisch, Muller, Krivokuca, & Winterholler, 2004)
  Thrombotic/Embolic (PFO)
  Hypersensitivity
Peripheral Nerve Injury
  Inflammation
  Scar
### Number of Complications according to Sclerosant Form

<table>
<thead>
<tr>
<th>Immediate</th>
<th>Liquid</th>
<th>Foam</th>
<th>Both</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anaphylactic shock</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Intra-arterial injections</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Vasovagal fainting alone</td>
<td>4</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>Headaches alone</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Paresthesias alone</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Nausea and vomiting alone</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Visual disturbances alone</td>
<td>4</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>Visual disturbance associated with 1 or more of headache, nausea,</td>
<td>0</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>vasovagal fainting</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Others</td>
<td>1</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td><strong>Delayed</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deep venous thrombosis</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Muscular venous thrombosis</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Muscular venous extension</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Perforating venous thrombosis</td>
<td>0</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Intense superficial thrombophlebitis</td>
<td>0</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Skin necrosis</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>12</td>
<td>37</td>
<td>0</td>
</tr>
</tbody>
</table>

### Number of Sessions, Sclerosing Agent, and Type of Injected Varicose Vein

<table>
<thead>
<tr>
<th>Type of Varicose Vein</th>
<th>Number of Sessions with Liquid</th>
<th>Number of Sessions with Foam</th>
<th>Number of Sessions with Both</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reticular and spider veins</td>
<td>3,631</td>
<td>2,293</td>
<td>40</td>
</tr>
<tr>
<td>Great saphenous vein trunk or junction</td>
<td>261</td>
<td>1,533</td>
<td>130</td>
</tr>
<tr>
<td>Small saphenous vein trunk or junction</td>
<td>109</td>
<td>492</td>
<td>4</td>
</tr>
<tr>
<td>Main tributaries</td>
<td>422</td>
<td>714</td>
<td>34</td>
</tr>
<tr>
<td>Small varices or nonsaphenous</td>
<td>717</td>
<td>332</td>
<td>37</td>
</tr>
<tr>
<td>Perforating veins</td>
<td>77</td>
<td>199</td>
<td>2</td>
</tr>
<tr>
<td>Postsurgical recurrences</td>
<td>217</td>
<td>832</td>
<td>97</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>5,434</td>
<td>6,395</td>
<td>344</td>
</tr>
</tbody>
</table>

**Australian TGA GUIDELINES**

- Aethoxysklerol maximum dosage: 2mg/kg body weight per day or 5 mls of 3% solutions (equals 3gm per 100ml)
- Sodium Tetradecyl Sulphate maximum dosage: 4 mls of 3% or 10mls of 1%
<table>
<thead>
<tr>
<th>Inflammation in Vascular system</th>
<th>Symptoms</th>
<th>Vasculitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small veins</td>
<td>Purpura - large purple circles on the skin.</td>
<td>Henoch-Schonlein.</td>
</tr>
<tr>
<td>Deep layers</td>
<td>Deep, painful red bumps on the arms and legs.</td>
<td>Erythema nodosum.</td>
</tr>
<tr>
<td>Medium-sized arteries</td>
<td>Kidney failure, heart complications, gastrointestinal problems and high blood pressure.</td>
<td>Polyarteritis nodosa.</td>
</tr>
<tr>
<td>Inflammation of arteries in the brain and head.</td>
<td>Severe headaches, blindness, and stroke</td>
<td>Temporal (giant cell) arteritis.</td>
</tr>
<tr>
<td>Inflammation of the large arteries around the heart.</td>
<td>Fever and night sweats, heart attack.</td>
<td>Takayasu's arteritis.</td>
</tr>
<tr>
<td>Blood vessels of respiratory tract.</td>
<td>Coughing, shortness of breath, nose bleeds, and ear infections</td>
<td>Wegener's granulomatosis.</td>
</tr>
</tbody>
</table>


**Complications and Risks**
If performed properly, sclerotherapy is an efficient treatment method with a low incidence of complications. Nevertheless, a series of adverse events may occur in the context of the therapy. These are, in particular: 14–17

- Allergic reaction; 18–20
- Skin necroses; 21
- Excessive sclerosing reaction (thrombophlebitis);
- Pigmentation; 4, 22–25
- Matting; 24
- Nerve damage; 16, 26
- Scintillating scotomas;
- Orthostatic collapse; and
- Thromboembolism. 15
6 Risk Management

6.1 ACP/NZCAM requires written informed patient consent before UGS.

6.2 The ACP/NZCAM approved sclerosants covered by this standard are
   - Sodium tetradecylsulphate (STS)
   - Polidocanol supplied as either Sclerovein™ or as Aethoxysclerol™

6.3 Duplex/Doppler Ultrasound equipment used in delivering this standard must include a high
   frequency linear array probe with colour flow and Doppler capabilities.

6.4 Under this standard, liquid and foam sclerosant formulations are approved within the maximum
   allowable limits (Refer 6.5, 6.6, and 6.7 below).

6.5 Under the NZ Medicines Act, Foam is an unapproved use of an approved/or unapproved medicine
   (STS is the only approved sclerosant) but permitted when administered by a Registered Medical
   Practitioner provided full written informed consent has been given by the patient.

6.6 This standard restricts the use of STS in concentrations of 3% to 4 mls liquid per day (≈ .12g per
   patient per day).

6.7 Use of polidocanol must not exceed 2mg/kg body weight per treatment day.

6.8 The minimum resuscitation equipment required is:
   - Oxygen, intravenous fluids, adrenalin, blood pressure and cardiac monitor, pulse oximetry,
   - defibrillator, and suction.
REFERENCES


Not available at time of printing (handouts available on-site)
Anaphylaxis/ anaphylactoid reactions

Anaphylaxis is a serious, potentially life-threatening reaction that involves multiple organs and usually rapid in onset. At its most severe, there is bronchospasm, upper airway angioedema and hypotensive shock. Anaphylaxis is an IgE mediated type 1 hypersensitivity reaction that results in mast cell activation and release of multiple mediators such as histamine, leukotrienes, TNF and various other cytokines. Anaphylaxis is the most serious and life-threatening form of systemic allergic reactions.

Anaphylactoid reactions refer to an identical clinical pattern that is however non-IgE mediated. Certain allergens including drugs can trigger the mast cell cascade directly without involving IgE as the initial mediator. Anaphylactoid reactions therefore do not require prior sensitization as they are direct mass cell releasers and may produce anaphylaxis-like reactions in a dose-dependent manner. By contrast, classic anaphylaxis is not dose-dependant as the immune system is primed to recognize even minute amounts of the allergen and able to amplify the reaction via IgE mediation. For practical purposes, we can consider the clinical effects and management of anaphylaxis and anaphylactoid reactions to be identical.

The incidence of anaphylaxis/ anaphylactoid reactions in commonly used sclerosants (STS and POL) ranges from 0.01% to 0.1%. A series of 2686 patients by Thibault revealed an incidence of 0.1% (4 cases) anaphylaxis/ anaphylactoid reaction to 3% STS sclerotherapy. These (non-fatal) reactions occurred within 30 minutes of the injection and included systemic systemic features of urticaria, dizziness (hypotension), wheezing, tachycardia, nausea, vomiting and abdominal pain. An internet based phlebology survey by Varcoe (pre-foam) revealed an allergic reaction rate of 0.03% to 0.3% (mild to severe).

It should be noted that although exceedingly rare, there have been documented deaths from anaphylaxis/ anaphylactoid reactons for both STS/ POL. The German POL network documented 35 cases of allergies from 1987 to 1993 (6yrs) where most were either vasovagal in nature or of unproven allergies. Of these 35 reported cases, 9 patients were given repeat challenges with POL resulting in 3 out of the 9 patients showing true POL allergy. Unfortunately one suffered a fatal anaphylactic reaction despite maximum intervention.

Other allergic reactions

Other (milder) allergic reactions may develop as a result of sclerosant exposure. These are usually confined to the skin as urticaria (type 1 IgE-type hypersensitivity) or other non-specific exanthema. Goldman reported an incidence of 0.3% (47 out of 14000 cases) of “non fatal allergic reactions” which includes generalized urticaria, erythema and other non-specific papulosquamous rash. The Australian polidocanol study involving 8000 patients over 2 years revealed a 0.2% incidence of allergic reaction that specifically noted the absence of anaphylaxis. Urticaria alone does not constitute anaphylaxis and should not be treated as such (with adrenaline) but should be monitored and treated with oral antihistamines if necessary.

Contact reactions can also occur from exposure to sclerotherapy paraphernalias such as adhesive tape, latex gloves, local anaesthetic (for release of trapped blood) and even the silicon component of thigh-high compression stockings. Many of these reactions are may be irritant rather than allergic in nature. Additional investigations such as patch testing or similar challenges may be necessary to confirm allergy. Most skin contact allergies are not immediate but delayed and may take up to 24-48 hours to manifest after sensitisation, and typically presents as an eczematous rash rather than urticaria/ hives characteristic of type 1 immediate type hypersensitivity.
The allergy controversy
Many physicians are skeptical about the purported frequency of allergic reactions in the commonly used sclerosants (STS/ POL). Goldman asserts that by contrast, he has not experienced any serious allergic reactions over 20 years involving over 20,000 patients. Weiss also claims no allergic reactions in over 100,000 injections since changing over to latex-free syringes in 1994. Weiss believes that many of the STS allergic reactions can be attributed to latex leaching from syringes. Allergies may also theoretically arise from impurities in sclerosants such as Carbitol, found in STS sclerosants. Anecdotally, foam sclerosants are associated with fewer allergic reactions. The author believes that many of the STS related allergic reactions are anaphylactoid in nature and dose dependent, hence, fewer reactions are now seen with the typically lower dosages (liquid amounts) of STS used in foam echosclerotherapy.

Clinical features of anapylaxis/ anaphylactoid reactions

<table>
<thead>
<tr>
<th>Mucocutaneous</th>
<th>Respiratory</th>
<th>Cardiovascular</th>
<th>Neurological</th>
<th>Abdominal</th>
</tr>
</thead>
<tbody>
<tr>
<td>● Urticaria</td>
<td>● Wheeze</td>
<td>● Tachycardia</td>
<td>● Vascular headache</td>
<td>● N&amp;V</td>
</tr>
<tr>
<td>● Angioedema</td>
<td>● SOB</td>
<td>● Bradycardia</td>
<td>● Dizziness</td>
<td>● Pain</td>
</tr>
<tr>
<td>● Flushing</td>
<td>● Cough</td>
<td>● ECG changes</td>
<td>● Confusion</td>
<td></td>
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<tr>
<td>● Itch</td>
<td>● Dysphagia</td>
<td>● Hypotension</td>
<td>● Feeling of doom</td>
<td></td>
</tr>
<tr>
<td>● Rhinitis</td>
<td>● Stridor</td>
<td>● Cardiac arrest</td>
<td>● Collapse</td>
<td></td>
</tr>
<tr>
<td>● Conjunctivitis</td>
<td>● Cyanosis</td>
<td></td>
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</tbody>
</table>

Emergency management of allergic reactions

● Urticaria (generalised)
  o Evaluate for wheezing/ stridor
  o Check vital signs
  o Antihistamines +/- corticosteroids (non-dermatologists)
● Anaphylaxis
  o IM adrenaline (lateral thigh) 0.25mg to 0.5mg (0.25ml to 0.5ml 1:1000 adrenaline)
  o IV access
  o Lay patient flat and elevate legs
  o O2 +/- airway ventilation/ support
  o Call ambulance
References:


Not available at time of printing (handouts available on-site)
Post sclerotherapy pigmentation and telangiectatic matting are the commonest adverse events seen after sclerotherapy to dilated lower limb veins. Fortunately these adverse effects are often relatively minor in degree, self-limiting, and generally only of a cosmetic nature. Never the less, good understanding of the causes of these probably, together with sound sclerotherapy technique, will minimise their occurrence and degree of severity.

**Post sclerotherapy Pigmentation**

The reported incidence is from 11% - 80% of patients. Pigmentation is defined as any brown staining of the skin following sclerotherapy with persistent pigmentation being defined as that, which has persisted over 1 year following treatment.

Histological examination has shown this pigmentation to be due to hemosiderin deposition in the dermis. The occurrence of haemosiderin pigmentation occurs as a result of extravasation of erythrocytes into the dermis and subcutaneous tissues during the period of time immediately following injection up until complete sclerosis is achieved. The erythrocytes are phagocytosed by dermal macrophages and the haemoglobin is rapidly broken down into particles identifiable as ferritin. The ferritin particles aggregate within the cytoplasm of the macrophage as haemosiderin and give the typical Prussian blue reaction with Perl’s reagent. However the haemosiderin seen by histologists is heterogeneous and contains forms of $\text{Fe}_2\text{O}_3$ as well as ferritin.

The mechanism of pigmentation explains why in the majority of patients there is gradual spontaneous resolution of the pigmentation with time as the intracellular ferritin is mobilised and utilised for future haemopoiesis and other Fe-protein synthesis.

The incidence of pigmentation is therefore related to multiple factors including:

1. Sclerosing solution type and concentration
2. Sclerotherapy technique
3. Intravascular pressures
4. Total body iron stores
5. Vessel fragility (age)
6. Post-sclerotherapy management (compression and release of post-sclerotherapy coagula)
7. Vessel diameter
8. Concomitant medication

Methods of avoiding and treating post-sclerotherapy pigmentation will be discussed in detail.

**Telangiectatic Matting**

Telangiectatic matting (TM) is the occurrence of previously unnoticed, fine red telangiectasia after treatment. The reported incidence varies from 5% to 75%.

There are multiple reasons for the development of TM:

1. Ischaemic injury may produce a hypoxia induced neovascularization
2. Injury to endothelial cells can release angiogenic stimulating substances
3. Oestrogen hormones can stimulate angiogenesis ($F > M$)
4. Persistent superficial venous pressure (untreated proximal reflux) is a common factor
5. Inadequate post-sclerotherapy compression may be a factor
The avoidance and management of TM will be discussed in detail.
Tues 18 Sept 1745-1800 Dr Andrew Stirling How to Write a Scientific Paper

Not available at time of printing
Tues 18 Sept 1800-1810 Dr Kurosh Parsi Certificate of Ultrasound in Phlebology

Not available at time of printing (handouts available on-site)
BASIC PHLEBOLOGY TRAINING
(Training Manual Excerpts)

Minimum Training Requirements
The following are the minimum requirements that need to be met before the candidate can apply to sit for the Part I Clinical Examination.
Certification in Sclerotherapy will NOT be awarded without evidence of compliance with ALL the following requirements. In exceptional circumstances some of these requirements may be deleted at the discretion of the Board of Censors.

Part I Written Examination.
A Pass in the Written Examination is required before the candidate is invited to sit the Clinical Examination.

Basic Phlebology Certificate Course
The course aims to prepare candidates for the Phlebology Part I Examination. One attendance is the minimum requirement. The Candidates are advised to attend the Basic Phlebology Certificate course before sitting the Written Examination.

Basic Phlebology Preceptorship
For practitioners with no prior experience in Sclerotherapy, the Basic Phlebology Preceptorship is a Basic Phlebology Training requirement. Participation in the Basic Phlebology Preceptorship can be done before or after the Written Examination (preferably before).

Vein School, Clinical Meetings & Journal Club
All candidates must attend at least 6 meetings and must have proof of attendance.

Logbook Requirements

40 cases of CW-Doppler examination
1. Logbooks to be completed.
2. 30 venous incompetence and 10 ABI measurements.
3. Supervisor’s signature required (can be an ACP certified sclerotherapist, phlebologist, vascular surgeon, or vascular physician).

100 direct vision sclerotherapy procedures
1. Logbooks to be completed.
2. 20 cases to be done under direct supervision.
3. Supervisor’s signature required (can be an ACP certified sclerotherapist, or a phlebologist, Fellow Australasian College of Phlebology).
**Phlebology Part I Examination**

**WRITTEN EXAMINATION**
The Written Examination tests the theoretical knowledge of the candidate in basic phlebology with an emphasis on sclerotherapy. The Written Examination is an MCQ paper. This paper consists of 100 multiple choice questions (5 parts each- total of 500 questions) over a 2-hour period in true/false format. There are no negative markings in the multiple choice question paper. This examination will cover a range of topics including venous anatomy, physiology and pathology, Doppler principles, CW-Doppler, duplex examination, telangiectatic conditions, principles of sclerotherapy, sclerosing agents and their mechanism of action, complications of sclerotherapy, thrombophilia, physical principles of compression and compression therapy. It is advisable that the Candidate has attended the Basic Phlebology Certificate course before sitting the Written Examination.

**CLINICAL EXAMINATIONS**
The Clinical Examinations are conducted approximately 2 months after the Written Examination. Only candidates who have successfully passed the Written Examination are invited to sit for the Clinical Examinations.

**OSCEs**
This includes examinations in diagnostic procedures and sclerotherapy techniques. These examinations follow the OSCE (Objective Structured Clinical Examination) format and are supervised by the Chief Censor together with members of the Board of Censors and qualified phlebologists from the particular State in which the examination is being held. Each station is manned by an examiner. A photograph, a model or a device with a written question is presented. The examiner will read the question to the candidate and the candidate is expected to answer the question. As this is an OSCE, no further elaboration or explanation regarding the question will be provided by the examiner. The bell will ring and the candidate will move to the next station. There are no patients in this examination.

**Short Cases**
This part encompasses a number of short cases which will be run along modified OSCE lines with individual stations manned by members of the Board of Censors and qualified phlebologists from the particular State in which the examination is being held. Two examiners will be present per short case station. The examination is done on real patients. Candidates are expected to do a general examination, perform a CW-Doppler examination, interpret any duplex findings and blood tests, be able to formulate the patient’s problem, suggest further investigations and a plan of management. The candidate must demonstrate competence in performing a CW-Doppler examination.
**Phlebology Part I Syllabus**

- Anatomy and physiology of the venous system
- Venous hypertension and its complications
- Pathophysiology of varicose veins and telangiectasias
- Diagnostic techniques
- CW-Doppler competency
- Principles of duplex ultrasound
- Thrombophilia and hypercoagulable states
- Diseases with telangiectatic manifestations
- Indications and contra-indications of sclerotherapy
- Sclerosing agents and their mechanism of action
- Pharmacokinetics and pharmacodynamics of sclerosing agents
- Adverse effects and complications of sclerotherapy
- Techniques of sclerotherapy
- Indications, contraindications, techniques of ultrasound guided sclerotherapy
- Indications, contraindications, techniques of endovenous laser ablation
- Indications, contraindications, techniques of venous surgery
- Principles of compression therapy
- Medicolegal and ethical aspects of sclerotherapy
- Infection control

**Reading List**

**Examinable Texts**

For all books listed, the latest edition is recommended. Any textbook which becomes available after 31 December of the year preceding the examination will not be used for questions. Outdated, erroneous or controversial information in recommended textbooks will be excluded and not examined.


**Journals**

Relevant articles for the two years up to 31 December of the year prior to the examination should be known in detail.

- Australian & New Zealand Journal of Phlebology
- Journal of Dermatologic Surgery

**Other**

Product information sheets for the most commonly used sclerosants.
Advanced Phlebology Training
In the 3-year Advanced Training Period, the minimum requirements for formal training in Australia and New Zealand are:

4 supervised phlebology clinics per week
a. Or equivalent of forty-eight supervised phlebology clinics (96 hours) per 3 months.
b. Clinics to be supervised by a College fellow.
c. The candidate is directly responsible for patient care.

1 diagnostic ultrasound session per week
a. Or equivalent of 12 sessions (24 hours) per 3 months.
b. Scanning to be supervised by a College fellow or vascular sonographer.
c. The trainee to personally perform the scanning.

1 supervised interventional phlebology session per week
a. Or equivalent of 12 sessions (24 hours) per 3 months.
b. The trainee to personally perform ultrasound guided sclerotherapy, endovenous laser therapy and other interventional procedures under supervision.

Vein School, Clinical Meetings & Journal Club
All candidates must attend at least 18 meetings and must have proof of attendance.

40 cases of CW-Doppler examination
a. Logbooks to be completed.
b. 30 venous incompetence and 10 ABI measurements
c. Supervisor’s signature required (can be an ACP certified sclerotherapist, phlebologist, vascular surgeon, or other specialist).

100 cases of duplex ultrasound examination
a. Logbooks to be completed.
b. Both superficial and deep venous systems.
c. Include both upper and lower limbs.
d. Include both incompetence and thrombotic studies.
e. Supervisor’s signature required (can be a phlebologist, vascular surgeon, or other specialist with formal training in venous ultrasound).

100 ultrasound guided sclerotherapy procedures
a. Logbooks to be completed.
b. Liquid and/or foam sclerosant (either assisted or non-assisted).
c. 20 cases to be done under direct supervision.
d. Supervisor’s signature required (must be a phlebologist, Fellow Australasian College of Phlebology, accredited in UGS).

20 endovenous laser ablation procedures
a. Logbooks to be completed.
b. 10 cases to be done under direct supervision

c. Supervisor’s signature required (must be a phlebologist, Fellow Australasian College of Phlebology, accredited in EVLA).

20 management cases of chronic venous disease
a. Logbooks to be completed.
b. Management of patients with CEAP stages C4-C6 is accepted.
c. Supervisor’s signature required (The supervisor can be a phlebologist, or another specialist in a relevant specialty with an active interest in management of leg ulcers).

20 vascular laser therapy procedures
a. Logbooks to be completed.
b. 10 cases to be done under direct supervision.
c. Supervisor’s signature required (can be a phlebologist, dermatologist, or another specialist with formal training in vascular laser therapy).
Observation or assistance in 5 cases of venous surgery
a. Logbooks to be completed.
b. Superficial veins or deep veins.
c. Ambulatory phlebectomy is accepted.
d. Supervisor’s signature required (can be a surgical phlebologist, venous or vascular surgeon).

At least one presentation at the Annual Scientific Meeting of ACP
a. The subject of this presentation can overlap with that of the publication requirement.
b. A poster presentation is acceptable.
c. Must be done during the period of Advanced Phlebology Training.

Two publications in the ANZ Journal of Phlebology
a. These may include research articles or case presentations.
b. As all articles are peer reviewed, it is recommended that 6 months should be allowed for acceptance of publication.
c. Those candidates who do not have their publications (or at least a letter of acceptance from the editor) by the closing date of applications for the Part II examination will not be eligible to sit for the Part II examination.
d. It is a requirement for papers to be accepted as valid for the candidate to be the primary author.
e. Application for admission to the Part II examination will not be accepted without evidence of compliance with publication requirements.

Laser Safety Course
a. At least one attendance required.
b. Copy of Certificate of Attendance to be submitted.
c. Must be done during the period of Advanced Phlebology Training.

Advanced Phlebology Course
a. At least one attendance required.
b. Usually held in conjunction with the annual Scientific Meeting of the College.
c. At least one attendance required.
d. Must be done during the period of Advanced Phlebology Training.

Pass in Level 5 (or higher) Advanced Cardiac Life Support (ACLS)
a. Copy of Certification to be submitted.
b. Must be done during the period of Advanced Phlebology Training.
Phlebology Part II Examinations
a. WRITTEN EXAMINATIONS

1 General Phlebology (MCQ True/False)
This paper consists of 100 multiple choice questions each consisting of 5 parts, over a 2-hour period in true/false format. There are no negative markings in this multiple choice question paper. This exam covers general phlebology.

2 Phlebological Medicine (Short Essays)
This examination consists of two papers each containing 6 questions. The allocated time per paper is 2 hours. Each paper encompasses 6 questions on general phlebology and general medicine pertaining to phlebology. Questions provide clinical scenarios which may require a plan of management.

3 Procedural Phlebology (MCQ Best Answer)
This paper consists of 100 multiple choice questions each consisting of 4 parts, over a 2-hour period in best answer format. There are no negative markings in this multiple choice question paper. This exam covers basic laser physics, laser safety and regulations, ultrasound guided sclerotherapy, endovenous laser therapy, ambulatory phlebectomy, venous surgery, vascular laser therapy and other interventional procedures covered by the curriculum.

4 Diagnostic Ultrasound in Phlebology (MCQ Best Answer)
This paper consists of 120 multiple choice questions each consisting of 4 parts, over a 2-hour period in best answer format. Marks will be deducted for incorrect responses: plus 1 mark for a correct answer, zero marks for no answer, and minus 1 mark for an incorrect answer.

5 Pharmacology in Phlebology (MCQ True/False)
This paper consists of 100 multiple choice questions each consisting of 5 parts, over a 2-hour period in true/false format. There are no negative markings in this multiple choice question paper.

b. CLINICAL EXAMINATIONS
The clinical examinations are conducted 4-8 weeks following the written exam. Only candidates who have successfully passed the written exam are invited to sit for the clinical exams.

1 OSCEs
The first section encompasses examinations in diagnostic procedures, imaging techniques, echosclerotherapy and endovenous ablative techniques. These examinations follow the OSCE (Objective Structured Clinical Examination) format and are supervised by the Chief Censor together with members of the Board of Censors and qualified phlebologists from the particular State in which the examination is being held. A photograph, a model or a device with a written question is presented. As this is an OSCE, no further elaboration or explanation regarding the question will be provided by the examiner. The bell will ring and the candidate will move to the next station. Scanning of patients, a DVT study, a venous incompetence study, upper limb studies and other venous studies may form part of this examination. The candidate must demonstrate competence in performing duplex examinations.

2 Short Cases
This part encompasses a number of short cases which will be run along modified OSCE lines with individual stations manned by members of the Board of Censors and qualified phlebologists from the particular State in which the examination is being held. Candidates are expected to do a general examination, perform a CW-Doppler examination, perform a duplex examination, interpret the duplex findings, be able to formulate the patient’s problem, suggest further investigations and a plan of management.
Reading List

a. Texts
For all books listed the latest edition is recommended. The reading list is offered as a useful source of information and is a basis for study.

Ultrasound Textbooks

• Myers K, Clough A. Making sense of vascular ultrasound: a hands-on guide. Arnold. Relevant VENOUS chapters only. Available from Alliance Distribution Services, 9 Pioneer ave. Tuggerah 2259. Phone: (02) 4390 1300 or (02) 4390 1355.

Thrombosis and Haemostasis


Phlebology

Laser Safety


Pharmacology of Phlebology

• College Notes.
• Product Information Sheets for all drugs.

Fellowship requirements are:

1. Possession of a medical degree and registration as a medical practitioner in Australia or New Zealand.
2. At least two years acceptable training in a teaching hospital (post-graduate year 1 and 2) or equivalent recognized by the ACP.
3. Successful completion of the Basic Phlebology Training program.
4. Successful completion of the Advanced Phlebology Training program.
5. Election to College.
6. Payment of annual subscription fee.
Tues 18 Sept 1810-1820 Dr Kurosh Parsi An Overview of the Examination Process

Not available at time of printing (handouts available on-site)
PART II
Advanced Phlebology & Refresher Course

- BASIC SCIENCES
- VENOUS INSUFFICIENCY
- VENOUS THROMBOEMBOLISM
- WIDER ASPECTS OF PHLEBOLOGY
- MANAGEMENT OF VENOUS INCOMPETENCE
MECHANISM OF ACTION OF SCLEROTHERAPY

GENERAL MECHANISM FOR PRODUCING ENDOTHELIAL DAMAGE

Sclerotherapy refers to the introduction of a foreign substance into the lumen of a vessel causing thrombosis and subsequent fibrosis. This procedure, when performed on telangiectasia, is referred to as microsclerotherapy.

The mechanism of action for sclerosing solutions is that of producing endothelial damage (endosclerosis) that causes endofibrosis. The extent of damage to the blood vessel wall determines the effectiveness of the solution.

Total endothelial destruction results in the exposure of sub-endothelial collagen fibers causing platelet aggregation, adherence, and release of platelet-related factors. This series of events initiates the intrinsic pathway of blood coagulation by activating factor XII. Ideally, sclerosing solutions otherwise should not cause activation or release of thromboplastic activity because this would initiate the extrinsic pathway of blood coagulation.

Excessive thrombosis is detrimental to the production of endofibrosis because it may lead to recanalization of the vessels as well as excessive intravascular and perivascular inflammation and its resulting sequelae. This can be prevented or at least minimized with post-sclerotherapy compression. However, thrombosis usually occurs to some degree as a result of sclerotherapy. If a thrombus is formed, it should be well anchored to the venous wall to prevent embolization. Wolf in 1920 established that effective sclerosis causes thrombosis that penetrated the full thickness of the adventitia of the vessel wall. Schneider has shown in histologic examinations of sclerosed varices that the strongest fixation of a thrombus occurs in areas where the entire endothelium is destroyed. Therefore endothelial damage must be complete and should result in minimal thrombus formation with subsequent organization and fibrosis. In addition, after sclerotherapy, maximum full-thickness fibrosis of the treated segment occurs after 6 weeks of compression. Therefore, in addition to limiting the extent of thrombosis, compression may facilitate endofibrosis.

For sclerotherapy to be effective without recanalization of the thrombotic vessel, the endothelial damage and resulting vascular necrosis must be extensive enough to destroy the entire blood vessel wall.

Destruction of the entire vessel wall and not just the endothelium is necessary. The reason may relate to the multifunctional nature of vascular smooth muscle cells. These cells, which are found in significant concentration within superficial veins, have a large number of functions including the synthesis of collagen, elastin, and proteoglycans. It is hypothesized that if they remain viable, they can regenerate a foundation that promotes migration of undamaged adjacent endothelial cells that allow recanalization of the treated vessel.

In addition, for effective destruction of a varicosity or telangiectasia, the entire vessel must be sclerosed to prevent recanalization. Recanalization occurs easily in vessels where only a section of endothelium is damaged. This is due to rapid endothelial regeneration, which has been measured at a turnover rate of 0.1% to 10% per day or higher.
CATEGORIES OF SCLEROSING SOLUTIONS

All sclerosing solutions can be placed into three broad categories based on their mechanisms for producing endothelial injury: detergent, osmotic, or chemical.

Detergent Solutions
Detergent sclerosing solutions commonly used to treat varicose and telangiectatic veins include sodium morrhuate (SM), ethanolamine oleate (EO), sodium tetradecyl sulfate (STS), and polidocanol (POL). They produce endothelial damage through interference with cell surface lipids. Strong detergents, such as STS and SM, produce maceration of the endothelium within 1 second of exposure. The intercellular “cement” is disrupted, causing desquamation of endothelial cells in plaques. Because the hydrophilic and hydrophobic poles of the detergent molecule orient themselves so that the polar hydrophilic part is within the water and the hydrophobic part is away from the water, they appear as aggregates in solution (micelles) or fixed onto the endothelial surface. Strong detergent sclerosants therefore have a low safety margin.

Detergents act as micelles when injected into a non-detergent environment (blood). Their destructive action on endothelial cells is enhanced when they act as aggregates rather than monomers. Thus the concentration of the sclerosing solution in the vessel is an important factor regarding endothelial destruction and activity. They have been found to aggregate to a significant extent at higher temperatures versus room temperatures.

Effective endosclerosis occurs through damage to endothelium and not through thrombosis induced by destruction or damage to red and/or white blood cells.

Osmotic Solutions

Hypertonic solutions such as hypertonic saline (HS) probably cause dehydration of endothelial cells through osmosis causing endothelial destruction. It is speculated that fibrin deposition with thrombus formation on the damaged vessel wall occurs through modification of the electrostatic charge of the endothelial cells. For the vessel wall to be completely destroyed, the osmotic solution must be of sufficient concentration to diffuse throughout the entire vein wall. In contrast to the immediate action of detergent sclerosing solutions, experimental studies have shown that endothelial destruction with HS 22% or glucose 66% occurs only after 3 minutes. The destroyed endothelial cells do not appear to be desquamated as with detergent sclerosing solutions.

Hypertonic solutions have a predictable destructive power that is proportional to their osmotic concentration. Ranked the solutions from strongest to weakest as the following:

1. Sodium salicylate 40%
2. Sodium chloride 10% + sodium salicylate 30%
3. Invert sugar 75%
4. Saccharose 5%
5. Phenoll%
6. Dextrose 66%
7. Sodium chloride 20%
8. Sodium salicylate 30%

The authors concluded that maximal endothelial destruction occurred as early as 30 minutes to 4 days after injection, after which time the injected vessel went through either a reparative or a fibrotic process.
Because dilution occurs with intravascular serum and blood osmotic solutions, have their greatest effect at or near the site of injection. In contrast, detergent-sclerosing solutions can exert effective sclerosis for 5 to 10 cm along the course of the injected vessel. HS 23.4% is more potent (about two to three times more) than POL 0.5%. Detergent solutions have about twice the therapeutic efficacy of osmotic solutions. A better comparison would have been with HS 11.7%.

Chemical Solutions
Chemical irritants also act directly on endothelial cells to produce endosclerosis. 4% polyiodinated ions. The chemical destruction is in part related to the dissolution of intercellular cement, which has been demonstrated to occur after 30 seconds of exposure.

**Aethoxysklerol**

**Composition**
- **Active.** Laureth-9 (polidocanol).
- **Inactive.** Ethanol, dibasic sodium phosphate dehydrate, monobasic potassium phosphate, water for injections. Aethoxysklerol is buffered to pH 6.5 to 8.0.

Pharmacokinetics. Following the IV administration of 14C-laureth-9 solution into the saphenous vein of six healthy male volunteers, blood was collected over 12 hours and urine and faeces collected over 96 hours. After 12 hours 89% of the dose was eliminated from the blood. A total of 48% of the radioactive dose was recovered in urine (21%) and faeces (27%).

**Contraindications** Known allergy to laureth-9 or any of the excipients; Bed-ridden patients and patients unable to walk;

Arterial disease such as severe artherosclerotic peripheral vascular disease;

Patients with thromboembolic disorders and patients with high risk of thrombosis (those with multiple risk factors such as taking oral contraceptive tablets, adiposis, smoking, longer periods of immobilization).

Other contraindications include acute superficial thrombophlebitis; significant valvular or deep vein incompetence; huge superficial veins with wide open communications to deeper veins; acute cellulitis; allergic conditions; acute infections; varicosities caused by abdominal and pelvic tumours unless the tumour has been removed; uncontrolled systemic disease such as diabetes, toxic hyperthyroidism, tuberculosis, asthma, neoplasm, sepsis, blood dyscrasias and acute respiratory or skin diseases.

Depending on severity, sclerotherapy may be contraindicated in: leg oedema (if it cannot be influenced by compression); Symptoms of diabetic microangiopathy;

Inflammatory skin reactions in the injection area;

Acute severe cardiac diseases (endocarditis, myocarditis). Note that heart failure, if stabilized by previous treatment, is not a contraindication to sclerotherapy. The same applies to arterial hypertension if it has been adequately managed by previous treatment;

Febrile states;

Advanced age with impaired mobility or very poor general condition.

**Carcinogenesis, mutagenesis, impairment of fertility.** The carcinogenic potential of laureth-9
has not been adequately assessed in long-term animal studies. In short-term studies investigating the genotoxic potential of laureth-9, no evidence of mutagenecity was noted; however, a concentration dependent increase in the incidence of chromosomal abnormalities (polyploid cells) was observed in cultured Chinese hamster fibroblasts, suggesting a possible genotoxic effect of the drug.

**Impairment of fertility.** No effect on fertility was observed when male and female rats were treated intermittently with laureth-9 at intravenous doses up to 10 mg/kg (once a week exposure in rats was about 80% of the maximum human dose in terms of surface area prior to mating.

**Use in pregnancy.** There are no adequate and well controlled studies in pregnant women. Therefore, laureth-9 should not be used in pregnant women.

**Use in lactation.** It is not known whether laureth-9 is excreted into human milk. Because many drugs are excreted in human milk, caution should be exercised when Aethoxysklerol is administered to a breastfeeding woman.

**Use in children.** There are no data and therefore Aethoxysklerol is not recommended for use in children.

**Interactions** There are no data on drug interactions with laureth-9.

**Adverse Reactions Pivotal clinical trial.** Aethoxysklerol and sodium tetradecyl sulfate injection caused reactions that were expected based upon the known pharmacological properties of the drugs and/or the mode of application of the drug (needle injection). Immediate local reactions (pain on injection, inflammation swelling and local allergic reactions) and delayed local reactions (hyperpigmentation, vein thrombosis, ecchymoses and neovascularisation) were all related to the injected agent and to the known effects on vascular endothelium. In both groups the most common repeated adverse events were hyperpigmentation, vein thrombosis, ecchymoses and pain on injection. (See Table)

**Aethoxysklerol**

<table>
<thead>
<tr>
<th>All adverse events (independent of the number of treatments per patient)</th>
<th>OHIO substudy</th>
<th>MICA substudy</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>75</td>
<td>75</td>
</tr>
<tr>
<td>Any</td>
<td>89%</td>
<td>88%</td>
</tr>
<tr>
<td>Hyperpigmentation</td>
<td>72%</td>
<td>65%</td>
</tr>
<tr>
<td>Skin necrosis</td>
<td>4.0%</td>
<td>2.7%</td>
</tr>
<tr>
<td>Rash</td>
<td>-</td>
<td>8.8%</td>
</tr>
</tbody>
</table>

| Vein thrombosis* | 59% | 61% | 46% | 42% |
| Neovascularisation | 5.3% | 9.3% | 11% | 7.2% |
| Ecchymosis | 63% | 49% | 70% | 58% |
| Pain | 31% | 45% | 41% | 43% |
| Local allervav | 4.0% | 6.7% | 36% | 23% |
**Inflammation**

<table>
<thead>
<tr>
<th></th>
<th>4.0%</th>
<th>0.0%</th>
<th>59%</th>
<th>41%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Swelling</strong></td>
<td>-</td>
<td>-</td>
<td>40%</td>
<td>19%</td>
</tr>
<tr>
<td><strong>Oedema</strong></td>
<td>-</td>
<td>-</td>
<td>2.2%</td>
<td>0.0%</td>
</tr>
<tr>
<td><strong>Taste disturbance</strong></td>
<td>0.0%</td>
<td>2.7%</td>
<td>1.1%</td>
<td>0.0%</td>
</tr>
<tr>
<td><strong>Visual field defect</strong></td>
<td>-</td>
<td>-</td>
<td>1.1%</td>
<td>0.0%</td>
</tr>
<tr>
<td><strong>Paraesthesia</strong></td>
<td>0.0%</td>
<td>1.3%</td>
<td>2.2%</td>
<td>3.6%</td>
</tr>
<tr>
<td><strong>Dizziness</strong></td>
<td>-</td>
<td>-</td>
<td>2.2%</td>
<td>0.0%</td>
</tr>
</tbody>
</table>

* It should be noted that while vein thrombosis is reported as an adverse event, it is often part of the pharmacological mechanism of action of laureth-9 and is expected with sclerotherapy. STS = sodium tetradecyl sulfate

**Postmarketing.** Spontaneous reporting worldwide of adverse events associated with the use of Aethoxysklerol are as follows.

**Uncommon (> 1,100, < 1/100).** Skin. Pigmentation at injection site: discolouration (hyperpigmentation, less frequently haematomas) and neovascularisation in the sclerosed area.

Vascular. Pain or phlebitis along the injected vein. Vascular. Thrombosis formation, locally at the injected vein. Rare (≥ 1/10,000, < 1/1,000). Skin/vascular. Superficial venous inflammation (periphlebitis, thrombophlebitis) and local tissue death (necrosis), particularly after inadvertent injection into adjacent tissue (perivascular injection); the risk increases in proportion to the Aethoxysklerol concentration.

Skin/body as a whole. Allergic/sensitivity reactions: local allergic and non-allergic skin reactions, very rarely systemic allergic reactions such as anaphylactic shock, angioedema or asthmatisform reactions.

Very rare (≤ 1/10,000). Vascular. Deep vein thrombosis of unknown aetiology, that may have been due to the underlying disease.


Special senses. Locally impaired sensitivity and taste sensations (metallic or furry taste). Body as a whole. Fever and headache.

**Dosage and Administration** Dosage and selection of the Aethoxysklerol concentration depends on the size of the varices to be sclerosed. The maximum dosage 2 mg/kg bodyweight per day must not be exceeded. Two weeks may be necessary, depending on the severity and extent of the varices and on the success of the previous treatments.

**Overdosage** Overdose (caused by injection of an excessive amount of Aethoxysklerol for the vein size being injected) may result in local necrosis, especially if extravasation occurred. No serious sequelae were observed in patients who received Aethoxysklerol doses in excess of the recommended maximum dose of 2 mg/kg bodyweight per day.

**Fibro-Vein**

Composition **Active.** Sodium tetradecyl sulfate **Inactive.** Benzyl alcohol 2%; buffered to pH 7.6.

Actions The action of sodium tetradecyl sulfate in compression sclerotherapy is considered to be that of irritation to the intima of the vein wall, so that on compression of the vein, fibrosis takes
place and the vein is permanently occluded by the development of fibrosis in the wall and across the lumen of the compressed vein.

Contraindications Allergy to sodium tetradecyl sulfate or to any component of the preparation.

Patients unable to walk due to any cause.

Patients currently taking oral contraceptives. Significant obesity.

Acute superficial thrombophlebitis. Local or systemic infection. Varicosities caused by pelvic or abdominal tumours. Uncontrolled systemic disease, e.g. diabetes mellitus. Significant valvular incompetence requiring surgical treatment. Precautions

Allergy and anaphylaxis
A higher incidence of allergic reaction is thought to result from repeated treatment involving sodium tetradecyl sulfate injection and may involve intervals of several years between courses of injection. Where special caution is indicated a test dose of 0.25 to 0.5 mL. FibroVein should be given up to 24 hours before any further therapy.

Special care should be exercised when injecting above and posterior to the medial malleolus where extravascular injection is in danger of being close to the posterior and tibial artery. Pigmentation can result if blood is extravasated at the injection site, particularly when treating the smaller surface veins, and compression is not used.

Extreme caution in use is required in patients with an arterial disease such as severe peripheral atherosclerosis of thromboangiitis obliterans (Buerger’s disease).

Use in pregnancy. Safety for use in pregnancy has not been established. Use only when clearly needed for symptomatic relief and when the potential benefits outweigh the potential hazards to the foetus.

Use in lactation. It is not known whether sodium tetradecyl sulfate is distributed into human milk. Caution should be exercised when used in breastfeeding mothers.

Adverse Reactions Local. Pain or burning. Skin pigmentation. Tissue necrosis and ulceration may occur with extravasation. Paraesthesia and anaesthesia may occur if an injection affects a cutaneous nerve.

Vascular. Superficial thrombophlebitis. Deep vein thrombosis and pulmonary embolism are very rare. Inadvertent intraarterial injection is very rare, but may lead to gangrene. Most cases have involved the posterior tibial artery above the medial malleolus.

Systemic reactions. Allergic reactions are rare, presenting as local or generalized rash, urticaria, nausea or vomiting, asthma, vascular collapse. Anaphylactic shock, which may potentially be fatal, is extremely rare.

ANTICOAGGULANTS

Clexane

Actions. In comparison with natural heparin, Clexane is characterized by a clear increase in the ratio between anti-Xa and anti-ha activities, which is always greater than four. It has several actions on the coagulation pathway through binding to antithrombin III. The antithrombotic activity is related to inhibition of thrombin generation and inhibition of two main coagulation factors: factor Xa and thrombin.

Pharmacokinetics. The pharmacokinetic parameters of Clexane were studied from the changes in plasma anti-Xa activity.

After injection of Clexane by the subcutaneous route, the product is rapidly and completely absorbed. The absolute bioavailability is over 90%.

The maximum plasma activity is observed after three hours and is, on average, 1.6 microgram/mL after the injection of a 40 mg dose.

The elimination of enoxaparin (based on anti-Xa activity levels) is characterized by a half-life of approximately 4.4 hours for a dose of 40 mg. Following a 40 mg dose, anti-Xa activity may persist in the plasma for 24 hours.

Elimination of Clexane at prophylactic dosages is not significantly modified in patients with mild (creatinine clearance 50 to 80 mL/minute) to moderate (creatinine clearance 30 to 50 mL/minute) renal insufficiency. It is slightly reduced in the elderly (t1/2 = six to seven hours). This modification has no effect on the doses or the frequency of injection, as there is no plasma accumulation in elderly subjects. The anti-Xa activity generated by Clexane does not cross the placental barrier during the second trimester of pregnancy.

Anti-Xa activity is generated by Clexane is focalized within the vascular space. Metabolic breakdown of Clexane is slight and takes place mainly in the liver (desulfation and depolymerisation). Small amounts of the product are eliminated by the kidneys in an intact or slightly degraded form.

Renal impairment. A linear relationship between anti-Xa plasma clearance and creatinine clearance at steady state has been observed, which indicates decreased clearance of Clexane in patients with reduced renal function.

Weight. There is a lower weight adjusted clearance (L1hour/kg) in obese subjects.

Elderly. Based on the results of a pharmacokinetic analysis, the Clexane kinetic profile is not different in elderly subjects compared to younger subjects when renal function is normal. However, since renal function is known to decline with age, elderly patients may show reduced elimination of Clexane.

Indications

Prevention of thromboembolic disorders of venous origin in patients undergoing orthopaedic and general surgery.

Prophylaxis of venous thromboembolism in medical patients bedridden due to acute illness.

Prevention of thrombosis in extracorporeal circulation during haemodialysis.

Treatment of established deep vein thrombosis.

Treatment of unstable angina and non-Q-wave myocardial infarction, administered concurrently.
with aspirin.

**Contraindications** Allergy to Clexane, heparin or its derivatives including other low molecular weight heparins. Acute bacterial endocarditis.

Conditions with a high risk of uncontrolled haemorrhage including major bleeding disorders, focal lesions, haemorrhagic stroke, active ulcerative conditions showing a tendency to haemorrhage (e.g. peptic ulcer, ulcerative colitis).

**Precautions**

Clexane is to be used with extreme care in patients with a history of heparin induced (including low molecular weight heparins) thrombocytopenia with or without thrombosis. The risk of heparin induced thrombocytopenia may persist for several years. If history of heparin induced thrombocytopenia is suspected, in vitro platelet aggregation tests have limited predictive value. The decision to use Clexane in such a case must be made only in consultation with an expert in the field.

Not to be administered by the intramuscular route.

Clexane should be used with care in patients with the following conditions: hepatic insufficiency, uncontrolled arterial hypertension, a history of gastrointestinal ulceration, impaired haemostasis, recent ischaemic stroke, diabetic retinopathy, recent neurological or ophthalmological surgery. Pharmacokinetics of enoxaparin are altered in renal impairment.

**Haemorrhage.** As with other anticoagulants, bleeding may occur at any site.

**Monitoring of platelet count.** The risk of antibody mediated heparin induced thrombocytopenia also exists with low molecular weight heparins. Should thrombocytopenia occur, it usually appears between the 5th and the 21st day following the beginning of Clexane treatment. Therefore, it is recommended that the platelet counts be measured before initiation of therapy with Clexane and then regularly thereafter during the treatment. In practice, if a confirmed significant decrease of the platelet count is observed (30 to 50% of the initial value), Clexane treatment must be immediately discontinued and the patient switched to another therapy.

**Low weight.** An increase in exposure of Clexane with prophylactic dosages (non-weight adjusted) has been observed in low weight women (<45 kg) and low weight men (<57 kg), which may lead to a higher risk of bleeding. Therefore, careful clinical monitoring is advised in these patients.

Impaired renal function. In patients with renal impairment, there is an increase in exposure of Clexane which increases the risk of bleeding. Since exposure of Clexane is significantly increased in patients with severe renal impairment (creatinine clearance <30 mL/minute), a dosage adjustment is recommended for therapeutic and prophylactic dosage ranges. Although no dosage adjustment is recommended in patients with moderate (creatinine clearance 30 to 50 mL/minute) and mild (creatinine clearance 50 to 80 mL/minute) renal impairment, careful clinical monitoring is advised (see Dosage and Administration). Pharmacokinetics of enoxaparin are altered in renal impairment. The extent to which a defect in platelet function in severe renal failure might further contribute to bleeding risk is unknown.

**Use in the elderly.** No increased bleeding tendency is observed in the elderly with the
prophylactic dosage ranges. Elderly patients (especially 80 years or older) may be at an increased risk for bleeding complications with the therapeutic dosage ranges. Careful clinical observation is advised. A dosage adjustment may be necessary in elderly patients due to age related impairment of renal function (see Dosage and Administration, Renal impairment).

**Use in pregnancy.** (Category C) Animal toxicity studies have shown that Clexane may have some effect on rat and rabbit reproduction. There is no information available concerning the use of Clexane during the first and third trimesters. As there are no adequate and well controlled studies in pregnant women and because animal studies are not always predictive of human response, this drug should be used during pregnancy only if the doctor has established a clear need.

**Use in lactation.** Studies performed in female rats demonstrated that Clexane has no effect on lactation or milk composition. Effects of Clexane on breastfeeding women have not been studied. As a precaution, women should be advised not to breast feed while using Clexane.

**Interactions.** Clinical trials revealed no adverse effects that could be caused by drug interactions. It is recommended that agents which affect haemostasis should be discontinued prior to Clexane therapy unless strictly indicated. These agents include medications such as anticoagulants, thrombolytics, nonsteroidal anti-inflammatory drugs (NSAIDs) (including ketorolac), preparations containing aspirin (acetylsalicylic acid), systemic salicylates, ticlopidine, dextran 40, clopidogrel, other antiplatelet agents including glycoprotein lib/ilia antagonists or systemic glucocorticoids. If the combination is indicated, Clexane should be used with careful clinical and laboratory monitoring of the haemostatic factors where appropriate.

**Adverse Reactions clinical trial data.** The following information relates to adverse events observed in controlled clinical trials with patients given Clexane prophylactically or for the treatment of deep vein thrombosis (n = 1,170) or with patients given Clexane for the treatment of unstable angina or non-Q-wave myocardial infarction, administered concurrently with aspirin (n = 1,578).

Reported adverse events are presented at the following frequencies. Common: 2>:1/100 (1%) and < 1/10 (10%); uncommon: 2>:1/1,000 (0.1%) and < 1/100 (1%); rare: 2>:1/10,000 (0.01%) and < 1/1,000 (0.1%); very rare: < 1/10,000 (0.01%).

**Haematologidal.** Common. Haemorrhage. Bleeding may occur in the presence of associated risk factors such as organic lesions liable to bleed, invasive procedures or the use of medications affecting haemostasis (see Precautions and Interactions). Major haemorrhage including retroperitoneal and intracranial bleeding has been reported. Some of these cases have been fatal.

**Blood disorders.** Uncommon. Thrombocytopenia. Mild, transient, asymptomatic thrombocytopenia has been reported during the first days of therapy.

**Hepatic.** Uncommon. Asymptomatic and reversible increases in the levels of liver enzymes (e.g. transaminases) have been reported.

**Postmarketing data.** The following information related to events observed following the marketing of Clexane. Voluntary reports of adverse events that have been received since market
introduction (without causal relationship) that are not listed previously are cited below.

**Haematological.** Very rare. There have been rare reports of neuraxial haematomas with the concurrent use of Clexane and spinal/epidural anaesthesia and postoperative indwelling catheters. These events have resulted in varying degrees of neurological injuries including long-term or permanent paralysis (see Precautions).

Rare cases of immunoallergic thrombocytopenia with or without thrombosis have been reported. In some cases, thrombosis was complicated by organ infarction or limb ischaemia. Asymptomatic and reversible increases in platelet count levels have been reported.

**Hypersensitivity and skin.** Injection site. Very rare. Pain, haematoma and mild local irritation may follow the subcutaneous injection of Clexane.

Hard inflammatory nodules, which are not cystic enclosures of Clexane, have been observed at the injection site. They resolve after a few days and should not cause treatment discontinuation.

Cases of skin necrosis at the injection site have been reported with both unfractionated and low molecular weight heparins. These phenomena are usually preceded by purpura or erythematous plaques, infiltrated and painful. Treatment must be discontinued immediately.

**Systemic allergic reactions.** Very rare. Cutaneous (bullous) or systemic allergic reactions (such as pruritus, rash and urticaria), including anaphylactic/anaphylactoid reactions, may occur. In some cases discontinuation of the treatment may be necessary.

Cases of hypersensitivity cutaneous vasculitis have been reported.

**Dosage and Administration** Do not mix Clexane with other injections or infusions.

**Prophylaxis of venous thrombosis.** Prophylaxis against thromboembolism should be tailored according to the patient’s risk. Risk factors include age over 40 years, history of deep vein thrombosis or pulmonary embolism, surgery and other trauma, prolonged immobilization, cardiac disease, obesity, malignancy, varicose veins, hyper-coagulable states, pregnancy and post partum, oral contraceptives, severe infection, inflammatory bowel disease.

High risk patients. In patients with high risk of thromboembolism, a dosage of

Moderate risk patients. In patients with a moderate risk of thromboembolism, the recommended dosage is Clexane 20 mg (0.2 mL; 2,000 IU anti-Xa activity) subcutaneously once daily. In moderate risk patients undergoing surgery, the initial dose should be given approximately two hours preoperatively.

**Prophylaxis of venous thromboembolism in medial patients.** The recommended dose should be 40 mg once daily by subcutaneous injection for a minimum of six days, continuing for a maximum of 14 days or less if the patient returns to full ambulation earlier than 14 days.

Treatment of deep vein thrombosis. The recommended dosage for treatment of established deep vein thrombosis with Clexane is 1.5 mg/kg bodyweight once daily (150 IU anti-Xa activity/kg bodyweight) or 1 mg/kg bodyweight (100 IU antiXa activity/kg bodyweight) twice daily subcutaneously.
Warfarin sodium therapy should be initiated when appropriate (usually within 72 hours of commencing Clexane initiation). Clexane should be continued for a minimum of five days and until a therapeutic oral anticoagulant effect has been achieved.

In the analgesics and anti-rheumatics group, phenylbutazone interacts, however; only a few cases of interactions have been reported with phenilacetic acid derivatives such as fenbufen, indomethacin and sulindac. The arylpropionic acid derivatives ketoprofen and fluriprofen have shown interactions with the coumarins. Piroxicam, isoxicam and paracetamol also interact.

With antimicrobial agents interactions also occur with Rifampicin and erythromycin and there have been isolated reports of interactions with the fluroquinonolones, ciprofloxacin, norfloxacin, ofloxacin, and nalidixic acid. There have been some case reports of interactions with naftcilin, Dicloxacillin, cefamandole, cefazolin, amoxicillin, several Cephalosporins, Griseofulvin, Metronidazole, miconazole, fluconazole, Itraconazole, Ketoconazole, sulfamethoxazole and cotrimoxazole. There have been reports of a possible interaction with roxithromycin. No interactions have been seen with temafloxacin and fleroxacin.

Interactions with the following immunosuppressives and anticancer agents have been reported; tamoxifen, Azathioprine, fluorouracil, aminoglutethimide and a combine of ifosfamide and mesna.

The barbiturates Phenobarbital (phenobarbitone) secobarbital (secobarbitone), heptobarbital (heptabarbitone) butobarbital (butabarbitone) and barbital (barbitone) interact with coumarins. No such interaction has been reported for chloral durate.

The antidepressants mianserin, amitriptyline and nortriptyline have been reported not to interact with Warfarin.

**Coumadin**

**Composition.** Warfarin sodium.

**Actions.** Vitamin K dependent factor anticoagulant.
Pharmacology. Coumadin and other coumarin anticoagulants act by inhibiting the synthesis of vitamin K dependent coagulation factors. The resultant in vivo effect is a sequential depression of factors VII, IX, X and II. The degree of depression is dependent upon the dosage administered. Anticoagulants have no direct effect on an established thrombus, nor do they reverse ischaemic tissue damage. However, once a thrombosis has occurred, anticoagulant treatment aims to prevent further extension of the formed clot and prevents secondary thromboembolic complications, which may result in serious and possibly fatal sequelae.

**Pharmacokinetics.**
Absorption. Coumadin is essentially completely absorbed after oral administration with peak concentration generally attained within the first four hours.

**Distribution.** There are no differences in the apparent volumes of distribution after intravenous and oral administration of single doses of Warfarin solution.

**Metabolism.** The elimination of Warfarin is almost entirely by metabolism. Coumadin is selectively metabolised by hepatic microsomal enzymes (cytochrome P450) to inactive hydroxylated metabolites (predominant route) and by reductases to reduced metabolites (Warfarin alcohols). The Warfarin alcohols have minimal anticoagulant activity. The metabolites
are principally excreted into the urine and, to a lesser extent, into the bile.

**Excretion.** The terminal half-life of Warfarin after a single dose is approximately one week; however, the effective half-life ranges from 20 to 60 hours with a mean of about 40 hours. Studies with radiolabelled drug have demonstrated that up to 92% of the orally administered dose is recovered in urine. Very little Warfarin is excreted unchanged in urine. Urinary excretion is in the form of metabolites.

**Renal dysfunction.** Renal clearance is considered to be a minor determinant of anticoagulant response to Warfarin. No dosage adjustment is necessary for patients with renal failure.

**Hepatic dysfunction.** Hepatic dysfunction can potentiate the response to Warfarin through impaired synthesis of clotting factors and decreased metabolism of Warfarin.

**Intravenous administration.**

**Indications.** Prophylaxis and treatment of venous thrombosis and its extension, and pulmonary embolism. Prophylaxis and/or treatment of the thromboembolic complications associated with atrial fibrillation, coumadin is not indicated in patients with lone atrial fibrillation who are less than 60 years of age with no risk factors, e.g. previous thromboembolism (TIA, ischaemic stroke), diabetes mellitus, hypertension, and an otherwise normal heart.

**Contraindications.** Threatened abortion, eclampsia and pre-eclampsia.

Inadequate laboratory facilities or unsupervised senility, alcoholism, psychosis, or lack of patient cooperation. Spinal puncture and other diagnostic or therapeutic procedures with potential for uncontrollable bleeding.

Major regional, lumbar block anaesthesia. Malignant hypertension.

**Use in pregnancy.** (Category D) Coumadin is contraindicated in women who are or may become pregnant because the drug passes through the placental barrier and may cause fatal haemorrhagic to the fetus *in utero.*

**Interactions.** Coumarins (mainly Warfarin) are known to interact with approximately 250 different drugs. Drugs that interact include the antiarrhythmic agents quinidine, amiodarone, propafenone and moricizine.

Some studies have shown an interaction with diuretics while others have not. Antiplatelet drugs such as aspirin at high dosages can interact whereas dipridamole does not appear to have an effect.

In the lipid lowering agents, cholestyramine and fibrates interacts with coumarins but colestipol and the HMG-CoA reductase inhibitors do not.

Antidiabetic drugs have also been shown to interact with coumarins. Regarding gastrointestinal drugs, the H$_2$-antagonists Cimetidine and ranitidine can interact but the proton pump inhibitors and antacids do not appear to do so.
**Cardiprin 100**

**Composition** Active, Aspirin. Inactive. Glycine (aminoacetic acid) as a solubilising agent; saccharin. Actions Antiplatelet, antithrombotic.

**Pharmacology.** Metabolism of arachidonic acid via the enzyme cyclooxygenase produces mainly thromboxane (TXA₂) in platelets, and prostacyclin (PGI₂) in the vascular endothelium. TXA₂ causes vasoconstriction and induces platelet aggregation; PGI₂ causes vasodilation and has a platelet antiaggregatory effect. Platelet cyclooxygenase is more sensitive to aspirin inhibition and can only be regenerated with the formation of new platelets. Aspirin can therefore have a selective inhibitory effect on thromboxane production and hence on platelet aggregation. In vitro and ex vivo studies have shown that at low doses (100 to 300mg) there is a differential effect between the inhibitory action of aspirin on platelet cyclooxygenase and the cyclooxygenase in the blood vessels. At these doses there is complete inhibition of platelet aggregation induced by collagen, ADP and arachidonic acid. The anti-inflammatory, antipyretic and analgesic actions of aspirin are also though to be mediated via inhibition of prostaglandins biosynthesis.

**Pharmacokinetics.** Absorption. After oral administration, aspirin is generally well absorbed from the gastrointestinal tract, partly from the stomach and mainly from the small intestine. Time to peak plasma levels is plasma levels are within 15 minutes. A small amount of aspirin is absorbed as the metabolite salicylic acid, after hydrolysis in the gastrointestinal mucosa. The rate of drug absorption is enhanced by the formulation of aspirin in a soluble form and can be significantly altered by factors delaying gastric emptying time (e.g. food). Distribution. Salicylates are rapidly distributed through all body tissues and most transcellular fluids including plasma, spinal and synovial fluids, breast milk, saliva and peritoneal fluid. The placental barrier is readily crossed. Only small amounts of salicylate are present in sweat, bile and faeces. In normal patients the average volume of distribution is 150 mUkg.

Protein binding. Salicylate is 80 to 90% bound to plasma protein, especially albimim. Metabolism. Aspirin is converted to salicylic acid (salicylate) in many tissues, but primarily in the gastrointestinal mucosa and the liver. Salicylate is converted mainly in the liver to three main metabolic products: salicylic acid, salicylic phenolic glucuronide and salicylic acyl glucuronide. A small amount of gentisic acid is formed. The metabolism of salicylate normally follows first order kinetics. However, after very large doses, the metabolic pathways become saturated (zero order kinetics) and small dosage increments result in large increases in aspirin levels.

Excretion. Salicylates are excreted predominantly by the kidneys. Most of an administered dose can be recovered in the urine as free salicylate (10%) or metabolites (75% as salicyluric acid). Excretion of free salicylate is extremely variable; 85% of ingested aspirin in alkaline urine, 5% in acidic urine. Patients with impaired renal function require dosage adjustment.

Half-life. The plasma elimination half-life for aspirin is approximately 30 minutes. The half-life of salicylate is therapeutically more important and is dose dependent, increasing as the plasma concentration increases. Allow doses, the elimination half-life is two to three hours and at high dose.
ANAESTHETICS

Xylocaine Plain and Xylocaine with Adrenaline

Composition Xylocaine plain. Active. Lignocaine hydrochloride. Inactive. Sodium Chloride, sodium hydroxide to adjust pH, water for injections.

Xylocaine with adrenaline. Active. Lignocaine Hydrochloride, adrenaline acid tartrate. Inactive. Sodium chloride, sodium hydroxide, water for injections, sodium metabisulfite 0.5 mg/mL as antioxidant.

Actions Injections solutions for the production of local or regional anaesthesia. Lignocaine is classed as a membrane stabilising agent and is a local anaesthetic of the amide type.

Pharmacology. Lignocaine, like other local anaesthetics, causes a reversible blockade of impulse propagation along nerve fibres by preventing the inward movement of sodium ions through the nerve membrane. Local anaesthetics of the amide type are thought to act within the sodium channels of the nerve membrane.

Local anaesthetic drugs may have similar effects on excitable membranes in the brain and myocardium. If excessive amounts of drug reach the systemic circulation rapidly, symptoms and signs of toxicity will appear, emanating mainly from the central nervous system and cardiovascular system.

Central nervous system toxicity usually precedes the cardiovascular effects as it occurs at lower plasma concentrations. Direct effects of local anaesthetics on the heart include slow conduction, negative inotropism and eventually cardiac arrest.

Pharmacokinetics. Lignocaine has a rapid onset and a medium duration of action. The onset of action is one to five minutes following infiltration and 5 to 15 minutes following other types of administration.

The addition of adrenaline considerably slows the absorption of Lignocaine, although the rate also depends on the site of injection. Peak plasma concentrations are reduced by 50% following subcutaneous injection.

Contraindications. Allergy or hypersensitivity to amide type local anaesthetics or sodium metabisulfite in solutions with adrenaline. Detection of suspected hypersensitivity by skin testing is of limited value.

Local anaesthetic techniques must not be used when there is inflammation and/or sepsis in the region of the proposed injection and/or in the presence of septicaemia.

Solutions containing adrenaline. Conditions where the production or exacerbation of tachycardia may prove fatal, e.g. thyrotoxicosis or severe heart disease, or in obstetrics when maternal blood pressure exceeds 130/80 mmHg.

Local analgesia in parts of body with compromised blood supply or supplied by end arteries, e.g. fingers, toes, nose, ears or penis. There is a possibility of producing arterial vasoconstriction and subsequent ischaemic gangrene distal to the site of injection. Intravenous regional techniques.
Known sensitivity to sympathomimetic amines. Use in most patients with cerebral arteriosclerosis.

**Precautions.** Careful and constant monitoring of cardiovascular and respiratory vital signs and the patient’s state of consciousness should be accomplished after each local anaesthetic injection. It should be kept in mind that at such time restlessness, anxiety, tinnitus, dizziness, blurred vision, tremors, depression or drowsiness may be early warning signs of CNS toxicity. Elderly, young or debilitated patients, including those with advanced liver disease or severe renal dysfunction, should be given reduced doses commensurate with their age and physical condition.

Lignocaine should be given with great caution to patients with epilepsy, impaired cardiac conduction, bradycardia, severe shock or digits intoxication. Lignocaine should also be administered with great caution to patients with impaired cardiovascular function.

Solutions with adrenaline should be used with extreme caution in patients with severe or untreated hypertension, arteriosclerotic heart disease, cerebral vascular insufficiency, heart block, advanced diabetes, poorly controlled thyrotoxicosis or any other pathological conditions that might be aggravated by the effects of adrenaline. Adrenaline may induce anginal pain in patients suffering from ischaemic heart disease.

Solutions containing adrenaline should be used with caution in patients with ventricular fibrillation, prefibrillatory rhythm, tachycardia, myocardial infarction; phenothiazine induced circulatory collapse and prostatic hypertrophy.

**Impaired renal function.** Since Lignocaine is metabolised in the liver excreted via the kidneys, the possibility of drug accumulation should be considered in patients with hepatic and/or renal impairment.

**Use in lactation.** Lignocaine passes into breast milk. The amount of Lignocaine appearing in breast milk from a breastfeeding woman receiving parenteral Lignocaine is unlikely to lead to a significant accumulation of parent drug in the breastfed infant.

**Solutions containing adrenaline.** *Drugs acting on the central nervous system.* Solutions containing adrenaline should be used with extreme caution in patients receiving MAOIs or tricylic antidepressants as severe sustained hypertension may result. The effects of adrenaline may be potentiated by tricyclic antidepressants, some antihistamines and thyroid hormones. Phenothiazines and butyrophenones may reduce or reverse the pressor effects of adrenaline, which may lead to a hypotensive response and tachycardia.

**Oxytocic drugs of the ergot type.** Adrenaline containing solutions should not be used in the presence of Oxytocic drugs of the ergot type, as they are known to interact to produce severe, persistent hypertension and its subsequent sequelae.

**Adrenergic neuron blocking agents.** Solutions containing adrenaline neuron blocking agents (e.g. guanethidine, debrisoquine, bethanidine).

**Inhalational anaesthetics.** Serious cardiac arrhythmias and acute pulmonary oedema if hypoxia is present may occur if preparations containing adrenaline are employed in patients during or following the administration of chloroform, halothane, cyclopropane, trichloroethylene or other
halogenated compounds.

Cardiac glycosides. Solutions with adrenaline may interact with cardiac glycosides resulting in cardiac arrhythmias.

Beta-blockers. Noncardioselective P-blockers such as propranolol enhance the pressor effects of adrenaline, which may lead to severe hypertension and bradycardia. Quinidine. Solutions with adrenaline may interact with quinidine, resulting in cardiac arrhythmias.

Hypoglycaemics. Adrenaline induced hyperglycaemia may lead to loss of blood sugar control in diabetic patients treated with Hypoglycaemic agents.

Adverse Reactions.
Central nervous system. CNS manifestations are excitatory and/or depressant and may be characterized by lightheadedness, nervousness, apprehension, euphoria, confusion, dizziness, drowsiness, Tinnitus, hyperacusis, blurred vision, vomiting, sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness, respiratory depression and/or arrest, agitation, difficulty swallowing, paraesthesia circumoral, numbness of the tongue and slurred speech.

The excitatory manifestations maybe very brief or may not occur at all, in which case the first manifestation of toxicity may be drowsiness merging into unconsciousness and respiratory arrest. Drowsiness following administration of Lignocaine is usually an early sign of a high blood level of the drug and may occur as a result of rapid absorption. Monitor unconscious patients for circulatory collapse, as CNS effects may not be apparent as an early manifestation of toxicity, and may in some cases progress to frank convulsions, ultimately leading to respiratory depression and/or arrest. It is crucial to have resuscitative equipment and anticonvulsant drugs available to manage such patients.

Cardiovascular. Cardiovascular manifestations are usually depressant and are characterized by bradycardia, Hypotension and cardiovascular collapse, which may lead to cardiac arrest. Cardiac arrhythmias and hypertension have also been observed.

Methaemoglobinaemia can occur following intravenous administration.

Allergic. Allergic reactions are characterized by cutaneous lesions, urticaria, and oedema or anaphylactoid reactions/shock.

Allergy to amide type local anaesthetics is rare. Sodium metabisulfite (a sulfite), which is not included in solutions with adrenaline, may also cause allergic type reactions including anaphylactic symptoms and life-threatening or less severe asthmatic episodes in certain susceptible people. The overall prevalence of sulfite sensitivity in the general population is unknown and probably low. Sulfite sensitivity is seen more frequently in asthmatic than in non-asthmatic people. If such a reaction occurs, it should be managed by conventional means.

The detection of sensitivity by skin testing is of doubtful value.

Neurological. The incidence of adverse reactions associated with the use of local anaesthetics may be related to the total dose of local anaesthetic administered and are also dependent on the particular drug used, the route of administration and the physical status of the patient. Neurological reactions following regional nerve blocks have included persistent numbness,
paraesthesia and other sensory disturbances. In a prospective review of 10,440 patients who received Lignocaine for spinal anaesthesia, the incidences were reported to be about 3% each for positional headaches, Hypotension and backache; 2% for shivering; and less than 1% each for peripheral nerve symptoms, nausea, respiratory inadequacy and double vision. Many of these observations may be related to local anaesthetic techniques, with or without a contribution from the local anaesthetic.

**Impaired hepatic function.** Although Lignocaine is metabolised by the liver, dosage reduction for local anaesthetic is probably not warranted. However, caution should be exercised with repeated doses.

**Impaired renal function.** Impairment of renal function is unlikely to affect Lignocaine clearance in short term (24 hours). However, toxicity due to accumulation may develop with prolonged or repeated administration.

**Overdosage.** Acute emergencies associated with the use of local anaesthetics are generally related to high plasma levels or to unintended subarachnoid injection of the local anaesthetic solution (see Adverse Reactions and Precautions).

With accidental intravascular injections, the toxic effect will be obvious within one to three minutes. With Overdosage, peak plasma concentrations may not be reached for 20 to 30 minutes depending on the site of injection, and toxic signs will be delayed. Toxic reactions mainly involve the central nervous and cardiovascular systems.

In children, early signs of local anaesthetic toxicity may be difficult to detect in cases where the block is given during general anaesthesia.

**Symptoms.** **Acute toxicity.** CNS toxicity is a graded response with symptoms and signs of escalating severity. The first symptoms are circumoral paraesthesia, numbness of the tongue, lightheadedness, hyperacusis and Tinnitus. Visual disturbance and muscular tremors are more serious and precede the onset of generalized convulsions.

These signs must not be mistaken for neurotic behaviour.

Unconsciousness and grand mal convulsions may follow. These may last from a few seconds to several minutes. Hypoxia and hypercarbia occur rapidly following convulsions due to the increased muscular activity, together with the interference with normal respiration and loss of the airway. In severe cases, apnoea may occur. Acidosis increases the toxic effects of local anaesthetics.

Recovery is due to redistribution of the local anaesthetic drug from the central nervous system and metabolism. Recovery may be rapid unless large amounts of the drug have been injected.

**Cardiovascular toxicity.** Cardiovascular toxicity indicates a more severe situation. Hypotension, bradycardia, decreased cardiac output, heart block, arrhythmia and even ventricular arrhythmias, ventricular fibrillation and cardiac arrest may occur as a result of huge systemic concentrations of local anaesthetics.

Cardiovascular toxic effects are generally preceded by signs of toxicity in the central nervous system, unless the patient is receiving a general anaesthetic or is heavily sedated with drugs such as a benzodiazepine or a barbiturate. In rare cases, cardiac arrest has occurred without
prodromal CNS effects.

**Treatment.** If signs of acute systemic toxicity appear, injection of the local anaesthetic should be stopped immediately. If convulsions occur, immediate attention is required for the maintenance of a patient airway and assisted or controlled ventilation with oxygen via a positive airway pressure delivery systemic mask. Adequacy of the circulation should then be evaluated, bearing in mind that drugs used to treat convulsions depress the circulation when administered intravenously. Should convulsions persist despite adequate respiratory support, and if the status of the circulation permits, appropriate anticonvulsant medication such as an ultra short acting barbiturate (e.g. thiopentone) or a benzodiazepine (e.g. diazepam) may be administered intravenously. The clinician should be familiar with these anticonvulsant drugs prior to use of local anaesthetics. Suxamethonium will stop the muscle convulsions rapidly but will require tracheal intubation and controlled ventilation, and should only be used by those familiar with these procedures. If ventricular fibrillation or cardiac arrest occurs, effective cardiovascular resuscitation treatment must be instituted and maintained for a prolonged period if necessary. Optimal oxygenation and ventilation, and circulatory support as well as treatment of acidosis are of vital importance. Dialysis is of negligible value in the treatment of acute Overdosage with Lignocaine.
Superficial and deep venous systems of lower limbs

The return of the blood to the heart is the primary function of the venous system. This is achieved via a pumping mechanism which relies on a unidirectional valvular system. Valve-containing vessels pass from the deep dermis into the superficial layer of the fat. These vessels range from 70 to 120 microns in diameter. Post-capillary venules join these larger vessels from all directions. Valves are found at most places where the small vessels join the larger ones, but valves are also present within the large vessels not associated with tributaries. Venous valves have two cusps with associated sinuses. The free edges of the valves are always directed away from the smaller vessel and toward the larger ones. In other words, valves utilized in the venous system are ‘one-way’ valves. In normal state, they let the blood get through but not back.

The peripheral venous system is divided into superficial and deep compartments. The superficial and deep venous systems communicate via the ‘perforating’ veins. The perforating veins also contain valves which in normal physiological state allow flow from the superficial to deep system. Thus, the orientation of the valves allows a unidirectional flow of blood from small tributaries into the main trunks, from the superficial venous system into the deep venous system and from the peripheral venous system into the central venous system. Backflow from deep to the superficial veins and backflow from proximal to distal segments of any given truncal vein is prevented by the orientation of these ‘one-way’ valves. The deep and superficial veins of the lower limb occupy two distinct compartments separated by the deep fascia. The deep compartment functions as the ‘ventricle’ of the calf pump. The deep veins can be divided into two groups: inter-muscular and intra-muscular veins. The soleal sinuses and gastrocnemius veins lie within the muscles. The posterior tibial, anterior tibial and peroneal veins lie between the muscles. All the deep veins of the calf join to form the popliteal vein which is the calf pump outflow tract. This outflow tract continues through the thigh pump in the subsartorial canal that protects it from compressive forces generated by the thigh. This outflow tract continues through the abdomen and the thorax where it is subject to intermittent positive and negative pressures associated with respiration.
Basic Venous Physiology

The calf muscle pump generates systolic pressures of 200-300 mmHg. The foot pump also plays an important role in venous return in that it contains a pump powerful enough to propagate a column of blood to the right atrium. It also coordinates the actions of the venous pump of the calf. The large gastrocnemius and soleal veins form the main chamber of the pump but all other deep veins play a role. Similar to flow of blood from the left atrium to the left ventricle during diastole, blood flows from the superficial to deep venous system when the calf muscle pump relaxes via a pressure gradient of 100-110 mmHg. This flow occurs through the perforating veins. It is therefore normal to have a low pressure in the superficial system during exercise and when supine. Superficial vein pressure only rises when standing still and when the calf pump fails. Venous hypertension during exercise is the ultimate cause of almost all venous pathology.

The main physiological functions of the venous system include: (1) Thermoregulation (2) Storage of blood –70% of blood volume is contained within the venous system at any time and (3) Regulation of cardiac output. The primary function of the superficial venous system is thermoregulation. Blood vessels in human skin are under dual vasomotor control, involving separate nervous signals for vasoconstriction and for vasodilatation. In the superficial venous system, vasoconstrictor fibres are the predominant vasomotor innervation. Removal of this vasoconstrictor signal allows vasodilatation which is important in dissipation of heat. Response to cold is also influenced by the superficial venous system. Constriction of arterioles and the superficial veins allows heat conservation by diverting venous blood through the perforators into the deep veins, which lie closer to arteries. In the deep veins, the cool venous blood returning to the core can take up heat from the warm blood in the adjacent deep limb arteries. Thus, some of the heat contained in the arterial blood as it enters the limbs takes a ‘short circuit’ back to the core, and when the arterial blood reaches the skin, it is already cooler than the core. Superficial veins also act as alternative output channels for the foot pump and are called into use when the deep veins are obstructed by muscular contraction, thrombosis or injury.

Valvular failure leads to reversal of flow in the vessel. Turbulent flow below a leaking valve causes a saccule to develop. Best known example is the formation of a saphena varix below the terminal valve of the long saphenous vein. Venous incompetence and calf pump failure lead to venous stasis and venous hypertension. Incompetent veins deal with the consequent hypertension by progressive enlargement and hypertrophy. This leads to the tortuous appearance of ‘varicose’ veins. Blood in incompetent reticular veins can flow back into post-capillary venules causing the appearance of ‘telangiectasias’.

Skin changes of chronic venous disease- haemosiderin pigmentation, lipodermatosclerosis, atrophie blanche, stasis dermatitis, ulceration- are secondary to the phenomenon of venous hypertension. A thorough understanding of these pathological processes is fundamental in planning appropriate management for these patients. This is especially important in management of chronic venous insufficiency as any attempt at the treatment of venous stasis and ulceration must first address the issue of the underlying venous hypertension.
The Superficial Venous System

The superficial venous system can be divided into three interconnecting medial, posterior and lateral systems. The great saphenous vein (GSV) and its tributaries form the medial system, the small saphenous vein (SSV) and its tributaries form the posterior system and the lateral thigh and the lateral calf vein and their tributaries form the lateral system. The proximal GSV and the proximal 2/3 of SSV is under the superficial fascia and hence ‘invisible’. The word ‘saphenous’ itself is derived from the word ‘saphena’ meaning invisible and was first coined by the Persian Physician, Mathematician and Philosopher, Avicenna (Ibn Sina), in the 10th century AD.

The GSV and SSV normally receive blood only from the foot and the low pressure dermal and subcutaneous plexi. They drain directly into the outflow tract via the saphenofemoral (SFJ) and saphenopopliteal junctions (SPJ) respectively. They also have numerous other communications with the deep compartment via the perforating veins.
Anatomy of the great saphenous vein (GSV)

The GSV originates in the medial foot and passes upward anterior to the medial malleolus, then crosses the medial tibia in a posterior direction to ascend in the medial line across the knee. Above the knee it continues anteromedially above the deep fascia to the thigh, where it passes through the foramen ovale and joins the common femoral vein at the groin crease. Large tributaries of the GSV are easily mistaken for the main trunk. Most patients have at least two major tributaries below the knee (the anterior superficial tibial vein and the posterior arch vein) and at least two above the knee (the anterolateral vein of the thigh and the posteromedial vein of the thigh). Up to 20% of patients have a duplicated main GSV trunk in the thigh. The GSV terminates at the SFJ. The SFJ is at the level of the groin crease and is covered by the superficial fascia. That ends proximally at the inguinal ligament. The term ‘confluence of the superficial inguinal veins’, also known as the ‘crosse’, corresponds to the veins of the SFJ. The GSV has a constant terminal valve 1-2 mm distal to the SFJ. A pre-terminal valve is located 2 cm distal to the terminal valve. The most important tributaries join the GSV between the two valves.

Proximal tributaries of GSV drain venous blood from the abdominal wall and pudendal areas and from lateral to medial. The flow in superficial abdominal veins (superficial epigastric vein and superficial circumflex iliac vein) is bidirectional and a ‘downward flow’ into these veins into the SFJ is also normal.

There are many intercommunications between the three superficial systems. The GSV and SSV communicate via a number of ‘inter-saphenous’ veins the most prominent of which is the vein of Giacomini. The normal direction of blood flow is from the small saphenous system to the GSV via these intersaphenous veins.

The GSV communicates with the lateral system as well. The anterior thigh circumflex vein (anterolateral vein of the thigh, ALVT) joins the lateral thigh vein with the GSV at or near the SFJ. The normal flow would be from the lateral thigh vein to the GSV via the ALVT. Please note that the normal direction of flow in the lateral thigh vein is from proximal thigh to the lateral knee perforator and into the deep system. In other words, a ‘downward’ flow in that vein is physiological and normal.

The named tributaries of the GSV in proximal thigh are the anterior accessory great saphenous vein of the thigh (AAGSV), previously called the pre-saphenous arch vein and the posterior accessory great saphenous vein of the thigh (PAGSV), previously called the post-saphenous arch vein. The AAGSV runs parallel to the GSV in the thigh but located anteriorly within a fascial compartment in the thigh in its own fascial compartment. In most cases, there is a constant lymph node in the angle between the GSV and AAGSV before they merge. The venous network of the lymph node that surrounds the AAGSV may be large and incompetent and form a source for reflux. The PAGSV ascends parallel to GSV but located posteriorly within a fascial compartment but is not as often present as AAGSV and its connection to GSV is not constant.

The posterior thigh circumflex vein (posteromedial vein of the thigh), is a tributary of GSV or PAGSV which ascends obliquely in the posterior thigh. It may originate in the SSV, its thigh extension, or the lateral venous system.
The named tributaries of the GSV below the knee are the posterior arch vein (PAV) (new terminology: posterior accessory great saphenous vein of the leg) and the superficial anterior tibial vein (SATV) (new terminology: anterior accessory saphenous vein of the leg). The PAV is important as it contains the important Cockett perforators which communicate between this vein and the posterior tibial veins. The Gastrocnemius perforators are also important as quite often they are incompetent and contribute to lower leg varicosities. These perforators communicate between the PAV or intersaphenous veins and the gastrocnemius veins.

The valves in the GSV vary from ten to twenty in number; they are more numerous in the leg than in the thigh.
Anatomy of the small saphenous vein (SSV)

The SSV originates in the lateral foot as a continuation of the lateral marginal foot vein. It passes posteriorly lateral to the Achilles tendon in the lower calf. The SSV usually lies directly above the deep fascia in the midline as it reaches the upper calf. It travels in an inter-fascial compartment defined by the deep muscular fascia and the superficial fascia. The distal compartment appears on transverse ultrasound scan as an ‘Egyptian eye’.

SSV enters the popliteal space between the two heads of the gastrocnemius muscles. In two thirds of cases, it joins the deep popliteal vein above the knee joint, and in one third of cases, it joins with other veins (most often the GSV or the deep muscular veins of the thigh). The saphenopopliteal junction (SPJ) most often lies 2-4 cm above the popliteal skin crease but its exact location is variable. Gastrocnemius veins may join the popliteal vein, upper SSV, or their confluence at the SPJ. The SSV may merge with the gastrocnemius vein before joining the popliteal vein in 10-30% of the limbs.

The SSV possesses from nine to twelve valves, one of which is always found near its termination in the popliteal vein. In the lower third of the leg the SSV is in close relation with the sural nerve, in the upper two-thirds with the medial sural cutaneous nerve.

The **thigh extension of the SSV (TE)**, courses in groove between the biceps femoris and semimembranosus muscles and is present in 95% of the limbs. It has been called the ‘femoropopliteal vein’ or the ‘cranial extension. The TE is recognized on ultrasound by its intra-fascial position into a triangle shaped compartment and is defined by the semitendinosus muscle medially, the long head of the biceps laterally and the superficial fascia that stretches over the intermuscular groove. This vein does not terminate in GSV and usually terminates in a perforator of the post thigh joining the profunda femoris, or continue straight up into the gluteal area as a single vein or divided into multiple veins, or divide into many muscular branches in the post thigh. If the TE joins the posterior thigh circumflex vein and join the GSV, it is termed **the vein of Giacomini**.

The **sciatic nerve vein** accompanies the sciatic nerve. Symptoms of 'sciatic' pain may be present when this vein is incompetent.

The **small saphenous artery** in its varied forms has been described by M. Schadeck (Schadeck, M. Sclerotherapy of Small Saphenous Vein: How to Avoid Bad Results. Phlebologie 2004, 57, No2, 165-169). This artery can easily be mistaken for a vein and injected during ultrasound guided sclerotherapy. The diagram below demonstrated the frequency of the location of this artery in relation to the SSV. Injection of this artery can lead to skin necrosis.
Relationship of Small Saphenous Artery to SSV
Perforating Veins

Many superficial collecting veins deliver their blood into the GSV and SSV, which deliver most of their blood into the deep system through the saphenofemoral junction (SFJ) and the saphenopopliteal junction (SPJ). However, the SPJ and SFJ are not the only pathways from the superficial system to the deep system. Both the superficial collecting web and the superficial truncal veins are also connected to a variable number of perforating veins that pass through anatomic defects in the deep fascia to join directly with the deep veins of the calf or thigh. Perforating veins usually contain venous valves that prevent reflux of blood from the deep veins into the superficial system. A few named perforating veins are fairly constant in location and are named only as vague groupings.

Every single perforator has an accompanying artery and care should be taken during UGS not to inject these arteries inadvertently.

GSV System Perforators

- **Perforators of the femoral canal** (former Dodd perforators) connect the GSV to the FV. Hunterian perforators are located distal to Dodd and connect the GSV to the FV.
- **Para-tibial perforators** (formerly Sherman and Boyd) connect the main trunk of GSV or its tributaries to the PTV or calf muscle plexus. These perforators lie close to the medial surface of the tibia.
- **Posterior tibial perforators** (formerly Cockett’s) connect the PAV to PTV. The three groups are the ‘upper, middle, and lower.
- **Anterior leg perforators** pierce the ant. tibial compartment fascia to connect the ant GSV tributaries to the ATV.
- **Medial gastrocnemius perforator** connects the PAV to the medial gastrocnemius vein.

SSV System Perforators

- **Soleal (intergemellar) perforator** (former perforator of May) connects the SSV with the soleal veins.
- **Para-Achillean perforators** (former perforator of Bassi) connect the SSV with peroneal veins.
- **Perforator of the popliteal fossa** runs subcutaneously along the posterior aspect of the calf, sometimes parallel to the SSV and typically forms a junction with the popliteal vein lateral to the SPJ.
- **Posterior thigh perforators** include posteromedial thigh perforators that pierce the adductor muscles, sciatic perforators lying along the midline of the posterior thigh, posterolateral thigh perforators piercing the biceps femoris and semitendinosus muscles (former Hach perforators) and pudendal perforators.
Important Lower Limb Perforators

GSV Perforators

Saphenofemoral Junction

Dodd's

Hunterian

Boyd's

Med. Gastrocnemius

Cockett's

Posterior Perforators

Medial Gastrocnemius Perforator

Saphenopopliteal junction

Post. thigh perforators

Lateral gastrocnemius perforator

Perforator of May

Perforator of Bassi
**Lateral System Perforators**

Lateral calf perforators connect veins of the lateral venous plexus with the peroneal veins. **Lateral gastrocnemius perforator** connects the lateral calf vein to the peroneal veins.

Lateral thigh perforators pierce the muscles of the lateral thigh.

**Other Perforators**

Perforators of the knee are designated medial or lateral knee perforators, suprapatellar and infrapatellar perforators.

Perforators of the foot are divided into dorsal, medial, lateral and plantar perforators. Ankle perforators are divided into medial, anterior and lateral.

Perforators of the gluteal muscle are divided in superior, mid, and lower perforators.
Anatomy of foot veins

The dorsal venous arch and the medial and lateral marginal veins are the anatomic origins of the GSV and SSV. These are placed under the superficial fascia. On the dorsum of the foot the dorsal digital veins receive, in the clefts between the toes, the intercapitular veins from the plantar cutaneous venous arch and join to form short common digital veins which unite across the distal ends of the metatarsal bones in a dorsal venous arch. Proximal to this arch is an irregular venous net-work which receives tributaries from the deep veins and is joined at the sides of the foot by a medial and a lateral marginal vein, formed mainly by the union of branches from the superficial parts of the sole of the foot. On the sole of the foot the superficial veins form a plantar cutaneous venous arch which extends across the roots of the toes and opens at the sides of the foot into the medial and lateral marginal veins. Proximal to this arch is a plantar cutaneous venous net-work which is especially dense in the fat beneath the heel; this net-work communicates with the cutaneous venous arch and with the deep veins, but is chiefly drained into the medial and lateral marginal veins.

Lateral venous system

This system is a fairly independent superficial system although it may receive reflux from SFJ via the ALVT. The lateral system includes the lateral thigh and lateral calf veins. It may represent the embryonic lateral marginal vein. Telangiectasias of the lateral upper thigh and lateral lower calf are usually due to an incompetent lateral venous system. The lateral knee, lateral thigh and calf perforators are usually incompetent leading to the incompetence of the lateral system, and hence the telangiectasia.
The Deep Venous System

All venous blood eventually is received by the deep venous system on its way back to the right atrium of the heart. The principal deep venous trunk of the leg is called the popliteal vein (PV) from below the knee until it passes upward and anteriorly through the adductor canal in the distal thigh, where it is called the femoral vein (FV) for the remainder of its course in the thigh. In most cases there are five major named tributaries to the deep venous system, three below (posterior tibial, anterior tibial and peroneal veins) which form the popliteal vein and two above the knee (femoral and profunda femoris veins) which form the common femoral vein.

Deep Veins of the Calf

In the lower leg, three groups of *inter-muscular* deep vein. These veins are all paired.

- The **anterior tibial veins** are the upward continuation of the venæ comitantes of the dorsalis pedis artery. They leave the front of the leg by passing between the tibia and fibula, over the interosseous membrane, and unite with the posterior tibial, to form the *popliteal vein*.
- The **posterior tibial veins** accompany the posterior tibial artery pass upward posteromedially beneath the medial edge of the tibia.
- The **peroneal vein** passes upward posteriorly through the calf.

*Intra-muscular* deep veins include the **gastrocnemius** and the **soleal** groups. Venous sinusoids within the calf muscle coalesce to form soleal and gastrocnemius intramuscular venous plexi, which join the peroneal vein in mid-calf. In most patients, each one of these is actually a pair of veins flanking an artery of the same name; thus there are actually six named deep veins below the knee in a typical patient. Just below the knee, the four anterior and posterior tibial veins join with the two peroneal veins to become the single large popliteal vein.

The **gastrocnemius veins** are situated within the medial and lateral gastrocnemius muscles. Lateral gastrocnemius veins have a much smaller caliber than medial gastrocnemius veins. A dense intramuscular venous plexus unites to form a common extra-muscular trunk which travels for 1 to 4 cm in the loose adipose connective tissue of the popliteal fossa. This common extra-muscular trunk of the gastrocnemius vein terminates:

- directly in the popliteal vein, very obliquely, forming an acute angle, almost vertically above the popliteal vein; this allows the gastrocnemius veins to ensure a shock-absorbing role in the case of sudden pressure variations in the popliteal vein;
• in the saphenopopliteal junction at the level of the concavity of the short saphenous vein: this type of termination makes saphenopopliteal junction ligation flush with the popliteal vein more difficult;

• simultaneously in the popliteal vein and short saphenous vein via a lambda-shaped termination.

In the popliteal fossa, the gastrocnemius veins and small saphenous veins have a different macroscopic appearance. The gastrocnemius veins are bluish and have a fine, poorly muscular, but highly elastic wall. In contrast, the small saphenous vein, situated more superficially, retains the pearly appearance of veins of the subcutaneous plexus. The intramuscular gastrocnemius veins form a dense venous plexus, which branches and anastomoses within the mass of muscle tissue. In the lower part of the calf, one of the largest branches emerges from the medial gastrocnemius muscle, where the muscle mass gives way to membranous tissue, this constitutes the gastrocnemius perforating vein or Gillot's lower pole perforating vein.

Incompetence of a gastrocnemius vein, usually the medial, may cause swelling and discomfort within the calf yet nothing is apparent. Awareness may be precipitated by attempting to wear tight fitting boots or trousers when the difference in calf circumference is recognised yet there is no ankle oedema. Next a venous flare or dilated venules appear over a perforator site, usually the mid-calf perforator, but sometimes the Boyd's perforator, filling the posterior arch tributary of the greater saphenous vein.

Incompetence of a gastrocnemius vein is suggested by the history and clinical examination. Reflux is demonstrated by Doppler ultrasound and accurately localized by duplex ultrasound with colour-flow imaging. The anatomy is clearly visualized by venography. Large gastrocnemius veins are seen in athletes and ballerinas with well-developed calf muscles and such veins are physiological and should not be interrupted. It is imperative that reflux is demonstrated before surgical treatment is offered. Incompetence of gastrocnemius veins contributes to the so called 'restless leg syndrome'.

_The soleus veins_ are situated in a deeper muscle plane. They unite to form one or several main trunks, which terminate in either the posterior tibial vein or the peroneal vein. In contrast with the gastrocnemius veins, their extramuscular portion is very short. However like the gastrocnemius veins, they receive a perforating vein at the lower pole of the muscle, which communicates with the gastrocnemius venous plexus or directly with the short saphenous superficial network.

The general arrangement of the intramuscular plexus of soleus veins is variable. It can be predominantly vertical and terminate via a single common trunk in the posterior tibial or peroneal network, or it can be predominantly horizontal and comprise an anastomosis with the posterior tibial or peroneal network via multiple intramuscular arches at different levels.
Deep Veins of the Thigh

The *popliteal vein* (PV) courses proximally behind the knee and then passes anteromedially in the distal thigh through the adductor canal, at which point it is called the *femoral vein* (FV). The PV and the FV are one and the same, and this is the largest and longest deep vein of the lower extremity. The *profunda femoris vein* (PF) is a short, stubby vein that usually has its origin in terminal muscle tributaries within the deep muscles of the lateral thigh, but may communicate with the popliteal vein in up to 10% of patients. FV usually lies posterolaterally to the femoral artery in the thigh and then moves medially at the groin. In approximately 25% of people the femoral vein is directly posterior to the artery at the groin.

In the proximal thigh, the FV and the PF join together to form the common femoral vein (CFV), which passes upward above the groin crease to become the iliac vein. The FV is sometimes incorrectly referred to as the "superficial femoral vein" despite the fact that it is not a part of the superficial venous system.

The *external iliac vein*, the upward continuation of the femoral vein, begins behind the inguinal ligament, and, passing upward along the brim of the lesser pelvis, ends opposite the sacroiliac articulation, by uniting with the hypogastric vein to form the common iliac vein. On the right side, it lies at first medial to the artery; but, as it passes upward, gradually inclines behind it. On the left side, it lies altogether on the medial side of the artery. It frequently contains one, sometimes two, valves. The external iliac vein receives the inferior epigastric, deep iliac circumflex, and pubic veins.

The *inferior epigastric vein* is formed by the union of the venae comitantes of the inferior epigastric artery, which communicate above with the superior epigastric vein; it joins the external iliac about 1.25 cm. above the inguinal ligament. The *deep iliac circumflex vein* is formed by the union of the venae comitantes of the deep iliac circumflex artery, and joins the external iliac vein about 2 cm above the inguinal ligament. The *pubic vein* communicates with the obturator vein in the obturator foramen, and ascends on the back of the pubis to the external iliac vein. The *hypogastric vein* begins near the upper part of the greater sciatic foramen, passes upward behind and slightly medial to the hypogastric artery and, at the brim of the pelvis, joins with the external iliac to form the common iliac vein.

The *common iliac veins* are formed by the union of the external iliac and hypogastric veins, in front of the sacroiliac articulation; passing obliquely upward toward the right side, they end upon the fifth lumbar vertebra, by uniting with each other at an acute angle to form the inferior vena cava. The right common iliac is shorter than the left, nearly vertical in its direction, and ascends behind and then lateral to its corresponding artery. The left common iliac, longer than the right and more oblique in its course, is at first situated on the medial side of the corresponding artery, and then behind the right common iliac. Each common iliac receives the iliolumbar, and sometimes the lateral sacral veins. The left receives, in addition, the middle sacral vein. No valves are found in these veins.
Deep Inter-muscular Veins of Lower Limbs

- CFV
- SFJ
- GSV
- FV
- POP
- SSV
- PTV
- ATV
- PER
Important nerves of lower limbs

The **saphenous nerve** is the largest cutaneous branch of the femoral nerve. It approaches the femoral artery where this vessel passes beneath the Sartorius, and lies in front of it, behind the aponeurotic covering of the adductor canal, as far as the opening in the lower part of the Adductor magnus. Here it quits the artery, and emerges from behind the lower edge of the aponeurotic covering of the canal; it descends vertically along the medial side of the knee behind the Sartorius, pierces the fascia lata, between the tendons of the Sartorius and Gracilis, and becomes subcutaneous. The nerve then passes along the tibial side of the leg, accompanied by the GSV, descends behind the medial border of the tibia, and, at the lower third of the leg, divides into two branches: one continues its course along the margin of the tibia, and ends at the ankle; the other passes in front of the ankle, and is distributed to the skin on the medial side of the foot, as far as the ball of the great toe, communicating with the medial branch of the superficial peroneal nerve. The saphenous nerve, about the middle of the thigh, gives off a branch which joins the subsartorial plexus. At the medial side of the knee it gives off a large infrapatellar branch, which pierces the Sartorius and fascia lata, and is distributed to the skin in front of the patella. This nerve communicates above the knee with the anterior cutaneous branches of the femoral nerve; below the knee, with other branches of the saphenous; and, on the lateral side of the joint, with branches of the lateral femoral cutaneous nerve, forming a plexiform net-work, the plexus patellæ. The infrapatellar branch is occasionally small, and ends by joining the anterior cutaneous branches of the femoral, which supply its place in front of the knee. Below the knee, the branches of the saphenous nerve are distributed to the skin of the front and medial side of the leg, communicating with the cutaneous branches of the femoral, or with filaments from the obturator nerve.

The **sciatic nerve** lies in the space between the popliteal vein and the SSV. It supplies nearly the whole of the skin of the leg, the muscles of the back of the thigh, and those of the leg and foot. It is the largest nerve in the body, measuring 2 cm. in breadth, and is the continuation of the flattened band of the sacral plexus. It passes out of the pelvis through the greater sciatic foramen, below the Piriformis muscle. It descends between the greater trochanter of the femur and the tuberosity of the ischium, and along the back of the thigh to about its lower third, where it divides into two large branches, the tibial and common peroneal nerves. This division may take place at any point between the sacral plexus and the lower third of the thigh. When it occurs at the plexus, the common peroneal nerve usually pierces the Piriformis.
The **common peroneal nerve** is derived from the dorsal branches of the fourth and fifth lumbar and the first and second sacral nerves. It descends obliquely along the lateral side of the popliteal fossa to the head of the fibula, close to the medial margin of the biceps femoris muscle. It lies between the tendon of the biceps femoris and lateral head of the gastrocnemius muscle, winds around the neck of the fibula, between the Peronæus longus and the bone, and divides beneath the muscle into the superficial and deep peroneal nerves. Previous to its division it gives off articular and lateral sural cutaneous nerves.

In most instances (80%), the **sural nerve** is formed in the distal portion of the leg by the union of the medial sural cutaneous nerve and the peroneal communicating branch. In 20% of cases, the peroneal communicating branch is absent. In such cases, the sural nerve is derived from the medial sural cutaneous nerve alone. The lateral sural cutaneous nerve is laterally situated and usually divides into medial and lateral branches. In a few cases, its medial division may contribute to the sural nerve through the peroneal communicating branch.

The sural nerve passes downward near the lateral margin of the tendo calcaneus, lying close to the SSV, to the interval between the lateral malleolus and the calcaneus. It runs forward below the lateral malleolus, and is continued as the lateral dorsal cutaneous nerve along the lateral side of the foot and little toe, communicating on the dorsum of the foot with the intermediate dorsal cutaneous nerve, a branch of the superficial peroneal. In the leg, its branches communicate with those of the posterior femoral cutaneous. The medial calcaneal branches perforate the laciniate ligament, and supply the skin of the heel and medial side of the sole of the foot.

Though the sural nerve is considered to be a sensory nerve, motor fibres have been found in 4.5% of nerves. In the current case, since the nerve passed through the gastrocnemius muscle, it is likely that it gave motor branches to the muscle as it passed through it. Presence of motor fibres may play important role in sural nerve biopsy and pathological findings. This abnormal course of the sural nerve can produce pain up on the contraction of the gastocnemius or altered sensation over the area of its distribution. Pain associated with sural nerve entrapment in athletes and in scar tissue after the injury of gastrocnemius has already been reported. In the former case, the sural nerve was entrapped in the superficial sural aponeurosis. In the case reported here, the nerve passed through the fleshy part of gastrocnemius.
Venous system of upper limbs, neck and chest

Cephalic and basilic veins are the superficial veins of the arm. On the dorsum of the hand and in front of the wrist superficial venous plexuses are easily seen through the skin. From these the blood passes up the forearm chiefly on its flexor surface by the radial, median and anterior and posterior ulnar veins. Just below the crease of the elbow the median vein communicates with the deep veins and then divides into two branches like the limbs of a Y. Of these the inner is the median basilic and is noticeable as the vein from which patients were usually bled, while the outer is the median cephalic. After a course of an inch or two the median basilic is joined by the anterior and posterior ulnar veins and the median cephalic by the radial. After this junction the median basilic is continued up the inner side of the arm as the basilic vein. Basilic vein is the largest arm vein measuring 6 – 8 mm. Its course is along the medial (ulnar) aspect of the arm. It pierces the deep fascia about the middle of the arm and in the axilla joins the venae comites of the brachial artery to form the axillary vein, which lies on the inner side of its artery.

The median cephalic vein after joining the radial runs up the outer side of the arm as the cephalic vein. Cephalic vein, measuring 4 – 6 mm, runs along the lateral (radial) aspect of the arm emptying into the axillary vein. Although the basilic vein is larger, the cephalic vein is more superficial and easier to dissect out. Therefore it is often the preferred vein for dialysis fistulas or grafts. A little below the clavicle it passes through the costocoracoid membrane to enter the upper part of the axillary vein. Conversely, it may take an acute angle before it enters the axillary vein sometimes making negotiation with a catheter or wire difficult.

Deep forearm veins

These are 2 or 3 veins each that course with and are named like the corresponding arteries of the forearm (radial & ulna). Brachial veins are the deep veins of the upper arm, usually paired and smaller than the superficial veins. They travel in the upper arm parallel to (on either side) the brachial artery and join with the basilic vein to form the axillary vein. Axillary Vein

The axillary vein begins at the junction of the basilic and brachial veins. It runs medial, anterior and caudal to the axillary artery, to the lateral border of the first rib where it becomes the subclavian vein. It lies caudal to the lateral half of the clavicle, slightly medial to and partly overlies the axillary artery. The artery and vein are within the axillary fascia. The brachial plexus (nerves) runs between artery and vein. Valves may be present near the junction of the brachial and basilic veins.
Subclavian Vein

The subclavian vein runs from the lateral border of the first rib to the sternal end of the clavicle where it joins the internal jugular vein and becomes the brachiocephalic vein. It lies posterior and superior to the subclavian artery. A pair of valves is not uncommon at the termination of the subclavian vein. The subclavian vein becomes the axillary vein at the lateral margin of the first rib. The thoracic ducts enter the superior aspect of the subclavian vein near its junction with the internal jugular vein.

Superficial veins of the right upper limb
**Internal jugular vein (IJ)**

The internal jugular vein exits the jugular foramen of the skull base and courses inferiorly along with the carotid artery and vagus nerve. The IJ begins posterior to the carotid artery at the cranium but spirals around the artery and ends up anteriorly at the level of the chest. It lies between the two heads of the sternocleidomastoid muscle in the mid portion and finally lies posterior to the clavicular head of the muscle. It joins with the subclavian vein to become the brachiocephalic vein.

**External jugular (EJ)**

The external jugular vein (EJ) begins approximately at the level of the angle of the mandible (from the junction of the posterior auricular and the retromandibular veins) and courses posterior to the sternocleidomastoid muscle to enter inferiorly into the subclavian vein. Occasionally both the IJ and EJ may have incomplete or valve like structures.

**Intercostal veins**

There are both anterior and posterior intercostal veins. The posterior division enters into the azygous and hemiazygous systems. The anterior division empties into the internal mammary vein. They lie beneath the ribs and serve as a collateral pathway in the presence of central deep venous thrombosis (DVT).
Facial veins

The venous drainage of the face largely parallels the arterial supply. In general, the named arteries are anterior to the veins. The veins are straight whereas the arteries have a more tortuous course. The facial veins lack valves, therefore a two-way flow of blood is possible.

The supratrochlear vein (frontal vein) begins on the forehead in a venous plexus which communicates with the frontal branches of the superficial temporal vein. The veins converge to form a single trunk, which runs downward near the middle line of the forehead parallel with the vein of the opposite side. The two veins are joined, at the root of the nose, by a transverse branch, called the nasal arch, which receives some small veins from the dorsum of the nose. At the root of the nose the veins diverge, and, each at the medial angle of the orbit, joins the supraorbital vein, to form the angular vein. Occasionally the frontal veins join to form a single trunk, which bifurcates at the root of the nose into the two angular veins.

The supraorbital vein begins on the forehead where it communicates with the frontal branch of the superficial temporal vein. It runs downward superficial to the Frontalis muscle, and joins the frontal vein at the medial angle of the orbit to form the angular vein. Previous to its junction with the frontal vein, it sends through the supraorbital notch into the orbit a branch which communicates with the ophthalmic vein; as this vessel passes through the notch, it receives the frontal diploic vein through a foramen at the bottom of the notch.

The angular vein formed by the junction of the frontal and supraorbital veins, runs obliquely downward, on the side of the root of the nose, to the level of the lower margin of the orbit, where it becomes the anterior facial vein. It receives the veins of the ala nasi, and communicates with the superior ophthalmic vein through the nasofrontal vein, thus establishing an important anastomosis between the anterior facial vein and the cavernous sinus. The angular vein anastomoses with the ophthalmic vein. Also it anastomoses with the venous systems of the eyelid and the forehead as well as the infraorbital vein.

The facial vein commences at the side of the root of the nose, and is a direct continuation of the angular vein. It lies behind the facial artery and follows a less tortuous course. As it travels downward along the medial cheek and the lower face, it is in communication with blood from the lateral nasal branches and the labial veins. As with its corresponding artery, it is deep to and thus covered by the superficial facial muscles. Over the lower cheek it is connected to the deep facial vein that parallels the buccal branch of the internal maxillary artery. It anastomoses with the pterygoid plexus located medial to the upper ramus of the manible. The facial vein crosses over the lower border of the manible anterior to the masseter muscle and passes over the submandibular glands (in contrast with the anterior facial artery that passes behind the gland) and enters the internal jugular vein. It communicates with the retromandibular vein that connects with the external jugular vein. The so-called dangerous triangle of the medial face- involving the upper lip and the paranasal area- is a result of the communications of the facial vein with the cavernous
sinus, either directly through the ophthalmic vein or indirectly through the pterygoid plexus.

The facial vein has no valves, and its walls are not so flaccid as most superficial veins. 

Tributaries.—The facial vein receives a branch of considerable size, the deep facial vein, from the pterygoid venous plexus. It is also joined by the superior and inferior palpebral, the superior and inferior labial, the buccinator and the masseteric veins. Below the mandible it receives the submental, palatine, and submaxillary veins, and, generally, the vena comitans of the hypoglossal nerve.

The superficial temporal vein parallels the artery and supplies venous drainage to the scalp and the forehead. It begins on the side and vertex of the skull in a plexus which communicates with the frontal and supraorbital veins, with the corresponding vein of the opposite side, and with the posterior auricular and occipital veins. From this net-work frontal and parietal branches arise, and unite above the zygomatic arch to form the trunk of the vein, which is joined in this situation by the middle temporal vein, from the substance of the Temporalis. It then crosses the posterior root of the zygomatic arch, enters the substance of the parotid gland, and unites with the internal maxillary vein to form the posterior facial vein. It may end within or below the gland as it empties into the internal jugular vein. It also communicates with the anterior jugular vein along with the facial vein. 

Tributaries.—The superficial temporal vein receives in its course some parotid veins, articular veins from the temporomandibular joint, anterior auricular veins from the auricula, and the transverse facial from the side of the face. The middle temporal vein receives the orbital vein, which is formed by some lateral palpebral branches, and passes backward between the layers of the temporal fascia to join the superficial temporal vein.
The posterior auricular vein begins upon the side of the head, in a plexus which communicates with the tributaries of the occipital and superficial temporal veins. It descends behind the auricula, and joins the posterior division of the posterior facial vein to form the external jugular. It receives the stylomastoid vein, and some tributaries from the cranial surface of the auricula.

The occipital vein begins in a plexus at the back part of the vertex of the skull. From the plexus emerges a single vessel, which pierces the cranial attachment of the Trapezius and, dipping into the suboccipital triangle, joins the deep cervical and vertebral veins. Occasionally it follows the course of the occipital artery and ends in the internal jugular; in other instances, it joins the posterior auricular and through it opens into the external jugular. The parietal emissary vein connects it with the superior sagittal sinus; and as it passes across the mastoid portion of the temporal bone, it receives the mastoid emissary vein which connects it with the transverse sinus. The occipital diploic vein sometimes joins it.

Veins of the eyelids

The veins of the eyelids are divided into two major sections: the pretarsal and posttarsal veins. The pretarsal veins are superficial veins that are connected Medially to the angular vein and laterosuperiorly to the superficial temporal and lacrimal veins. The posttarsal division is composed of the deeper veins that connect the orbital veins with the deeper branches of the anterior facial vein and the pterygoid plexus. These veins also anastomose medially and laterally with the veins of the pretarsal division.

The major venous drainage of the eyelids is to the superficial temporal and the angular and facial veins. The angular vein is on the superficial surface of the medial canthal tendon and approximately 8mm medial to the medial canthus. It connects with the frontal-supraorbital system superiorly and the facial vein inferiorly. As in the arterial system, there are venous arcades that drain into these major venous drainage systems.

Sclerotherapy of temple and orbital veins

From time to time, patients request removal of veins around the orbit and on the temples. These veins seem to become more prominent especially after a face lift procedure. This procedure seems to restrict the drainage of these veins in some patients, hence making the veins more prominent after surgery. Ambulator phlebectomy can be attempted, however some of these veins may be too small to be hooked out. Care should be taken during sclerotherapy as the drainage of the angular vein is to the cavernous sinus via the superior opthalmic vein and hence there is a theoretical risk of cavernous sinus thrombosis and blindness.
Dorsal nasal vein
Superior ophthalmic vein
Angular vein
Supratrochlear vein (vein of the forehead)
Supraorbital vein
Zygomatico-orbital vein
Temporal tributary of Zygomatico-orbital vein
Peripheral arcade of upper eyelid
Supraorbital vein
Supratrochlear vein
Superior ophthalmic vein
Dorsal nasal vein
Angular vein
Facial vein
Peripheral arcade of lower eyelid
Margina arcade of lower eyelid
Zygomatico-orbital vein
Lateral palpebral vein
Anterior tributary of superficial temporal vein

Temporal tributary of Zygomatico-orbital vein

Angular vein

Peripheral arcade of lower eyelid

Zygomatico-orbital vein

Parietal tributary of superficial temporal vein

Superficial temporal vein

Supraorbital vein

Anchial vein

Lacrimal gland

Facial vein

Cavernous sinus

Inferior ophthalmic vein

Superior ophthalmic vein
**Intra-abdominal veins**

The common femoral vein, after passing deep to Poupart's ligament, becomes the **external iliac** which runs along the brim of the true pelvis and, after a course of some three inches, joins the **internal iliac** which drains the pelvis and so forms the **common iliac vein**. In front of the body of the fifth lumbar vertebra the common iliac veins of the two sides unite to form the **inferior vena cava**.

**Inferior Vena Cava (IVC)**

The IVC is formed from the junction of the common iliac veins at approximately the L5 level. It courses upwards towards the heart anteriorly and to the right of the spine. It lies to the right of the aorta and is oval in shape. It runs up to an opening in the diaphragm. On its way it receives spermatic or ovarian veins from the genital glands, **renal veins** from the kidneys, and **lumbar veins** from the abdominal walls. Before reaching the diaphragm it lies in a groove in the back of the liver and receives the **hepatic veins** from that organ. The hepatic portal system which lies in the abdomen will be treated later.

The renal veins enter the IVC at approximately the lower third of L1. The left renal vein enters slightly higher than the right. Both can be duplicated. IVC duplication .2%, Left renal vein duplication 11 - 17%. The **renal veins** are of large size, and placed in front of the renal arteries. The left is longer than the right, and passes in front of the aorta, just below the origin of the superior mesenteric artery. It receives the left spermatic and left inferior phrenic veins, and, generally, the left suprarenal vein.

**Hepatic and portal veins**

These consist of a right, middle and left. All three may join and enter the IVC as a common trunk but most commonly the right enters by itself and in 80% the middle and left hepatic veins have a common trunk. The middle hepatic vein lies in the interlobar fissure separating the R and L lobes of the liver. Caudate veins (usually 2-3) drain separately into the IVC, several cm’s below the main hepatic veins.

The veins which drain the blood from the stomach, intestines, spleen and pancreas unite to form a large vein which begins behind the head of the pancreas and ends by dividing into right and left branches in the transverse fissure of the liver. This is the **portal vein** which lies in front of the inferior vena cava and is about three inches long. Its formative tributaries are the **superior** and **inferior mesenteric** and the **splenic veins**. These accompany the arteries of the same name, and their most usual method of termination is that the inferior mesenteric runs up and joins the splenic to the left of the middle line of the body, and this, after running horizontally to a point a little to the right of the middle line, joins the superior mesenteric, and so the portal vein is formed.
There are two marked characteristics of the portal system; one is that it has no valves and the other that it begins and ends in capillaries, since the two terminal branches of the portal vein branch and rebranch. In the lower part of the rectum the veins run partly into the portal and partly into the general system, and in this dependent position they are liable to become varicose and to form haemorrhoids. The histology of the veins corresponds very closely to that of the arteries (q.v.); their walls are, however, much thinner and there is less muscular and elastic tissue. At certain places, especially where tributaries come in, the endothelial lining is raised to form semilunar pocket-like valves. In most cases there are two cusps to each valve, but three or one are sometimes found. The opening of the pocket is of course arranged so that it shall only be filled when there is a tendency to regurgitation of the blood.
Veins of the thorax and pulmonary veins

Veins of the Thorax

The IVC, after piercing the diaphragm, has a very short thoracic course and opens into the lower and back part of the right auricle of the heart. The right and left innominate veins are formed behind the sternal end of the clavicle by the union of the subclavian and internal jugulars of their own side. The left vein is much longer than the right and runs nearly horizontally behind the upper half of the manubrium sterni to join its fellow on the right side of that bone just below the first rib. By the junction of these the superior vena cava is formed, which runs down to the right auricle of the heart. The chief tributaries of the innominate veins are the vertebral, the internal mammary and the inferior thyroid. The intercostal veins open into the azygos veins, which begin in the abdomen sometimes by a vertical trunk joining the lumbar veins known as the ascending lumbar, sometimes on the right side by a communication with the inferior vena cava. The right azygos vein is known as the vena azygos major and passes through the aortic opening of the diaphragm. Entering the thorax, it rubs up in front of the thoracic vertebrae, to the right of the aorta and thoracic duct, and receives the intercostal veins of the right side. At the level of the fourth thoracic vertebra it arches forward to open into the posterior surface of the superior vena cava.

On the left side, the upper intercostal veins join to form the left superior intercostal vein, which opens into the left innominate. Lower down the intercostal veins from the fourth to the seventh spaces form the superior hemiazygos vein or hemiazygos accessoria, which runs down on the left of the spinal column and, crossing it about the level of the eighth or ninth thoracic vertebra, opens into the vena azygos major. The lower intercostal veins on the left side join the inferior hemiazygos vein which runs up and opens either into the superior hemiazygos or into the azygos major below the opening of that vein.

Pulmonary veins

The veins emerging from the lungs bring back the oxygenated blood from those organs to the left ventricle of the heart and also the greater part, if not all, of the blood carried by the bronchial arteries to nourish the lungs. The existence of bronchial veins is asserted, but they are extremely difficult to demonstrate, and if present are quite incapable of returning all the blood which the bronchial arteries carry to the lungs. There are three pulmonary veins coming out of the right lung, while on the left there are only two. On the right side, however, two of the three veins usually unite in the root of the lung, so that there are, as a rule two pulmonary veins entering the left auricle of the heart on each side, but it is not uncommon to find three on the right side or one on the left. The pulmonary veins have no valves and return the blood carried to the lungs by the pulmonary arteries as well as most, if not all, of that carried by the bronchial arteries.
Pelvic veins

The **ovarian veins** correspond with the spermatic in the male; they form a plexus in the broad ligament near the ovary and uterine tube, and communicate with the uterine plexus. They end in the same way as the spermatic veins in the male. Valves are occasionally found in these veins. These veins ascend in the retroperitoneum adjacent to the psoas muscle in pairs, which combine to form a single vein prior to termination. The right ovarian vein terminates in the inferior vena cava at an acute angle. The left ovarian vein terminates in the left renal vein at a right angle. Occasionally, valves are present in the ovarian veins. The veins enlarge greatly during pregnancy to accommodate increased blood volume. Following childbirth, a period of venous stasis occurs. Incompetence of these veins contributes to **pelvic congestion syndrome**.

Other Important Veins

With the exception of the fetal umbilical vein which passes upward and backward from the umbilicus to the liver, and the iliolumbar vein which usually joins the common iliac vein, the tributaries of the hypogastric vein correspond with the branches of the hypogastric artery. It receives (a) the gluteal, internal pudendal, and obturator veins, which have their origins outside the pelvis; (b) the lateral sacral veins, which lie in front of the sacrum; and (c) the middle hemorrhoidal, vesical, uterine, and vaginal veins, which originate in venous plexuses connected with the pelvic viscera.

1. The **superior gluteal veins** are venæ comitantes of the superior gluteal artery; they receive tributaries from the buttock corresponding with the branches of the artery, and enter the pelvis through the greater sciatic foramen, above the Piriformis, and frequently unite before ending in the hypogastric vein.

2. The **inferior gluteal veins** (sciatic veins), or venæ comitantes of the inferior gluteal artery, begin on the upper part of the back of the thigh, where they anastomose with the medial femoral circumflex and first perforating veins. They enter the pelvis through the lower part of the greater sciatic foramen and join to form a single stem which opens into the lower part of the hypogastric vein.

3. The **internal pudendal veins** are the venæ comitantes of the internal pudendal artery. They begin in the deep veins of the penis which issue from the corpus cavernosum penis, accompany the internal pudendal artery, and unite to form a single vessel, which ends in the hypogastric vein. They receive the veins from the urethral bulb, and the perineal and inferior hemorrhoidal veins. The deep dorsal vein of the penis communicates with the internal pudendal veins, but ends mainly in the pudendal plexus.
4. The **obturator vein** begins in the upper portion of the adductor region of the thigh and enters the pelvis through the upper part of the obturator foramen. It runs backward and upward on the lateral wall of the pelvis below the obturator artery, and then passes between the ureter and the hypogastric artery, to end in the hypogastric vein.

5. The **lateral sacral veins** accompany the lateral sacral arteries on the anterior surface of the sacrum and end in the hypogastric vein.

6. The **middle hemorrhoidal vein** takes origin in the hemorrhoidal plexus and receives tributaries from the bladder, prostate, and seminal vesicle; it runs lateralward on the pelvic surface of the Levator ani to end in the hypogastric vein. The hemorrhoidal plexus (plexus haemorrhoidalis) surrounds the rectum, and communicates in front with the vesical plexus in the male, and the uterovaginal plexus in the female. It consists of two parts, an internal in the submucosa, and an external outside the muscular coat. The internal plexus presents a series of dilated pouches which are arranged in a circle around the tube, immediately above the anal orifice, and are connected by transverse branches.

   The lower part of the external plexus is drained by the inferior hemorrhoidal veins into the internal pudendal vein; the middle part by the middle hemorrhoidal vein which joins the hypogastric vein; and the upper part by the superior hemorrhoidal vein which forms the commencement of the inferior mesenteric vein, a tributary of the portal vein. A free communication between the portal and systemic venous systems is established through the hemorrhoidal plexus. The veins of the hemorrhoidal plexus are contained in very loose, connective tissue, so that they get less support from surrounding structures than most other veins, and are less capable of resisting increased blood-pressure.

   The **pudendal plexus** lies behind the arcuate public ligament and the lower part of the symphysis pubis, and in front of the bladder and prostate. Its chief tributary is the deep dorsal vein of the penis, but it also receives branches from the front of the bladder and prostate. It communicates with the vesical plexus and with the internal pudendal vein and drains into the vesical and hypogastric veins. The prostatic veins form a well-marked prostatic plexus which lies partly in the fascial sheath of the prostate and partly between the sheath and the prostatic capsule. It communicates with the pudendal and vesical plexuses.

   The **vesical plexus** envelops the lower part of the bladder and the base of the prostate and communicates with the pudendal and prostatic plexuses. It is drained, by means of several vesical veins, into the hypogastric veins.

   The **dorsal veins of the penis** are two in number, a superficial and a deep. The superficial vein drains the prepuce and skin of the penis, and, running backward in the subcutaneous tissue, inclines to the right or left, and opens into the corresponding superficial external pudendal vein, a tributary of the GSV. The deep vein lies beneath the deep fascia of the penis; it receives the blood from the glans penis and corpora cavernosa penis and courses backward in the middle line between the dorsal arteries;
near the root of the penis it passes between the two parts of the suspensory ligament and then through an aperture between the arcuate pubic ligament and the transverse ligament of the pelvis, and divides into two branches, which enter the pudendal plexus. The deep vein also communicates below the symphysis pubis with the internal pudendal vein.

The uterine plexuses lie along the sides and superior angles of the uterus between the two layers of the broad ligament, and communicate with the ovarian and vaginal plexuses. They are drained by a pair of uterine veins on either side: these arise from the lower part of the plexuses, opposite the external orifice of the uterus, and open into the corresponding hypogastric vein.

The vaginal plexuses are placed at the sides of the vagina; they communicate with the uterine, vesical, and hemorrhoidal plexuses, and are drained by the vaginal veins, one on either side, into the hypogastric veins.

The spermatic veins emerge from the back of the testis, and receive tributaries from the epididymis; they unite and form a convoluted plexus, called the pampiniform plexus, which constitutes the greater mass of the spermatic cord; the vessels composing this plexus are very numerous, and ascend along the cord, in front of the ductus deferens. Below the subcutaneous inguinal ring they unite to form three or four veins, which pass along the inguinal canal, and, entering the abdomen through the abdominal inguinal ring, coalesce to form two veins, which ascend on the Psoas major, behind the peritoneum, lying one on either side of the internal spermatic artery. These unite to form a single vein, which opens on the right side into the inferior vena cava, at an acute angle; on the left side into the left renal vein, at a right angle. The spermatic veins are provided with valves. The left spermatic vein passes behind the iliac colon, and is thus exposed to pressure from the contents of that part of the bowel.
<table>
<thead>
<tr>
<th>Vein</th>
<th>Tributaries</th>
<th>Drains Into</th>
<th>Regions Drained</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>deep dorsal v. of the clitoris</td>
<td>many unnamed tributaries</td>
<td>vesical venous plexus</td>
<td>erectile tissue of the clitoris</td>
<td>deep dorsal v. of the clitoris passes anterosuperior to the urogenital diaphragm (between the arcuate pubic ligament and the transverse ligament of the perineum) to enter the pelvic cavity; an unpaired vein</td>
</tr>
<tr>
<td>deep dorsal v. of the penis</td>
<td>many unnamed tributaries</td>
<td>prostatic venous plexus</td>
<td>erectile tissue of the penis</td>
<td>deep dorsal v. of the penis passes anterosuperior to the urogenital diaphragm (between the arcuate pubic ligament and the transverse ligament of the perineum) to enter the pelvic cavity; an unpaired vein</td>
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<tr>
<td>deep external pudendal v.</td>
<td>part of the drainage of the superficial dorsal v. of the penis/clitoris</td>
<td>femoral</td>
<td>skin and superficial fascia of the penis/clitoris; pubic region</td>
<td>deep external pudendal v. shares its region of drainage with the superficial external pudendal v.</td>
</tr>
<tr>
<td>internal pudendal v.</td>
<td>deep dorsal v. of the penis/clitoris, v. of the bulb, posterior labial/scrotal v., inferior rectal v.</td>
<td>internal iliac</td>
<td>crus and bulb of the clitoris/penis; urogenital region, anal region</td>
<td>internal pudendal v. passes through the pudendal canal</td>
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<td>of clitoris, deep dorsal</td>
<td>many unnamed tributaries</td>
<td>vesical venous plexus</td>
<td>erectile tissue of the clitoris</td>
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</tr>
<tr>
<td>of clitoris, superficial dorsal</td>
<td>no named tributaries</td>
<td>superficial external pudendal v.</td>
<td>skin and superficial fascia of the clitoris</td>
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</tr>
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<td>of penis, deep dorsal</td>
<td>many unnamed tributaries</td>
<td>prostatic venous plexus</td>
<td>erectile tissue of the penis</td>
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<td>of penis, superficial dorsal</td>
<td>no named tributaries</td>
<td>superficial external pudendal v.</td>
<td>skin and superficial fascia of the penis</td>
<td>superficial dorsal v. of the penis is located superficial to the deep fascia of the penis</td>
</tr>
<tr>
<td>ovarian v.</td>
<td>no named tributaries</td>
<td>right: inferior vena cava; left: left renal v.</td>
<td>ovary and the distal part of the uterine tube; ureter</td>
<td>connects with the uterine v.; a pampiniform plexus occurs, but is not as well developed as that seen in the male</td>
</tr>
<tr>
<td>pampiniform venous plexus</td>
<td>no named tributaries</td>
<td>becomes the testicular vein at the deep inguinal ring</td>
<td>testis, epididymis, ductus deferens</td>
<td>pampiniform venous plexus surrounds the testicular a. to cool arterial blood before it reaches the testis</td>
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</tr>
<tr>
<td>plexus, pampiniform venous</td>
<td>no named tributaries</td>
<td>becomes the testicular vein at the deep inguinal ring</td>
<td>testis, epididymis, ductus deferens</td>
<td>pampiniform venous plexus surrounds the testicular a. to cool arterial blood before it reaches the testis</td>
</tr>
<tr>
<td>plexus, prostatic venous</td>
<td>deep dorsal v. of the penis</td>
<td>internal iliac v.</td>
<td>penis and the prostate gland</td>
<td>prostatic venous plexus is connected with the vesical venous plexus</td>
</tr>
<tr>
<td>plexus, rectal venous</td>
<td>no named tributaries</td>
<td>superior, middle &amp; inferior rectal vv.</td>
<td>rectum and anal canal; anus</td>
<td>rectal venous plexus is a site of portal-caval anastomosis</td>
</tr>
<tr>
<td>plexus, uterine venous</td>
<td>multiple tributaries from the uterus; deep dorsal v. of the clitoris</td>
<td>uterine vv. to the internal iliac v.</td>
<td>uterus &amp; uterine tubes</td>
<td>connects with the ovarian v. and the vaginal venous plexus</td>
</tr>
<tr>
<td>plexus, vaginal venous</td>
<td>multiple tributaries from the vagina</td>
<td>vaginal v. to the internal iliac v. or uterine v.</td>
<td>vagina</td>
<td>connects with the uterine venous plexus, the vesical venous plexus and the rectal venous plexus</td>
</tr>
<tr>
<td>plexus, vertebral venous, external</td>
<td>intervertebral vv.</td>
<td>adjacent segmental vv.; vertebral v. in the cervical region</td>
<td>vertebral column and associated muscles</td>
<td>two plexuses are described: anterior and posterior; connects with the internal vertebral venous plexus</td>
</tr>
<tr>
<td>Vein</td>
<td>Tributaries</td>
<td>Drains Into</td>
<td>Regions Drained</td>
<td>Notes</td>
</tr>
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<td>-----------------------------------------</td>
<td>-----------------------------------------------------------------------------</td>
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</tr>
<tr>
<td>plexus, vertebral venous, internal</td>
<td>anterior and posterior longitudinal vertebral sinuses</td>
<td>adjacent segmental vv.</td>
<td>spinal cord, meninges, vertebral column</td>
<td>connects with the external vertebral venous plexuses; valveless; a route for potential spread of metastases from the pelvis to the brain</td>
</tr>
<tr>
<td>plexus, vesical venous</td>
<td>multiple tributaries from the bladder in both sexes</td>
<td>superior and inferior vesical vv. to the internal iliac v.</td>
<td>urinary bladder</td>
<td>in the male - connects with the prostatic venous plexus and the rectal venous plexus; in the female - connects with the rectal venous plexus, uterine venous plexus and vaginal venous plexus</td>
</tr>
<tr>
<td>portal v.</td>
<td>formed by the union of the superior mesenteric v. and the splenic v.; tributaries: posterior superior pancreaticoduodenal v., right gastric v., left gastric v.</td>
<td>divides into right and left branches before entering the liver; into the liver sinusoids</td>
<td>all of the gut and its glands</td>
<td>portal v. connects with the vena caval drainage at 1) esophagus, 2) rectum, 3) umbilicus, 4) retroperitoneal gut structures; portal v. courses between two capillary beds (gut and liver)</td>
</tr>
<tr>
<td>Vein</td>
<td>Tributaries</td>
<td>Drains Into</td>
<td>Regions Drained</td>
<td>Notes</td>
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<tr>
<td>prostatic venous plexus</td>
<td>deep dorsal v. of the penis</td>
<td>internal iliac v.</td>
<td>penis and the prostate gland</td>
<td>prostatic venous plexus is connected with the vesical venous plexus</td>
</tr>
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<td>pudendal, deep external</td>
<td>part of the drainage of the superficial dorsal v. of the penis/clitoris</td>
<td>femoral</td>
<td>skin and superficial fascia of the penis/clitoris; pubic region</td>
<td>deep external pudendal v. shares its region of drainage with the superficial external pudendal v.</td>
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<td>pudendal, internal</td>
<td>deep dorsal v. of the penis/clitoris, v. of the bulb, posterior labial/scrotal v., inferior rectal v.</td>
<td>internal iliac</td>
<td>crus and bulb of the clitoris/penis; urogenital region, anal region</td>
<td>internal pudendal v. passes through the pudendal canal</td>
</tr>
<tr>
<td>pudendal, superficial external</td>
<td>part of the drainage of the superficial dorsal v. of the penis/clitoris</td>
<td>GSV</td>
<td>skin and superficial fascia of the penis/clitoris; pubic region</td>
<td>superficial external pudendal v. shares its region of drainage with the deep external pudendal v.</td>
</tr>
</tbody>
</table>

GSV: greater saphenous vein
<table>
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<tr>
<th>Vein</th>
<th>Tributaries</th>
<th>Drains Into</th>
<th>Regions Drained</th>
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</tr>
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<tbody>
<tr>
<td>rectal venous plexus</td>
<td>no named tributaries</td>
<td>superior, middle &amp; inferior rectal vv.</td>
<td>rectum and anal canal; anus</td>
<td>rectal venous plexus is a site of portal-caval anastomosis</td>
</tr>
<tr>
<td>superficial dorsal v. of the clitoris</td>
<td>no named tributaries</td>
<td>superficial external pudendal v.</td>
<td>skin and superficial fascia of the clitoris</td>
<td>superficial dorsal v. of the clitoris is located superficial to the deep fascia of the clitoris</td>
</tr>
<tr>
<td>superficial dorsal v. of the penis</td>
<td>no named tributaries</td>
<td>superficial external pudendal v.</td>
<td>skin and superficial fascia of the penis</td>
<td>superficial dorsal v. of the penis is located superficial to the deep fascia of the penis</td>
</tr>
<tr>
<td>superficial external pudendal v.</td>
<td>part of the drainage of the superficial dorsal v. of the penis/clitoris</td>
<td>GSV</td>
<td>skin and superficial fascia of the penis/clitoris; pubic region</td>
<td>superficial external pudendal v. shares its region of drainage with the deep external pudendal v.</td>
</tr>
<tr>
<td>testicular v.</td>
<td>pampiniform plexus</td>
<td>left: left renal v.; right: inferior vena cava</td>
<td>testis, ureter</td>
<td>left testicular v. is longer than the right testicular v.</td>
</tr>
<tr>
<td>uterine venous plexus</td>
<td>multiple tributaries from the uterus; deep dorsal v. of the clitoris</td>
<td>uterine vv. to the internal iliac v.</td>
<td>uterus &amp; uterine tubes</td>
<td>connects with the ovarian v. and the vaginal venous plexus</td>
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<tr>
<td>vaginal venous plexus</td>
<td>multiple tributaries from the vagina</td>
<td>vaginal v. to the internal iliac v. or uterine v.</td>
<td>vagina</td>
<td>connects with the uterine venous plexus, the vesical venous plexus and the rectal venous plexus</td>
</tr>
<tr>
<td>vena cava, inferior</td>
<td>formed by the union of the paired common iliac vv; tributaries: lumbar vv. 1-4, right ovarian/testicular v., renal vv., right suprarenal v., inf. phrenic v., hepatic vv.</td>
<td>right atrium</td>
<td>all of the body below the level of the respiratory diaphragm</td>
<td>the inferior vena cava is longer than the abdominal aorta</td>
</tr>
<tr>
<td>vertebral venous plexus, external</td>
<td>intervertebral vv.</td>
<td>adjacent segmental vv.; vertebral v. in the cervical region</td>
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<td>two plexuses are described: anterior and posterior; connects with the internal vertebral venous plexus</td>
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</tr>
<tr>
<td>vesical venous plexus</td>
<td>multiple bladder tributaries</td>
<td>Sup. and inf. vesical vv. to the internal iliac v.</td>
<td>urinary bladder</td>
<td>in the male - connects with the prostatic venous plexus and the rectal venous plexus; in the female - connects with the rectal, uterine and vaginal venous plexus</td>
</tr>
</tbody>
</table>
Wed 19 Sept 0820-0850 Dr David Gibson Overview of Ultrasound and Laser Physics

POWERPOINT (see next page)
Abstract

Background:
The deciding parameters concerning hemodynamic efficacy of compression devices are the interface pressure and the stiffness of the compression material.

Aim:
To discuss the relationship between the exerted pressure of different compression devices on venous diameter and intravenous pressure in different body positions.

Methods:
The diameter of leg veins in different body positions has been measured by Duplex under increasing pressure produced by pneumatic cuffs containing ultrasound-permeable windows.
The difference of the interface pressure between standing and supine measured proximal to the inner ankle is a parameter for stiffness.

Results:
In the upright position a pressure of about 50-70 mmHg is necessary to narrow and to occlude veins of the lower leg. Intermittent pressure peaks of this magnitude may be obtained during walking under compression material of high stiffness.

Conclusion:
To obtain hemodynamic improvement in patients with severe venous insufficiency the pressure peaks of a compression device should intermittently exceed the local intravenous pressure on the leg during walking. This can preferably be achieved by compression devices with high stiffness resulting in a tolerable resting pressure in the supine position and a high working pressure.

Compression pressure and stiffness

Compression is defined by a force exerted to an area on the body surface. The compression pressure, expressed in Pascal (Pa) is the force of 1 Newton per square meter. In the medical field mainly mm Hg is used as the unit of pressure. (1 mm Hg = 133,3 Pascal).

According to the law of Laplace the pressure will be zero over completely flat areas, while it will be high over sharp edges. The compression pressure $P$ is directly proportional to the tension of the textile ($T$) but inversely proportional to the radius of the curvature to which it is applied. ($P \sim T/R$).

The pressure developed beneath a bandage is also governed by the width and number of layers applied.

Stiffness is defined as the increase in sub-bandage pressure per centimetre increase in the circumference of the leg (1). This parameter characterizes the elastic property of a compression device and defines the relationship between resting and working pressure. When the muscle contracts, inelastic material will produce a higher increase of interface pressure than elastic, yielding material. To achieve the same pressure peaks elastic material would need to be applied with a much higher pressure which would not be tolerated in the resting position (Fig. 1). Stiffness may be measured in the laboratory where it corresponds to the slope of the hysteresis curve. The fact that it can also be assessed by in-vivo measurements on the individual leg will certainly be of increasing practical importance in future trials (2).

Compression material
The compression devices most commonly used are summarized in Table 1.

Table 1: Overview on low and high stiffness compression materials

<table>
<thead>
<tr>
<th>Low stiffness</th>
<th>High stiffness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Compression stockings (single layer)</td>
<td>Rigid bandages (e.g. zinc paste), Velcro-band devices, pumps</td>
</tr>
<tr>
<td>Single component elastic bandages</td>
<td>Short stretch bandages, adhesive, cohesive material</td>
</tr>
<tr>
<td></td>
<td>multi-layer bandages</td>
</tr>
</tbody>
</table>

Several layers of bandages or stockings applied over each other make bandages stiffer. As shown in Fig. 1 inelastic material produces a much higher pressure increase in the upright position than elastic material. When several layers of elastic material are applied over each other stiffness of the final bandage will increase. This is also true for elastic stockings applied over each other (3).

Adhesive and cohesive materials increase stiffness.

**How can we assess interface pressure and stiffness in vivo?**

Several devices to measure interface pressure of compression devices are commercially available. The prerequisites of a good sensor and the ideal measuring sites on the leg have been described in an international consensus paper (2).

The classification of compression stockings varies widely between different countries. The pressure ranges given by the producers are entirely based on vitro-testing. Therefore it was proposed not to use the terminology of compression classes but rather indicate the pressure exerted to the distal lower leg in mmHg. It could be shown that for high quality stockings there is a satisfactory agreement between the pressure data measured in vitro and in vivo (3).

For compression bandages in vivo measurements are the only possibility to assess the interface pressure. In a recent consensus meeting it was proposed to classify bandages with a pressure on the distal lower leg of less than 20 mm Hg as mild, 20-40 mm Hg as moderate, 40-60 mm Hg as strong and >60 mmHg as very strong (4).

In order to obtain valuable information on the elastic property of a compression device which may be quite complex when several materials are combined, the so called "static stiffness index (SSI)" may be a useful parameter: A calibrated pressure sensor is fixed to the medial aspect of the leg about 12 cm above the inner ankle. This is the area where the muscular part of the gastrocnemius muscle changes into the tendinous part showing the most extensive changes in local curvature and leg-circumference by changing the body position between supine and standing. The difference between the interface pressure in the standing and in the lying position (mmHg), called SSI, is a valuable parameter for the stiffness of the compression system (2).

The pressure peaks and the pressure amplitudes during walking are also parameters for stiffness and correlate well with SSI. However, these parameters depend on the walking ability of the patient and require measuring systems that allow dynamic pressure readings.

It is important to note that different stiffness indices may be obtained with differently sized sensors. Therefore reliable comparisons will only be possible testing different compression devices by using the same sensor on the same site (2).

**Therapeutic aim**

The main target of any effective treatment of severe venous disease is to lower ambulatory venous hypertension. This can be achieved by abolishment of venous refluxes by venous surgery, sclerotherapy or by compression treatment.
One of the main intentions of adequate compression therapy of the lower extremities is to counteract gravity.

Compression is able to affect venous hemodynamics if the interface pressure is high enough to overcome intravenous pressure, always adjusted to the body position. The ideal compression device would exert a low sub-bandage resting pressure in the supine position that is well tolerated during night-time and would show a pressure increase when the patient stands up in order to counteract the increasing intravenous pressure. While walking the external compression should reduce venous refluxes by intermittent narrowing of the veins and should increase the amount of blood pumped up towards the heart with every single step.

Table II summarizes the most important treatment goals.

<table>
<thead>
<tr>
<th>Clinical indication</th>
<th>Intended effect</th>
<th>Pressure required</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oedema</td>
<td>Prevention (long sitting)</td>
<td>10-20 mmHg</td>
</tr>
<tr>
<td></td>
<td>Therapy</td>
<td>20-60 mmHg *</td>
</tr>
<tr>
<td>Thromboprophylaxis</td>
<td>Acceleration of venous flow</td>
<td>10-15 mmHg</td>
</tr>
<tr>
<td>(lying position)</td>
<td>(lying position)</td>
<td></td>
</tr>
<tr>
<td>Venous occlusion after surgery,</td>
<td>Occlusion of dissected branches, „empty</td>
<td>Lower leg &gt;70 mm Hg,</td>
</tr>
<tr>
<td>endovenous therapy</td>
<td>vein“ Standing:</td>
<td>Thigh 30-60 mmHg</td>
</tr>
<tr>
<td>Chronic venous insufficiency (refluxes)</td>
<td>Intermittent narrowing of veins during</td>
<td>50-80 mmHg **</td>
</tr>
<tr>
<td></td>
<td>walking</td>
<td></td>
</tr>
</tbody>
</table>

*) adjusted to the severity of oedema, limb circumference, consistency of tissue, mobility.
**) adjusted to the degree of ambulatory venous hypertension

**Which compression pressure do we need?**
In order to prevent leg swelling after prolonged sitting compression stockings exerting a pressure between 10 and 20 mmHg are sufficient (5). Existing oedema can be reduced in a shorter period of time using higher pressures (Table II).

In order to narrow superficial and deep leg veins the external compression pressure should be higher than the intravenous pressure. This can be shown by observing the venous diameter with a Duplex probe through a fenestrated, pneumatic cuff that is gradually inflated (6). The pressures needed to narrow and then to occlude a vein depend on the body position. They correspond to the physiological values; this is about 20 mm Hg in the supine and 50-70 mmHg in the upright position at lower leg level. So called thromboprophylactic stockings are able to accelerate venous blood flow velocity in the supine but not in the upright position.

**How to achieve a compression pressure in the range of 50-70 mmHg?**
Such high compression pressures may mainly be achieved with strongly applied compression bandages. Ideally these high values should be exerted only during standing and walking and should fall immediately when the patient lies down. The superposition of compression stockings may come close to these pressure ranges.

**What are the hemodynamic consequences of higher stiffness?**
The effects of compression do not depend only on the interface pressure.
With the same resting pressure inelastic compression material reduces venous refluxes more effectively than elastic bandages (7). In contrast to compression stockings exerting a pressure of around 30 mmHg inelastic bandages applied with a pressure of more than 50 mmHg are able to reduce ambulatory venous hypertension, even in patients with deep vein incompetence (8).

What is the hemodynamic mechanism of high stiffness material?
As it can be demonstrated by Duplex measurement of the venous diameter on the lower leg using a fenestrated inflatable cuff, stiff material can lead to an intermittent occlusion of the lower leg veins with each muscle contraction during walking. For a short moment the sub-bandage pressure peaks during muscle systole will overcome the intravenous pressure and will thereby occlude the vein. Stiff bandages may therefore act like an artificial valve suppressing refluxes during each muscle systole (9). At the same time the muscle veins will be squeezed out and the blood volume expelled towards the heart during walking will be increased. As we know from several experiments using pneumatic compression pumps intermittent pressure waves also have marked effects on the release of vasodilating, anticoagulatory and anti-inflammatory mediators from the endothelial cells (10).

Disadvantages of high stiffness bandages
The application of stiff bandages exerting high pressure is not easy and should be trained. Usually these bandages are applied to the lower leg in the sitting patient whose ankle is in maximal dorsiflexion. In general such bandages should be applied using several layers with a considerably higher resting pressure compared to elastic material. Just by lying down and relaxing the ankle interface pressure will show an immediate pressure drop. This pressure drop will continue in the first minutes and hours when the patient is walking to values that are 30-40% lower compared to the initial pressure, mainly due to an immediate reduction of leg volume. For the next few days only a mild further pressure drop occurs. Depending on the amount of oedema removed the bandages will get loose and have to be renewed accordingly.

Compression stockings show only a minor pressure loss.

High stiffness and arterial occlusive disease
Sustained external compression should never exceed the intra-arterial pressure which can be assessed by measuring the systolic ankle pressure using a Doppler probe. Up to now a systolic ankle pressure of 50-70 mmHg was a clear contraindication for any kind of compression therapy. However, some recent experiments using specially designed intermittent pneumatic compression devices have shown that short pressure pulses with peak values of more than 100mm Hg followed by long intervals without pressure may increase arterial blood flow and produce beneficial clinical effects even in severe stages of peripheral arterial occlusive disease (11).

Bandages with high stiffness, applied with intentionally low resting pressure which should be adjusted to the systolic ankle pressure, will produce intermittent pressure peaks in a similar way when the patient is walking or moving the ankles. Especially in patients with oedema and mixed, arterial and venous disease the resulting massage effect of inelastic compression material will reduce the swelling and increase the arterial blood flow.

Elastic bandages exerting a sustained external resting pressure should not been applied in any kind of peripheral arterial disease.

Some practical considerations concerning measurement of pressure and stiffness in vivo
In future trials comparing different compression devices it will be mandatory to measure the “dosage” of the exerted pressure on the individual leg. This is especially important when multi-component bandages are developed, for which the elastic property of the final bandage is unpredictable (4). The pressure that is exerted during standing and walking is more relevant than the resting pressure in the supine position. Measurement of interface pressure is also an ideal educational tool for teaching correct bandaging.
Experienced bandagers will adapt the strength of the applied bandage to the circumference of the leg, to the amount of oedema and to the walking ability of the patient. It is rather unrealistic to adjust the bandage pressure to the degree of ambulatory venous hypertension in an individual case. However, based on the relationship between external compression pressure and intravenous pressure explained above, it seems reasonable to use strong compression in cases with severe venous pathology while light or medium pressure may be enough to treat milder cases and to counteract oedema.

In conclusion there is a need to measure interface pressure and stiffness of the final bandage in future trials comparing the clinical outcome by using different compression products.

In vivo measurements of these parameters are also recommended when new compression devices are developed.
References

Figures:
Figure 1: Measurement of interface pressure in the medial gaiter area with an inelastic bandage system (Rosidal sys®) and with an elastic bandage applied in several layers (Perfekta®). In the sitting position both bandages exert a pressure of 50 mm Hg. The inelastic bandage system shows pressure peaks up to more than 80 mmHg during dorsiflexion and an increase of pressure to 69 mm Hg by standing up. The elastic bandage produces only very mild pressure increase during ankle movement and standing (below).
Fig. 2: Schematic drawing of the intravenous pressure in the distal leg in a patient with ambulatory venous hypertension (green) in comparison to the interface pressure under an elastic (red) and an inelastic bandage (yellow). The intravenous pressure is around 90mm Hg during standing, fluctuates between 75 and 90 mm Hg during walking (ambulatory venous hypertension) and falls down to 10-20 mmHg in the supine position. Short intermittent occlusions of the leg veins will occur when the external bandage pressure exceeds the intravenous pressure. This occurs during walking (muscle systole) with the inelastic bandage (yellow) but not with the elastic bandage (red). In the supine position the pressure of the elastic bandage will be too high to be tolerated.
Patients with venous disease often exhibit dermatological changes. Sometimes these skin changes are the only clue to an appropriate list of differential diagnoses. Venous insufficiency is the most common venous disease which presents with a range of skin changes. Most people are familiar with venous eczema, lipodermatosclerosis and venous ulcers as manifestations of long-term venous insufficiency. The less familiar are changes such as acroangiodermatitis and pigmented purpuric dermatoses. Dilatation of venous structures is not always due to venous incompetence and could be primary or secondary to systemic disease, or environmental influences such as actinic damage and radiation. This paper will discuss the dermatological manifestations of venous insufficiency as well as other forms of vascular ectasias that may present in a similar fashion to venous incompetence. The second instalment will focus on dermatological manifestations of haematologic disease and in particular thrombo-inflammatory conditions. The third instalment will discuss vascular anomalies.

Skin Manifestations Of Venous Insufficiency
Common Manifestations
Common manifestations of venous insufficiency include oedema, corona phlebectasia paraplantaris (Figure 1), stasis dermatitis, pigmentary changes (hyper- and hypo-), atrophie blanche, lipodermatosclerosis, and skin ulceration. Less common manifestations include pigmented purpuric dermatoses, and acroangiodermatitis. Superficial thrombophlebitis (STP) can also occur in association with venous incompetence but will be discussed in the second instalment of this paper (Figure 2).

Venous hypertension is responsible for many manifestations of chronic venous insufficiency (CVI) including oedema, red cell extravasation, perivascular fibrin deposition, impaired arterial inflow, and other locally mediated disturbances. The local tissue sequelae of CVI are due to impaired clearance of cellular metabolites secondary to concurrent damage to the lymphatic system.

One of the earliest manifestations of venous hypertension is lower limb oedema. Oedema is defined as a clinically apparent increase in the interstitial fluid volume. Oedema that retains an indentation when pressed with a finger is called pitting oedema. Systemic causes of oedema include congestive cardiac failure, hepatic insufficiency, renal failure, pretibial oedema in myxoedema, hypoproteinemia, and other systemic disorders. Lymphoedema may be a sign of primary lymphatic outflow obstruction, or it may be secondary to the overproduction of lymph due to severe venous hypertension (the so-called phlebolymphoedema). Usually, lower leg oedema is symmetrical in patients with systemic disorders.

Oedema of venous insufficiency is quite often associated with skin changes. Venous eczema (Figure 3) is usually the earliest cutaneous manifestation of venous insufficiency, and it may be a precursor to hyperpigmentation and lipodermatosclerosis. The medial ankle is most frequently involved revealing erythematous, scaling, and sometimes exudative, weeping patches and plaques (Figure 4).

Secondary bacterial infection with staphylococcus aureus can cause typical crusting. Pseudomonal colonisation is a common complication. In long-standing cases, lichenification may occur as a consequence of chronic scratching and rubbing.

The development of contact dermatitis is especially problematic in the treatment of patients with venous eczema. Topical treatments, including neomycin, bacitracin and some topical corticosteroids, have been reported to cause contact sensitization in patients with venous eczema.1 Contact dermatitis should be considered when venous eczema becomes clinically worse or does not improve despite appropriate topical treatment. Patch testing will be indicated. Topical antibiotics such as neomycin and popular preparations such as Kenocomb should never be used on open venous ulcers.

Venous hypertension leads to red cell extravasation. Breakdown of red cells leads to haemosiderin deposition and cutaneous pigmentation (Figure 5). Hyperpigmentation secondary to red cell extravasation is difficult to treat and not responsive to hydroquinone or other standard bleaching
agents used to treat hypermelanosis. Treatment of the underlying venous hypertension can help in lightening the pigmentation but in general once haemosiderin pigmentation is present, it is mostly irreversible. Post-inflammatory hypopigmentation may occur which can even result in depigmentation. This should be differentiated from atrophie blanche.

Atrophie blanche is a specific type of dermal scarring usually presenting as a small reticulated porcelain white patch. Dermoscopic examination of atrophie blanche demonstrates dilated capillary loops interspersed within the dermal scars. Atrophie blanche is not specific for venous hypertension and can be a manifestation of livedoid vasculopathy as discussed below. Atrophie blanche is caused by obstruction of dermal arterioles and infarction of the skin supplied by these vessels (Figures 6-8). This obstruction can be secondary to venous hypertension or thrombo-occlusive vasculitis involving dermal arterioles. Skin in this condition is avascular and prone to ulceration and necrosis. Vasculitis can also lead to identical white scars of atrophie blanche on the legs. Other systemic diseases, such as systemic lupus erythematosus (SLE), rheumatoid arthritis, and Klinefelter syndrome can result in skin ulcerations that heal with atrophie blanche-like lesions.

Lipodermatosclerosis (LDS) is the induration of skin and the underlying fat in the lower legs (Figure 9). It usually extends from the ankles to the lower border of the calf muscle. Histologically, there is a septal panniculitis and sclerosis of the overlying skin.2 A less-known form is the so called ‘acute lipodermatosclerosis’ which is primarily an acute panniculitis. Acute lipodermatosclerosis can be confused with acute cellulitis or even acute venous eczema. Eventual induration of skin and the underlying adipose tissue leads to the more familiar clinical picture of chronic lipodermatosclerosis. Progression of disease leads to the classic inverted champagne bottle appearance. The induration and lack of perfusion of the tissue makes it prone to ulceration.

Most chronic lower leg ulcers have a venous aetiology.3 Most venous ulcers are caused by superficial venous incompetence.4 Only a minority are caused by deep vein incompetence or obstruction. Non-healing ulcers of the medial ankle are most likely due to underlying venous hypertension. These areas are especially prone to venous hypertension because their drainage largely depends on the competence of the great saphenous system and its associated perforating veins. The incompetent Cockett perforators along the posterior arch tributary of the great saphenous vein (GSV) play a crucial role in the altered haemodynamics of this area.

Venous ulcers are usually larger but shallower than other ulcers, have a moist granulating base and an irregular border (Figure 10). Calcification is a common finding. The tissue surrounding these ulcers may exhibit signs of stasis dermatitis and other changes such as atrophie blanche and hyperpigmentation.

Non-healing, nodular areas of venous ulcers should be biopsied to exclude neoplastic change. The most common tumour is squamous cell carcinoma, however rarely basal cell carcinomas may develop too.5 Malignant melanoma may present as a lower leg ulcer. Other important differentials include arterial, neuropathic, traumatic, pyoderma gangrenosum, vasculitis, infection, and self-induced artefact. Biopsies should be taken from the edge of the ulcer and not from its necrotic base. Multiple biopsies may be required for the diagnosis. Care must be taken in providing haemostasis. It is usually not possible to close the biopsy site with suture as the tissue is friable. Steri-strips can be applied but compression should be used to prevent bleeding from the biopsy site.

Livedoid Vasculopathy
Atrophie blanche is not always a manifestation of CVI but can be a sign of livedoid vasculopathy (LV). This condition is characterized by ulceration associated with a segmental pattern of reticulate purpura and atrophie blanche affecting the lower limbs (Figure 11). It has been falsely referred to as livedoid vasculitis, although true vasculitis is not a feature of this condition. It has also been described as ‘livedo reticularis with summer ulcerations’. LV is a thrombo-occlusive process. Histologically, it shows fibrin deposition within both the wall and the lumen of affected vessels but there is no evidence of a true vasculitis.6
LV has been associated with Raynaud’s phenomenon and acrocyanosis. Patients with LV may have a history of recurrent leg ulcerations. Such patients may have thrombophilic abnormalities such as factor V Leiden mutation, protein C deficiency, hyperhomocysteinemia, abnormalities in fibrinolysis, and increased platelet activation.

Pseudo-Kaposi’s Sarcoma (Acroangiodermatitis)
Another unique feature of CVI is the development of violaceous plaques and nodules on the legs and dorsal aspects of the feet (Figures 12-13). These lesions frequently undergo painful ulceration and can be clinically indistinguishable from classic Kaposi’s Sarcoma (KS). This clinical appearance has led this entity to be called pseudo–KS or acroangiodermatitis. Pseudo-KS is a hyperplasia of pre-existing vasculature. In contrast, KS is a vascular tumour associated with Human Herpes Virus type 8 (HHV 8). Vascular proliferation in KS is independent of the existing vessels.

Pseudo-KS is usually seen as a complication of severe CVI. Less commonly, congenital or acquired arteriovenous malformations, and arteriovenous fistulas can result in localised venous hypertension and the development of pseudo–KS. Pseudo-KS can also occur distal to the site of arterio-venous fistulas for haemodialysis. These lesions may resolve after closure, thrombosis or surgical elimination of the shunt. Pseudo-KS has also been described in association with hepatitis C and in some vascular malformations such as Klippel-Trenaunay syndrome (KTS). Bilateral lesions are usually associated with CVI, whereas unilateral lesions suggest an underlying arterio-venous malformations or a high flow lesion. Patients occasionally experience pruritus and pain. Ulceration and bleeding may also occur.
Pigmented Purpuric Dermatoses

Pigmented purpuric dermatoses (PPD) are a group of conditions characterized by extravasation of red cells and marked haemosiderin pigmentation of the skin. This condition has been wrongly called ‘capillaritis’, although there is no evidence of a true vasculitis. The hallmark of PPD is its characteristic orange-brown, speckled, cayenne pepper–like discoloration. The aetiology of PPD is currently under investigation. Venous hypertension, contact dermatitis, and certain drugs have been implicated in the pathogenesis of PPD. Other associations are hypersensitivity to food dyes and preservatives such as tartrazine, and clothing dye dermatitis (especially khaki dyes). Drugs such as aspirin, glipizide, thiamine and interferon-alfa have been implicated. The author’s recent research has discovered coagulation abnormalities and in particular platelet function abnormalities in this group of patients (in press). Differential diagnoses include angiocentric mycosis fungoides, venous hyperpigmentation, leukocytoclastic vasculitis, petechial haemorrhage and scurvy.

A number of clinical patterns are recognized all demonstrating a similar histologic appearance. These include Schamberg’s disease (or progressive pigmented purpuric dermatosis) (Figure 14), itchy purpura of Lowenthal (Figure 15), lichen aureus (Figures 16-17), purpura annularis telangiectodes (Majocchi disease) (Figure 18), pigmented purpuric lichenoid dermatosis of Gougerot and Blum (Figures 19), unilateral PPD (Figures 20-21) and eczematoid-like purpura of Doucas and Kapetanakis. A granulomatous variant of PPD has also been reported.

These subclass all share common histopathologic features of red cell extravasation, haemosiderin deposition and a lymphocytic infiltrate. A lichenoid inflammatory infiltrate is present in the Gougerot-Blum sub-type. Spongiosis is present in the Doucas and Kapetanakis sub-type. Older lesions tend to be less inflamed and extravasated red cells may no longer be present. In the author’s experience, these sub-classifications are somewhat artificial and one patient may exhibit multiple sub-types.
Lichen aureus is commonly found in association with venous disease and sometimes directly over an underlying incompetent perforator. Treatment of the underlying localised venous hypertension can help in the resolution of lichen aureus (Figures 16-17). Topical steroids are possibly helpful only if pruritus is present but otherwise have a limited role. The underlying condition and in particular coagulation abnormalities should be investigated and excluded. Patch testing, other forms of allergy testing and food elimination diets may be appropriate. Aspirin, vitamin E, fish oil and other forms of drugs and supplements interfering with platelet function should be stopped, if appropriate.

Cutaneous Vascular Anomalies

Cutaneous vascular anomalies may be classified into the following categories of tumours, malformations and ectasias. Tumours may be congenital or acquired, benign or malignant and hyperplastic or neoplastic. The most common tumour of childhood is haemangioma. Haemangiomas are not present at birth and typically undergo involution. Malformations are errors of morphogenesis and are present at birth. They do not spontaneously involute. Vascular ectasias are dilatations of preexisting normal vessels. This paper will discuss vascular ectasias not necessarily associated with venous incompetence. The third installment of this paper will discuss vascular tumours and malformations.
Vascular Ectasias

Dilatation of blood vessels can be due to increased intravascular pressure or loss of elasticity of the vessel wall. Varicose veins are dilated vessels demonstrating increased muscularity and loss of elastic fiber. Vascular ectasia could also be due to actinic damage resulting in elastolysis of the vessel wall.

Venous Lake
Venous lakes are probably the most common vascular ectasias. These are dark blue phlebectasias that tend to occur on sun-exposed skin, especially on the lower lips or ears of elderly patients (Figure 22). Chronic actinic damage injures the vascular adventitia and the dermal elastic tissue, causing dilatation of normal venous structures. Treatment with sclerotherapy for larger lesions and vascular laser therapy for smaller lesions is usually successful. Malignant melanoma is a differential diagnosis and should be excluded. Venous lakes, as against melanomas, are compressible and empty with compression. Venous lakes are benign lesions but can bleed or thrombose causing pain.

Angiokeratomas
Angiokeratomas are a group of vascular ectasias that involve the papillary dermis. Angiokeratomas are not true angiomas but rather ectasias of pre-existing vessels. Overall, altered haemodynamics typically caused by trauma appear to produce telangiectatic vessels of the papillary dermis with an overlying reactive hyperkeratosis to the epidermis. These lesions may also produce papillomatosis, and acanthosis of the epidermis. 11

Angiokeratoma of Fordyce. These are typically asymptomatic, 2-5 mm, blue-red papules with a scaly surface located on the scrotum, shaft of penis, labia majora, inner thigh, or lower abdomen. They are dilated ectatic capillaries in the superficial dermis with overlying epidermal hyperplasia. These lesions bleed easily. Angiokeratomas occur in the presence of varicoceles or vulvar varices and venous hypertension has been implicated in its pathogenesis. 12 If treatment is needed, then locally destructive methods have been used. Light hyfrecation may not provide adequate haemostasis and bleeding may occur. Vascular laser photocoagulation usually provides adequate treatment.

Angiokeratoma Circumspectum (AC). This is a rare type of angiokeratoma with a unilateral distribution of discrete papules and nodules localized to a small area of the leg or trunk. Due to its unilateral and truncal distribution, AC has also been called angiokeratoma corporis neviform. AC has been classified as a capillary malformation. It has been reported to coexist with angiokeratoma of Fordyce and caviar spots (angiokeratomas of the tongue). It can be found in association with other vascular malformations and haemangiomas. Recurrent bleeding can occur. Women are affected more commonly than men, in a ratio of approximately 3 to 1. 13 These lesions respond well to vascular laser treatment.

Angiokeratoma Corporis Diffusum (Fabry’s disease). This is an X-linked disorder caused by a deficiency of the lysosomal enzyme alpha-galactosidase. This inborn error of metabolism results in deposition of neutral glycosphingolipids in the lysosomes of most cells. The most affected cell is the vascular endothelium. 14 Angiokeratomas form and tend to concentrate between the umbilicus and
the knees but usually spare the head. Lymphoedema and varicose veins are present. Renal failure, cardiac failure and cerebrovascular disease are the major causes of death. Heterozygous females may develop angiokeratomas but experience a milder clinical course.

Telangiectasias
Telangiectasias are dilated venules, arterioles or arteriovenous malformations visible on the skin or mucosal surfaces. Telangiectasias that develop in the absence of any preceding or coexisting cutaneous or systemic disease are considered to be primary or essential. Telangiectasias resulting from or in association with a known disease are classified as secondary.

Leg telangiectasias (spider veins). These vessels are dilated post-capillary venules demonstrating backflow from incompetent reticular (feeder) veins or directly from incompetent tributary or even truncal veins (Figure 23). Doppler examination may demonstrate reflux in even very small telangiectatic vessels. Telangiectasias associated with venous incompetence are usually dark red. Larger bluish vessels are termed venulektasias. Not all leg telangiectasias are secondary to venous incompetence and essential telangiectasias are commonly found near the feet and ankles. Gold standard for treatment of leg telangiectasias is sclerotherapy. The underlying venous incompetence must be treated first, before any attempt is made at sclerosing the telangiectasias. Vascular laser therapy is unlikely to be effective if underlying venous incompetence is present.

Facial telangiectasias. Telangiectasias seen on the face and trunk are usually due to actinic damage. Facial telangiectasias may also be associated with flushing and vascular instability as seen in rosacea or carcinoid syndrome (Figure 24). Best treatment for these lesions is vascular laser therapy. Peri-alar telangiectasias usually demonstrate higher flow volumes and are more difficult to treat. They are more common in people suffering with sinus disease and chronic nasal congestion. This can lead to recurrent peri-alar telangiectasias despite adequate treatment. Vascular laser therapy may need to be repeated a number of times for larger peri-alar telangiectasias or sclerotherapy can be attempted.

Post-radiation telangiectasias. Radiotherapy can lead to appearance of telangiectasias up to 20 years after the procedure. The radiation damage causes destruction of elastic fibers within vessels and leads to formation of the telangiectasias (Figure 25). Malignancy and in particular squamous cell carcinoma can develop in these areas.

Mat telangiectasias. These are patches of telangiectasias seen in patients with scleroderma and CREST syndrome (Figure 26). They present as flat and non-pulsatile vessels appearing as discrete mats. This should not be confused with telangiectatic matting which is a complication of surgery and sclerotherapy.

Telangiectatic matting. This is a form of neovascularisation which occurs as a complication of sclerotherapy or surgery (Figure 27). Patches of very small telangiectasias appear over the sites of recanalized vessels. Inflammation seems to play a role in its pathogenesis. Inflammatory cytokines with angiogenic properties probably play an important role in the pathogenesis of this condition. Other associations include concurrent use of female hormonal supplements such as oral contraceptives and hormone replacement therapy. There are usually underlying small incompetence veins in direct association with these vessels.
Poikiloderma of Civatte. Poikiloderma of Civatte refers to a reticulate erythematous pigmentation seen on the sides of the neck (Figure 28). Areas shaded by the chin are spared. The term poikiloderma refers to the combination of atrophy, telangiectasias, and pigmenary changes (both hypo- and hyper-pigmentation). It occurs most commonly in fair-skinned middle-aged or elderly females. Chronic exposure to ultraviolet light appears to be a primary aetiologic factor but photosensitizing chemicals in perfumes or cosmetics have also been implicated.  

Periungual telangiectasias. These are telangiectasias along the proximal nailfold. They are visualized dermoscopically as dilated capillary loops. These are pathognomonic signs of connective tissue diseases, such as lupus, scleroderma, and dermatomyositis.  

Generalised Essential (GET). This refers to the syndrome of widespread primary telangiectasias of unknown aetiology. There is no reported association with venous disease or actinic damage. Familial cases have been reported with an autosomal dominant pattern of inheritance. It is termed ‘generalised’ because of the widespread anatomic distribution of the telangiectasias. However, GET usually starts as light pink linear or punctuate telangiectasias on the feet, ankles, and distal legs and may gradually spread (Figures 29-30). When the lesions become confluent, the skin appears diffusely erythematous. Involvement of the oral mucosa and conjunctiva has been reported. GET is usually asymptomatic, but tingling, burning or numbness is occasionally reported. The age of onset is usually in the fourth or fifth decade, but it may be observed in younger adults. 

Hereditary Benign Telangiectasias. This condition resembles GET. It commences in early childhood, and is usually present on sun exposed skin. 

Hereditary Haemorrhagic Telangiectasia (HHT- Osler-Rendu-Weber disease). These lesions are arteriovenous malformations (AVM) of the microvasculature. They usually appear as punctuate dark red elevated lesions on mucous membranes, face and distal limbs. Lesions may involve the tongue, retina, lung and the brain (Figure 31). HHT lesions appear during the third or fourth decade of life. Recurrent epistaxis is usually the presenting symptom. Coagulation factor deficiency such as Von Willebrand’s disease may be present. Family screening may be indicated. 

Unilateral Naevoid Telangiectasia (UNT). In this condition, patches of telangiectasia present in a unilateral linear distribution. The segmental pattern suggests a somatic mosaicism. Pregnancy, chronic liver disease and other states of oestrogen excess are implicated in its pathogenesis. The third and fourth cervical dermatomes are the most common sites, but the thoracic dermatomes and scattered distant sites may also be involved. 

Telangiectasia Macularis Eruptiva Perstans (TMEP). This is the telangiectatic form of cutaneous mastocytosis. The lesions appear as brown telangiectatic macules that may be generalized in distribution. When a lesion of other forms of mastocytosis such as urticaria pigmentosa or mastocytoma is stroked, it typically urticates, becoming pruritic, oedematous, and erythematous. This change is referred to as the Darier sign, which can be explained on the basis of mast cell degranulation induced by physical stimulation. The Darier sign is usually negative in TMEP. This is because the lesions of TMEP are paucicellular and, therefore, mast cells may not be present in sufficient numbers for significant degranulation to occur. Diagnosis of TMEP is made on skin biopsy. Anaesthetic should be injected without adrenalin adjacent to, and not directly into, the lesion to avoid mast cell degranulation, which would make histologic examination difficult.
Ataxia Telangiectasia (AT). This is an autosomal recessive immunodeficiency characterized by progressive neurologic impairment and cerebellar ataxia. Patients are susceptible to sinopulmonary infections, impaired organ maturation, and a predisposition to malignancy. Oculo-cutaneous telangiectasias have a later onset than ataxia, first noticed in childhood and sometimes not until adolescence. Ocular telangiectasias extend from the lateral canthus in the equatorial region of the conjunctivae towards the corneal limb. Telangiectasias may also involve the ears, eyelids, the cubital and popliteal fossae. The telangiectases are predominantly of venous origin and are not AV malformations.

Spider Naevi. Spider naevi are dilated ascending arterioles. They usually exhibit radiating thin-walled vessels in association with a central arteriole (Figure 32). These lesions are also referred to as spider angiomas and naevus araneus. Spider naevi are not vascular proliferations but occur as a result of the dilation of preexisting vessels. They demonstrate arterial flow on Doppler examination. Compression of the central arteriole produces blanching. When released, the lesion quickly refills from the central arteriole. Spider naevi are acquired lesions present in 10-15% of healthy adults and young children. Lesions are found most commonly on the face, neck, chest and arms. In young children, spider naevi are common on the backs of the hands and fingers. Hormonal changes play a role in pathogenesis of these lesions. Many women develop lesions during pregnancy or while taking oral contraceptives. The lesions usually resolve spontaneously 6 to 9 months after delivery or after discontinuing oral contraceptives. Numerous lesions are seen in patients with significant hepatic disease such as advanced hepatitis C.

References
THE DIAGNOSTIC PATHWAY OF ULCER DIAGNOSIS
A “hole in the skin” secondary to destruction of the epidermis and at least the upper papillary dermis.

DIFFERENTIAL DIAGNOSIS (NOT VEINS)
- Neoplasm (SCC, BCC, MM, KS, lymphoma)
- Occlusive (arterial, embolic, Buerger’s)
- Trauma (DA, decubitus, physical)
- Vascular (vasculitis, haematological)
- Endocrine (DM, myxoedema, nutritional)
- Infection (deep fungal, mycobacterium)
- Neuropathic (DM, syphilis, leprosy, spinal)
- Skin (pyoderma gangrenosum, NLD)

LEG ULCERS: TOP SIX
- Venous
- Arterial
- Squamous cell carcinoma
- Infection
- Diabetic neuropathy
- Vasculitis (leucocytoclastic, pyoderma gangrenosum, PAN, livedoid, ec)

ULCER DIAGNOSIS
- HISTORY
  - Patient profile
  - Pre-existing conditions
  - Rapidity of onset
  - Symptoms (pain)
  - Systemic features
  - Immunosuppression
  - Medications
  - Previous treatment
  - Social
- EXAMINATION
  - Site
  - Size
  - Shape
  - Border
  - Base
  - Peripheral pulses
  - Surrounding skin
  - General

VENOUS ULCER
- Gaiter region (medial lower third of leg)
- Uneven edges, shallow and exuding, granulation tissue
- Surrounding reddish-brown pigmentation and oedema
- Varicose eczema, LDS, atrophic blanche, healed ulcers
- Absent to mild pain, relieved by elevation
- Normal pulses, no neuropathy

ARTERIAL ULCER
- Lateral ankle or foot
- Well demarcated edges, deep, necrotic base, poor quality or no granulation tissue
- Atrophic skin, dystrophic nails, absence of hair growth, limb may be cool
- Very painful at rest (relieved by lowering leg to a dependant position, dependent rubor)
- Diminished or absent pulses
- Possible neuropathy
SQUAMOUS CELL CARCINOMA
- Any site (often below knee)
- Heaped (crater) firm edges, necrotic centre or crusted and easily traumatised
- Surrounding skin often sun damaged (solar keratosis, Bowen’s, BCC etc)
- Not painful unless very large
- Normal pulses, no neuropathy

“INFECTIVE” LEG ULCER
- Bacterial, deep fungal, mycobacterium
- Immunosuppressed, penetrating injury
- Arms or legs
- Absent to moderate pain
- Purulent, erythematous margins, raised edges
- Maybe multinodular/ulcerative
- Lymphatic distribution

DIABETIC ULCER
- “Neurotropic ulcers”
- Over metatarsal arch, heel, toes
- Often deep and infected; surrounded by thick callus
- Painless
- Diminished pulses, neuropathy

PYODERMA GANGRENOSUM
- A/w with inflammatory bowel disease, haematological, connective tissue, arthritis
- Calves and thighs
- Irregular raised border (dusky red-purplish), necrotic base may extend to fascia
- Very painful
- Often severe (rapidly growing)

INVESTIGATION OF LEG ULCERS
- Biopsy (ulcer edge, histology and microbiology)
- Duplex US, ABI
- FBC, ESR, EPG/EPG, BSL, etc
- Wound swab
- Refer appropriately
Wed 19 Sept 1130-1150 Dr Abdullah Omari Medical Management of Leg Ulcers

Not available at time of printing
Introduction

The haemostatic system involves a delicate balance between both procoagulant and anticoagulant reactions. The coagulation system can be considered a biological amplifier, whereby trace amounts of collagen, von Willebrand Factor and tissue factor exposed on the subendothelial matrix leads to the formation of large amounts of cross-linked fibrin from fibrinogen through a series of proteolytic cleavages by plasma coagulation factors.

Traditionally, haemostasis has been modelled by a “waterfall” or “cascade” model, whereby it is split up into two pathways, namely the intrinsic and extrinsic pathways. More recently, this model has been refined into a cell-based model [1] whereby the contributions of both coagulation factors and cells such as platelets play an important role in clot formation.

The cell-based model of haemostasis

The cell-based model of haemostasis can be divided into three phases, namely initiation, amplification and propagation. The model involves the formation of trace amounts of thrombin which in turn stimulates platelet activation, allowing for the exposure on the platelet surface for large scale thrombin generation for subsequent fibrin generation.

The initiation phase follows vascular insult and the exposure of active tissue factor to the circulation. Tissue factor combines with activated Factor VII to form a complex responsible for the cleavage and subsequent activation of Factor IX and Factor X. Activated Factor X combines with Factor V to stimulate the production of trace amounts of thrombin from prothrombin.

The amplification phase commences following the adherence of platelets to exposed von Willebrand Factor at the site of vascular injury. The trace levels of thrombin generated during the initiation phase stimulates platelet activation, leading to the surface exposure of phosphatidylserine as well as the release of procoagulant molecules such as factor V from alpha and dense granules. Thrombin also activates Factor V as well as Factor VIII and Factor XI which then further stimulate the activation of Factor X, which binds with factor V on platelet phosphatidylserine.

The propagation phase involves the large scale thrombin generation from prothrombin by activated Factors V and X. Thrombin then stimulates the cleavage of fibrinogen into fibrin. These fibrin monomers spontaneously form hydrogen bonds with other fibrin monomers allowing the formation of long fibrin polymers to produce a fibrin clot.

Platelets

The primary role of platelets in haemostasis is to support coagulation by the formation of a platelet plug comprised of aggregated platelets, and to provide a catalytic surface for the generation of thrombin for fibrin clot formation.

Platelet activation occurs following exposure to a number of agonists, such as thrombin, ADP, collagen and von Willebrand Factor. This instigates platelet degranulation, whereby vasoactive mediators (such as ADP) are released into the circulation assisting in the recruitment and activation of further platelets. The platelet glycoproteins IIb and IIIa undergo a conformation change which allows for the binding of fibrinogen. Further activated platelets also adhere to this fibrinogen forming large platelet aggregates. Furthermore, platelets support coagulation by providing a catalytic surface for the assembly of the prothrombinase complex between activated Factors X and V. This catalytic
surface is comprised of negatively charged membrane phospholipids, in particular phosphatidylserine, which becomes exposed on the platelet surface following platelet activation. In addition, there is now increasing data to suggest that fragments of the platelet membrane (termed microparticles) are released following platelet activation and possess a high concentration of exposed phosphatidylserine.

**Fibrinolysis**
Finally, fibrinolysis is concerned with the breakdown of fibrin clots. Fibrin is degraded by plasmin, derived from its precursor plasminogen. This plasminogen proteolysis is mediated by tissue-plasminogen activator (tPA), primarily located on endothelial cells. The activity of tPA is regulated by plasminogen activator inhibitor (PAI-1).

**References**
Ultrasound has replaced all other modalities for confident diagnosis of deep or superficial venous thrombosis. Deep vein thrombosis (DVT) most commonly commences in below-knee veins and approximately 10% propagate to above-knee veins. If a scan for DVT is positive then some 20% are bilateral. If the initial scan is negative, conversion to a positive scan is extremely unlikely. A large proportion of patients with DVT are asymptomatic and presentation may be with a pulmonary embolus (PE) although some 40% of patients with PE confirmed by CT angiography have a negative lower limb DVT scan. It is acceptable to perform a D-Dimer test as the first investigation, for a negative result can allow ultrasound examination to be deferred to a convenient time without the need for anticoagulation in the interim.

A DVT may recanalise with restoration of valve function or destruction of valves, or the vein may remain permanently occluded. Approximately 50% of patients with a major DVT develop symptoms of the post-thrombotic syndrome within 10 years and about 5-10% eventually develop venous ulceration. If a scan is positive for DVT then serial scans are required to determine whether or not there is early propagation and later recanalisation. Later recurrence of symptoms requires repeat scanning to determine whether or not there is further fresh thrombosis. The duration for treatment with anticoagulation after deep vein thrombosis is arbitrary with warfarin usually discontinued at 3-6 months. A D-Dimer test shortly before proposed cessation may help to determine whether to continue or not depending on whether it is positive or negative. A final ultrasound scan should be performed when treatment is ceased to allow a baseline to compare for findings if there is suspected later recurrence.

Normal veins are easily compressible, larger than the corresponding artery, and smooth walled with an echo free lumen, while colour Doppler shows flow with distal augmentation and phasic flow with respiration or cessation of flow with the Valsalva maneuver in proximal veins. Acute thrombosis causes loss of compressibility, increased vein diameter and echogenic thrombus in the lumen, while colour Doppler shows loss of flow with augmentation or loss of phasic flow with respiration. Chronic thrombosis is demonstrated by more marked thrombus echogenicity and wall thickening, reduced vein diameter, and recanalisation or collateral flow. Deep vein occlusion following ultrasound-guided sclerotherapy has more the appearance of chronic thrombus with marked hyperechoic material in the lumen.
Wed 19 Sept 1340-1400  Dr Abdullah Omari  Management of Superficial Thrombophlebitis

Not available at time of printing
Venous Thromboembolism: Management of Acute DVT

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Initial phase of DVT Management:
- Ensure correct diagnosis has been made
- Assess patient’s general health and suitability for receiving anti-coagulant therapy – in particular, decide if there are any potential clinical contra-indications to anti-coagulation and if so, consider what alternatives to anti-coagulant therapy are available and effective
- Perform baseline blood testing to assess patient’s safety for receiving anti-coagulation - renal function, liver function, full blood count and coagulation times
- Testing for laboratory thrombophilia is not indicated at this stage and in some assays, test results will be affected by acute thrombotic state making their interpretation difficult
- Main objective of initial therapy is to prevent thrombus extension and early and late recurrences

Outpatient versus Inpatient Management:
- Assess patient’s suitability for being managed as an outpatient, which is generally preferable
- Patient groups that are unlikely to be suitable for outpatient treatment include those with co-existent serious medical problems, patients in significant pain, patients with significant renal impairment, those unlikely to be compliant with treatment and patients with a significant risk of bleeding.
- Network of integrated care must be set-up in community for ongoing support and management – administration of low molecular weight heparin (usually patient); commencement, monitoring and appropriate dose-adjustment of warfarin (usually general practitioner); is there a hospital in the home program available for those who need assistance?

Immediate Treatment:
- If there are no contra-indications, patient should be anti-coagulated initially with low molecular weight heparin (using once daily or twice daily dosing regimen)
- In some circumstances, unfractionated heparin may be preferable (as in severe renal failure)
- Initiation of oral anti-coagulants should commence on the first treatment day
- Low molecular weight heparin should be given for a minimum of 5 days and not ceased until INR has been > 2.0 for at least 2 consecutive days
- Monitoring of low molecular weight heparin with anti-Factor Xa levels are not recommended routinely
- In general there is no evidence to support the use of thrombolytic agents for initial treatment of DVT
- Progress venous ultrasound at around one week may be helpful to assess for possible clot extension
- Ambulation as tolerated is recommended

Long-term Treatment:
- The dose of warfarin should be adjusted to maintain a target INR of 2.5 (range of 2.0-3.0)
- Duration of anti-coagulation depends upon whether DVT is provoked or idiopathic
- For patients with a first episode of DVT secondary to a transient (reversible) risk factor, usual treatment duration is 3 months
- For patients with a first episode of idiopathic DVT, the usual treatment duration is at least 6-12 months
- If recurrent, idiopathic DVT, long-term anticoagulation is usually given
- The America College of Chest Physicians (ACCP) recommendations are similar for both proximal and calf vein DVT – ?contentious issue
- For patients with DVT and cancer, low molecular weight heparin (rather than warfarin) is recommended for long-term therapy
Use of Grade II elastic compression stockings during 2 years after episode of DVT are recommended

**References:**
Introduction to Thrombolysis

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:

A. active internal bleeding
B. disseminated intravascular coagulation
C. recent (< 3 months) cerebrovascular event, neurosurgery, or intracranial trauma
D. presence of an absolute contraindication to anticoagulation.

Strong relative contraindications include:

A. recent CPR
B. recent major surgery, delivery, biopsy, trauma
C. bleeding event
D. intracranial lesion or seizure
E. uncontrolled hypertension
F. thrombocytopenia
G. known right-to-left cardiopulmonary shunt
H. renal failure
I. severe hepatic dysfunction
J. septic thrombophlebitis
K. diabetic hemorrhagic retinopathy.

A. Lower Extremity and Inferior Vena Caval 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 axillosubclavian venous thrombosis is largely dependent upon the degree of symptoms and overall patient condition. In general, symptomatic younger patients without major co-morbidities 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 pharamcomechanical 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 regimens 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.

**Introduction to IVC Filters**

Although systemic anticoagulation remains the cornerstone of venous thromboembolism treatment, not all patients are candidates for this therapy, some fail the therapy, and some patients on anticoagulation suffer complications from the treatment. Fortunately, inferior vena cava (IVC) filtration is available for these selected patients as an adjunctive treatment for venous thromboembolism, or as an effective prophylactic measure in selected high-risk patients.

**Indications for Infrarenal IVC Filtration**

The three classic indications for IVC filtration include the presence of venous thromboembolic disease (pulmonary embolus or IVC, iliac, or femoropopliteal deep venous thrombosis) combined with one of the following:

(a) contraindication to anticoagulation
(b) complication of anticoagulation
(c) failure of anticoagulation (including recurrent PE despite adequate anticoagulation and inability to achieve therapeutic systemic anticoagulation).

The contraindications to anticoagulation have been cited as a bleeding complication of anticoagulation, known recent hemorrhage, recent major trauma or surgery, hemorrhagic stroke, thrombocytopenia, heparin-associated thrombocythemia thrombosis syndrome, and a known central nervous system neoplasm, aneurysm, or vascular malformation.
Additional well-accepted indications include:

A. massive pulmonary embolism with residual deep venous thrombus (DVT) in a patient at risk for further PE; free floating iliofemoral or IVC thrombus.

B. Severe cardiopulmonary disease and deep venous thrombosis (e.g. cor pulmonale with pulmonary hypertension); and poor compliance with anticoagulant medications.

“Extended” indications include prophylactic IVC filter placement in selected, high-risk patients without documented PE or deep venous thrombosis.

A. Severe trauma victims with closed head injury, spinal cord injury, and/or multiple long bone or pelvic fractures.

B. Other high-risk patients include those that are immobilized or subjected to prolonged intensive care.

C. Prophylactic filters preoperatively in patients who have multiple risk factors for venous thromboembolism.

D. Filtration for protection during iliofemoral DVT thrombolysis for prevention of significant PE.

Indications for Suprarenal IVC Filtration

A. Renal vein thrombosis.

B. IVC thrombosis extending up to or above the level of the renal veins, renal cell carcinoma with renal vein or IVC involvement, thrombus extending above a previously placed infrarenal filter.

C. Pulmonary embolism after gonadal vein thrombosis.

D. Anatomic variants, such as a duplicated IVC and low insertion of the renal veins.

E. Pregnant women.

Contraindications to IVC Filtration

Absolute contraindications:
Complete thrombosis of the IVC.
Uncorrectable Coagulopathy.

Bacteremia/Sepsis are relative contraindications, and clinical judgement must be used.

Success and Efficacy of IVC Filtration

The technical success for IVC filter placement should be equal to or exceed 97% in experienced hands.

The primary indicator of efficacy of an IVC filter is the recurrent PE rate. Generally speaking, all available IVC filters have comparable recurrent symptomatic PE rates, which range roughly between 2 and 5%. However, it is important to understand that the true incidence of recurrent PE following IVC filtration is probably higher, since most asymptomatic PE remain undiagnosed.

Decousus et al. compared IVC filters to a control group. This study showed a significant benefit of filters at 12-day follow-up, with the control group experiencing a more than four-fold increase in PE rate compared to the filter group (4.8% without filter vs. 1.1% with filter). This difference was even greater when only patients with PE at enrollment were considered (8.6% without filter vs. 1.1% with filter).
filter). However, there was no significant difference in mortality between these two groups. In addition, at 2-year follow-up, there was no significant difference between the two groups with respect to PE rate, but the filter group did experience more recurrent deep venous thrombosis than the control group (20.8% and 11.6%, respectively). These findings persisted in a subsequent paper reporting on 8-year follow-up of these patients. There was a significantly lower rate of symptomatic PE in the filter group compared with the no filter group (6.2% vs. 15.1%, p=0.008) while there was a slightly higher rate of DVT (35.7% vs. 27.5%, p=0.042).

**General IVC Filter Placement Procedure**

Prior to placement of an IVC filter, objective documentation of venous thromboembolism is essentially best performed with either ultrasonography, radionuclide scintigraphy, contrast enhanced computed tomography.

The procedure may be performed via a jugular, femoral, subclavian, or sometimes an upper extremity peripheral vein route.

Inferior vena cavography is performed to analyse the status of the IVC with regard to patency and the presence or absence of thrombus, to include measurements of the diameter of the IVC and the location of the renal veins, and to exclude the presence of a venous anomaly such as a megacava, duplicated IVC, and circumaortic or retroaortic left renal vein.

**Complications of IVC Filtration**

Complications of IVC filter placement vary among the specific filters, but for simplicity, they can be considered collectively for all filters as a group.

Thromboembolic events following IVC filter placement, such as recurrent PE.

**IVC thrombosis**

Recurrent deep venous thrombosis at the access site are not uncommon, with occurrence rates reported to be, 0.5-6%, 2-30%, 20.8%, and 2-28%, respectively. Most partial IVC thrombosis complications, often diagnosed incidentally on ultrasound or CT examinations, remain asymptomatic, and might be better interpreted as evidence of efficient embolus trapping by the filter. Complete IVC thrombosis can result in phlegmasia cerulea dolens, which can sometimes be treated with venous thrombolysis. Filter migration (0-18%) and embolisation to the right heart or pulmonary arteries (2-5%) occasionally occur spontaneously, but may be precipitated by entrapped exchange guide-wires used during bed-side central venous catheter placement procedures. Both guide-wire entrapment and filter embolisation have been successfully treated using Interventional Radiology transcatheter techniques.

IVC penetration, which may or may not cause a retroperitoneal hematoma or perforation into the aorta or gastrointestinal tract, has been documented to be as high as 41% in one series, but clinically significant penetration is believed to be a rare event. Filter fracture, which can be a late occurring event, can be detected on plain radiographs, and has been reported to occur with a rate of 2-10%.
Retrievable Filters

Because of the risk of the above-mentioned long-term complications of IVC filtration, retrievable filters have been introduced. Currently available temporary or retrievable filters might be better classified as "optional" filters, since they can function as permanent or temporary filters. An IVC filter that gives the option for use as a permanent or temporary filter is an attractive alternative for patients with a time-limited need for IVC filtration. This would include patients such as severely injured trauma patients at high risk for pulmonary thromboembolism and patients with venous thromboembolism and a temporary contraindication for anticoagulation, who subsequently can undergo anticoagulation. These patients may only require a filter for the short term, and could benefit by having the filter removed percutaneously at a later time.

The Günther Tulip Retrievable Filter has been used in Europe since 1992, and was introduced in the U.S. in 2000. It is manufactured from conichrome, a non-ferromagnetic alloy and thus it is MRI compatible up to 1.5 T.

The Recovery Retrievable Filter received FDA clearance as a permanent filter in 2002 and was redesigned in 2005 and renamed G2. Since there is no indicated retrieval time limit, extended implantations have occurred, with some filters successfully removed up to six months post insertion. Both of these filters are MRI-compatible and MRI-safe, and do not cause an artifact on MRI.

Selected References for IVC Filters;


Selected references for Thrombolysis:


Vascular Anomalies

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Vascular Anomalies

Can be divided into two types:
- Tumours
- Malformations

Vascular Tumours

Can be divided into two groups:
- Congenital- present at birth
- Non-congenital- appear later

Vascular Tumours Classification
Mulliken 1982

Congenital vascular tumours include:
- Haemangioma
- Kaposiform Haemangioendothelioma (KHE)
  - Tufted angioma
  - Haemangiopericytoma

Vascular Tumours Classification
Mulliken 1982

Acquired vascular tumours include:
- Pyogenic granuloma
- Kaposi’s Sarcoma
- Angiosarcoma
- Unilateral nevoid telangiectasias
- Angiolymphoed hyperplasia with eosinophilia
- Haemangioendotheliomas

Vascular Tumours

Haemangiomas:
- Proliferative vascular tumour
- Not present at birth
- Grow disproportionate to the child’s growth
- Evolution→ involution (when involuting develops areas of grey discoloration)
- Many sub-types: RICH, NICH, Segmental, etc

Rapidly Involuting Congenital Hemangioma (RICH)

- Vascular tumor
- Fully developed at birth
- Undergoes rapid involution and regression
- Rare entity
- Has positive glucose transporter (Glut-1)

**Non-Involuting Congenital Hemangioma (NICH)**

- Present before birth
- Grows in proportion with patient
- Does not involute
- If removed early (2-4 years) is similar to RICH
- Has negative glucose transporter (Glut-1)

**Haemangiomas**

- Can be life-threatening
- Treatment with oral steroids, supportive therapy, excision
- Managed by Pediatric Dermatologists, pediatric plastic surgeons
- ‘Strawberry haemangioma’ should not be used.

**Kaposiform Haemangioendothelioma (KHE)**

- Found in infants, less common in adults
- Close association with Kasabach-Merritt syndrome
- Tumour size may be large (>5cm)
- Localized:
  - Retroperitoneum 50%
  - Proximal thigh and groin 20%
  - Arms 20%
  - Other areas 10%
Kasabach-Merritt Syndrome

- Present from birth to first few weeks old
- Key Characteristics on skin:
  - Large
  - Swelling
  - Rapidly growing
  - Tender
  - Bruising reddish-purple soft-tissue
- Management
  - Prednisone
  - Interferon alpha
  - Vincristine

Tufted Angioma

- Tumour found presence:
- At birth 14%
- First year of life 49%
- Characteristics
  - Firm
  - Flat
  - Hyperhidrosis
  - Hard
  - Painful
- Associated with pregnancy and liver treatment

Haemangiopericytoma

- Found most often in the pelvis, proximal femur, vertebrae and humerus
- Affect patients ranging from 12 to 50
- Grow slowly and may be 20 years before diagnosis made
- Symptoms
  - Painless swelling/lump
  - Difficulties using limbs
  - Breathing difficulties
  - Coughing

PHACE Syndrome

- Posterior fossa brain malformations, large facial Hemangioma, Arterial anomalies, Cardiac anomalies and aortic coarctation, Eye abnormalities, and Sternal clefting and/or Supraumbilical raphe
- Rare, more common in females
- Present from birth to first few weeks of life
- Unilateral left-side most common, can be bilateral

Vascular Malformations

- Structural abnormality
- Errors of morphogenesis (4th-10th week)
- Present at birth
- Grow proportionately to the child
- Do not involute
Vascular Malformations can be a combination of:
- Capillary
- Venous
- Arterial
- Lymphatic

Terms NOT to use to describe VM:
- Port-wine stain
- Cherry angioma
- Salmon patch
- Campbell de Morgan spots
- Cavernous haemangioma
- Hepatic/vertebral/intramuscular haemangioma
- Naevus flammeus
- Lymphangioma
- Cystic hygroma

Vascular Malformation
Mulliken Classification 1982
- Capillary Malformation (CM)
- Venous Malformation (VM)
- Lymphatic Malformation (LM)
- Arteriovenous Malformation (AVM)
- Any combination of the above

Doppler Findings
Predominantly a collection of vessels
High flow lesions
- Prominent arterial feeders
- AVM
- AVF

Low flow lesions
- Capillary/Venous/Lymphatic

AVM vs. NICH

AVM
- Mostly vessels
- High flow
- Arterial feeder
- Will not go away
- Surgery:
  - Wide excision
  - Deeper excision
NICH

- Soft tissue mass
- High flow
- Arterial feeder
- Will not go away
- Surgery:
  - Narrow margins
  - Superficial fat

Capillary Malformation (CM)

- Dilated mature capillaries
- Present at birth
- No involution
- Uni or bilateral
- Pink in infancy → purple with age
- Raised and nodular after puberty

Capillary Malformation (CM)

- PWS
- Phacomatosis pigmentovascularis

Sturge Weber Syndrome

- Facial CM
  - Trigeminal nerve distribution (V1 ± V2 + V3)
  - Unilateral but could be bilateral
- Glaucoma
- Convulsions
- Hemiparesis
- Mental retardation

Venous Malformations

- Bluish phlebectasias
- Overlying skin may be normal or CM
- STP may occur

Usually localized around

- Face
- Trunk, limbs
- Brain, spinal cord, lungs etc

Generalized:

- Blue Rubber Bleb Syndrome
- Glomovenous Malformation (Glomangioma)
Blue Rubber Bleb (BRB) Syndrome

- Inherited AD
- Generalised distribution
- Multiple venous malformations
- Large draining veins
- DD
  - venous compression syndromes esp in UL
  - Multiple Hereditary Glomangiomas

BRB Syndrome

- Multiple cutaneous and GI lesions
- 0.1 to 5 cm nodules
- Lesions bleed causing Fe def
- Spontaneous thrombosis
- GI complications eg bowel infarction
- Other organs involved:
  - Brain, heart, lung, nasopharynx, etc

Maffucci Syndrome

- VM
- Enchondromas
- Bony abnormalities
  - Usually asymmetric
  - Cause secondary fractures

Glomovenous Malformation: (Glomangiomas)

- Arise from glomus cells
- Glomus cells are of smooth muscle origin
- Glomus bodies:
  - arteriovenous shunts present on acral structures
  - Neuromyoarterial receptors responsible for thermoregulation

- Glomus tumours are:
  - Usually solitary- nail bed
  - Painful
  - Arise later in life
  - Not inherited

- Generalised
- Not tender
- Present in childhood
- Strong family history
Lymphatic malformations

- Can be classified as:
  - Microcystic
  - Macrocystic

- Complications involves:
  - Bleeding
  - Infection
  - Lymphedema when extensive
  - May be associated with bony hypertrophy

- Old terminology examples:
  - Lymphangioma
  - Lymphangioma circumscriptum
  - Cystic hygroma
Capillary-lymphatic Malformation (CLM)

Includes:
- Hypertrophic naevus flammeus
- ‘ verrucous lymphangioma’
- Haemangiomalymphangioma
- Angiokeratomas
- CLM- Angiokeratoma of Fordyce
- CLM- Angiokeratoma Corporis Diffusum (Fabry’s Disease)
- CLM- Angiokeratoma of Mibelli

AVM Descriptions

- First appear as red or skin colored swelling
- Grow in proportion to the child’s growth
- At puberty may enlarge and bleed

AVM Classification

- Stage I: lesion has a pink-bluish stain and warmth. Doppler ultrasonography reveals arteriovenous shunting.
- Stage 2: the lesion has pulsations, thrill and bruit.
- Stage 3: the patient has dystrophic skin changes, ulceration, bleeding and pain.
- Stage 4: the patient has high-output cardiac failure.
AVMs

- May be confused with severe vv
- Chronic Aorto-caval fistulae
  - Venous dilatation in both lower limbs
  - Venous pulsation
  - ‘Machinery’ abdominal murmur
  - Abdominal pain, haematuria
- With macrofistulae may lead to
  - Limb hypertrophy
  - Tachycardia
  - Skin atrophy, ulceration

Complex Malformations

- Klippel Trenaunay Syndrome
  - C + V + L
- Parkes Weber Syndrome
  - C + V + A + L

KTS Identification

- CVL malformation
- Commonly presents as varicose veins
- Present at birth
- Usually unilateral
- Single limb or an arm and a leg on the same side
- Limb hypertrophy
- Subcutaneous fat is thickened
- May be painful
- Deep veins are normal
- Anterolateral vein of the thigh is usually involved

Proteus Syndrome

- Also known as Riley-Smith and Bannayan Syndromes
- Condition of abnormal growth
- Occurs bones, skin and head
- Symptoms includes:
  - Rough and raised skin
  - Increased size of an organ or bone
  - Capillary malformations

Cobb Syndrome

- Also known as Cutaneomeningospinal angiomatosis
- Rare, non-inherited condition
- Presented at birth
- Neurologic complications develop in early adulthood
Treatment for Vascular Malformations

**Alcohol Sclerotherapy/Embolization**

- Traditional method for treatment of VM
- Absolute ethanol (95-98%) is an aggressive sclerosant, causes degeneration of proteins in body and rapid thrombosis
- Advantage: most effective especially for large VMs
- Disadvantage: painful procedure, serious side effects e.g. peripheral tissue damages, respiration difficulties, CNS depression, cardiac arrest, etc. (1-4)
- Requires the use of tourniquets and compression to minimize the passage of alcohol into the systemic circulation.
- Performed under general anaesthesia, with careful monitoring of cardiovascular intra-operatively (too much ethanol in circulation becomes toxic, hemolysis and cardiac arrest).
- Local complications: blistering, full-thickness cutaneous necrosis and damage to local nerves. If ethanol reached the capillary bed of any given tissues (e.g. skin), it may cause significant soft tissue swelling and subsequently compartment syndrome (5).

**Ultrasound Guided Sclerotherapy**

- In foam format it has improved efficacy and is comparable with alcohol in potency. It is also safer than alcohol.
- UGS involves using duplex imaging to guide and monitor the intraluminal introduction and the subsequent flow of the sclerosant in the vascular space and the vasospastic reaction of the vascular lesion.
- Acts by removing the endothelial lining of the vessels and inducing fibrosis. The foam format has the advantage of being more efficient in displacing intra vascular blood and increasing the contact surface area of the endothelial layer with the sclerosant. The foam is produced by mixing the detergent sclerosant with air using a 3 way stopcock.
- Advantage: no need for monitoring (taking into account when the optimum volume of sclerosant is used), decreased side effects and complication rate. Also, ultrasound allows visualization so one can stop the injection at the appropriate time once optimal response seen, thereby increasing safety.
- Disadvantage: not suitable for very deep structures as ultrasound tissue penetration diminishes with depth and visualization becomes a problem.
- Suitable for treatment of low flow lesions e.g. VMs and lymphatic malformations.
- High flow lesions e.g. arteriovenous malformations are still probably best treated with embolization.
**Surgery** (6)

- Aim: to remove the whole lesion, prepare the lesion for other treatments or rectify whatever remains following involution.
- Depends on the child’s age, weight and degree of impairment the VM is causing. E.g. VM near the eye is removed at an earlier time than a VM located at the back.
- Can be performed in phases to achieve a better outcome.
- Remains a controversial treatment option because of a high recurrence rate (7, 8) of 18% (9).

**Fluroscopic Ultrasound Guided Sclerotherapy (FUGS)**

- Fluroscopic monitoring remains useful in monitoring the draining of feeding vessels. For certain patients, both imaging modalities (fluoroscopy and ultrasound) may be used simultaneously (10).
- Procedure:
  1) Ultrasound guidance is used to canulate patients.
  2) Omnipaque contrast medium is injected allowing visualization of the lesion and the normal draining veins.
  3) The detergent sclerosant (e.g. Sodium Tetradecyl Sulphate 3%) is prepared as foam using the Tessarri technique (1:3 liquid to gas ratio).
  4) The Foam is introduced slowly into the lesion and appears radio-lucent on fluoroscopy. The injection is stopped when the draining veins appear to take up the foam.
  5) Compression is applied and maintained for 7 days post-operatively.
Treatment of Haemangiomas

**Oral corticosteroids (11)**

- Aim: slow the growth of haemangiomas.
- Example of treatment course: Prednisolone 2mg/kg per day for 2 weeks, 1mg/kg for a further 2 weeks, and 0.5mg/kg for the last 2 weeks. Repeat courses should be avoided.
- Since high doses of it are needed to be effective, the patient must be carefully monitored (6). In children, the child’s blood pressure and sugar levels need to carefully monitored as anti-hypertensives may need to be administered (renal department).
- Use of Interferon-alpha is controversial and mostly abandoned.
- When loose tissue remains, plastic surgery can be used to make the appearance look better.
- Upper respiratory tract lesions can be treated with laser therapy instead of surgery.
References

Introduction:

- Diagnose vascular anomalies by clinical exam and MRI, not by diagnostic angiography.
- If you need to do a diagnostic angiogram, image the venous phase.
- Check coagulation parameters on patients with large VMs.
- Anticoagulation for two weeks helps reverse consumption coagulopathy.
- Never use ethanol in the orbit.
- Never use sodium tetradecyl intra-arterially.
- Never coil a proximal feeding artery.

Classification of Vascular Anomalies

Vascular anomalies are divided into two groups:

A. Tumors e.g. hemangiomas
B. Vascular malformations.

Malformations are categorized according to channel type and flow characteristics. High-flow vascular anomalies include arteriovenous fistula (AVF) and arteriovenous malformations (AVM).

Low-flow malformations include capillary malformations (CM), venous malformations (VM) and lymphatic (LM) malformations.

Some patients have combined channel anomalies (e.g. Klippel–Trenaunay syndrome (CLVM)).

Techniques

Endovascular techniques for treating vascular malformations consist predominantly of embolisation and sclerotherapy. Arterial embolisation is reserved for high-flow vascular malformations.

Emboliisation Agents

1. Polyvinyl Alcohol Foam Particles (PVA)

Polyvinyl alcohol was first utilised in foam form in the early 1970’s. Currently, the material is available as particles ranging in size from approximately 50 microns to 2,000 microns. As such, the level of
embolisation can be tailored to the clinical scenario at hand. As with other very small particulate agents, the smallest size of PVA may result in significant tissue ischémia and should be reserved mostly for tumor embolisation. PVA is delivered through the catheter in suspension form. The particles are mixed in a combination of saline and contrast either in a bowl or in syringes with mixing occurring across a three-way stop cock. PVA has a tendency to flocculate or settle out of solution. Immediately prior to injection, the material should be resuspended so that uniform injection can be obtained. Non-uniform delivery of the material can result in occlusion of the delivery catheter. Aggregation of PVA may result in a more proximal occlusion than intended.

PVA causes direct mechanical obstruction and induces a foreign body type reaction with permeation of the particles by granulation tissue. Over time this reaction subsides and months to years later, the vessel may recanalise. Although PVA is considered to be a permanent agent, it will recanalise over time.

PVA is utilised predominantly for tumor embolisation as well as pre-operative devascularisation of other lesions. PVA is the preferred embolic agent for bronchial artery embolisation and the 500-700 micron diameter particles are utilised extensively for fibroid embolisation.

Mechanism of Action: cause occlusion of the vascular bed and obstructs inflow.

2. **Embospheres**

Embosphere particles are a new addition to the list of approved materials for arterial embolisation. These particles are hydrophilic, acrylic based spheres. Available sizes are similar to PVA and they come in sterile vials, which are easy to use. EmboGold particles are a highly visible form, which comes in sterile prefilled syringes.

Advantages of these particles are their compressibility. Less catheter clogging leads to less catheter exchanges due to occlusion. Better penetration of particles into the vascular bed allows for a more uniform embolisation. They have been extremely well received for uterine fibroid embolisation.

A disadvantage may be a slightly higher per vial cost. The end-point for embolisation is somewhat different than with PVA.

3. **Gelfoam**

Absorbable and should not be used in AVM’s.

Mechanism of Action: cause a combination of vascular thrombosis and endothelial ablation, resulting in fibrosis.

4. **NBCA – Histoacryl (Glue)**

Cyanoacrylate materials have been used as adhesive embolic agents for more than 20 years. Their role in the embolisation of high-flow vascular lesions has been well established. Indications of NBCA are numerous, including the embolisation of various types of cerebral and spinal vascular malformations, as well as a growing number of peripheral vascular lesions, such as systemic AVMs.

Mechanism of Action: The obliteration of a blood vessel embolised with a cyanoacrylate agent results from a polymerization process induced by the contact of the glue with an ionic environment such as blood or normal saline. Hence the importance of flushing the delivery microcatheter with dextrose 5%, a nonionic solution, prior to NBCA injection. In addition to their immediate mechanical effect, cyanoacrylate agents also induce a chronic inflammatory response, which is believed to play an
important role in the permanency of the occlusion obtained with NBCA. This response can range from a mild histiocytic reaction confined to the vessel lumen, sparing the vessel walls or contiguous parenchymal tissues to more intense inflammatory reactions, including the occurrence of patchy mural angionecrosis. The permanency of the vascular obliteration obtained with cyanoacrylate agents depends in part to the homogeneity of the glue cast. In other words, it is preferable to obtain a continuous NBCA column rather that a mélange of glue and trapped blood that potentially opens the door to recanalization processes. When treating vascular lesions characterized by the presence of arteriovenous shunts, such as arteriovenous malformations or fistulas, it is necessary for the glue to reach the actual shunt site rather than just sit more proximally in the arterial feeders.

5. **OnyxTM Liquid Embolic System**

Ethylene-vinyl alcohol copolymer or EVOH consists of a polymer mixed with tantalum in a dimethyl sulfoxide (DMSO) base. This material can be injected through a catheter and once it comes into contact with the bloodstream, the DMSO diffuses out of the solution allowing the EVOH polymer to precipitate creating a cast of the vessel with direct mechanical occlusion. The operational principles are similar to using glue. With the current agent, it is difficult to predict the exact depth of penetration and the time to polymerization. One significant advantage when compared to glue is that this is not an adhesive agent and there is no risk of gluing the catheter in place.

**Mechanism of Action:** cause occlusion of the vascular bed and obstructs inflow.

6. **Coils.**

Coils should rarely be used in mid to low flow AVM’s as it will not occlude the nidus and prevent the interventionalist from getting to the AVM nidus at a later stage. Coils can be beneficial in high flow AVM’s or AVF’s.

Coils are available in almost all sizes from approximately 2 - 30 mm in diameter and are constructed of both stainless steel and platinum. Dacron fibres are woven into the coil to increase the thrombogenicity of the coil. In addition to direct mechanical occlusion by the coil, the Dacron fibers act as a nidus for thrombus formation. Extensive inflammatory changes are induced in the occluded arteries as well as in the periadventitial tissue. Coils produce a permanent focal occlusion leaving the vessel distal to the coils patent. The use of coils is analogous to a focal surgical ligature of the vessel.

The size of the desired coil should be matched to the size of the vessel. If the coil is smaller than the vessel, the coil may embolise too far distally or through an AV fistula. Alternatively, if the coil is oversized, the coil may cause recoil of the delivery catheter with subsequent reflux of the coil back into the parent vessel.

Platinum/Fiber coils are MRI compatible.

7. **Balloons**

Detachable balloons have recently been reapproved for vascular use and are extremely efficacious for high flow arterial venous fistulas. Balloons cause mechanical occlusion of the vessel with formation of thrombus proximal and distal to the balloon. Advantages include precise deployment and retrievability. Although the current balloons are made of silicon and may deflate over time, it is assumed that the vessel will remain permanently occluded because of the associated thrombus formation. Balloons are suitable for treating high flow pulmonary AVM’s as well as a large vessel AVF’s.

**Mechanism of Action:** cause mechanical obstruction are reserved for AVF and venous occlusions.
Sclerosing Agents

A. Ethanol

Ethanol preparations suitable for sclerotherapy include 95% or 98% dehydrated forms, generally available through hospital pharmacies for neurolysis. Ethanol rapidly denatures proteins in the endothelial lining of vessels and, in stagnant channels, results in immediate thrombosis.

Having a low viscosity, it passes readily through arteriovenous shunts making it suitable for embolisation of AVM in conjunction with supra-selective catheterization of the nidus. It should never be injected into a proximal feeding artery, as penetration of the capillary bed will result in severe tissue necrosis.

Ethanol also causes neurolysis locally. Systemic effects include CNS depression, hypoglycemia, hypertension, hyperthermia, cardiac arrhythmias, pulmonary vasoconstriction and pulmonary hypertension, and electromechanical dissociation.

Cardiovascular collapse, sometimes fatal, can result from effects on the pulmonary vasculature or myocardium. To avoid this, ethanol should be administered in small aliquots, with adequate time for recovery between injections. Consideration should be given to monitoring of pulmonary artery pressure. A total dose of 1 ml/kg or 60 ml should not be exceeded in one procedure.

B. Detergent Sclerosants

This category includes sodium tetradecyl, the most commonly used sclerosant, ethanolamine and polidocanol. Like alcohol, these sclerosants damage the endothelial cells and cause coagulation of the intraluminal blood products. Detergent sclerosants can be opacified with water-soluble contrast medium or with oily contrast medium. Addition of air results in a microfoam which is felt to be more effective than the bland solution. A reasonable dose limit for image-guided sclerotherapy is 0.5 ml/kg or 30 ml.

C. Other Sclerosants

Doxycycline is available as a powder that can be suspended in saline or contrast medium and is often used for sclerotherapy of lymphatic malformations. It is painful to inject, but effective and relatively nontoxic. An advantage in treating large lymphatic malformations is that large volumes can be used in a single session.

Bleomycin has been used for decades in the sclerotherapy of lymphatic malformations (LM). It has some systemic side effects including pulmonary fibrosis, hair loss and pigmentation and therefore the quantity of this drug used in each session must be carefully limited.

Mechanism of Action: Intravascular use of sclerosants results in thrombosis, swelling and hemolysis. Large quantities of sclerosants result in hemoglobinuria requiring aggressive hydration and urine alkalisation.

Embolisation of High-flow Vascular Malformations (AVF & AVM):

Preoperative embolisation aims to occlude selectively the arterial supply to the lesion, in order to reduce bleeding during operative resection. The embolic material should be chosen to occlude the most distal feeding arteries (Onyx or Glue), avoiding proximal occlusion especially with permanent
devices (Coils or Balloons), and avoiding embolisation of normal tissue with excessively small particles (PVA).

PVA particles greater than 250 microns are relatively safe in terms of avoiding damage to adjacent skin or nerves.

Primary embolisation usually involves serial embolisation using ablative agents.

Intranidal injections of Ethanol may sometimes be helpful to achieve more permanent occlusion.

Sodium tetradecyl should not be used intra-arterially as it causes excessive arterial spasm and reflux.

**AVM’s**

Direct arteriovenous communications are curable if the actual shunt or the immediate draining vein is occluded with a permanent device e.g. Detachable Balloons, Coils or NBCA.

**AVF**

In occluding large AFV, some type of flow control may be necessary to prevent migration of the Balloons or Coils. Upstream or downstream angiographic balloon catheters and tourniquets are often used.

Side-to-side fistulas can be occluded using covered stents.

**Occlusion of Low-flow Vascular Malformations (VM)**

**VM**

VM is the most common symptomatic vascular malformation referred for treatment. The basic defect in this lesion is maldevelopment of the vessel wall, especially the muscular media, resulting in abnormalities of shape and size of the affected vessels. There are a large variety of morphological types and venous malformations that can involve all tissue planes. VM can be sequestered, in which case sclerotherapy is safe and simple to perform, or non-sequestered, involving channels that communicate directly with major conducting veins. In the latter situation, precautions must be taken to isolate the treated segments of veins, to retain the sclerosant and subsequent thrombus. Failure to do so not only carries a risk of systemic complications and pulmonary embolus, but usually results in prompt recannalization. Outflow occlusion can be achieved temporarily by the use of an occluding tourniquet (sterile orthopedic automatic tourniquet), or permanently by occluding the outflow veins with Coils, Balloons or NBCA.

Variceal masses protruding from conducting veins can be sclerosed effectively with the combination of intraluminal fibered coils and sclerosant.

Sclerotherapy of VM is usually performed with ethanol or sodium tetradecyl.

Endovenous ablation has been applied to venous malformations and appears to be safe and effective, but the longterm outcome is not yet known.

**LM**

Lymphatic Malformations (LM) are generally classified as macrocystic, microcystic and mixed forms.
Macrocystic forms respond best to resection or sclerotherapy. Intracystic fluid should be aspirated prior to the injection of the sclerosant. Aspiration of fluid and injection of sclerosant can then be monitored by continued ultrasound imaging. This technique is also helpful in injecting microcysts, where fluid aspiration and contrast injection generally are not feasible.

**Complications:**

**Tissue necrosis**

Damage to skin or more extensive tissues is the most common complication of liquid embolisation or sclerotherapy of vascular malformations. Approximately 5-10% of patients having sclerotherapy of venous malformations have some skin necrosis resulting in scarring. In many of these cases such scarring is inevitable since the malformation involves the skin. Ulcers are treated with topical antibiotic ointment or polymer dressings and most heal well.

Tissue damage caused by reflux of sclerosant into nourishing arteries can be extensive, resulting in loss of digits or limbs or requiring debridment and skin grafting. Sclerosing agents injected into the venous component of AVM can enter the arterial circulation when the venous outflow is completely occluded.

**Nerve Injuries**

Neurologic injury can result from three mechanisms. Extravasation of ethanol around a nerve trunk can result in a demyelinization and necrosis. Penetration of arteries supplying nerve trunks can result in ischemic necrosis. Severe swelling caused by intravascular sclerotherapy can cause compartment syndrome and subsequent nerve injury.

**Hematologic Effects**

Endovascular treatment of large vascular malformation results in intravascular thrombosis and consumption of clotting factors. In patients with extensive venous malformations, this may be significant. Patients with preexisting consumption coagulopathy can be pretreated with Heparin to build up their fibrinogen stores, thus minimizing this complication. Such patients also often require infusion of plasma or cryoprecipitate.

**Pulmonary Embolism**

Massive pulmonary embolism is a rare complication of embolisation of malformations involving large conducting channels. Migration of microthrombi during sclerotherapy can also affect the pulmonary vascular reserve and result in cardiovascular collapse. In neonates, right-to-left shunting across a patent foramen ovale can cause systemic embolisation sometimes resulting in stroke or death due to myocardial infarction.

**Hemoglobinuria**

This complication is frequent and generally not significant if the patient is well hydrated and the urine is alkalinised. However in the presence of poor hydration, it can result in acute renal failure requiring hemodialysis.
Consumption Coagulopathy

Consumption of fibrinogen during sclerotherapy can be significant.

Systemic Effects of Ethanol

The most common systemic effect of ethanol is CNS depression. This can be significant in patients having general anesthesia or receiving narcotic analgesia. Cardiovascular affects include cardiac arrhythmias during and after the procedure, severe hypertension following the procedure, and intraprocedural pulmonary vasoconstriction and cardiovascular collapse.

Acute desaturation with bradycardia can also occur during ethanol sclerotherapy. Usually, this is reversible with cessation of injection, administration of atropine and 100% oxygen.

Other systemic adverse affects include hyperthermia, severe hypertension and hyperglycemia.

REFERENCES:


Treatment of Venous Malformations in Children

Venous malformations are the most common type of vascular malformation. They are usually present at birth but may not be visible until much later. They comprise of abnormal, dilated, thin walled vessels that lack smooth muscle. They tend to grow slowly and steadily with or slightly faster than the child. However, added factors such as trauma, infection and hormonal changes may cause rapid expansion. Large or multiple malformations may be associated with clotting dysfunction.

Venous malformations may involve any organ system. They may be superficial, deep, or both. The presentation and management depends on the site, size and extent of the malformation, with major disfigurement and proximity to vital structures such as the orbit or major cervical vessels necessitating early intervention. Small lesions can be observed indefinitely or treated at a more appropriate time.

Diagnosis is primarily on clinical grounds, with imaging used to confirm the diagnosis, exclude other vascular components, determine the extent and evaluate progress and response to treatment. Ultrasound is the mainstay of imaging diagnosis as it is a safe, dynamic, noninvasive modality without the use of ionising radiation. Doppler is vital to exclude high flow components. Ultrasound however has a limited role in determining the extent. MRI is the mainstay of staging particularly for deep lesions, helping confirm the diagnosis and determining the margins and extent. Direct puncture venography during endovenous sclerotherapy is useful to confirm the diagnosis, and evaluate the draining veins. It can also be used to titrate the sclerosant, thereby minimising local, pulmonary and systemic sequelae. Serial and end of treatment MRI is useful for monitoring progress and confirming resolution.

Treatment options include:
- Observation for small lesions with minimal cosmetic or functional impact,
- Conservative therapy with compression garments, particularly in the extremities.
- Sclerotherapy: Until recently using absolute alcohol, but currently with STS foam. This can be performed under ultrasound guidance alone, or with a combination of ultrasound and venography. Risks include recurrence, injury to adjacent tissue including overlying skin loss and a small risk of haemolysis. Multiple treatments are usually necessary.
- Laser therapy: Both for the skin and large deep venous components.
- Surgical excision: For poorly responsive or localised lesions. Best when localised to a single compartment such as a muscle. May need to sacrifice adjacent tissue particularly if margins indistinct. High risk of recurrence.
Lymphoedema reducing its risk though early detection and treatment

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Abstract

Lymphoedema occurs when the lymph load is greater than the lymphatic transport capacity. The major causes are damage associated with surgery and or radiotherapy or a malformation of the lymphatic system. Between 10 and 30% of men and women develop lymphoedema with the prevalence relating to the extent of surgery and use of radiotherapy. Lymphoedema does not always develop immediately, on average it takes 3 years to become clinically manifest. On occasions however surgical, radiotherapeutical or tissue trauma oedema may be replaced by lymphoedema in a continuum. Assessment of risk level and its sub-clinical detection using bioimpedance are crucial to reduce the risk of the development of chronic lymphoedema. Once formed, treatment and management is a life-time issue although some self management strategies and improving patient awareness of what they can do can make a big difference. Of crucial importance however is the concomitant diagnosis and remediation of cardio vascular and other non lymphatic issues which impact on lymphatic load. Included in these is an improved awareness of the impact that vascular related interventions such as surgery, ligation and harvesting may have on the nearby lymphatic system. The vascular specialist has an important role in early detection and differential diagnosis of lymphoedemas as well as ensuring referral to specialist lymphoedema therapists to assist in the sequencing and targeting of treatment.

Introduction

One of the biggest surprises for the patient is that the slight swelling that has appeared recently in the leg associated with surgery/vascular ligation/radiotherapy or other tissue trauma failed to resolve and continued to worsen. The reason is because the ability of the lymphatic system to remove fluids and their contents is exceeded by the amount of fluid and materials awaiting removal. The further surprise for the patient is that its unlikely to resolve without some significant attention involving often a plethora of treatment (health professional based) and management (self/partner based) programs. Once clinically manifest at best, the swelling may disappear, but on average it is reduced and at worst, it progresses insidiously. The third big surprise often the surgical/radiotherapeutical oedema resolves for a varying period of time but then returns with vengeance after some stimulus usually associated with some event which has added load to the lymphatic system such as an infection. On the positive side however, is that early treatment when there is early detection of the early stage of the lymphoedema is likely to be relatively effective. (Piller and O’Connor, 2006, reviewed Piller 2006)

However for those without lymphoedema, its prevention by recognition and targeted response to the early pre-clinical signs of its appearance by education and some relatively simple management options are crucial. Phlebologists can play an important role in this process by using simple techniques for the early detection of lymphoedemas (Reviewed Piller, 2007)

What’s most important?

For every person, not matter what the nature of the intervention, there is a risk of overloading a damaged or dysfunctional lymphatic system and of the development of chronic lymphoedema. There is always a delicate balance between making a person’s life miserable through a continual focus on the “at risk” limb, trying to ensure that lymphoedema does not develop and allowing the person to live a normal life and not become fearful or paranoid about possible lymphoedema development. That is why it’s important to gauge who is at high, medium, low and no risk and to inform the patient appropriately.
Incidence of lymphoedema
To develop swelling, there has to be a fairly significant damage to the lymphatic system in the affected area. It is believed that it is working at about 10% of its capacity at rest and that to show signs of failure, about 90% of the transport capacity must have been destroyed (as long as the lymph load is within normal range). Foeldi et al (2003) has divided lymphatic failure into three categories. Mechanical insufficiency, in which there is a normal lymph load on a damaged (due to surgery/radiotherapy) lymphatic system, Dynamic insufficiency in which there is an increased load (due to high vascular permeability or high venous pressures for whatever reason) on a normal system and the very serious problem of an increased load on a damaged lymphatic system.

While figures are possibly a little inaccurate due to variable diagnostic criteria, about 10%-15% of women who have an axillary clearance and mastectomy, develop arm lymphoedema, but when radiotherapy is added it jumps to around 20-30%. Lymphoedema is a problem for men also and this is a very understudied area. When they have had bowel or prostate cancer, melanoma or other lower abdominal cancers requiring the removal of nodes and/or radiotherapy then they too are at a similar risk level. Leg lymphoedemas are generally more of a problem and harder to manage. The same applies to women with cervical and other reproductive system cancers. (Commonwealth Government Medical Services Advisory Committee Report 2004)

Some patients develop lymphoedema subsequent to varicose vein stripping or incompetent vein ligature if large lymph collectors in their adventitial layer are inadvertently damaged others develop it when veins are harvested for cardiac repair work.

What is Lymphoedema?
Lymphoedema, like venous oedemas, is generally a swelling of the compartment above the deep fascia of the musculature. The swelling initially is a protein rich fluid (which attracts further fluids due to the protein’s osmotic action). As time progresses, there is an increase in the density and number of blood vessels, increases in adiposites and changes in the populations of macrophages and fibroblasts and then finally an increasing fibrotic induration. Additionally the higher than normal levels of protein mean a chronic sub-clinical inflammatory process and further swelling in the epifascial tissues. There are also associated problems of limb heaviness, discomfort, tension, aches and pains, loss of normal range of movement and muscle strength, which often are more of an issue for the patient than the swollen limb.

All of the above can be avoided if patients and practitioners are more aware of the risk factors leading to the swelling and its sequelae in the first place. Early recognition of pre-clinical changes in the at risk limb are crucial, especially when we consider the long average latent period prior to the appearance of clinically manifest lymphoedema.

The forms of lymphoedema
About 3-10% of lymphoedemas are primary in nature and are caused by some congenital malformation (generally hypoplasia but sometimes hyperplasia) of the lymphatic system which can become apparent at birth (Nonne Milroy), puberty (Meige or Praecox) or in later life (Tardum). An exploration of family history may expose this underlying lymphatic hypoplasia.

When the swelling is not lymphoedema
When a patient presents with a swollen leg there are often reasons other that a disruption of the lymphatic system in the immediate area, why it might be swollen.

If there is damage or constriction of the lymphatic collectors and or thoracic duct any where along that pathway to its exit point at the subclavian/jugular junction, drainage from distal areas may be compromised. Examples include: radiotherapy to the supra-clavicular area or neck, diaphragmatic hernias, peritonitis (where the resulting adhesions may impede lymphatic drainage), radiotherapy in the abdominal/groin area.
Diet (especially longer chain fatty acids) may result in such large flow from the mesenteric area that lymph flow from the legs is impeded or worse, there is retrograde flow from the mesenteric area into the limbs called chylous reflux. The area of diet and its role in exacerbating lymphoedema needs further research but there are relatively strong links.

When there is excessive accumulation of fluids in the tissues is due to high venous pressure (due to CVI or CCF), hypertension etc may excessively load the lymphatics.

The excessive accumulation of fluids associated with the problems of vascular fragility, phlebitis and often-similar problems with the lymphatic system such as lymphangitis or lymphadenitis can also excessively load the lymphatics.

In the main, irrespective of the reason for changes in the amount of composition of the extracellular fluids the end point is the same, that is, excessive extracellular fluids, changes to their contents (most noticeably an increase in protein and other inflammatory and signaling molecules), changes in the ratios of the cells within it, and changes in their migration rates. As will be obvious, this may be due to an increased load on the lymphatic system (generally related to vascular problems) or a reduced transport capacity of the lymphatic system (generally related to its damage or destruction by surgery or radiotherapy or though its congenital malformation).

**Risk factors**

Some of the observed factors will be within the patient’s control and others outside of it. Those under their control are body mass, skin integrity, and activity levels – (with inactivity being the worst) and constrictive clothing (particularly underwear that has elastic across the line of the groin and bra’s which are under-wired and have narrow straps). In fact, any garment which exerts an external force on the lymphatics should be regarded as a potential risk factor simply because most of the lymph collectors are relatively close to the surface and the pressure of lymph within them generally low. Since intra-lymphatic pressures are far lower than venous ones (in the range 5-15 mm hg) the impact of external local area compression can often have a very significant influence on lymph clearance from an area.

Factors out of the patient’s immediate control are the extent (level) of axillary or groin clearance and radiotherapy to the root of the extremity are certainly well established. Others also outside of their control but less well established are age, whether the intervention was on the dominant arm and to a lesser extent dominant leg, seroma duration, the number or drains and wound infection.

**Signs**

If there is no swelling which can be measured objectively by a tape measure or some other means then its worth testing if any part of the limb (particularly the distal part) shows signs of pitting. This is a sign of local area free fluid accumulation. Pitting may be observable before any circumference change is detected.

There may also be signs of tissue changes associated with the buildup of fatty tissue and fibrotic induration. These changes can often be detected by conducting a pinch and roll test by holding the affected tissues between the thumb and forefinger and gently rolling the tissues between them. Both of these tests are best done in consideration of the major lymphatic territories of the limbs. Even if these two tests show little or are equivocal then it’s often worth asking the presenting patient if they have any subjective changes to the limb ie heaviness, tension, bursting pains and the like as these are signs of impending lymphoedema.

**Assessment of Fluids**

The major early sign (apart from the patient often commenting that the limb feels different) is the appearance of fluids in the limb or a part of it. There are hand held bio-impedance meters which are made in Queensland (Impedimed) which will give an objective measurement of extracellular fluids and
their differences between the limbs to an accuracy of about 5 ml. Ratios of change of impedance are also used in the more basic instruments but all can inform of subtle tissue fluid changes up to 10 months before they can be detected by other detection means. (reviewed Piller 2007)

**Assessment of Fibre**

If you do not wish to use the pinch and roll test in which the suspected tissue is held between the thumb and forefinger and gently rolled another objective means is by using a tissue tonometer (BME, Flinders Medical Center) which can record the resistance to compression to within 1 mm. (reviewed Piller 2007)

**Other Assessment forms**

Often in difficult cases it may be useful to get a larger picture of the problems facing the fluid removal from the affected limb. Among the other useful tools are Ultrasound to indicate the extent and spread of induration and assess changes to the fascias and depths of the compartments, MRI, to assess areas of fluid pooling, lymphoceles and the larger lymph vessels as well as the distribution and spread of adipose tissues which are a characteristic of later stage lymphoedemas.

**Assessment of the Functional Status of the Lymphatic System**

All the signs and symptoms we have discussed about are a consequence of functional changes in the lymphatic system but non of them actually measure it. The only tool in this respect is lymphoscintigraphy which can indicate the residual lymphatic transport capacity. It is able to do this through an examination of the rate of arrival of the injected radio-opaque tracer in the root of the extremity and the rate of clearance from the depot site. It’s also useful for showing areas of dermal back-flow and for often suggesting pathways that remain open which might be focussed on in massage treatment.

For some patients knowing what their risk is of developing lymphoedema can be determined by examining the transport index as indicated by lymphoscintigraphy. If the report shows little or no influence of the surgery/radiotherapy on the lymph transport then the risk of them developing lymphoedema is low and they do not perhaps have to be as concerned as a person in which the transport index is significantly lowered. (reviewed Keeley 2006)

**When lymphoedema is detected or a patient is at high risk**

As a Specialist, often there is little that one can do to become actively involved in the treatments, other than as mentioned before in helping determine if there are other disorders or diseases (especially the vascular ones) which generally contribute to an increased load on the system. The role of the specialist can best relate to continuity of care in terms of a holistic monitoring of the patient’s progress and possibly some staging, direction and coordination of the treatment program.

Those best to refer to are massage therapists, physiotherapists, nurses or occupational therapists who have trained in lymphatic massage.

Lymphatic massage is very different from all other forms of massage, its light, gentle and works by clearing accumulated lymph by helping load it into the lymph capillaries and then along the lymph collectors. Always the proximal parts of the limb are cleared first.

The core treatments and management strategies for lymphoedema revolve around education and awareness which are linked to the major factors which are known to facilitate a good control of the lymphoedema such as improving skin and wound care, reducing excess weight which reduce the load on the remaining lymphatic system, reducing exudation from the vascular system, through the use of sleeves and compression bandages and helping lymph to enter and move along the lymph collectors, through encouraging mild exercise and manual lymphatic drainage. The names of these programs are Complex Physical Therapy, Complex Decongestive Therapy, but the core underlying action is the
massage – called Manual Lymphatic Drainage. There are a number of major lymphatic massage training groups in Australia including the Dr Vodder School which trains internationally.

Treatments

There are a plethora of treatments and many review of their efficacy (Badger, et al 2004). However, not all of which have been well trailed and so it’s hard to make strong recommendations about some of them (Bernas et al 2001). But if it’s kept in mind that the strategies are to either increase the lymphatic transport capacity or reduce the lymph load then reasonable results are likely.

Staging

An accurate and differential diagnosis which will eliminate non-lymphatic disorders/diseases will set the basis for and appropriate staging of the treatment for the lymphoedema.

One of the first aspects of the staging will be to determine if there are any regions of fibrotic induration (due to the surgery, radiotherapy or the progress of lymphoedema), which will not only greatly impair lymph drainage through the area but slow or prevent the regeneration of new lymphatic pathways through those regions. New lymph capillaries do not bud well though any form of fibrous tissue.

If fiber is detected, then an attempt to conservatively lessen the impact of the induration must be made. This will be likely to have a strong effect on improving lymphatic transport capacity. This may range from specific frictional massage to low level scanning or hand held laser treatment.

How do you/your patient know that the treatment is working?

At first the limb may feel better (less heaviness and tension and bursting pain) and may become softer and later it may show a decrease in circumference or volume. There may be improvements in range of movement.

Treatment options

The range is large and often confusing, but perhaps necessarily so. Each patient is different and each lymphoedema may have different factors determining its development and progression. Often EBM findings do not fit into the individual patient treatment picture, but a strategy based on your experience or on the advice of other experts can help provide a balanced treatment pathway.

Antibiotics – useful when an infection is present – and infections are common in lymphoedema patients, as often their specific and general defense systems are compromised in the affected limb.

Benzopyrones (Flavonoids) - experimental and some clinical trial evidence indicates that some members assist in the removal of accumulated protein and fluid while others may slow the leakage from the vascular system or improve lymphatic function. The most common ones are Paroven and Lymphodran although there are certainly others including Bruise and Vein Capsules marketed through a range of CAM groups.

Compression Bandaging - Low stretch bandages are commonly applied between intensive lymphatic massage treatment programs and often worn overnight. Often uncomfortable but they do help reduce exudation of additional fluids from the vascular system. (Badger et al 2004)

Compression Garments – for most patients whose lymphatic transport capacity cannot be improved significantly these are the mainstay of lymphoedema management. These reduce the exudation of fluids from the vascular system. There are two basic groups of garments – made to measure (for unusual shaped limbs) and off the shelf (for the average type of swollen limb). The later are often available from chemists while the former are made on order through garment manufacturers. Often it’s better to refer to a physiotherapist or massage therapist for garment measurement. Subsequent to
fitment, care has to be made to ensure a pressure gradient from distal to proximal and the exerting of an appropriate pressure on the limb. It’s important to watch for distal limb swelling though the wearing of poorly fitted garments. Garments seem to be more effective when combined with mild forms of exercise.

**Compression pumps** – These generally administer variable external pressure supplied by an air pump. Pumps may have between 1 and 12 chambers. Care must be taken if they are to be used since there is a small risk of pushing fluids from the affected limb into its root or worse, into the genital area. Often fibrous bands can develop around the root of the extremity. These may compromise future drainage from the limb.

**Diet** - Long chain triglycerides (Fatty acids) are dependant on the lymphatic system for their absorption from the intestine and thus an attempt to reduce or at least monitor their intake may firstly help reduce lymph load in this area and reduce the risk of the retrograde flow of chyle into the lower limbs producing chyle filled papillomati and reduce the risk of chylous ascites.

**Diuretics** – Often wrongly prescribed for pure lymphoedemas. Do have benefit if there is an underlying oedema however. If a withdrawal of diuretics from a patient with lymphoedema is contemplated then it should be done slowly over time. Diuretics have no effect on the proteins accumulated in the tissues or on lymph flow.

**Elevation**- Valuable at any time, especially in the earlier stage lymphoedemas when the limb is predominantly fluid rich. Works more effectively when combined with exercise and variation in intra-thoracic pressure associated with deeper than normal breathing. This pressure variation seems essential to facilitate optimal clearance of lymph from the capacitance vessels in the abdominal and thoracic areas thus establishing a better pressure gradient to the limbs (particularly the legs).

**Exercise** – Whatever the form, can be very beneficial. Exercise will generally help vary the tissue pressures and so help fluids and their contents to enter the lymph capillaries and to move along them into the lymph collectors which normally pulsate at 6-10 beats per minute. Clearing materials from the interstitial spaces effectively is crucial in all lymphoedemas (and oedemas) so that optimal oxygen levels and effective waste product clearance are achieved.

Exercises do however need to be planned and it’s very important for the patient to warm up and cool down appropriately. Some exercise programs have been clinically trialled and most convey some benefit and certainly none have been shown to cause harm. Often the question is asked – how much exercise? Well there is no answer – some women with lymphoedema play A grade tennis, others participate in dragon boat races. The important point is to warm down slowly and in some instances to wear a support sleeve or stocking if the lymphatics are unable to handle the additional lymph load. Recent studies have shown that even strenuous exercise does not always worsen lymphoedemas. (Johansson and Piller 2006)

One exercise program which has been shown to be beneficial is that of a combination of a Tai Chi and Qi Gong like regimen. These have been shown to be particularly beneficial for upper limb lymphoedemas. One trial has shown that the combined variation in tissue pressure brought about by variation in muscular tone and the establishment of a pressure gradient to the major lymph ducts though increased depth of respiration significantly reduces arm lymphoedema. (Moseley, Piller and Carati, 2005)

**Exercise facilitating machines** – For some patients who do not wish to undertake a formal group exercise program there are a range of home user friendly exercise machines. Few have been clinically tested, although one trial was able to show a machine which helped change tissue pressures by passive movement to be of significant benefit for those with lower limb lymphoedema. (Moseley et al 2002)
Hydrotherapy – This water based exercise program is useful especially for lower limb lymphoedemas although the YMCA based Encore program for women who have had breast surgery is very beneficial and available Australia wide. The gentle supported movement and external pressure gradient from the surrounding water assist lymph flow to the root of the extremity. Hydrotherapy programs are also beneficial to improve range of movement and limb mobility. (Box et al 2004)

Low Level Laser – There is considerable experimental and some clinical evidence in the form of a double blind cross over trial (which tested the Rian Corp Hand held laser), that low level laser treatment is useful in reducing lymphoedemas. The exact mode of action is under investigation but some known effects include stimulatory action on macrophages, antibacterial effects and a tendency to make lymph collectors pump faster. It is hypothesized that laser also helps break up collagen’s tissues allowing better flow through of fluids and materials through the tissues thus improving lymph drainage.(Carati et al, 2003,

Massage – The arms and legs and body superficially are divided up into a number of lymph territories. Specialized forms of lymphatic massage have been developed to help move lymph from territories which are blocked to those which have still a patent system. The major type of lymphatic massage is called Manual Lymphatic Drainage. Its often combined with other treatments listed here such as bandaging and skin care and exercise programs where it is called complex lymphatic therapy or complex decongestive therapy or one of a multitude of other names. Lymphatic massage is gentle and light. Often the massage is also combined with essential oils such as lavender which has evidenced antibacterial effects. Tea tree oil has also similar properties but trials on lymphoedema are still underway.

Massage pads and massage tools – For the patient who cannot afford the necessary professional massage or for times between professional massage, often the alternate is a home based equivalent. There are a variety of massage pads and aids not all of which have been clinically tested but anecdotally most seem to have some benefit and none do harm. A clinical trial on a Niagara pad has shown it to be beneficial in chronic secondary leg lymphoedemas.

Electrical stimulation of lymphatic and skeletal smooth musculature – This recent innovation exploits the fact that there are two components (myogenic and neurogenic) to the determination of lymph flow. Electrical stimulation of the smooth musculature of the lymphangions encourages more forceful (and regular) contraction and thus is believed to improve lymph flow. A recent clinical trial at Flinders Medical Centre has shown this treatment to be beneficial for patients with secondary leg lymphoedemas and will be discussed in more detail at this congress. (Piller et al 2008 in press)

Skin care –One of the significant leverage points in the treatment of lymphoedema is to reduce the load on the compromised lymphatic system. Improving the quality of the skin as a barrier through improved skin care by whatever appropriate means is crucial. A commonly used beneficial cream is Sorbolene although any with aloe vera and vitamin E are often as effective. Care must be taken as many creams significantly reduce the life of support garments.

Other treatments - There are a plethora of other treatments and just because they are not presented here does not mean they do not work. Most are conservative and many have not been clinically trialled but have strong anecdotal evidence of their effectiveness. When conservative treatment fails there are a range of surgical techniques, including, microsurgery, excisional operations and various forms of liposuction. Recent developments in the later techniques which avoid damaging further the lymphatic system collectors have shown promising results (Brorson 2005).

If there are problems finding the right therapists then a call to the local patient support group in your state may help as might a check with web sites of the Lymphoedema Association of Australia, the Australian Lymphology Association, the Dr Vodder School web site or your nearby hospital which may have a lymphoedema clinic.
Gaining a good outcome for your patient

Review the patient’s prior surgical, medical and family history for factors which may have impacted on lymphatic transport

For Those with Clinically Manifest Lymphoedema

Exclude or include other reasons for the apparent changes in the limbs
Measure the limb - how big and different is it?
Encourage self-measurement of the limb
Ask for comments about the limb - how different does it feel?
Separate factors that are a consequence of the surgery and radiotherapy from those due to lymphoedema development or its progression
Treat (or refer for treatment) non-lymphatic related reasons for limb swelling
Indicate the range of treatments for their lymphatic issues
Refer to experts for further lymphoedema assessment and treatment
Be aware that Lymphoedema massage techniques are very different from normal massage
Review at 6 to 12 month intervals – be alert for events which may have changed load on or transport capabilities of the lymph system

For those at risk of lymphoedema

Assess and review risk factors – particularly those which may increase lymphatic load
Measure the limb – this may be a useful future baseline measurement
Encourage self-measurement of limb – perhaps monthly
Encourage attendance at Lions free screening programs
Provide educational material relevant to risk reduction management
Reduce the impact of any factors which may be increasing lymphatic load
Encourage early reaction to the perception of any changes in size or how the limb feels
Encourage review at intervals between 6 and 12 months with a higher frequency for higher risk category patients

Major factors affecting lymph load and transport

Factors which may increase lymph load

- Elevated Blood pressure
- Inflammatory events
- Poorly functioning vascular system especially on the venous side
- Being obese or overweight
- Poor skin care
- Over activity, especially repetitive actions
- Infections which are not treated
- A diet rich in long chain triglycerides (Fatty Acids)
- Sunburn
- Becoming overheated

Factors which may reduce lymphatic transport

- Being obese or overweight
- Being immobile
- Constrictive clothing or undergarments (especially in the groin or axillary areas)
- Chronic inflammatory disorders and skin conditions
Factors which may increase lymph transport capacity

- Gentle or moderate exercise – with warm down period
- Occasional deeper respiration than normal
- Water based exercise programs
- T’ai Chi and Qi Gong type of exercise programs

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Other useful information


Journal of Lymphoedema www.journaloflymphoedema.com
Lymphoedema is a life-long disease which may be improved but not healed by our therapy. Therefore it seems to be desirable to ascertain the clinical suspicion of lymphoedema or to rule out this diagnosis at least once in the life of a patient suffering from chronic swelling of the extremities. The most appropriate methods for that purpose are lymphoscintigraphy and indirect lymphography. Radionuclide lymphography and indirect lymphography are based on a similar principle: lymphatic drainage is assessed after „indirect“ application of a tracer into the tissue, in contrary to the „direct“ injection of contrast-material performed in conventional lymphangiography. Labelled compounds are used for scintigraphy, and newly developed, water-soluble contrast media for indirect lymphography. While radionuclide lymphography has become the most important diagnostic tool for diagnosing compromised lymph-drainage, indirect lymphography has been established only in few centres, mainly for scientific reasons.

Scintigraphy and radionuclide lymphography

The essential function of the lymphatics to clear the interstitium from large molecules can be tested by introducing labelled proteins or colloids into the tissue. Scintigraphy by gamma camera allows the assessment of the distribution of the tracer in the leg and its storage in the lymph nodes. Using new high-resolution techniques ("lymphangioscintigraphy LAS") images of lymph nodes and also of lymphatics may be obtained. The function of the lymphatic drainage can be assessed by comparing the uptake of radioactivity in the lymph nodes with the injected dose. Nodal scintigraphy has gained some new importance for the assessment of sentinel lymph nodes in different areas of cancer-surgery.

Labelled tracers

It has been suggested that the optimal particle size for a lymphatic tracer is in the range of 10 nm. After injection into the tissue these particles enter the initial lymphatics, some part being incorporated into macrophages, and are transported towards the lymph nodes.

Commonly used compounds are antimony sulphide colloid, rhenium sulphate, human serum albumin nanocolloid and DTPA serum albumin, all labelled with technetium (99m Tc).Labelled gold colloids are not used anymore.

Injection

Injection volume should be small (0.1-0.2 ml) and should have high specific activity. For routine diagnosis of lymphoedema subcutaneous injections are recommended into the web space between the first and second finger of the hand and between first and second toe of the foot. Several subcutaneous injections may improve diagnostic accuracy.

For special information injections into the area of interest, e.g. around leg ulcers, scrotum etc can be performed.

Subfascial lymph transport can be assessed after intramuscular injection of the tracer into the distal third of the calf.

Stress-tests

In order to enhance lymph -transport active movement, e.g. exercise with a foot ergometer or walking on a treadmill is recommended. This is essential if the assessment of the lymphatic drainage function is mainly based on the amount of radioactivity recovered in the regional lymph nodes. It has been shown that sensitivity and specificity of static images obtained one hour after injection are increased by muscular exercise. However, some authors prefer to assess static images taken in different time-intervals after injection without any stress test.
Measurement of radioactivity
Different kinds of techniques have been described:

The registration of time-activity curves over the injection site reflects the local clearance. It has been shown that the correlation between the clearance of the tracer from the depot and its uptake in the lymph nodes is very poor. However, this parameter has been used especially to demonstrate therapeutic effects.

Pictures taken from the whole extremity by a gamma camera and whole body images may show pathological distribution of the injected substance due to dermal back-flow or a block of lymph collectors.

The uptake of radioactivity in the lymph nodes is the most reliable single parameter for assessing the lymph-transport. It can be documented by simple scintigraphy, measured with a scintillation probe or a gamma camera, or using a combined transmission- emission-procedure in order to correct for different depth of the inguinal and iliac lymph nodes. By comparing the injected dose with the corrected lymph node activity quantitative information can be given. The appearance-time after the injection of the tracer alone, which should be shorter than 40 minutes under normal conditions, is an unreliable parameter.

Semiquantitative scoring systems considering different parameters of tracer kinetics and calculating a transport index have been proposed. For routine diagnosis visual interpretation of images may be sufficient in the majority of cases.

Results
Based on visual interpretation the sensitivity of isotope lymphography in diagnosing lymphoedema was reported to be 97% and its specificity 100%.

Intracutaneous tracer-injection is not able to separate lymphoedema from non-lymphoedema. The normal storage values after intracutaneous tracer-application in patients with lymphoedema, even caused by metastases in the lymph nodes, demonstrate the fact that intracutaneous networks of initial lymphatics may act as an effective collateral pathway. Therefore intracutaneous application of the tracer is useful to detect sentinel lymph nodes or to visualize pathological skin-lymphatics filled by dermal backflow as it is intended by the LAS-technique. When quantitative measurement of the lymph-node activity is taken as the deciding parameter subcutaneous tracer administration is to be preferred. Using quantitative lymphoscintigraphy we were able to diagnose subclinical forms of lymphatic damage e.g. on the normal appearing contralateral leg of patients with hereditary lymphoedema.

It has been shown that lymphatic function declines with age, markedly so after the age of 65 years. Table I summarizes the results of lymphoscintigraphic studies in several pathologic conditions.

Table I: Lymph-drainage in different conditions. Summarized findings reported in the literature (DVT = deep vein thrombosis, PTS = postthrombotic syndrome). ↓=decreased, ↑=increased

<table>
<thead>
<tr>
<th>Lymph transport</th>
<th>Praefascial</th>
<th>Subfascial</th>
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<tr>
<td>Lymphoedema</td>
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<td>Venous oedema</td>
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<td>DVT</td>
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<td>Leg-ulcers</td>
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<td>Lipoedema</td>
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**Practical consequences**

Today isotope lymphography is the most appropriate method to ascertain the clinical suspicion of lymphoedema. This method is also important to rule out lymphatic involvement in different forms of swollen extremities with unknown origin. It is easy to perform and not invasive. However, its routine use is mostly restricted to nuclear medical departments with clinical partners in the background who are interested in lymphology and who admit their patients to scintigraphic investigations.

Unfortunately up to now no broadly accepted consensus has been reached concerning a standardized protocol for lymphoscintigraphy. It has to be underlined that “normal findings” very much depend on methodological details and are not always able to rule out lymphoedema.

**Indirect lymphography**

Skin lymphatics may be opacified by intradermal injection of newly developed contrast media. This method gives a valuable picture of the local dermal lymphatics in a region of interest and may provide important clues about the lymphatic drainage of the limb as a whole. However, lymph nodes are opacified only rarely so that this method cannot substitute conventional, direct lymphography.

**Method**

Non-ionic, water soluble, dimeric, hexaiodinated contrast media such as Iotasul or Iotrolan are constantly infused subepidermally by a motor pump (0.12 ml/min) using thin, butterfly needles with a total amount of 2-4 ml per injection site. The tip of the needle has to be situated in the uppermost part of the dermis so that a bluish, glassy-appearing wheal will develop. Under normal conditions, lymphatics begin to fill after a few minutes that can be observed by an image converter and documented on mammography films at 5-minute intervals. Xeroradiography may be a valuable alternative; computer-tomography with three-dimensional reconstruction gives impressive pictures.

**Indications**

This method may be helpful regarding a differentiation of various forms of lymphoedema like in hypoplasia of distal lymphatics versus proximal hypoplasia with distal distension. It has a high sensitivity for diagnosing lymphoedema (97%) with a moderate specificity (89%). Pathological changes of skin lymphatics can be demonstrated in areas of lipodermatosclerosis due to chronic venous insufficiency and in the vicinity of leg-ulcers. Localized lymphoedema as e.g. after trauma, inflammation or in morbidly obese patients is characterized by pathological initial lymphatics in certain skin areas while large collectors may appear normal.

Patients with lipoedema show a typical flame-like pattern of the dye distribution, which is one of the very few pathognomonic features of this entity.

Fascinating results may be obtained after replantation-surgery.

**Results**

The infused contrast agent forms growing depots, which taper into peripheral lymph-collectors. Under normal conditions these collectors can be followed for an average length of 10-30 cm. Retrograde flow may fill small skin lymphatics as a sign of dermal backflow due to incompetence of lymphatic valves.

In principle, four different patterns have been described in lymphoedematous skin:

Type I: No lymphatics are opacified. Instead, contrast may spread in a cuff-like pattern obviously marking the adventitial space of blood vessels. This feature is found especially in congenital lymphoedema and fits well with findings obtained by microlymphangiography.

Type II: A dense network of small skin lymphatics corresponding to precollectors, but only sparse lymph collectors are opacified. This pattern is found mainly in patients with peripheral lymphoedema praecox and tardum.

Type III: Precollector skin lymphatics and lymph collectors are both enlarged, possibly due to proximal obstruction. Most patients with descending form of lymphoedema due to primary proximal
obstruction and with involvement of the entire limb tend to show this constellation. This pattern is also seen after infusion of the dye into skin-areas with papillomatosis cutis.

Type IV: Neither precollectors nor lymph collectors can be seen. This pattern may be explained by a „die-back“-mechanism of lymphatics in the presence of proximal abnormalities. However, also technical problems in patients with extremely dense and thick skin can also yield a similar picture. In patients with chronic venous insufficiency morphological changes of dermal lymphatics may also be demonstrated in pathological skin regions. Precollectors are fragmented, filled by dermal backflow and show extravasations of the dye into the tissue. Prefascial collectors are enlarged and show often increased contractility. Even lymph nodes in the groin may be opacified. These features of wide and well-filled lymph-collectors correlate with an increased praefascial lymph transport demonstrated by radionuclide lymphography.

Injecting the dye into the border of venous ulcers can fill no lymphatics.

Lipoedema, which is often confused and sometimes combined with lymphoedema, shows a very characteristic and quite specific pattern.. The depot of the dye injected into the oedematous parts of the lower leg forms a typically flame-like structure.

Practical consequences

Indirect lymphography is a kind of radiological patent blue-test, but with a much better resolution. Exact injection technique is a prerequisite for adequate results. The value of this method is rather restricted to scientific than to practical information.

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Background
Chronic pelvic pain, defined as non-cyclic pelvic pain of greater than 6 months duration, is a common presenting problem to the Gynaecologist. A third of all patients worked up for chronic pelvic pain with laparoscopy, have no obvious etiology. Pelvic congestion syndrome (PCS) has long been recognized as a cause of chronic pelvic pain, caused by retrograde flow down incompetent gonadal veins resulting in pelvic varicosities. This is anatomically analogous to the male varicocele, but because the pelvic varicosities are not externally visible or palpable, the diagnosis is most often elusive. The treatment of choice is the same as for a male varicocele, transcatheter gonadal vein embolization.

Clinical Diagnosis
The symptom complex can be best understood as the result of gravity related filling of the pelvic varicosities. The classic and almost pathognomonic presentation includes varying degrees of pelvic and lower back pain that is worsened with standing and exercising, and is therefore most severe at the end of the day. It is also often exacerbated with intercourse. Patients who usually describe the pain to be diminished or relieved in the supine position have the most relief upon awakening in the morning. The above presenting complaints are predictive of clinical success after transcatheter embolization of the varices and gonadal veins.

The visualization of incompetent gonadal veins and associated pelvic varices has been difficult without performing invasive gonadal vein venography to demonstrate spontaneous reflux. Clinical pelvic examination is insensitive to recognise pelvic varicosities, unlike in the male of an enlarged scrotum with palpable varices.

Transvaginal color Doppler ultrasound performed in a supine and upright positions with and without Valsalva is the best screening modality. This often reveals an increase in pelvic venous channels and provides confirmation that the pelvic varices are affected by gravity-dependent filling. Laparoscopy is usually unrewarding in making the observation of pelvic varices due to the compression of the varices from the peritoneal CO₂ insufflation and the resultant draining of the varices while the patient is in a Trendelenburg position. There has therefore been no satisfactory accurate non-invasive modality up until recently to confirm the clinical suspicion without performing venography.

Pelvic MRI and gadolinium enhanced 3D magnetic resonance venography (MRV) is almost 100% sensitive in detecting large gonadal veins and associated varices. The MRV combined with pelvic MRI allows us to exclude other common causes of pelvic pain such as fibroids, endometriosis, adenomyosis, ovarian masses and lower lumbar intervertebral disc abnormalities. It is thus an excellent modality for the workup of chronic pelvic pain. The anatomic information garnered from the MRV also provides an excellent roadmap for the Interventional Radiologist prior to venography and embolisation.

Who Should Be Involved With The Decision To Treat?
With the ability to screen for the syndrome with an accurate non-invasive test using Doppler Ultrasound patients may present to General Practitioners, Phlebologists, Surgeons and Gynaecologist alike. It is important to state on the request form that the suspected clinical diagnosis is pelvic varices and gonadal vein reflux. It is our recommendation that these patients are worked up in partnership with a Gynaecologist who understands and appreciates the syndrome. It is imperative that the patients have a complete pelvic examination and, if warranted, a laparoscopy prior to embolisation of the varices by the Interventional Radiologist. The Interventional Radiologist will only proceed with transcatheter embolisation when there is consensus between the patient and physicians involved. This team approach engenders trust and comfort for the patient but due to the protean causes of chronic pelvic pain, consultation with an Urologist, Neurologist, Gastroenterologist, Orthopedic Surgeon, Physiotherapist and Psychiatrist is sometimes
necessary.

**Pre-Procedure Patient Preparation**

The procedure is performed on an outpatient basis but the patient is admitted into the day stay unit. Informed consent is obtained. It is preferred to perform the procedure after menses and prior to ovulation. A Beta-HCG should be performed if there is any chance of pregnancy prior to the procedure. A peripheral IV is started. It is our practice to administer 1g of Cephazolin or if allergic, 80mg of Gentamycin prior to initiating the puncture.

**Anatomy of the Gonadal Veins**

The left ovarian vein usually drains into the left renal vein at a 90° angle. The right gonadal vein empties most often into the IVC at a 45°. Both ovarian veins usually have multiple tributaries, which are of significance when embolising as there may be communication with the pelvic venous plexus and the internal iliac veins. Rarely is communication seen with inferior mesenteric veins.

**Treatment Of The Incompetent Gonadal Veins And Pelvic Varices**

Transcatheter embolisation of varicoceles in males is a well established technique. Given the identical anatomic and physiologic setting in the female with PCS, similar therapeutic techniques are embraced for managing the female pelvic varices.

Access to the gonadal veins is traditionally gained via the transjugular or transfemoral route. Access to the right gonadal vein is more challenging via the transfemoral route.

After access is obtained a left renal venogram is performed with the patient in a 10-30 degree upright position on a tilt table. If there is no spontaneous reflux down the left renal vein, embolisation is not pursued. If spontaneous reflux down the left renal vein is present, in the appropriate clinical setting, cannulation of the left gonadal vein is performed. A venogram of the left gonadal vein is then performed to visualise all the tributaries/collaterals of the main gonadal vein. These are critical to map out and embolise for a successful long-term clinical result. The catheter is then advanced to the level of the pelvic varicosities and a hand injection of the pelvic varices is then performed to assess rate of flow and pathways of outflow from the varices. Cross filling to the contralateral side, and or outflow via the internal iliac veins and into the common iliac vein and IVC is critical to evaluate prior to initiating the embolisation especially if sclerosing agents are used.

**Where To Begin & End the Gonadal Vein Embolisation, & Which Embolic Agents To Utilize?**

The goal of the embolisation is to terminate the pressure head within the gonadal vein that is transmitted to the pelvic veins and varicosities by occluding the gonadal vein and all its tributaries. Most veins are occluded by using embolisation coils alone or in combination with sclerosing agents.

Sclerosants are not uniformly employed for the procedure. 2cc of 3% Sotradecol mixed with 0.5cc of contrast in a 3cc syringe is gently administered by hand under continual fluoroscopic vision into the distal gonadal veins and varices to prevent inadvertent reflux or flow into the systemic venous system. The catheter is then retracted and the main gonadal vein and each of its tributaries is embolised using stainless steel coils ranging from 5-12mm in diameter to the level of approximately 3-5cm from the left renal vein. The gonadal vein embolisation is then followed by another left renal venogram from the renal hilum to confirm occlusion of the gonadal vein as evidenced by no spontaneous reflux.

Attention is then turned to the right gonadal vein that usually presents more of a challenge to cannulate and is situated just below the right renal vein. Embolization is then carried out in the same fashion as on the left. The catheter is then used to select both internal iliac veins to confirm that no further large tributaries or varices are being fed via reflux from the pudendal veins. The
sheath and catheters are then removed and the patient is transferred to the same day unit for routine observation for two hours.

**Post Procedure Care and Follow-up**

In our experience, the patients are usually pain free for the first 24 hours after the procedure. Almost all patients however, experience an onset of moderate to severe pain after 24-48 hours that crescendo's over 24 hours before subsiding over 2-3 days. The pain is usually controlled with Panadiine Forte and Voltaren, the pain often mimics the initial presenting pelvic pain in location and nature. Some patients develop a low-grade temp of 38°C which is usually controlled with Panadol. Patients are scheduled for a post procedure baseline transvaginal color doppler ultrasound to confirm thrombosis of the pelvic varices within 2 weeks of the procedure. This is then followed by a 1 month, 6 month and 1 year office visit and ultrasound to confirm continued clinical success.

**Clinical Results After Transcatheter Gonadal Vein Embolization**

A few studies have attempted to address this issue. These have demonstrated clinical effectiveness ranging from 58-78% Most patients experienced initial pain relief but some appeared to recur. A likely cause for failure is incomplete embolisation of the varices and tributaries of the gonadal vein. Over time the pressure head is recreated through the remaining tributaries. It is our experience that those patients who present with atypical symptoms, are those that tend to have partial relief of their pain post embolisation and are the subset of patients that appear to have the most recurrences. This underscores the importance of appropriate patient selection. In all cases that recur, it is imperative to perform repeat gonadal vein venograms together with interrogation of the internal iliac veins, to exclude possible recanalisation of gonadal veins and tributaries with reformation of pelvic varices.

**References**

5) Hobbs JT. The pelvic congestion syndrome. Pracitioner 1976; 41:41-46
7) Venbrux AC, Lambert DL. Embolization Of The Ovarian Veins As The Treatment For Patients With Chronic Pelvic Pain Caused By Pelvic Venous Incompetence (Pelvic Congestion Syndrome) Curr Opin Obstet Gynecol 1999 11:395-399
The aim of surgery for varicose veins is three-fold - the surgeon aims to abolish superficial reflux, minimise its chances for recurrence, and achieve this result in as cosmetic a fashion as possible, while aiming to avoid complications. To achieve these aims, all patients should be properly assessed by Duplex ultrasonography, performed by a sonographer experienced in the examination of venous reflux, and then have their operation individualised to their pattern of disease.

Saphenofemoral reflux is treated surgically by saphenofemoral ligation, aiming to divide second order branches in the groin to minimise recurrence. The great saphenous vein is stripped to the knee from above downwards via an inversion technique. Most reflux below the knee is via the posterior arch vein, and therefore stripping the GSV to this level has little effect on reflux, and inversion stripping to the knee minimises the risk of injury to the saphenous nerve. Experience has shown that if the GSV is not stripped it tends to increase the risk of recurrence; aiming to save the vein to use as a conduit for bypass surgery is usually unsuccessful in this scenario anyway. External banded valvuloplasty (Venocuff) is a surgical alternative for superficial reflux.

Reflux in the small saphenous vein may be treated satisfactorily by saphenopopliteal ligation. The value of stripping in this instance is less clear. The location of the junction must always be marked prior to operation using duplex scanning as the junctional anatomy is so variable. The sural nerve should be sought and preserved at surgery. A thorough knowledge of the anatomy of the popliteal fossa is essential in carrying out this surgery.

Treatment of perforator reflux via a surgical approach once again requires preoperative marking to localise incisions and minimise the extent of dissection required. Extensive operative approaches such as the Linton procedure involving large incisions through diseased skin are rarely carried out now. Other approaches have been sought to deal with this difficult problem, such as subfascial endoscopic perforator surgery (SEPS), to allow access to divide perforators without incisions in skin which is either ulcerated or recently healed.

The advent of less invasive procedures to deal with superficial venous reflux has led to a radical change in the treatment of patients with varicose veins. However, well performed surgery still has a place in the list of options to consider for these patients.
Ultrasound guided sclerotherapy was developed in France, Australia, the USA and Canada in the late 1980s. With the application of B mode ultrasound imaging, incompetent truncal veins could be seen and accessed which enabled the source of venous reflux to be treated. This offered a viable non-surgical alternative to surgery for large varicose veins.

Veins up to approximately 5mm in diameter (when the patient is standing) respond well to this relatively simple, non-invasive treatment. Larger veins (no size limit for many practitioners) have also been successfully treated, however a relatively high recanalization rate necessitates multiple treatments over a year or more to achieve total fibrosis that can now be obtained from a single session of endovenous laser ablation.

Following patient assessment and mapping of the superficial veins of the lower limb, the procedure is performed in the phlebologist's rooms. No anaesthetic is required and the patient is able to drive home afterwards. There are no scars, no time is lost from work and the patient can continue with normal daily activities.

Serious complications are a rare making UGS a simple, safe and effective treatment for truncal and branch varicosities. It has a high degree of patient satisfaction and acceptance.
New Advancement in Endovenous Radiofrequency Ablation

A/Prof Lourens Bester, St. Vincent’s Hospital, Sydney

Introduction:

Endovenous radiofrequency ablation (RFA) was introduced into clinical practice in 1998 in Europe and 1999 in the US. Since then, over 150,000 cases have been performed worldwide. The clinical benefit of RFA over traditional vein stripping surgery was demonstrated in four randomized trials. In these trials, patients who received RFA exhibited less post-operative pain and complications, quicker recovery, and better quality of life. The advantage of RFA versus endovenous laser (EVL) treatment is that the RFA patients experience less pain and have a better recovery profile.

Recently, a new generation of RFA catheter called ClosureFAST catheter was released to the market. It addressed the disadvantage of procedure speed and ease of use issues associated with the previous generation of RFA catheters. It uses a segmental ablation technique as opposed to the continuous pullback technique used by previous generations of RFA and EVL procedures. In addition to significantly improved ease of use, this eliminates the pullback speed variable and ensures consistent thermal dose and thus consistent treatment along the entire vein segment. Proebstle et al reported on 252 limbs in 194 patients treated with this catheter. Vein occlusion rate was 100% at 3 months (164 limbs) and 6 months (62 limbs). Over 70% of patients experienced no pain after the procedure, measured by a pain analogue scale. Average energy delivery time was 2.2 minutes and the average procedure time (from catheter insertion to catheter removal) was 16.4 minutes.

Patient Selection:

The indications for ablation of incompetent truncal veins are identical to those for surgical ligation and stripping.

Contraindications:

Contraindications for RFA:

1. pregnancy
2. inability to wear compression stockings secondary to inadequate circulation
3. contraindications to local anaesthetic use
4. uncorrectable coagulopathy or hypercoagulability
5. pacemaker (only for RFA)
6. inability to adequately ambulate post-procedure
7. treatment of competent vein segments below an incompetent vein segment, such as below the knee.

Technique Overview:

1. Mapping of the vein segment to be treated, tortuous segments, perforating veins and large tributaries.

2. US guided venous access into the lowest segment of the incompetent vein followed by positioning the catheter one to two centimetres below the junction of the Epigastric vein and GSVJ.

3. US guided perivenous tumescent anaesthesia. The purpose of the tumescent fluid is to empty the vein and to improve thermal transfer to the vein wall, to separate the vein from surrounding
structures to prevent thermal injury as well as for its anaesthetic effect.

4. Place patient in Trendelenberg positioning to empty vein.

5. Thermal energy is then delivered using protocols inherent to each device.

6. Manual compression is applied to the vein while energy is delivered.

7. After the procedure the patient is placed in a compression stocking (class 2) and ambulation is immediately initiated.

Follow-up:

Following RFA, patients return for clinical and DUS evaluation after one week, then six weeks later to confirm vein closure and exclude complications. Longterm follow-up with DUS should be performed for up to one year or until the treated vein is no longer visible, whichever comes first.

Success Rates:

Technical:

Technical success is defined as the ability to perform the procedure and to permanently occlude the target vein.

Certain clinical situations may predispose to lower success rates. These include morbid obesity and concurrent heparin anticoagulation.

Recurrence:

Recurrence is defined as recanalisation of a previously occluded vein segment. Most of these recanalisations occur less than 1-year following treatment. Recanalisation of < 5cm long previously closed vein segments occurs about 1-4% and recanalisations of > 5cm of previously closed segments in less than 1%.

Late clinical recurrences are more likely related to development of incompetence in previously untreated vein segments

Complications:

Adverse events following RFA are unusual. Bruising over the treated segment may occur and is most likely related to the liberal use of local anaesthetic injected along the course of the treated vein.

One week after treatment the vein will begin to cicatrize circumferentially and longitudinally and this may lead to the feeling of tightness similar to that after a strained muscle. This transient discomfort is self-limited and may be ameliorated with the use of non-steroidal anti-inflammatory drugs and graduated compression stockings. These are more commonly reported using existing laser protocols than for RFA.

Parasthesias/nerve injury, skin burns, superficial phlebitis and DVT have also been reported. The nerves at highest risk include the saphenous nerve, adjacent to the GSV below Boyd's perforating vein, and the sural nerve adjacent to the SSV in the mid and lower calf. Both of these nerves have
only sensory components. The most common manifestation of a nerve injury is a parasthesia, most of which is transient.

There is also a higher rate of nerve injury when treating the below-knee GSV as compared to the above-knee segment.

Skin burns have been reported in 2% of cases following RFA and laser. The use of tumescent anaesthesia is essential to minimize this complication.

DVT following RFA is unusual. Pooling data from several series suggests the incidence is, at most, approximately 1-2%. DVT after RFA is usually an extension of clot from the ablated vein across the junction into the deep vein and, less often, in more peripheral deep veins. Indistinct DVT after classical surgery is in the more peripheral deep veins. Starting treatment 1-2 cm below the Epigastric vein and early ambulation and compression should keep this complication to a minimum.

Comparisons with Surgery:

Two small comparisons between RFA and high ligation with inversion phlebectomy for the GSV have been performed. In these studies, advantages for RFA were seen in terms of recovery time, fewer missed days of work and patient satisfaction with equivalent clinical outcomes.
References:


4. Stoetter L, Schaaf I, Bockelbrink A. Invaginating stripping, kryostripping or endoluminal radiofrequency obliteration to treat GSV insufficiency: duplex ultrasound findings and clinical outcome postoperatively and at 1-year follow up. 17th annual meeting of American Venous Forum. San Diego, Feb, 2005


A review of approximately 380 veins personally treated by endovenous laser ablation (EVLA) over the past 4 1/2 years shows in excess of 85% primary success and 90% secondary success at three years determined by ultrasound surveillance. Recurrence was almost invariably by recanalisation of the proximal treated saphenous vein and was easily treated by ultrasound-guided sclerotherapy. Secondary success rates could have been higher but some patients elected not to have further treatment because there was no clinical recurrence. There were very few limbs in which reconnections were seen from the common femoral vein or abdominal / pelvic tributaries into thigh varices. These findings markedly differ from recurrence patterns after surgery. Success rates are higher for EVLA than for UGS or surgery in my practice although the studied series were not concurrent or randomized. Venous thromboembolic complications were seen in less than 2% of limbs as detected by routine ultrasound review at 3 -4 days after every procedure. All deep venous occlusions were asymptomatic, all above-knee lesions caused only partial obstruction and all rapidly resolved with short-term anticoagulation shown by serial ultrasound examinations. There was one symptomatic pulmonary embolus that resolved with anticoagulation. No systemic neurological events were observed apart from visual aura in patients with past migraine after secondary sclerotherapy. Two of 90 limbs treated for small saphenous reflux developed numbness in the sural nerve distribution that resolved within 6 months. The incidence of these complications is probably less than described by others after surgery.
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Introduction
Venous ulceration in the gaiter area of legs occurs as a consequence of unabated, persistent chronic venous insufficiency. This is due to valvular deficiency of superficial, perforator or deep veins alone or in combination. Most venous ulcers heal rapidly after superficial vein surgery if the deep venous system is not involved. However, the results are not good when deep veins are involved. Treatment options to correct deep venous insufficiency, then, have to be looked at.

There is evidence that surgical treatment of deep vein valvular reflux leading to severe chronic venous insufficiency provides long-term relief of symptoms and heals venous leg ulcers in 65 - 80% of patients at 5 years post-operation. Venous valve reconstruction for chronic venous insufficiency was introduced by Kistner as early as in 1968. However, deep venous valvular reconstructions have not become popular and maintain an aura of controversy due to a lack of comparative studies between conservative and surgical therapy. Furthermore, previous studies have included patients with valvular surgery performed with additional superficial and perforator vein surgery making it difficult to assess whether the benefits of such therapies were due to valve repairs or superficial/perforator surgery.

This study was undertaken to further justify the role of deep venous valvular reconstructions in chronic venous insufficiency, in patients who had recalcitrant non-healing leg ulcer as a ‘last resort treatment’, despite multiple superficial/perforator vein surgeries, compression therapy and medical management.

MATERIAL & METHODS:
Between October 1994 and November 1999, of 162 patients who underwent deep vein reconstructions in our department, 137 patients (169 limbs) were included in this prospective study. The mean age of this patient group was 38.7 years (range 17-75 yrs) with a male: female ratio of 2.18:1. In the 169 limbs, there were a total of 411 previous superficial/perforator surgeries performed in the past. 24 Limbs had single surgery performed, 83 limbs had 2 surgeries and 62 limbs had 3 or more surgeries performed before presenting as recalcitrant, recurrent venous ulceration. Majority of the procedures 284/411 (69%) were superficial vein + perforator surgeries in combination, Perforator surgery alone was for 96/411 (23.35%) and superficial surgery alone included 31/411 (7.54%).

Diagnosis of deep vein pathology was ascertained by ascending and descending venography and with colour duplex Doppler assessment in all patients.

Criteria for inclusion in this study was as follows:

1). Patients with CEAP C6 Ulceration of Leg greater than or equal to 3 cms. diameter.

2). Evidence of severe deep venous reflux - Grade III/IV reflux on descending venogram and Valve Closure Time (VCT) > 3 secs. by standing Duplex scan with patient performing Valsalva manoeuvre.
3). Failure of Conservative Therapy for more than 3 months – with Class II/III compression stockings + Daflon

4). Previous Superficial or perforator vein surgery(ies) with no current duplex recorded superficial or perforator vein incompetence

5). Open surgical demonstration of a repairable, refluxive valve

Patients with colour duplex scan or venographic (ascending or descending) findings of concomitant superficial vein reflux or perforator incompetence in association with deep venous reflux, existence of a coagulopathy (on pre-operative coagulopathy screening), presence of mixed disease (obstructions with reflux), fixed equinus deformity of the ankle or operative findings of valveless syndrome were excluded from this study.

Primary refluxive disease was present in 96 patients (118 limbs).

We performed external valvuloplasty in 12 limbs (19 valves) - 9 limbs (16 valves) by external technique as described by Kistner and 3 limbs (3 valves) by Trans-commissural technique.

Internal valvuloplasty was undertaken in 90 limbs (144 valves) – Kistner’s vertical venotomy technique5 was used in 52 patients (60 limbs / 97 valves), Raju’s transverse technique8 in 1 patient (1 limb / 2 valves), Sottiurai “T” technique7 in 3 patients (4 limbs / 4 valves) and the Tripathi ‘Trapdoor’ technique17 was employed in 17 patients (25 limbs / 41 valves).

External supports were used in 16 limbs (16 valves). In the beginning of our study we used Dacron (3 limbs / 3 valves) and PTFE grafts (5 limbs / 5 valves) as external supports but lately we use the External Valve Support (W.L Gore and Associates, Flagstaff, Arizona) (2 limbs / 2 valves) and PTFE Pericardial membranes (W.L Gore and Associates, Flagstaff, Arizona) (6 limbs / 6 valves). Multi-level (2-3) reconstructions were performed in 37 limbs. 41 patients had secondary valvular defects involving 51 limbs.

Axillary-femoral vein or sapheno-femoral vein valve transplant was performed for 29 patients (35 limbs) and 3 patients (3 limbs) respectively (1-valve segment in 14 limbs and 2-valve segments in 24 limbs); sapheno-femoral venous transposition was performed in 3 patients (4 limbs), Femoral or popliteal vein ligation was carried out in 6 patients (9 limbs).

All patients were anticoagulated with Enoxaparine 1mg/kg body weight b.i.d for 3 days and oral anticoagulation was started on post-op. day 1 and continued for 3 months during which INR was maintained between 2.5 and 3.0.

All patients had regular clinical examinations for leg ulcer healing assessments and colour-duplex scans at follow-up at 1, 3, 6, 12 and 24 months after vein valve reconstruction. The repaired valve stations were evaluated for endpoints i.e. competence (valve closure time) and patency.

Post-operatively, all patients underwent calf muscle strengthening physiotherapy, and complied with compression therapy with 3-layer crepe bandaging or class II compression stocking until ulcer healed completely.

RESULTS:

Two-year results of external valvuloplasty showed ulcer healing in 6/12 (50%) legs with maintenance of competency at only 6/19 (31.5%) valve stations. Furthermore, external repairs had better outcomes with 5/9 (55.56%) leg ulcer healing compared to transcmmisural valvular repairs with 1/3 (33.3%) but the results were statistically not significant (p>0.05).
Overall, internal valvuloplasty was the most durable valve repair procedure with 2-year leg ulcer healing rates of 61/90 (67.7%) and valve station competency of 115/144 (79.8%). The new Trapdoor Valvuloplasty technique also showed 19/25 (76%) ulcer healing rates and valve station competency of 34/41 (82.9%). There were 7 valves in our early experience where valve leaflet trauma occurred during vertical venotomy and these were repaired with CV8 PTFE sutures. 3 of these valves had post-operative thrombosis and 4 (57.1%) valves remained competent at 2 years. It was also noted that single-level repairs had a lower ulcer healing rate 29/53 (54.7%) than multi-level repairs 27/37 (72.9%) (P=0.002). 65.5% of limbs whose ulcers healed had one or more valves competent at two years. Of the 105 valves that underwent single level repair, 62 (59.04%) valves were competent (VCT<0.5 secs.) with an ulcer healing in 54.7% limbs. Of the 74 valves that had multilevel repairs, 59 (79.7%) valves were competent (VCT<0.5 secs) with ulcer healing in 72.9% limbs (P<0.05).

For secondary incompetence, valve transplants showed a significant early deterioration in valve patency and competence which at 2-years were 57.8% and 47.3% respectively with 55.3% leg ulcer healing. Also, valve transplants with multiple valve stations had better leg ulcer healing (57%) than vein valve segments with single valve (46.1%) (P=0.002). 43.1% of limbs whose ulcers healed had one or more valves competent at two years. Of the 18 valve segments that underwent single level repair, 7 (38.9%) valves were competent (VCT<0.5 secs.) with an ulcer healing in 46.1% limbs. Of the 20 valve segments (43 valves) that had multilevel repairs, 24 (55.81%) valves were competent (VCT<0.5 secs) with ulcer healing in 57% limbs (P<0.2).

We were able to identify only 8% of valves in the secondary valvular incompetence group that underwent Axillary/Saphenous-femoral vein valve transplant procedure, which were amenable to internal valvuloplasty. The patency in this group was 63% and competency was 28%, with leg ulcer healing rate of only 33.3% indicating that when valves are damaged with thrombotic process, further operative trauma may not produce results comparable with those of primary incompetence.

The correlation between ulcer healing and duplex findings of valve patency and competence was stronger in the internal valvuloplasty group with multi valve repairs 72.9% ulcer healing with 78% valve competency compared with single valve repairs 54.7% ulcer healing with 63% valve competency.

The cumulative rate of clinical success at two years, defined by ulcer recurrence free survival was 63.5% for primary refluxive disease and 47% for secondary refluxive disease, when all procedures are taken into account.

**COMPLICATIONS:** Wound hematomas occurred in 17 limbs. Post-operative serosanguineous drainage more than 500 ml. in first three post-operative days requiring blood transfusion occurred in 9 patients. Overall rate of post-operative thrombosis in the operated limb was 12.4% (21/169 limbs). There was a significant difference (P=0.001) between patients with post-operative DVT which occurred in 6.7% (8/118) limbs with procedures for primary refluxive disease and in 25.4% (13/51) limbs with secondary reflux surgeries. One patient in the venous ligation group also had contralateral limb DVT, despite adequate anticoagulation. Valve resorption was seen in 11 valve stations undergoing repair for primary reflux. Wound infections occurred in 12 limbs (7.1%). All healed with conservative management. There was no mortality in this study.
**DISCUSSION:** Volume of reflux is one of the most important determinants of severity of chronic venous insufficiency\(^\text{12}\). Stasis induced symptoms and signs in chronic venous hypertension in deep vein refluxive disease are more than likely due to large volume reflux. The compensatory mechanisms like calf-pump action and perforator valve function gradually deteriorate with increased deep vein reflux\(^\text{2}\). Perforator incompetence is nearly always a result of deep valve reflux\(^\text{2}\). In chronic venous insufficiency, deep vein reflux occurs in 98% of patients, either alone or in combination of superficial or perforator vein incompetence\(^\text{13}\). Surgery of the insufficient superficial or perforator venous systems, in the presence of deep venous reflux, leads to poor healing of venous ulcers in a majority of patients\(^\text{3}\). Hence, this group in whom recurrence of leg ulceration is significant even after superficial and perforator vein surgery and the group which has deep venous reflux without superficial vein involvement, constitute a significant number of venous leg ulceration patients in whom deep venous valve reconstruction surgery becomes a last option.

This study presents results expressed at two years in a continuing long-term follow-up of all patients with venous leg ulcerations due to deep venous reflux. These patients CEAP C6 had non-healing venous ulceration despite superficial and perforator vein surgery and trial of conservative management for more than 3 months on venotropic drugs and compression therapy.

In our study we demonstrated ulcer healing of 63.5% limbs in the primary refluxive disease and 47% in secondary refluxive disease. Sottiurai\(^\text{3}\) has shown 80% ulcer healing in primary valvular reflux patients who underwent valvuloplasty and superficial venous surgery. He further showed 75% ulcer healing in secondary reflux patients who underwent vein valve transplant in combination with superficial venous surgery. These comparative data suggest that when deep vein reflux is associated with superficial venous incompetence, superficial venous surgery alone for these patients will result in non-healing or recurrence of a majority of these leg ulcers.

The ideal site for repair of valve is still debated. Sottiurai and others believe that popliteal vein is the gatekeeper of the leg veins and recommend popliteal level repair. Kistner and Raju have recommended repair of common femoral vein or termination of superficial femoral level. In our study, we based the site of valve reconstruction at valve stations with maximum reflux. We used 2-level repairs in patients with grade III / IV reflux according to Kistner. We also found that patients who underwent multi-level repairs (irrespective of site chosen) had superior results to single-level repairs, irrespective of sites of repair. The gatekeeper concept may therefore not be as important as has been emphasized in the past.

The benefits of valvular reconstructions are superior in the primary reflux group compared to the secondary (post-thrombotic) reflux group. In the primary reflux group, in our series, the ulcer healing rate has been 63% which is lower than that reported in other series\(^\text{5,10,14,15,16}\). It is likely that the results of our series are not augmented by the contribution of the effects of superficial vein/perforator surgery, which have been done in conjunction with valvuloplasty in other series.

Regarding external valvuloplasty, our results of external repair have been better than trans-commisural repairs; contrary to other reports and this we believe is due to learning curve with Transcommisural valvuloplasty, which we have done as a blind guess procedure rather than with angioscopic control. External cuffing has now been abandoned in our practice as the results are not satisfactory and yield a high leg ulcer recurrence and often end up with fibrotic / thrombotic occlusion of vein valve stations.
Valve cusp injuries or defects can be effectively repaired with CV8 PTFE sutures as has been our experience. Out of the 7 valve cusps repaired, more than half of these remained competent at two years. The most common internal valvuloplasty technique we employed was the Kistner vertical venotomy valvuloplasty \(^9\) in the early part of this study. After the development of “Trapdoor” valvuloplasty technique \(^1\), this has become our exclusive technique due to its technical advantages. “Trapdoor” valvuloplasty technique yields a ulcer healing rate of 76% and valve competency of 83% at two years. This is in concordance with reported results of internal valvuloplasty in the literature.\(^{18}\)

A curious phenomenon was observed on duplex follow-up scan in 11 valve stations of patients who developed loss of competence following internal valvuloplasty. These valves showed complete absence of valves or ‘valve resorption’. This may be due to collagenolysis following trauma to valve leaflets or due to persistent distal reflux \(^1\). The real cause is however not known. In our study, multiple level repairs yielded better outcomes than single level repairs ( p <0.002) for primary reflux , in agreement with observations made by Raju in support of back-up repair \(^2\).

We observed post-operative thrombosis in 6.7% limbs in the primary reflux group and 25.4% limbs in the secondary reflux group. In the former group, DVT occurred at the site of valve repair in 62.5% limbs and remote site in 25% limbs and involving the whole femoro-popliteal system in 12.5%. In the latter group, DVT occurred at the site of valve repair in 53.85% limbs and remote site in 15.3% limbs and involving the whole femoro-popliteal system in 30.76%. The reporting of post-valve reconstruction DVT in literature is not common. According to Raju \(^1\) this occurs in 4.5% where as Perrin \(^18\) has reported 8.8% DVT after valvuloplasty and 29.7% after valve transplants. Our experience is therefore more in concordance with the Perrin experience. No pulmonary embolism was reported in our study. This thrombosis occurred in spite of adequate anticoagulation.

For secondary valvular reflux, valve transplants have been used from axillary / brachial venous segments or from the opposite GSV. An attempt was been made to harvest a segment of vein with at least two competent valve stations, wherever possible. When the valves were incompetent, external or internal valvuloplasty was employed. The results of ulcer healing in our study improved (p<0.003) when multiple valve segments were used compared to single valve segment transplants. There was also a marked reduction of early and mid (3-9 months) deterioration of valve function, especially total or partial occlusion of valve stations when ultra thin PTFE pericardial membrane was used compared to our previous experience with using the PTFE / Dacron sleeve. It was possible to perform trabeculectomy in some deep veins to improve outflow where the DVT was extensive. Overall, valve transplantation results in our study showed 58% patency, 47% competency and 55.3% ulcer healing which favourably compares to other series.\(^2,18,21,22,23,24\)

There was a correlation between ulcer healing and duplex findings of valve patency and competence only in the primary reflux group which underwent internal valvuloplasty- with multi valve repairs (72.9% ulcer healing with 78% valve competency) compared with single valve repairs (54.7% ulcer healing with 63% valve competency) p<0.05. The other deep venous valve repair groups did not show statistically significant correlation. The early results of this series indicate that valvular reconstruction is more durable when performed for primary refluxive disease and if undertaken at multiple levels or using multiple valve segments. Most patients with successful valve reconstruction have successful healing of their venous ulcers and they return back to active life without having the need to use compression stockings in the leg.
**Recent Advances**
We have recently introduced a new concept in treating very severely redundant valves which are difficult to plicate at the commissures because the current valvuloplasty techniques result in bulky valve remnants left behind at the comissures causing increased thrombogenic potential at the valve station level.
To obviate this problem, we have embarked upon excision of redundant valve and stitching of the valve border to the vein valve and we have been technically able to provide a water tight repair with good competency following this repair. This technique is called “Reduction Valvuloplasty”(Figure below).
REFERENCES


25) Burnand KG, O'Donnell TF, Lea Thomas, Browse NL: The relative importance of incompetent communicating veins in the production of varicose veins and venous ulcers. Surgery, 1977, 82, 9
<table>
<thead>
<tr>
<th>Fig. 1</th>
<th>Strip Test a</th>
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<tbody>
<tr>
<td>Fig. 2</td>
<td>Strip Test b showing incompetent deep vein valve with reflux</td>
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<tr>
<td>Fig. 3</td>
<td>Identification of Valve Station</td>
</tr>
<tr>
<td>Fig. 4</td>
<td>Adventitial dissection of Valve station</td>
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Fig. 5  Transverse Venotomy

Fig. 6  Diagrammatic Schema of Transverse Venotomy

Fig. 7  Vein Valve Retractor Forceps

Fig. 8  Use of Vein Valve Retractor
Fig. 9   Opened “Trapdoor” with Vein Valve exposed

Fig. 10   Diagrammatic Schema of “Trapdoor”

Fig. 11   Posterior Commissure Apical Stitch

2   Valve cusps plicated till valve rim is straightened out
Fig.13  Valve Cusp abnormalities e.g hole in the valve cusp medially noted here and repaired

Fig.14  Completed valve plication

Fig.15  Open Technique of Competency Check

Fig.16  Closed Technique of Competency Check (Strip Test)
Fig. 17  Vein Lumen after closure of “Trapdoor”

9 months- Post-operative Duplex showing abolition of reflux at valvuloplasty site

Fig. 18
preoperative descending venogram showing incompetent common femoral vein valve
Fig.1b-post valve repair descending venogram showing valve competence with outline of competent cusps
Graph I

<table>
<thead>
<tr>
<th>Months</th>
<th>3</th>
<th>6</th>
<th>12</th>
<th>18</th>
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<tr>
<td>Primary Valve Reflux</td>
<td>89.8</td>
<td>86.4</td>
<td>79.6</td>
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<tr>
<td>Secondary Valve Reflux</td>
<td>72.5</td>
<td>56.8</td>
<td>56.8</td>
<td>52.9</td>
<td>47.05</td>
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<tr>
<td>Total</td>
<td>84.6</td>
<td>77.5</td>
<td>72.7</td>
<td>62.7</td>
<td>58.5</td>
</tr>
</tbody>
</table>
Graph II

**Ulceral Healing Vs Procedure**

- Internal Valvuloplasty
- Valve Transpl.
- External Valvuloplasty

% leg ulcer healing

Time in months

0 3 6 12 18 24
Graph III

Ulcer Healing Vs. Valve Competency

% ulcer healing

Primary Valve Reflux

Secondary Valve Reflux

■ 1 valve competent

□ > 1 valve competent

(P<0.05)