Outline of Venous Disease Management

Manifestations of Superficial and Deep Venous Disease

1. Varicose veins
2. Spider veins (also known as venous telangiectasias, venous hemangiomas)
3. Edema
4. Chronic Lymphovenous Edema
5. Hemorrhage (recurrent bruising of the skin or frank bleeding)
6. Venous ulceration
7. Superficial thrombophlebitis
8. Deep Venous Thrombosis
9. Postphlebitic syndrome
10. Pulmonary embolism
11. Restless legs and chronic venous insufficiency

Investigation:

1. Complete examination
2. Venous Duplex scan
   a) Assessment of deep venous system to detect DVT and Venous Insufficiency
   b) Complete examination of superficial venous system including:
      1) Great Saphenous and Small Saphenous veins
      2) Accessory Saphenous veins
      3) Perforating veins (Thigh, Paratibial, and Posterior Tibial Perforators)
3. Venogram, CT Venogram (CTV), Magnetic Resonance Venogram (MRV)-for abdominal and pelvic veins
4. Lymphography

Treatment of Superficial Venous Disease:

Conservative (only for symptomatic relief while waiting for definitive treatment)

1) Compression stockings
2) Leg elevation
3) Leg exercises
4) Daily walks
5) Avoidance of high heals
6) Drugs and Dietary Supplements

Definitive

1) Treatment of Reflux: Venous closure of incompetent superficial veins (Saphenous Veins, Accessory Saphenous Veins, Perforators)--Endovenous Laser or Radiofrequency Venous Closure
2) Varicose Veins: Ambulatory stab-phlebectomy for varicose veins in multiple sessions (or Foam Sclerotherapy)
3) Spider Veins: Laser treatment (or Foam Sclerotherapy) for symptomatic spider veins in multiple sessions (even asymptomatic spider veins should be considered for treatment-AVF Guideline)
4) Perforators: Laser or Radiofrequency Closure (or Foam Sclerotherapy)
5) **Lymphovenous Edema**: Compression therapy, (graduated compression stockings, Intermittent Pneumatic Compression pump therapy)

6) **Venous ulcer management**- surgical debridement, skin grafting, antibiotics, specialized compression therapy e.g., Una Boot applications, CircAid stockings, hyperbaric oxygen therapy.

**Treatment of Deep Venous Thrombosis:**

**Conservative:**

1. Anticoagulation therapy
2. Compression therapy—graduated compression stockings, ICP therapy (for post-phlebitic syndrome)

**Definitive:**

1. Thrombectomy
2. Intravenous thrombolysis
3. Catheter thrombolysis
4. Catheter thrombectomy with mechanical devices

Patients with venous disease may present with following signs and symptoms:

**Symptoms**

1. Pain (heaviness, aching, throbbing, cramping, itching, numbness, and, burning of legs)
2. Swelling
3. Hemorrhage (frequent bruising of skin of legs with minor trauma or frank bleeding episodes)
4. Skin discoloration (Dyschronia, Pigmentation)
5. Ulcer (active or healed)
6. Restless legs

**Signs**

1. Varicose veins
2. Spider veins
3. Edema
4. Tenderness of lower legs (*assessed at the medial surface of the tibia*)
5. Pigmentation
6. Induration of the skin of lower legs
7. Lipo-dermatosclerosis
8. Eczema at the lower legs
9. Atrophy blanche
10. Corona phlebectatica
11. Ulcer (active or healed)
Varicose and Spider Veins

Varicose Veins develop due to the dilatation of the previously existing veins, present in the subcutaneous tissue, under the influence of venous insufficiency, which is due to the diseased incompetent venous valves. The varicose veins are greater than 2 mm in diameter. Clusters of varicose veins always have an associated feeding vein or veins connected to the deeper veins causing significant reflux of blood under pressure in the wrong direction causing distention of these veins. If left untreated, these veins lead to dilatation of the deep veins due the reflux over-flow of blood causing the deep venous insufficiency.

Spider Veins develop due the proliferation of new capillaries caused by venous hypertension due to incompetent venous valves and are located in the dermis of the skin. The spider veins are less than 1 mm in diameter. This new growth of capillaries is called vascular neogenesis, and is due to proliferation endothelial stem cells present in the dermal layers of the skin. These stem cells are embryonic remnants left over during the early stages of venous development. Spider veins always drain into to the deeper veins through feeding veins and can be responsible for significant reflux, which produces pressure in these tiny thin-walled vessels and can cause pain with varying intensity and can result in bleeding. If left untreated, these veins lead to dilatation of the deep veins due the reflux over-flow of blood causing the deep veins to enlarge; thus, the venous valve become incompetent and cause venous insufficiency.

Reticular Veins are one to two millimeter in diameter and are located in subcutaneous space and are frequently the feeding veins for clusters of spider veins located in the dermal layer of skin.

Spider Veins Connection to deep Veins--The following articles support the view that spider veins are connected to the deeper veins and the possible embryological origin:


This study demonstrated, direct connection of the telangiectasias with named veins such as greater saphenous and other superficial veins; and, also demonstrated communicating veins directly connecting the telangiectasias to the sapheno-femoral junction.
Telangiectasias are associated with elevated venous pressure. Telangiectasias are linked to varicose veins.

Telangiectasias may receive venous hypertension from sub dermal reticular network associated with reflux demonstrated by Doppler study.

Dermatologica 1963, 127; 321-329

Faria J.L., Moraes I.N., Histopathology of the Telangiectasias associated with Varicose Veins

Bohler-Sommeregger K., Karnel F., Schuller-Petrovic SS., Sautler R.,
"Do telangiectasias communicate with the deep venous system."

Telangiectasias may receive their pulse of venous hypertension directly through minute incompetent perforating veins.

Spider Veins--relation to growth factors and embryological origins

Embryological Basis of Varicose and Spider Veins


A Doppler study of the distribution of varicose veins confirmed the presence of a distinct group of non-saphenous varicosities on the lateral aspect of the thigh and calf that represented the lateral venous system.
Embryological Basis of Varicose and Spider Veins

Based on anatomical dissections by Hochstetter, the initial venous system of the leg is characterized by a network of superficial veins from which the lateral venous system is derived. The deep venous system then develops in a rudimentary way, accompanied by development of a superficial external saphenous vein that is connected by small perforators to the deep system. When the deep system becomes predominant, the external saphenous vein disintegrates at the thigh, but not without leaving a few perforators intact. Albanese et al. speculated that, in areas where the superficial veins do not involute, superficial embryonic veins remain and may become easily and prematurely varicose. This change occurs for two reasons: (1) because of their superficial location, these veins are poorly supported by surrounding connective tissue; and (2) direct transfascial perforators continue to connect these veins with the deep venous system.

Angiogenic Factors and Spider Veins

The vascular proliferation seen in the skin of patients with venous disease has been known for many years but has not been explained. In recent years many angiogenic factors that stimulate the growth of blood vessels have been recognized.

Angiogenesis is poised to initiate rapid proliferation of new vessels, with entire cell populations turning over every 3 to 5 days following exposure to a wide variety of stimuli. These stimuli include:

- Hypoxia
- Venous Hypertension
- Adaptive biochemical forces
- Tissue trauma
- Growth
The following articles from the medical literature show the symptoms caused by spider veins of the legs:

**Handbook of Venous Disorders**

1996 Edition

The symptoms may not be recognized by the patient as being due to the varicose veins so they must be asked for by the interested physician. Neither the patient nor the physician may understand that these symptoms arise from telangiectatic blemishes as well as venous varicosities. This is true and some 50% of patients with telangiectasias will have such symptoms and 85% will be relieved of them by appropriate therapy.

**Current Therapy in Vascular Surgery 2001**

Negative physician perception regarding availability and efficacy of treatment of varices deny the patient the precise care that is sought. Furthermore, symptoms of primary venous insufficiency may be present but not recognized by the patient until asked for during a thorough history taking.

**Chapter 4 VENOUS PHYSIOLOGY AND PATHOPHYSIOLOGY**

Page 28


Symptoms may be present in up to 98% of patients with "clinically relevant" alterations of venous circulation, but even small telangiectasias are often symptomatic: 53% of patients presenting with telangiectasias less than 1mm in diameter complain of symptoms that resolve with treatment.
**Chapter 23: Painful Telangiectasias: Diagnosis and Treatment**

Table 23-1. Symptoms of painful telangiectasias*

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Overall Incidence</th>
<th>Patients with post-treatment Improvement %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fatigue, general ache</td>
<td>32</td>
<td>85</td>
</tr>
<tr>
<td>Pain in region of telangiectasias</td>
<td>32</td>
<td>85</td>
</tr>
<tr>
<td>Pain in region of reticular vein</td>
<td>31</td>
<td>86</td>
</tr>
<tr>
<td>Focal burning</td>
<td>27</td>
<td>93</td>
</tr>
<tr>
<td>Night Cramping</td>
<td>21</td>
<td>70</td>
</tr>
<tr>
<td>Local Edema</td>
<td>19</td>
<td>83</td>
</tr>
<tr>
<td>Throbbing sensation (focal or general)</td>
<td>17</td>
<td>86</td>
</tr>
</tbody>
</table>


**Chapter 4: Venous Physiology and Pathophysiology**

Table 4-3

Symptoms of Pain from Telangiectatic Webs

- Fatigue
- Heaviness
- Focal burning or aching
- Focal pruritis
- Sharp intermittent stabbing pain (focal)
- Diffuse burning
- Night cramping
- Restless legs


Pain associated with telangiectasias may be a consequence of stretching caused by pressure transmitted during reflux through reticular veins and venulectases. "the largest varicosities sometimes cause no complaints; other veins of small caliber may give rise to surprising discomfort."
Symptoms Caused by Spider Veins

Many of our patients whose occupations involve long periods of standing have complained of muscle fatigue and aching or localized pain over group of telangiectasias and/or venulactases. Some relief of symptoms occurs by wearing lightweight support hose.

Isacs MN. Symptomatology of vein disease

Aching/pain, excessive tiredness/fatigue, and throbbing in the legs correlate well with patients presenting with nonbulging reticular veins and telangiectasias compared with a matching control group. Furthermore, these symptoms were independent of the size of the veins.

Why Minor Varicose Veins Cause More Leg Pain Than Larger Varicose Veins?


The largest varicosities sometimes cause no complaints; other veins of small caliber may give rise to surprising discomfort. One theory that attempts to explain this phenomenon is that compliance of small veins may be greater than that of larger veins, with more dispensability and greater stretch resulting in greater stimulation of neural pain receptors.
Venous Insufficiency—Superficial and deep
Following articles demonstrate the relation of superficial and deep venous insufficiency—superficial venous insufficiency leads to deep venous insufficiency; and, treatment of superficial venous insufficiency improves the deep venous incompetence:

**Ann Vasc Surg 1994;8:566-570**

**Femoral Venous Reflux Abolished by Greater Saphenous Vein Stripping**

In 27 of 29 limbs with preoperative femoral reflux, that reflux was abolished by greater saphenous stripping. In patients with popliteal reflux both femoral and popliteal reflux was abolished. Improvement of deep venous hemodynamics by ablation of superficial reflux supports the reflux circuit theory of venous overload.

**J Vasc Surg 1996;24:711-8**

**Hemodynamic and clinical improvement after superficial vein ablation in primary combined venous insufficiency with ulceration.**

Superficial and perforating vein incompetence accounts for a substantial and correctable component of venous insufficiency in limbs with combined deep and superficial vein reflux and venous ulceration. These data indicate that surgical correction of this component significantly improves clinical symptoms and venous hemodynamics. Superficial and perforator ablation is an appropriate initial step in the management of combined deep and superficial incompetence.


**Correction of Lower Extremity Deep Venous Incompetence by Ablation of Superficial Venous Reflux**

Postoperative interrogation of the venous system revealed that in 16 (94%) of 17 patients, coexistent femoral venous insufficiency completely resolved. Thus ablation of superficial venous reflux eliminated incompetence in the deep venous system in patients with combined disease. These preliminary results suggest that superficial venous incompetence may be a cause of deep venous insufficiency.
Improvement of deep venous hemodynamics by ablation of superficial reflux supports a reflux circuit theory of venous overload.

It would be logical to suppose that ablation of such a reflux by superficial vein removal would correct the deep venous volume overload and allow diminution of the diameter of veins, thus producing valvular competence.

Earlier study of Phlebograms had shown that deep venous diameter was greatest in limbs with superficial reflux. Deep veins in limbs with proven prior deep venous thrombosis were actually found to be thinner in diameter than normal and those limbs with superficial reflux [46]. Now Doppler ultrasonography has shown that deep venous reflux in limbs with varicose veins proven not to have been the site of previous thrombosis is a startling 20.6%. Such deep venous reflux correlates with the severity of superficial reflux [47].

Using indirect parameters of venous pathophysiology, hemodynamics of the deep venous system were observed to improve after treatment of superficial venous incompetence [44].

We have recently studied 58 limbs with class 3 venous reflux. Ten limbs (17%) exhibited only superficial reflux, and superficial reflux was a major contributor to chronic venous dysfunction in another 17 limbs. Of some importance is the fact that primary, nonthrombotic deep (superficial femoral and popliteal vein) incompetence may accompany superficial reflux. This is explained by reflux proceeding distally down the greater saphenous vein and overloading the deep venous system [40]. One would presume this causes dilatation and elongation of the deep vessels so that their valves become incompetent. Our own study of limbs following greater saphenous vein stripping in which superficial and popliteal venous incompetence was present has revealed correction of the deep reflux by superficial venous stripping in a vast majority of limbs [41].
Investigations of venous pathophysiology in continental Europe are
influenced by the observations of Friederich Trendelenberg, Professor
of Surgery in Bonn in the 1880s. In his 1891 publication advocating
greater saphenous vein ligation, he coined the term ‘private circulation’
to describe the gravitational reflux down the saphenous vein which
returns proximally through perforating veins and the deep venous
system. Later observers have noted that this private circulation is
associated with primary deep venous valvular incompetence and this
is the most important consequence of saphenous reflux.

Prevalence of deep venous reflux in patients with
primary superficial vein incompetence

The prevalence of deep venous insufficiency in
patients with primary superficial venous reflux
and without history of DVT is 22%.

Comparative Evaluation of Duplex Derived
Parameters in Patients with Chronic Venous
Insufficiency: Correlation with Clinical
Manifestations

This study has suggested the importance
of superficial venous insufficiency in the
development of advanced Chronic Venous
Insufficiency (CVI). Superficial
insufficiency is predominant in both early
and advance CVI. These reports suggest
that superficial incompetence produces
an overflow of venous return through
perforating veins into the deep system.
Superficial venous insufficiency might play
a major role in the development of
advanced CVI.
Chronic venous insufficiency: Clinical and duplex correlations. The Edinburgh Vein Study of venous disorders in the general population.


The prevalence of CVI rises steeply with age. There is a strong correlation between venous symptoms and the presence and severity of CVI. CVI is associated in approximately one third of the subjects with incompetence limited to the superficial system and in these a good therapeutic outcome could be expected from surgery to the superficial veins. The severity of clinical features correlates significantly with prevalence of valvular reflux in the deep and superficial systems.

J Vasc Surg 2003; 38:517-21

How often is deep venous reflux eliminated after saphenous vein ablation

In patients with concomitant deep and superficial venous reflux, saphenous vein ablation results in resolution of deep reflux in about a third of patients.

Deep Venous Thrombosis and Superficial Venous Reflux


Superficial venous thrombosis frequently accompanies DVT and is associated with development of superficial reflux in most limbs. However, a substantial proportion of observed reflux is not directly associated with thrombosis and develops at a rate equivalent to that in uninvolved limbs.

It can, therefore, be concluded that deep venous reflux is the result of superficial venous reflux.
Five-year outcome study of deep vein thrombosis in the lower limbs
Akram M. Asbeutah, MS., Andrea Z. Riha, MBBS., James D. Cameron, MD Barry P. McGrath, MD

An important finding of this study was an unexpectedly high incidence of venous reflux in the apparently unaffected limb. Although these non-DVT limbs were not investigated at presentation, our data is consistent with the hypothesis that DVT may result in a more systemic disorder of venous function.

This study points out the fact that deep venous insufficiency is usually the primary pathology that leads to deep venous thrombosis; therefore, it is very important to treat superficial venous insufficiency aggressively, including the treatment of saphenous venous reflux and complete elimination of all the varicose and spider veins, in order to improve DVI.

Endoscopic perforator vein division with ablation of superficial reflux improves venous hemodynamics
Jeffrey M. Rhodes, MD, Peter Gloviczki, MD, Linda Canton, RN, BSN, Tracy V. Heaser, RVT, Thom W. Rooke, MD
Rochester, Minn

SEPS with ablation of superficial reflux improved calf muscle pump function, reduced venous incompetence, and produced excellent midterm clinical results. However, functional improvement directly related to SEPS requires further investigation. This study supports adding SEPS to ablation of superficial reflux in patients with advanced chronic venous insufficiency. (J Vasc Surg 1998;28:83947.)

This study shows that elimination of superficial venous reflux by treating perforator veins, with SEPS, reduces deep venous incompetence.

Spider Veins Resulting in Ulceration and Bleeding

Bleeding Spider Vein
THE CONCEPT
Spider veins and their connection to the deep veins
Treatment Protocol

- Color Duplex Scan with the patient in an upright position
- High ligation and PIN stripping (or Endovenous Closure)
- Ambulatory micro-phlebectomy in multiple sessions
- Spider veins treated with trans-cutaneous intense pulse light and/or ND:YAG laser in multiple sessions 2-3 weeks apart
- Leg exercises—detailed instructions
- Compression stockings

- A thorough education in venous disease
- Diet and supplements—detailed instructions (optional)
- Every six months—a follow-up color Duplex scan
### Treatment algorithms for telangiectasias and varicose veins: Current Guidelines

(Guidelines 4.12.0 of American Venous Forum on treatment algorithms for telangiectasias and varicose veins)

<table>
<thead>
<tr>
<th>No.</th>
<th>Guideline</th>
<th>Grade of Recommendation</th>
<th>Grade of Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>4.12.1</td>
<td><strong>For class 1 venous disease</strong> we recommend treatment of symptomatic patients, but treatment can be considered in asymptomatic patients as well. We recommend sclerotherapy or laser for spider telangiectasias, and liquid or foam sclerotherapy for reticular veins.</td>
<td>1, We recommend</td>
<td>A, high quality</td>
</tr>
</tbody>
</table>

**Class 1 Venous Disease—Spider Veins**
Venous Duplex Scan—Criteria of reflux

Duplex Scan Criterion currently used for the diagnosis of Venous Reflux:

*Duration of reflux*—0.5 second or more, documented with Duplex scan

Reflux was considered significant if the duration of retrograde flow exceeded 0.5 second and was measured in the common femoral, superficial femoral, long saphenous, popliteal, and proximal and distal posterior tibial veins.
Subjects were examined on a tilt table in a reversed 15-degree Trendelenburg position with the legs slightly flexed in minimal external rotation for maximum comfort. The duplex probe was used to determine vein compressibility in all examined veins. An automatic cuff inflator (Hokanson, Bellevue, Wash) was connected to cuffs placed at mid-thigh to examine the common femoral vein, superficial femoral vein, and sapheno-femoral junction; at mid-calf to examine the popliteal vein and sapheno-popliteal junction; and at foot level to examine the posterior tibial vein. Standard rapid inflation (100 mm Hg) and deflation were performed, and response in the examined veins, ie, augmentation of flow velocity, was determined. Duration of inflation was 3 seconds, and reflux longer than 0.5 seconds was considered a positive result.

Positive Indicators of Reflux:

**GSV:** reflux >= 0.5 seconds size: > 6.0 mm

**SSV:** reflux >= 0.5 seconds size: > 3.0 mm

**CFV:** reflux >= 0.5 seconds

**Popliteal:** reflux >= 0.5 seconds

**Perforator:** reflux >= 0.3 seconds size: > 3.0 mm
Patient Positioning for Venous Duplex Scanning:

Pt Position vs. Vessel Size

Change of Vein Size with Patient Position

Patient Positioning for Venous Duplex Scan

Ideal Position

Incorrect Position

Correct Position (until recently)

Great Saphenous Veins

Small Saphenous Veins
Revised CEAP Clinical Classification of Venous Disease

C -- Clinical
E -- Etiology
A -- Anatomical
P -- Pathophysiology

Clinical Classification

C0: No visible or palpable signs of venous disease
C1: Telangiectasias or reticular veins
C2: Varicose veins
C3: Edema
C4a: Pigmentation or eczema
C4b: Lipo-dermatosclerosis or atrophie blanche
C5: Healed venous ulcer
C6: Active venous ulcer

S: Symptomatic (including ache, pain, tightness, skin irritation, heaviness, muscle cramps, and other complaints attributable to venous dysfunction)

A: Asymptomatic
VENOUS NOMENCLATURE

Old Terminology
Femoral Vein
Long Saphenous Vein
Greater Saphenous Vein
Superficial Femoral Vein
Lateral Accessory GSV
Medial Accessory GSV
Short Saphenous Vein
Lesser Saphenous Vein
Perforators (Hunter, Boyd, Dodd, Cockett-1,2,3)
Giacomini Vein

New Terminology
Common Femoral Vein
Great Saphenous Vein
Femoral Vein
Anterior Accessory GSV
Posterior Accessory GSV
Small Saphenous Vein
Perforators (Femoral, Paratibial, Posterior Tibial)
Intersaphenous Vein

Anterior Accessory
Posterior Accessory
Intersaphenous Vein
Thigh extension of Small
Great Saphenous Vein
Small Saphenous Vein
Classification of Saphenous Refluxes

Type 1: Varices with competent GSV

Type 2: GSV Reflux Without Varices

Type 3: Varices with Incompetent GSV and Competent Saphenous Junction

Type 4: Varices with Incompetent GSV and Saphenous Junction

Type 5: Varices with Reflux Limited at the Saphenous Junction

Femoral Vein

Great Saphenous Vein

Re-entry Perforator
Chapter 20: Laser And High Intense Pulse Light

No. of Treatments Needed For Each Area

Table 20-1
Simplified Comparison of Laser Therapy and Sclerotherapy

<table>
<thead>
<tr>
<th></th>
<th>Laser</th>
<th>Sclerotherapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>How it works</td>
<td>Heats blood to the boiling point of water</td>
<td>Chemical irritation</td>
</tr>
<tr>
<td>Pain</td>
<td>Intense heat for fraction of a second</td>
<td>Small pinprick. Only painful with hypertonic saline</td>
</tr>
<tr>
<td>Area treated per session</td>
<td>Small</td>
<td>Large</td>
</tr>
<tr>
<td>Risks of epidermal injury</td>
<td>1–10%</td>
<td>Less than 1%</td>
</tr>
<tr>
<td>Treatment of tanned legs</td>
<td>No (except 1064 nm)</td>
<td>Yes</td>
</tr>
<tr>
<td>Risks of pigmentation changes</td>
<td>Up to 50% with lower wavelengths, greatly reduced with epidermal cooling</td>
<td>Hyperpigmentation in 8–25%</td>
</tr>
<tr>
<td>Cost</td>
<td>Up to 100% more per treatment than sclerotherapy</td>
<td>Variable depending on total time of treatment</td>
</tr>
<tr>
<td>Number of treatments</td>
<td>2–10</td>
<td>1–5</td>
</tr>
<tr>
<td>Need for repeat treatments</td>
<td>Frequent</td>
<td>Less frequent</td>
</tr>
</tbody>
</table>
Chronic Venous Insufficiency

Sequelae from C.V.I.

1. Cosmetic disfigurement.
2. Leg pain.
3. Edema.
4. Pigmentation.
5. Stasis dermatitis.
6. Atrophie Blanche.
7. Ulceration.
8. Malignant degeneration.
10. Superficial thrombophlebitis
12. Pulmonary embolism.

Sclerotherapy

Sequelae from Sclerotherapy:

1. Failure of treatment
2. Post-sclerotherapy pain.
3. Post-sclerotherapy edema.
4. Post-sclerotherapy hyper-pigmentation
5. Post-sclerotherapy telangiectatic matting
6. Tape blister & folliculitis.
7. Post-sclerotherapy coagula.
8. Superficial thrombophlebitis.
9. Localized hursutism.
10. Vasospasm.
11. Extravasation & cutaneous necrosis.
12. Lymphatic injection.
13. Excessive compression
15. Vasovagal reaction.
17. Localized allergic reaction (urticaria).
18. Intra-arterial injection.
20. Deep venous thrombosis
21. Systemic allergic reaction.
22. Pulmonary Embolism.
23. Air embolism (theoretical risk if using foam sclerosants.)
Compression Therapy

<table>
<thead>
<tr>
<th>Compression Strength</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>8-15mm</td>
<td>Leg fatigue, mild swelling, stylish</td>
</tr>
<tr>
<td>15-20mm</td>
<td>Mild aching, swelling, stylish</td>
</tr>
<tr>
<td>20-30mm</td>
<td>Aching, pain, swelling, mild varicose veins</td>
</tr>
<tr>
<td>30-40mm *</td>
<td>Aching, pain, swelling, varicose veins, post-ulcer</td>
</tr>
<tr>
<td>40-50, 50-60mm *</td>
<td>Recurrent ulceration, lymphedema</td>
</tr>
</tbody>
</table>

* Requires a prescription

21st Annual Congress of the American College of Phlebology
8-11 November 2007
Paper 1.6

The effect of three–month mandatory conservative treatment with compression hose therapy on quality of life issues and great saphenous vein reflux.


Vein specialists of the South, LOLC, 556 Third Street, Suite A, Macon 31211; Mercer University, Macon, GA, USA
Presented by: Kenneth E. Harper

43 patients (77% female) with reflux and studied for the effect of three months of conservative therapy with compression hose.

There was no statistically significant improvement in the GSV reflux and other QOL issues to warrant mandatory conservative therapy prior to correction of venous reflux.