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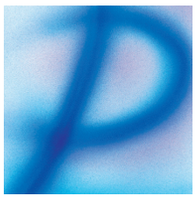
PHLEBOLYMPHOLOGY



# EDITORIAL

*Dear Readers,*

*Postthrombotic syndrome is a frequent and fascinating problem, which is still not fully understood. Severe clinical consequences, especially recalcitrant leg ulcers, are a considerable burden both for the patient and for the general health care budget.*

*As **Michel Perrin**, Lyon, points out in his article, most patients with postthrombotic syndrome are treated conservatively, and duplex ultrasound is sufficient for diagnosis. However, in recent years various surgical and endoscopic techniques have been developed in specialized centers to improve deep venous hemodynamics, and this requires more detailed information before and after such procedures. The first article in this issue of Phlebolymphology offers a kind of checklist of information that be gathered by various investigations and used to tailor treatment to the individual patient.*

***Waldemar Olszewski** from Warsaw, one of the leading pioneers in clinical lymphology, gives us some insight into his admirable work, which has considerable practical importance. He makes us keen to learn how much pressure is needed to move fluid effectively into non-lymphedematous regions of the body.*

*Chronic pelvic pain due to pelvic congestion syndrome can be relieved by the administration of Daflon 500 mg, as shown in two small, but carefully performed, randomized controlled trials, presented by **Omur Taskin** and co-workers from Antalya, Turkey.*

***Jean-Luc Gillet**, Bourgoin-Jallieu, France, presents a review of practical importance on how patients presenting with recurrent varicose veins should be investigated by duplex.*

*Two very informative reports on recent publications end this issue. The first deals with a useful catalogue containing definitions of phlebological terms, drawn up by an international consensus group, for which Bo Eklof and Michel Perrin were the driving forces. The second is on the Handbook of Venous Disorders, the bible of the American Venous Forum. Phlebologists who wish to explore things in greater depth will certainly want to own these two publications.*

*Enjoy your reading!*

**Hugo Partsch, MD**

# Investigations in postthrombotic syndrome according to clinical status

Michel PERRIN

*Vascular Surgery, France*

## INTRODUCTION

Many angiologists and vascular surgeons consider that postthrombotic syndrome (PTS) should only be managed noninterventional treatment. Unaware of the real possibilities provided by operational treatment, they limit investigation to color duplex scanning (CDS), which in this perspective is logical and reasonable. Compression, drugs, and lifestyle recommendations do not call for level III investigation according to the CEAP classification.[1]

Conservative treatment as far as etiology is concerned relies mainly on the signs and symptoms, independently of the location, extension of anatomical lesions, and pathophysiological abnormality. Conversely, when surgical or endovenous treatment is considered, the CEAP A and P descriptors must be defined precisely and the severity of PTS as a whole must be evaluated by accurate and informative investigations.

## AIM OF THE ARTICLE

To provide guidelines for undertaking investigations according to the patient's clinical presentation.

## CHRONIC VENOUS INSUFFICIENCY INVESTIGATIONS

These investigations have been recently defined in special issues of the Journal of Vascular Surgery and International Angiology.[2,3]

1. Investigations providing morphological and anatomical information:
   - Ultrasound investigations including color duplex scanning(CDS) and intravascular ultrasound (IVUS)
   - Ascending and descending phlebography
   - Spiral computed tomography
   - Magnetic resonance imaging

**Keywords:**
investigation, postthrombotic syndrome

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2. Investigations providing hemodynamic information and global information on chronic venous insufficiency (CVI) severity:
- CDS
- Pressure measurements: ambulatory venous pressure with and without tourniquet, arm/foot venous pressure differential, reactive hyperemia, foot venous pressure elevation, femoral vein pressure
- Plethysmography: photoplethysmography, air plethysmography (APG)

3. Investigations providing information on the microcirculation:
- They are not used in daily practice.

## INDICATIONS FOR INVESTIGATIONS ACCORDING TO CLINICAL STATUS

1. In patients presenting PTS with few symptoms and without moderate or severe edema according to venous clinical score[4] and/or skin changes, investigations can be limited to CDS. The same policy is recommended at follow-up visits when the patient is stable with or without compression.

2. When a patient compliant with suitable compression therapy is still symptomatic—persisting severe pain, venous claudication—or presents with severe edema, progressive worsening skin change, or recurrent ulcer, standard CDS should be completed by complementary investigations in the absence of severe systemic disease or when calf pump function cannot be improved (stiff ankle, calf muscular atrophy). The following investigations should be undertaken in order.

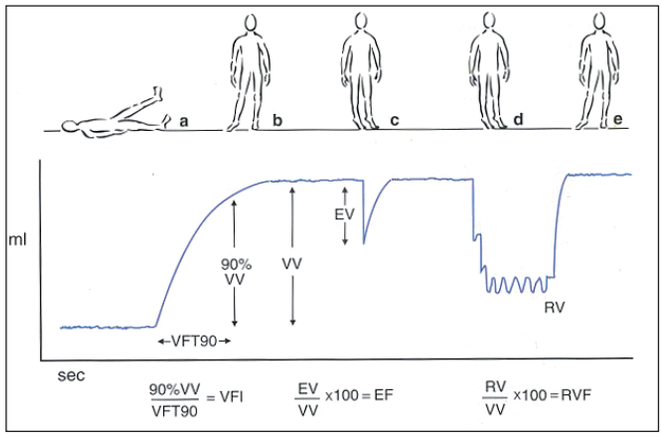



*Figure 1. Air plethysmography*
*The different maneuvers and the parameters*
*VV= venous volume; VFT= venous filling time;*
*VFI= venous filling index*
*EV= ejected volume; EF= ejection fraction; RV= residual volume*
*RVF= residual volume fraction*

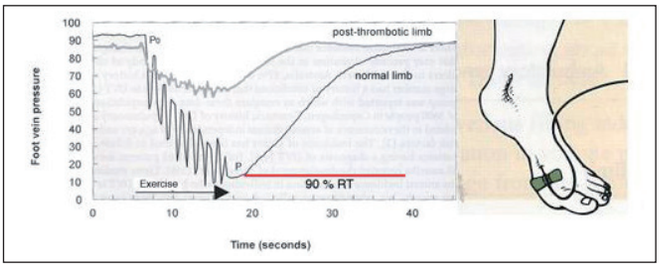



*Figure 2. Ambulatory venous pressure measurement*
*A needle is inserted in a vein on the dorsum of the foot with the patient standing.*
*Pressures are recorded before exercise and during a ten tiptoe exercise. The ambulatory venous pressure (AVP) is defined as the lowest pressure reached during the exercise.*
*P0= pressure before exercise; P= AVP; RT= refilling time*

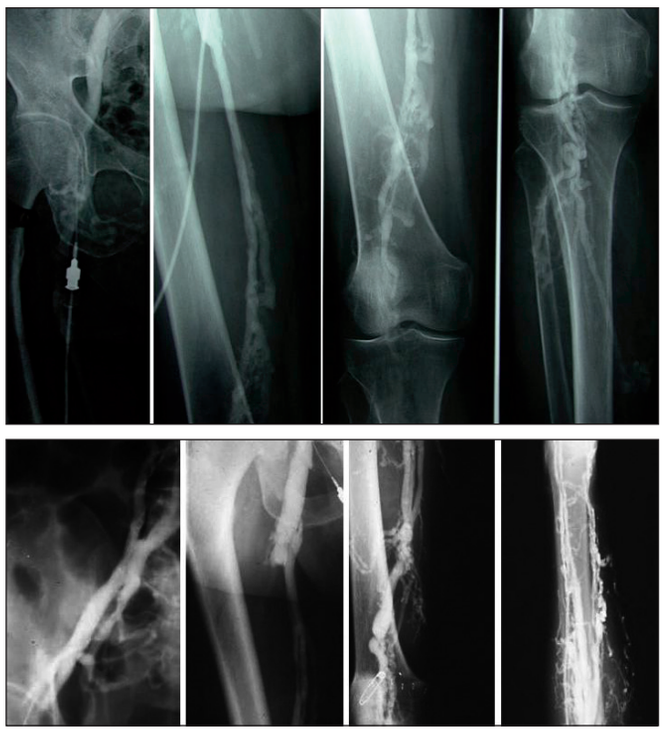


*Figure 3 and 3b. Descending venography*
*Deep axial reflux grade 4 according to Kistner*
*Kistner, RL, Ferris RG, Randhawa G, Kamida CB. A method of performing descending venography.* J Vasc Surg. *1986;4:464-468.*

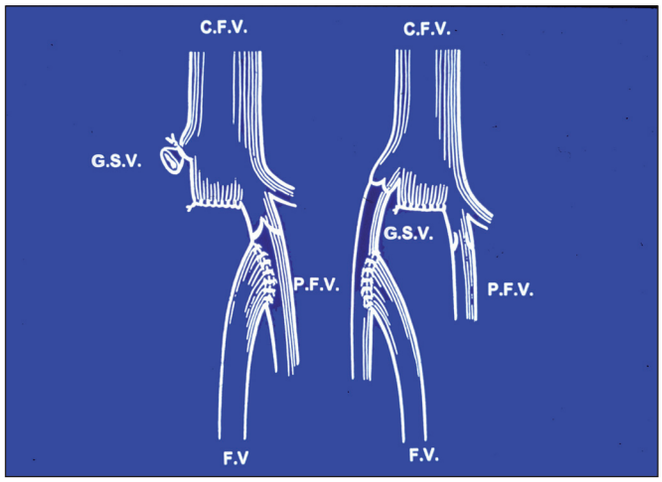



*Figure 4. Femoral vein transposition*
*GSV = great saphenous vein; FV= femoral vein; CFV= common femoral vein*
*PFV= profunda femoris vein*

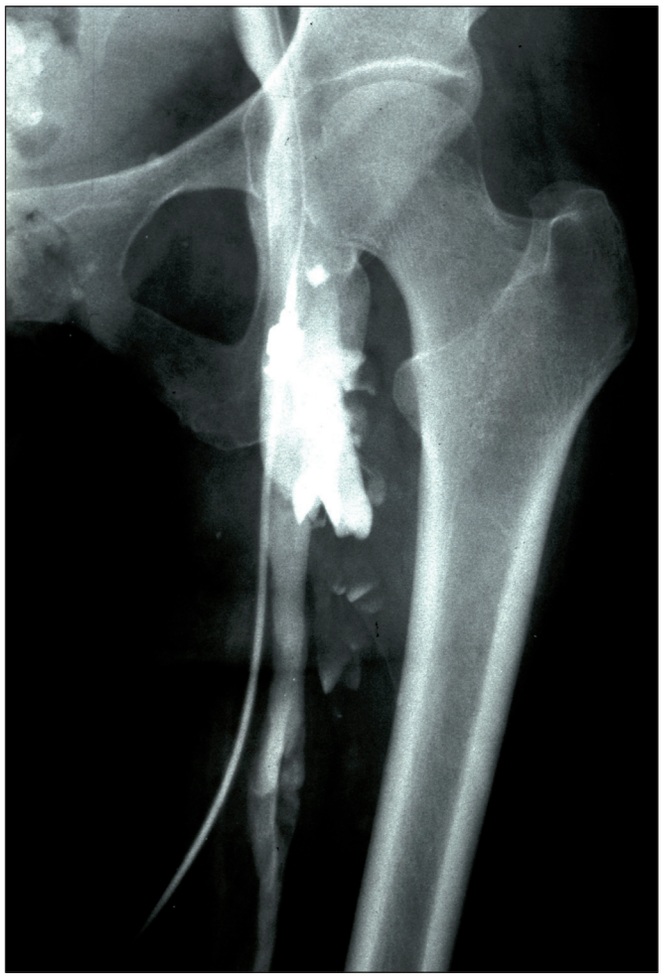


*Figure 5. Descending venography with Valsalva maneuver*
*Incompetent femoral vein. Competent profunda femoris vein*

• To evaluate hemodynamics by means of air plethysmography (*Figure 1*) and ambulatory venous pressure (*Figure 2*), but neither provides information on anatomical disorders or pathophysiological mechanisms.

• When deep reflux is identified by CDS and ascending and descending venography is decided, ascending phlebography identifies the postthrombotic changes in the deep system and the collateral patterns. Descending venography determines, first, the extent of the reflux, which is crucial for determining whether deep venous reconstructive surgery is needed as only axial reflux has to be treated (*Figures 3, 3b*), and, second, what kind of surgery is feasible:

- Femoral vein transposition (*Figure 4*) to the great saphenous vein or profunda vein, insofar as its proximal valve is competent[5] (*Figure 5*), knowing that valvuloplasty is rarely feasible in PTS
- If transposition is not feasible, phlebography combined with ultrasound scan provides information on the most suitable site for performing valve segment transplantation[6] (*Figure 6*) or neovalve construction[7] (*Figure 7*).

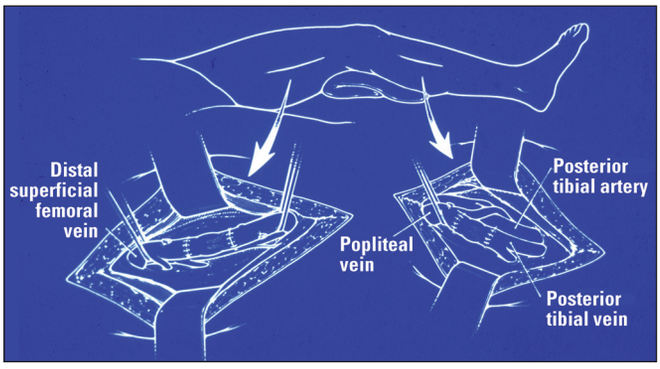
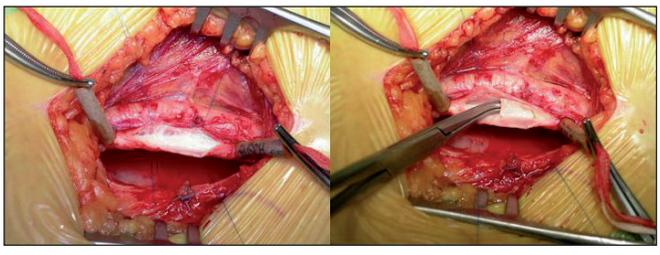



*Figure 6. Valve segment transplantation*
*Transposition to the popliteal vein above and below the knee.*



*By Courtesy Prof Maleti (Italy)*

*Figure 7. Neovalve construction according to Maleti's technique*
*Left: typical appearance of a postthrombotic vein after axial phlebotomy*
*Right: a monocuspid valve has been constructed with the thickened vein wall*

• Quantification of venous obstruction is not easy. Traditional methods measure arm-foot pressure differential,[8] outflow fraction,[9] and outflow fraction resistance by plethysmography (*Figure 8*),[10] but their specificity and sensitivity are inconsistent[11,12] and they do not quantify local anatomic distribution.

• Iliocaval obstruction is not always identified by femoral vein pressure measurement and phlebography, as both can underestimate its severity.[13] According to Neglen and Raju, intravenous ultrasound is more reliable (*Figures 9-11*),[14,15] but is invasive and expensive.

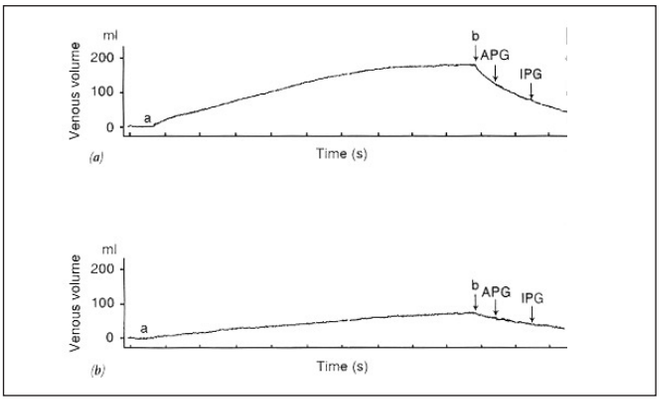



*Figure 8. Plethysmography*
*Vein obstruction is determined through calf venous capacitance and maximal venous outflow, the patient supine with the limb elevated. A thigh cuff is inflated (a) to prevent venous outflow. The thigh cuff is deflated (b). Capacitance is determined as the volume difference b-a.*
*Maximal venous outflow is determined as the difference in volume at 1 s for air plethysmography or at 3 s for impedance plethysmography.*
*Above: normal subject*
*Below: patient with venous obstruction*

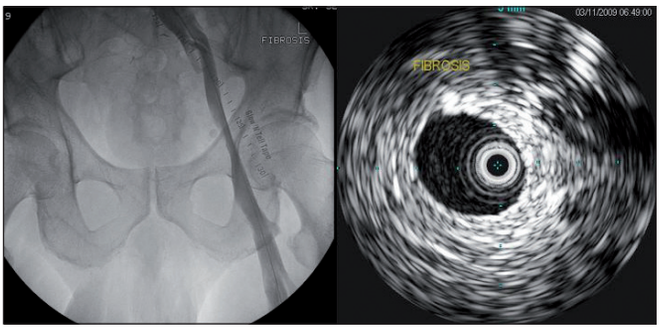



*Figure 9. Ascending venography and intravascular ultrasound (same patient)*
*Extensive wall fibrosis is evident on intravascular ultrasound which will not be suspected from the corresponding venogram.*
*(by courtesy Prof Raju, USA)*

The value of spiral computed tomography and magnetic resonance imaging is not yet clearly established.

• There is no reliable investigation for identifying infrainguinal obstructions since, as in iliocaval vein obstruction, phlebography is not reliable (*Figure 12*).

Some patterns need to be described:

- In severe PTS of the lower limb, the entire outflow seems to occur through the superficial system with nonvisualization of the deep system. This is a technical artifact despite the use of tourniquets.

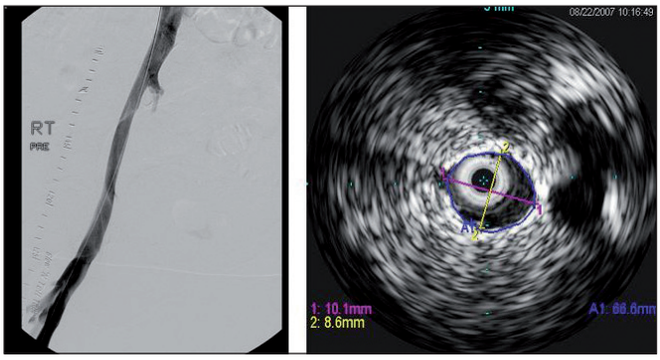



*Figure 10. Ascending venography and intravascular ultrasound (same patient)*
*Another case of diffuse postthrombotic stenosis. The venogram looked normal, but intravascular ultrasound showed a stenosed external iliac vein 10 mm x 8 mm (normal 14 mm x 14 mm).*
*(by courtesy Prof Raju, USA)*

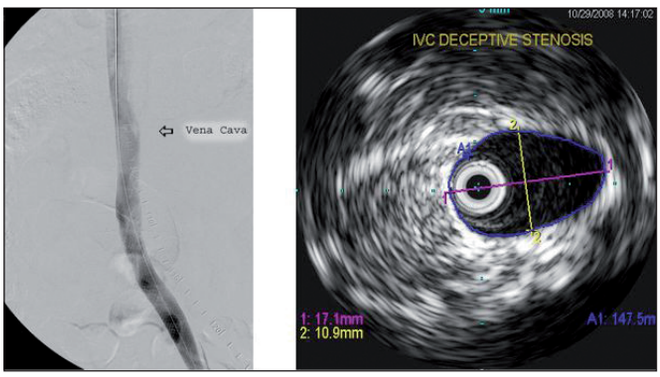



*Figure 11. Phlebography and intravascular ultrasound of inferior vena cava (same patient)*
*This patient had stenosis of the inferior vena cava, which was not picked up on venography. The inferior vena cava looks normal, but on intravascular ultrasound its maximum diameter was only 17 mm. For an adult male, this should be around 23 mm.*
*(by courtesy Prof Raju, USA)*

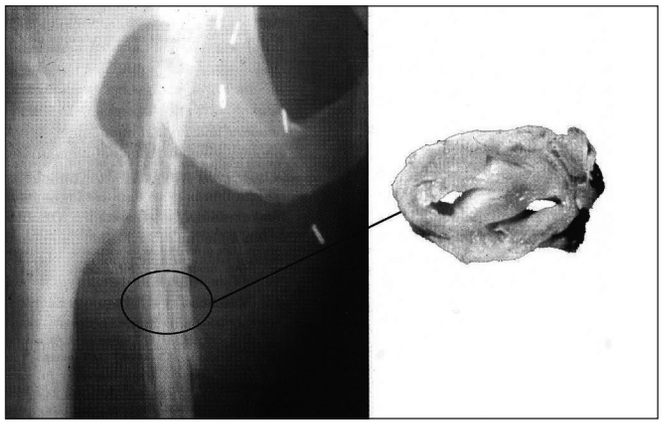


*Figure 12. Left: Ascending venography*
*Intraluminal trabeculations are apparent, although the vein appears to be patent.*
*Right: Venous pathology. The vein lumen is reduced to 2 narrow channels.*

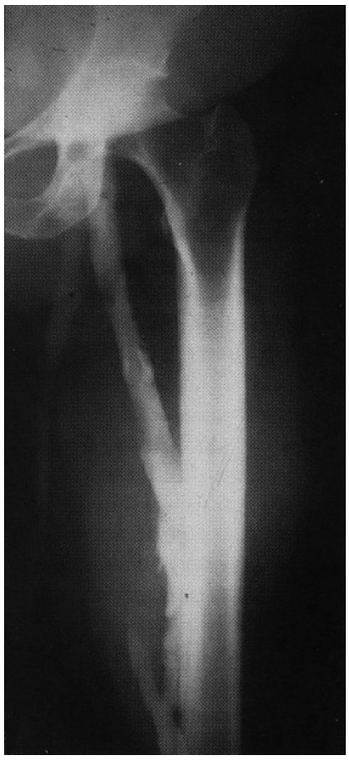


*Figure 13. Ascending phlebography*
*Axial transformation of the profunda vein.*
*(by courtesy Prof Raju, USA)*

- When the femoral vein is occluded, the profunda enlarges leading to axial transformation of the profunda femoris vein (*Figure 13*).[16]

Fortunately, collateral formation seems to compensate better for femoro-popliteo-crural venous obstruction than for obstruction of the iliac and common femoral veins. Furthermore, there is no efficient treatment of infra-inguinal venous obstruction, although good results with endophlebectomy have been reported in a small series of patients.[17]

3. In PTS, obstruction and reflux are frequently associated and unfortunately there is no investigation that identifies the most important pathophysiological factor. Nevertheless, the consensus is to treat first iliocaval obstruction, when combined with below inguinal reflux, as the successful treatment of the former generally improves the latter.

## CONCLUSION

Investigations in PTS must be tailored to the clinical situation and undertaken according to the planned treatment. Medical angiologists must realize that surgery and endovascular procedures can improve severe PTS not stabilized by conservative treatment.[7,15] knowing that the key for to successful operative treatment is thorough investigation performed by a skilled and trained unit.

**Address for correspondence**
Michel Perrin
Vascular Surgery
26 Chemin de Décines
F-69680 Chassieu
FRANCE

E-mail: m.perrin.chir.vasc@wanadoo.fr

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# Anatomical distribution of tissue fluid and lymph in soft tissues of lower limbs in obstructive lymphedema—hints for physiotherapy

Waldemar L OLSZEWSKI[1,2,3]
Pradeep JAIN [4]
Govinda AMBUJAM[4]
Marzanna ZALESKA[1]
Marta CAKALA[1]

1. *Department of Surgical Research and Transplantology, Medical Research Center, Polish Academy of Sciences, Warsaw, Poland*
2. *Department of Gastrointestinal and Transplantation Surgery, Central Clinical Hospital, Ministry of Internal Affairs, Warsaw, Poland*
3. *Rikshospitalet / Norwegian Radium Hospital, Oslo, Norway*
4. *Indian Lymphology Centers, BHU Varanasi and TMC Thanjavur*

## ABSTRACT

Knowledge of the exact location of tissue fluid (TF) and stagnant lymph (L) in lymphedema is indispensable to rational physiotherapy and specifically defines where to apply external pressure and how much. We visualized the "TF&L" space in the skin and subcutaneous tissue of the foot, calf, and thigh in various stages of lymphedema, using special staining techniques, in specimens obtained during lymphatic microsurgical procedures or tissue debulking. With the collecting trunks obliterated, L was present only in the subepidermal lymphatics, whereas the bulk of mobile TF accumulated in the spontaneously formed spaces in the subcutaneous tissue, around small veins, above and below the muscular fascia. Deformation of subcutaneous tissue by free fluid led to formation of multiple interconnecting tissue channels. Thus, massaging of tissues can propel TF through the spontaneously formed tissue channels, but not the partially or totally obliterated lymph collectors. The subepidermal lymphatic network conducts only a small fraction of L. Pneumatic compression therapy promoted formation of TF fluid channels.

## INTRODUCTION

Lymphedema is a symptom of morphologically or functionally insufficient lymph transport. There are several etiological factors damaging the lymphatic pathways. Infections and trauma of limb skin and deep tissues evoke reaction of peripheral lymphatics and lymph nodes.[1-4] Gradually, lymphatic structures become destroyed, tissue fluid transport toward and along lymphatics slows down, and edema of the dermis, subcutaneous tissue, as well as the muscular fascia and muscles gradually develops. Besides inflammation and trauma, the iatrogenic damaging factors for lymphatics are surgery and irradiation of lymph nodes in cancer therapy. Subsequently, changes in tissue and collecting lymphatics similar to those observed after infection and trauma develop (*Figure 1*).[5,6] In addition, the remaining inguinal lymph nodes atrophy due to lack of antigenic stimulation by antigens in afferent lymph. The degree of edema depends on whether obstruction affects the superficial or deep lymphatic system or both. Damage to the superficial collecting trunks

**Keywords:**
tissue fluid, lymph, lymphedema, lower limbs.

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is followed by edema of the skin and subcutaneous tissue, whereas obstruction of both drainage systems brings about fast and difficult to control accumulation of tissue fluid not only in superficial tissues, but also under the muscular fascia and between muscle fibers.

Our image of the limb lymphatic network in physiological conditions as well as lymphedema is based on lymphograms or lymphoscintigrams depicting the superficial and deep systems and lymph nodes.[7,8] These techniques do not visualize minor lymphatic structures under the epidermis where stagnant lymph accumulates. Direct lymphangiography with fluorescent tracers may help delineate minor dermal lymphatics, but is rarely used as it requires special equipment.[9] Ultrasonography, computer-assisted tomography, and magnetic resonance imaging (MRI) visualize tissue spaces filled with stagnant tissue fluid, but do not show lymphatics.[10-12] None of these methods provides a full view of the TF&L space. It is difficult to imagine how tissue fluid, in the areas with obstructed main lymphatics, finds its way to the normal uncongested tissue regions and is absorbed there. So far, only anatomical dissection and histological processing of biopsy material can visualize the tissue lymphatic network and the sites of accumulation of excess mobile tissue fluid.

In this study we visualized and calculated the volume of the TF&L space in skin and subcutaneous tissue of the foot, calf, and thigh in obstructive lymphedema stages III and IV in specimens obtained during lymphatic microsurgical procedures or tissue debulking. In order to follow the development of TF channels, lymphoscintigraphy and biopsies were performed in two patients before and after 100 sessions of pneumatic compression. The recorded observations provide useful hints for designing pneumatic devices and rational manual lymphatic massage to move stagnant tissue fluid toward unswollen regions.

## MATERIAL AND METHODS

### Tissue specimens

Groin, calf, and foot skin and subcutaneous tissue and inguinal lymph node specimens were obtained from 20 randomly selected patients with lower limb obstructive lymphedema stages III and IV, successively, as they came to our outpatient clinic for elective lymphovenous shunt or debulking surgery. Controls were specimens from 12 orthopedic patients with normal limbs operated upon for correction of fracture malunion. Fragments of inguinal lymph nodes were harvested during the lymphovenous shunt operations.

Lymphedema developed spontaneously or after an episode of dermatitis or following infected foot abrasion. At the time of admission, swelling had been present for an average of 7±1 years. Sixty percent of patients had experienced at least one attack of recurrent dermatolymphangioadenitis over the previous year and were treated with antibiotics. Staging of edema was based on our own classification.[13] Briefly, stage 1 corresponds to edema of foot, pitting subsiding after rest, stage 2 to edema of foot and up to the mid calf, only partly subsiding from the foot, stage 3 to nonsubsiding edema of foot and calf, hyperkeratosis of toe skin, and stage 4 to edema of entire limb, and hard foot and calf skin. All patients underwent limb lymphoscintigraphy performed with 99Tc labeled aggregated albumin (Nanocoll, Amersham, Switzerland). No superficial collecting trunks could be visualized in any case. MRI was performed to evaluate the thickness of the subcutis and its water content. We excluded specimens from patients with acute dermatolymphangioadenitis, skin ulcers, chronic venous insufficiency, limb ischemia, lipedema, and rheumatoid arthritis. The study was approved by the ethics committee of the Warsaw Medical University and the Indian Council for Medical Research. Oral informed consent was obtained.

### Soft tissue staining for visualizing the lymph and tissue fluid space

Sites of accumulation of stagnant lymph and tissue fluid in the interstitial space were visualized by injecting the composite skin, subcutaneous tissue, and fascia blocks with Paris blue dye in chloroform suspension.[14,15] Fragments of lymph nodes were injected under the capsule. Large particles of this dye specifically enter lymphatics, but not blood vessels. They are retained in dilated free tissue spaces and stain their walls. The injected tissue fragments were placed in 5% formaldehyde, treated with increasing concentrations of ethyl alcohol, and made translucent using methyl salicylate solution. One to three hundred thick fragments were sectioned and investigated under the light transmission microscope. The surface area of stained structures was measured under the light microscope,

magnification x100, using Olympus Microimage software (Olympus, Japan), and expressed as a percentage of the area of the microscopic field. The longitudinal and vertical lengths of stained spaces were measured to calculate their volumes and were expressed as a percentage of tissue fragment volume.

In order to prove that the stained spaces were not blood vessels, five-by-five mm thick fragments of Paris blue–injected tissues were snap frozen at 70°C and sectioned for immunohistochemical evaluation of the bluish stained structures. They were stained with monoclonal antibodies to lymphatic endothelial cell hyaluronan receptor LYVE-1 (R&D, Europe) and FVIII and CD31 (Dako, Glostrup, Denmark) to identify blood endothelial cells.

## RESULTS

Lymphoscintigraphy showed in most patients lack of patent superficial and deep lymphatic collecting trunks (*Figure 1*) and MRI displayed a "honeycomb" structure especially close to the muscular fascia (*Figure 2*). Skin, subcutaneous tissue, and muscular fascia specimens obtained from these patients, stained with hematoxylin-eosin, monoclonal antibodies, and Paris blue showed dilatation of the subepidermal lymphatics and tissue fluid spaces in the subcutaneous tissue, around small veins, and in the muscular fascia (*Figure 3*).

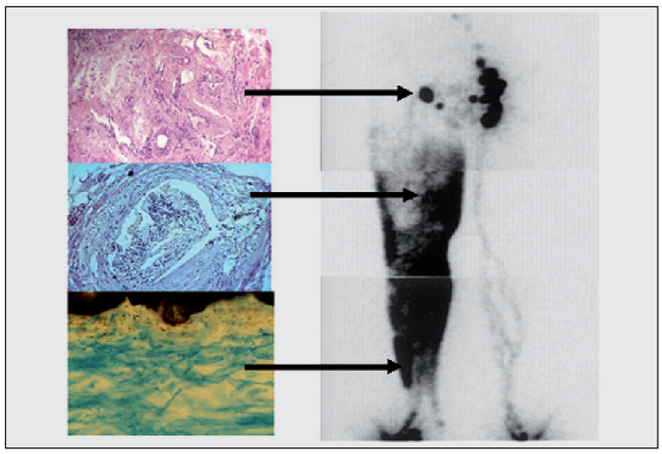


*Figure 1. Lymphoscintigraphy of damaged lymphatics and nodes and histology of biopsy material of these structures in obstructive lymphedema. Right panel: a lymphoscintigram of lower limbs in a patient 5 years after acute lymphangitis. The tracer injected into toe webs spreads in the subepidermal lymphatic plexus and tissue spaces. No collecting trunks are visible. Small solitary lymph nodes in the inguinal area. Left panel: histological picture of the biopsied node shows fibrosis and irregularly shaped lymph channels (x200). Below a picture of a collecting trunk almost totally obliterated, filled with a clot with mononuclear infiltrates (H-E staining, x200). Bottom picture depicts irregular network of subepidermal lymphatics (Paris blue staining, x100)*

*Figure 2. Magnetic resonance image of lower limb obstructive lymphedema stage III. Thickened skin and wide layer of subcutis of a honeycomb appearance. Biopsy material was taken from this region for histological evaluation of spontaneously formed "tissue channels" (frame).*

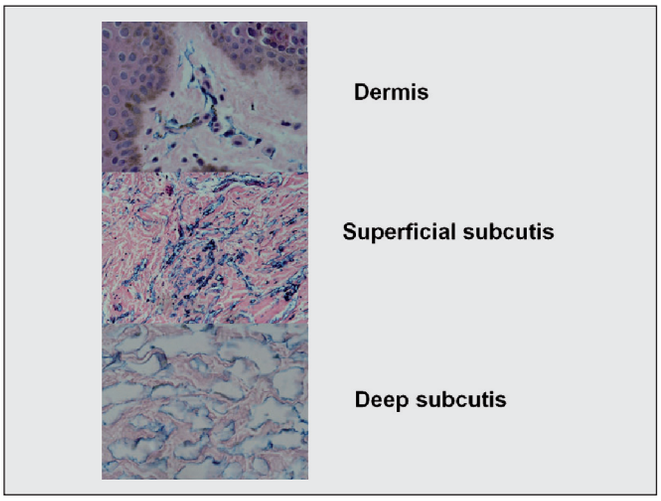



*Figure 3. Histological picture of calf epidermis, dermis, and subcutaneous tissue in obstructive lymphedema stage III. Specimen stained with Paris blue and H-E. Thick epidermis composed of 10-15 layers of keratinocytes. Bluish stained minor structures in the papillary dermis are multiple small dilated subepidermal lymphatics. In the subcutaneous tissue, bluish stained wide spaces filled with fluid are seen. Deeper in the subcutis these spaces become larger (x200).*

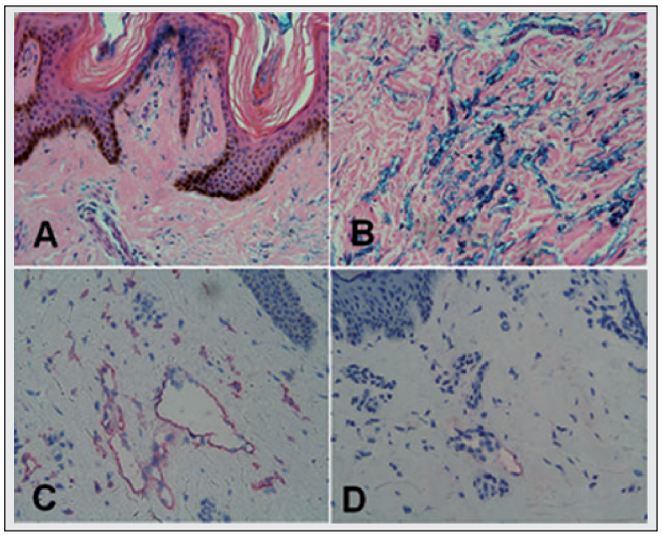



*Figure 4. Subepidermal lymphatics of calf skin of the patient as in Fig. 2. A. Thick epidermis, multiple small openings in papillae stained by intravascular injection of Paris blue (x100). B. Reticular layer of skin. Multiple bluish stained spaces—minor lymphatics and tissue spaces (x100). C. Dilated skin lymphatics stained with anti-LYVE-1 monoclonal antibody (x200). D. In other fragments they look partly obliterated, with some infiltrates (x200).*

## Subepidermal lymphatic plexus

The dilated subepidermal lymphatic plexus could be easily discriminated from blood vessels by positive LYVE-1 staining and by their shape (*Figure 4*). This plexus could be stereoscopically visualized by intradermal injection of Paris blue in chloroform suspension (*Figures 5A,B*). The venous pattern of the same skin region looked totally different from lymphatics (*Figure 5D*). In the course of lymphedema, the subepidermal plexus was undergoing gradual destruction, its deeper vessels becoming obstructed.

## Subcutaneous tissue and tissue fluid channels

Most stagnant tissue fluid accumulated in the deepest layers of the subcutaneous tissue composed of fibrous and fat tissue. Excess tissue fluid deformed tissue structures leading to formation of irregularly shaped channels (*Figures 6A,B*). Their walls were not lined with lymphatic endothelial cells and were not stained by monoclonal antibodies against LYVE-1. With progression of lymphedema, the subcutaneous space became richer in fibrous structures, newly formed channels closed

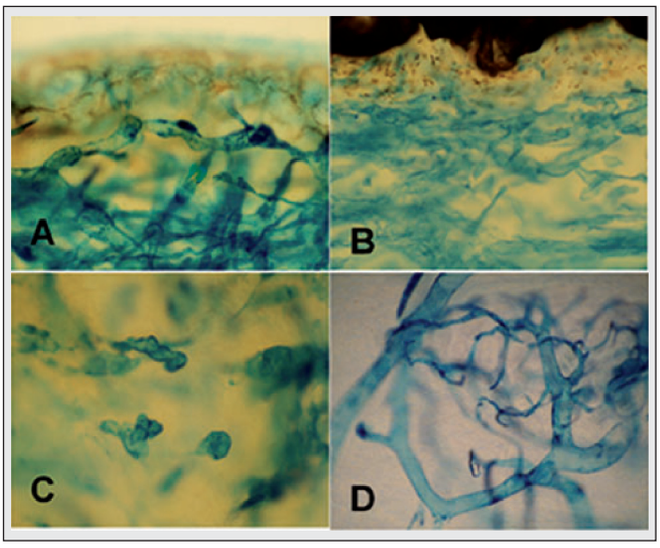



*Figure 5. Skin subepidermal lymphatics stained with Paris blue in A. Venous obstruction and B,C lymphedema stage IV (x100). A. Multiple dilated lymphatics. B. Underneath epidermis a dense network of small patent lymphatics. C In reticular layer most lymphatics are obliterated. D. Retrograde injection of the dye visualized veins forming a network of small vessels merging with larger vessels. Venous architecture is different from that of lymphatics.*

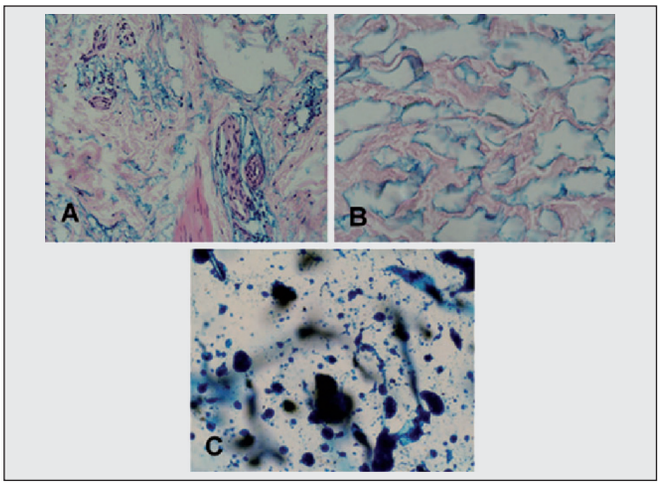



*Figure 6. Calf subcutaneous tissue at the border with muscular fascia (same patient as in Fig. 2). A. Large spaces around small veins and between collagen bundles (x100). B. Large spaces are dilated artificial tissue spaces. They are not lined by lymphatic endothelial cells (LYVE-1–negative) (Paris blue staining, x100). C. Following 100 1-hour 120 mm Hg compression episodes, new channels are being formed in the subcutis. These are multiple round spaces filled with Paris blue. They look different from those in B without massage therapy. (x100).*

down, and fluid accumulated in narrow spaces between collagen bundles.

### Perivascular spaces and muscular fascia

Mobile tissue fluid was also found in the perivascular areas (*Figure 6A*). Fluid accumulating in the thickened fascia formed multiple narrow longitudinal channels.

### Quantitative evaluation of TF&L volume

Quantitative evaluation of the surface and volume of dilated subepidermal lymphatics and spontaneously formed tissue spaces revealed that up to 60% of tissue volume is occupied by stagnant lymph and tissue fluid (58±6% in stage III and 32±6% in stage IV). In two stage III patients, compression therapy increased the TF&L space volume to 65±6%.

### Lymph nodes

Inguinal lymph nodes revealed obliterated lymphatic sinuses (*Figure 7*). Their endothelial cells did not stain with antibodies against LYVE-1. No perinodal accumulation of fluid was seen.

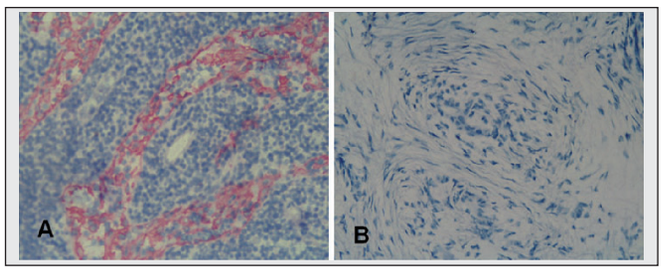



*Figure 7. Histological picture of an inguinal lymph node. A. Normal appearance, red stained node lymphatic sinuses (LYVE-1–positive) (x200). B. Fibrotic node without sinuses (x200). Lymph nodes create an obstacle for lymph flow from distal parts of the extremity.*

### The effect of pneumatic compression therapy on tissue fluid channels

On lymphoscintigraphy, performed in similar isotope recording conditions and after 100 sequential pneumatic compression sessions of one hour each at 120 mm Hg, new TF channels could be seen reaching groin level compared with pretreatment recordings (*Figure 8*). Comparison of calf subcutaneous tissue biopsies stained with Paris blue revealed after treatment round spaces not observed before massage (*Figure 6C*).

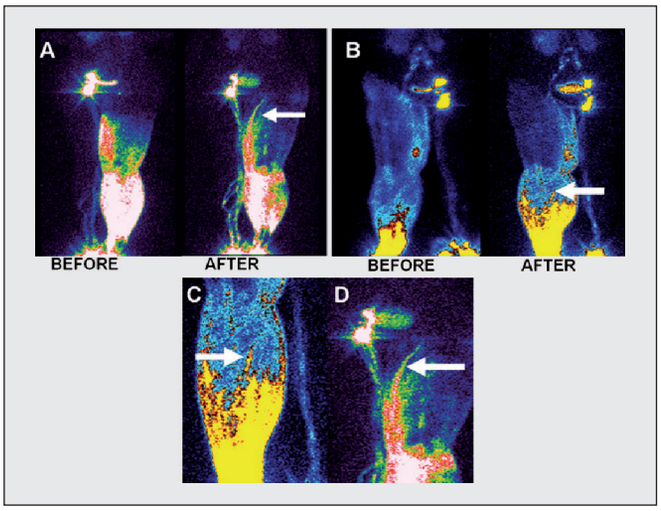



*Figure 8. Lymphoscintigrams showing formation of tissue channels after long-lasting compression therapy. A. A channel not seen before treatment reaching groin area (arrow). B. Multiple channels in the calf at higher level than before treatment (arrow). C, D. Under higher magnification, the structure of new channels differs from that of lymphatics (arrows).*

## DISCUSSION

Knowledge of where excess limb tissue fluid and lymph accumulate is indispensable for rational physical therapy. So far, this knowledge has been based on lymphoscintigraphy, ultrasonography, and magnetic resonance imaging. None of these techniques provide combined pictures of dilated lymphatics and tissue fluid–filled spaces in the dermis, subcutis, and muscles. Only anatomical dissection and histological processing of biopsy material can show the unobstructed fragments of lymphatic network and tissue spaces—sites of accumulation of mobile fluid. In our studies we visualized the sites of accumulation of stagnant lymph and tissue fluid and determined their volume. In patients with obstructed limb lymph collectors, lymph was present only in the subepidermal lymphatics, whereas mobile tissue fluid accumulated in the spontaneously formed spaces in the subcutaneous tissue, along small veins and above and below the muscular fascia. Foot, calf, and thigh skin and subcutaneous tissue of stages II-IV lymphedematous lower limbs contained similar calculated volumes of tissue fluid reaching on average 50% of the total tissue volume (*Figure 6*).

The most superficial tissue layer accumulating fluid was the subepidermal lymphatic plexus in a 200-300 µ thick papillary and reticular dermis. The volume of fluid in this plexus was negligible compared with that of the subcutaneous tissue and did not exceed 2% to 3% of total fluid retained in soft tissues (data not shown). It is unclear why subepidermal lymphatics remained patent while the collecting trunks were obstructed. However, it is known that the progressive fibrosis obliterates the plexus process in stage IV. The bulk of mobile tissue fluid accumulated in the subcutaneous tissue forming artificial interconnected spaces. These spaces were located between collagen bundles and fat globules, and around small veins. Formation of large perivascular spaces by tissue fluid could be explained by the presence of lax connective tissue in these regions, its high compliance and subsequently low resistance to fluid flow.

A new finding was formation of tissue fluid channels in and around the hypertrophic muscular fascia of the calf. This was observed in patients in whom both the superficial and deep systems of limb collectors were obstructed. There were narrow longitudinal spaces between the fascia and fibrous elements. The hydraulic conductivity of these structures was presumably high because of linear positioning of fibers.

The inguinal lymph nodes revealed major changes in the sinuses as obliteration and formation of blind spaces and depletion of lymphoid elements. High resistance to lymph flow in the fibrotic nodes could be a factor causing stagnation of lymph in the rudimentary and still patent afferent lymphatics. There was no perinodal accumulation of tissue fluid.

The volume of fluid accumulating in the tissue spaces calculated from densitometric readings of slides of the stained tissues reached 50% to 60% of tissue volume. Measuring tissue fluid content and its topographical distribution may be done using noninvasive methods as MRI. However, the resolution of MRI is still too low to show minor lymphatics, small tissue fluid "lakes", and thin fluid layers above and below fascia. Fumiere et al found that normal subcutaneous septa are seen as hyperechogenic lines in ultrasound and low signals in MRI and that hyperechogenic subcutis in ultrasound can be due to interlobular and intralobular water accumulation and/or to interlobular and intralobular fibrosis.[16] They recommended multiple imaging modalities to delineate precisely the nature of tissue water accumulation in lymphedema. Idy et al demonstrated retained water diffusely spreading over the entire dermis, and fluid retention in the interlobular spacing and beside the superficial fascia. Inside the subcutis, they identified superficial fat lobules, but not much fluid accumulation.[11] However, these images did not precisely depict the location and shape of tissue fluid–formed spaces and the structure of their walls. Our observations, based on microscopy studies of harvested tissue, supplement the knowledge obtained from noninvasive imaging on the topography of mobile fluid accumulation and the shape of channels in the edema-deformed tissue.

The recorded data provide hints on how to design the shape of pneumatic sleeves and where to press manually in order to effectively move fluid toward the unswollen regions. It can be inferred from the calculations of lymph volume in the subepidermal plexus that the superficial low-pressure massage would be ineffective in decreasing limb circumference. Lymph volume in the dermis is very low and the lymphatic network is interrupted by fibrous elements forming blind lakes. As most tissue fluid accumulates deep in the subcutis and above muscular fascia, high massage pressures should be applied to overcome resistance of fibrotic skin and reach deep limb compartments. Dilated tissue spaces do not contain valves. External pressure applied to the limb surface will also move fluid in a distal direction *(Figure 9)*. This would require special construction of sleeves preventing tissue fluid backflow. How high the massaging pressures should be depends on skin compliance and hydraulic conductivity of the subcutaneous tissue. This is the subject of our current studies.

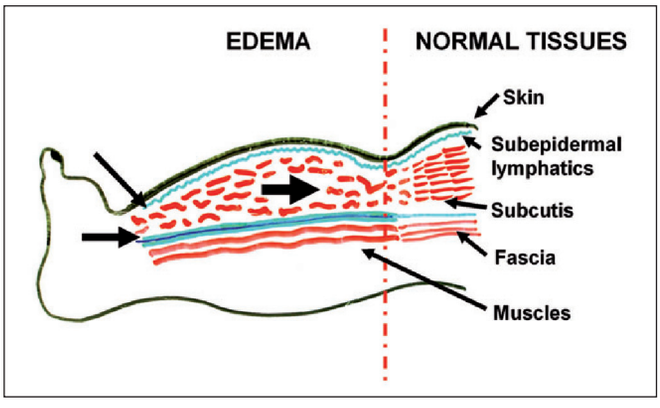



*Figure 9. Schematic presentation of the site of accumulation of lymph and free tissue fluid (subepidermal lymphatics, subcutis, and perifascial tissue spaces). Arrows show the direction of flow during pneumatic compression.*

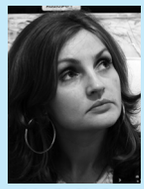
**Address for correspondence**
Waldemar L. OLSZEWSKI
Department of Surgical Research & Transplantology, Medical Research Centre
Polish Academy of Sciences
5 Pawi*f*skiego Str., 02-106 Warsaw
Poland

E-mail: wlo@cmdik.pan.pl, waldemar.l.olszewski@gmail.com


P. JAIN


G. AMBUJAM


M. ZALESKA


M. CAKALA

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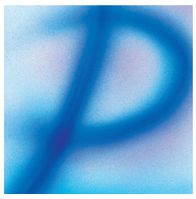

# Chronic pelvic pain associated with pelvic congestion syndrome and the benefit of Daflon 500 mg: a review*

*** This review is based on the following publication:** 1) Taskin O, Uryan I I, Buhur A, et al. The Effects of Daflon on Pelvic Pain in Women with Taylor Syndrome. *J Am Assoc Gynecol Laparosc.* 1996;3(4, Supplement):S49, and 2) Simsek M, Burak F, Taskin O. Effects of micronized purified flavonoid fraction (Daflon) on pelvic pain in women with laparoscopically diagnosed pelvic congestion syndrome: a randomized crossover trial. *Clin Exp Obstet Gynecol.* 2007;34(2):96-98.

Feza BURAK[1]
Tonguc GUNDUZ[2]
Mehmet SIMSEK[2]
Omur TASKIN[1,2]

*Departments of Obstetrics and Gynecology*
*Ìnonu University*
*1, Malatya and Akdeniz University*
*2, Antalya School of Medicine*

## SUMMARY

Chronic pelvic pain is common in women of childbearing age and causes disability and distress, which result in significant costs to health services. A specific diagnosis for the condition is often difficult because the pain may be of abdominal, neurogenic, or psychogenic origin, or may be caused by other pelvic conditions such as endometriosis, pelvic inflammatory disease, and ovarian cysts. Due to the possible interlinked factors, no diagnosis is made in 60% of patients.[1]

The pathogenesis of chronic pelvic pain is poorly understood. The vascular hypothesis offers the most likely explanation for chronic pelvic pain, which is thought to arise from overdistension of the pelvic venous system in which blood flow is markedly reduced. In pelvic congestion syndrome (PCS), pelvic varices are seen in a significant proportion of patients and may be the underlying etiology of chronic pelvic pain.[1]

Undetected severe diseases such as endometriosis, adhesions, interstitial cystitis, active chronic pelvic inflammatory disease, and irritable bowel syndrome may cause pain and should be excluded to confirm a diagnosis of PCS.

Investigation using laparoscopy is controversial since it often reveals no obvious cause of pain. Complementary investigation uses selective ovarian venography, which is commonly recommended by gynecologists. Newer, noninvasive methods such as magnetic resonance imaging and duplex ultrasound are gaining favor for the diagnosis of pelvic varices.

Using the example of micronized purified flavonoid fraction (MPFF, Daflon 500 mg, Servier, France), this review will provide an update on the diagnosis and management of PCS and on the benefits of MPFF 500 mg in the treatment of pelvic pain.

**Keywords:**
Daflon, laparoscopy, chronic pelvic pain, pelvic congestion

*Phlebolymphology*. 2009;16(3):290-294.

## INTRODUCTION

Chronic pelvic pain as part of a PCS is a common and significant disorder which affects 3.8% of women of childbearing age. The pathogenesis of chronic pelvic pain is poorly understood. Disorders of the reproductive tract, gastrointestinal system, urogenital apparatus, musculoskeletal system, and neurological system may be associated with chronic pelvic pain. Besides PCS, four other conditions account for most chronic pelvic pain: endometriosis, adhesions, interstitial cystitis, and irritable bowel syndrome. Since more than one source of pain can be found in the same patient, the diagnosis of PCS is a challenge for practitioners and is a diagnosis by exclusion of the other concomitant diseases.

At an individual level, chronic pelvic pain leads to years of disability and suffering, with loss of employment, marital discord and divorce, and numerous untoward and unsuccessful medical misadventures.[2-8] In many cases, despite extensive investigations, no obvious etiology is found, and therefore the workup begins with a good history and a thorough physical examination to make a diagnosis, so that a more tailored approach to investigations and treatment can then be provided. Combining this with a multidisciplinary approach has proven beneficial.[5-7]

## DESCRIPTION

The well-described clinical presentation is that of pain and fullness exacerbated by prolonged standing, coitus, and in the premenstrual period in multiparous women. PCS was early described as "a congestive vascular disturbance in the pelvis".[9] A more recent definition describes PCS as "chronic symptoms, which may include pelvic pain, perineal heaviness, urgency of micturition and post-coital pain, caused by ovarian and/or pelvic vein reflux and/or obstruction, and which may be associated with vulvar, perineal, and/or lower extremity varices."[10]
Pain in PCS usually begins with ovulation and lasts until the end of menses.

### Etiology and risk factors

PCS is the reflection of chronic venous disorders affecting the pelvic veins, whereby varicose dilations depending on the gonadal (uterus-ovaries) and hypogastric plexuses develop, producing pelvic varices or stasis or pelvic heaviness or a combination thereof. Clinically, it can be accompanied by vulvar varices (mainly during pregnancy), hemorrhoids, and varices in the small fossa between the pelvis and legs, on the inner thigh. The venous system is a network of preferential and secondary axes, which are all interconnected by a secondary system that can be used as a collateral system when these pressures are altered. Sometimes, this collateral system inverts the flow from the vena cava to the pelvis and lower limbs, forming escape points, and connects these systems and transmits this pressure change, so that the symptoms can be pelvic or in the legs.[11]

Pregnancy has been considered one of the major risk factors for PCS because of the relaxant effect progesterone has on the pelvic veins and because of the gravid uterus, which may increase their obstruction.[9]
In addition, there has long been an assumption that social and psychological factors play a part in PCS genesis. In the 1950s, Duncan and Taylor[4] measured vaginal blood flow at times when patients expressed anxiety, depression, or resentment, and found a decreased flow with relaxation during psychiatric interviews. They pointed to the intricacy of psychological factors in PCS, particularly in nulliparous women. Fry et al[12] reported significant associations between some social arrangements, paternal parenting, and patterns of hostility in patients with PCS. They considered that difficult father-daughter relationships and hostility patterns may have influenced the development of the condition in these patients.

### Diagnosis

The patient with chronic pelvic pain may be a candidate for laparoscopy if conservative forms of therapy (namely anti-inflammatory drugs, antibiotics, psychotherapy, and endocrine suppression) have been unsuccessful. However, objectively the decision for laparoscopy should be based on the patient's history, physical examination, and findings of noninvasive, including psychological, tests.

A detailed history and pelvic examination along with psychological interventions are complementary to the diagnosis. A bimanual pelvic examination looking for masses, tenderness, and nodularity usually reveals tenderness without induration or masses. [8,13,14]

Diagnostic laparoscopy is often carried out after referral to a gynecologist as an initial investigation to uncover pathological causes, such as endometriosis, adhesions, interstitial cystitis, and irritable bowel syndrome. Investigation by laparoscopy may show prominent enlarged broad ligament veins,[8,13,15-17] and may reveal pelvic varices. When laparoscopy findings are negative, which is not uncommon given the positional nature of the pelvic varices, further investigation using selective ovarian venography and duplex ultrasound is needed. Transuterine venography has become a standard method for the diagnosis of PCS,[5] although newer, noninvasive methods such as magnetic resonance imaging and duplex ultrasound are gaining favor for the diagnosis of pelvic varices.[1]

### Treatment

A multidisciplinary approach addressing environmental factors with physiotherapy, psychotherapy, and dietary modifications and incorporating medical management combining pain relief, hormonal therapy, and surgery is to be implemented in this challenging condition. Treatment options of presumptive PCS range from hormonal suppression, if the pain has a cyclical component, and cognitive behavioral pain management to hysterectomy. Combining this with a multidisciplinary approach has proven beneficial.[5-7]

Pelvic varices are eminently treatable, either using ovarian suppression (ligation of the gonadal and hypogastric veins, hysterectomy) or by the ligation or embolization of the pelvic veins. However, in cases of stasis or heaviness, surgical intervention is not necessary and therefore pharmacological therapy can be essential in order to treat the problem and relieve chronic pelvic pain. Thanks to their chemical composition, venoactive drugs such as MPFF 500 mg have been attributed a protective and tonic effect on the venous and capillary walls and a vein-specific anti-inflammatory effect[18] which account for their ability to effectively decrease chronic pelvic pain associated with PCS.

### Our experience with a venoactive drug

This is a review of the trials we have performed with MPFF (Daflon 500 mg, Servier, France), a drug widely used in the treatment of symptoms and edema related to chronic venous disease.

The choice of MPFF 500 mg was guided by its evidence base and its interesting characteristics. MPFF 500 mg is an oral phlebotropic and vascular protective agent consisting of an optimal ratio of 90% of diosmin (450 mg) and 10% of other active combined flavonoids expressed as hesperidin, isorhoifolin, linarin, and diosmetin (50 mg). It increases venous tone, improves lymphatic drainage, and reduces capillary hyperpermeability. MPFF 500 mg reduced reflux through pressurized veins in an animal model of acute venous hypertension, and has therefore been used clinically to treat chronic venous disease. By reducing the expression of some endothelial adhesion molecules, MPFF 500 mg inhibits the activation, migration, and adhesion of leukocytes, which leads to a reduction in the release of inflammatory mediators and thereby a reduction in capillary hyperpermeability, thus decreasing pain, heaviness, and edema.[19] Several indices of inflammation in the microcirculation are reduced by MPFF 500 mg. For these reasons, MPFF 500 mg seemed to be the most appropriate drug for our trials.

***Aim of our trials:*** to improve the clinical situation of patients with PCS, particularly chronic pelvic pain.

## PATIENTS AND METHODS

Patients with chronic pelvic pain were included after examination with transvaginal ultrasonography to exclude any disease responsible for pain, and then examined by laparoscopy during the follicular phase according to El-Minawi and El-Minawi.[20] The diagnosis of PCS in the selected patients was based on the strict criteria of prominent broad ligament and ovarian veins, without any other disease. In the case of negative laparoscopy,[20-23] a more sensitive approach such as venography or magnetic resonance imaging was performed.

## PILOT STUDY

In a pilot study, [22] we investigated the role of enlarged veins in the pathophysiology of PCS and evaluated the effects of MPFF 500 mg on pelvic pain in ten women aged 28 to 35 years. They all had prominent broad ligament and ovarian veins. The women were randomized to two groups in a double-blind trial. The treatment group received MPFF 500 mg twice a day for

4 months, and the control group a vitamin pill as placebo. Both groups were crossed over for another 4 months. The frequency and severity of lower abdominal pain and dyspareunia were scored on a scale from 0 to 6, and the results compared with pretreatment values. At the end of the fourth month, the frequency and severity of pelvic symptoms began to decrease with Daflon 500 mg compared with pretreatment and control. The mean scores were significantly less at the end of 4 months (3.9 $\pm$1.1 vs 4.2 $\pm$ 1.4, respectively, $P<0.05$). [22]

In a further trial of 20 patients,[23] the randomization was performed in two groups of 10 patients. They received either MPFF 500 mg twice a day (treatment group) for 6 months, or vitamin pills as placebo (control group) twice a day over the same period. Treatments were then crossed over for a further 6 months.[23]

Patients were asked to assess their chronic pelvic pain monthly on a 6-point scale (0 for no pain to 6 for intense pain).

## RESULTS

The demographic characteristics of patients at the inclusion are shown in Table I.[23] No difference between groups was seen regarding age, BMI, number of pregnancies, presence of pelvic varices, or pain score and duration at inclusion.

Pelvic varices were seen in more than 70% of patients at laparoscopy (*Table I*).

| | Treatment group (Mean±SD) | Control group (Mean±SD) |
|---|---|---|
| **Age (years)** | 32.3±1.7 | 33.7±2.1 |
| **Body weight (kg)** | 65.3±4.7 | 64.9±5.1 |
| **Gravida** | 3.4±1.2 | 3.1±1.9 |
| **Pelvic scores** | 5.1±0.9 | 4.9±0.7 |
| **Duration of pain (months)** | 18.1±4.5 | 19.3±6.1 |
| **Presence of peripheral varicosities** | 70% | 80% |

*Table I: Demographic characteristics of patients studied during initial randomization to either Daflon 500 mg (treatment group) or vitamins (control group) (*P>0.05)*

Right from the second month of trial 2, there was an improvement in pelvic pain scores in the treatment group compared with the control group which was significant ($P<0.05$) at the time of the crossover, ie, at month 6 (*Figure 1*).[23]

This confirms the preliminary results of the pilot study, [22] which showed a significant decrease in the frequency and severity of pelvic symptoms in the treatment group compared with the control group at month 4.

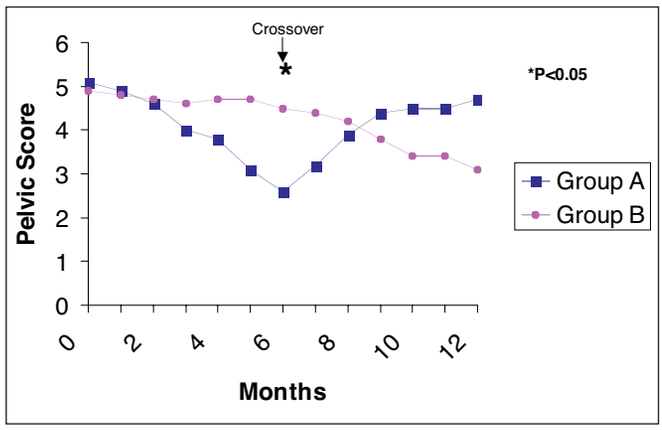


*Figure 1. Group A: Started with Daflon 500 mg and then switched to vitamins for placebo effect*
*Group B: Second arm of the study group received vitamins for 6 months and switched to Daflon 500 mg in the second half*

## CONCLUSION

Compared with placebo in two randomized, double-blind, placebo-controlled trials in 10[22] or 20 patients[23] with chronic pelvic pain diagnosed at laparoscopy with PCS with no other severe disease, MPFF 500 mg twice daily for 4 months or 6 months significantly reduced pelvic pain.

Based on our past experience, we conclude that venous dysfunction and stasis may be pathophysiologic components of pelvic pain in women with PCS. In cases of PCS, initial pharmacologic enhancement of venous tone may restore pelvic circulation and relieve pelvic symptoms such as pain and heaviness.

**Address for correspondence**
Omur TASKIN
Akdeniz University School of Medicine
Antalya
Turkey

E-mail: omurtaskin@hotmail.com

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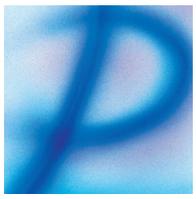

# Duplex ultrasonography protocol for investigation of patients presenting with recurrent varicose veins after surgery

Jean-Luc GILLET

*Vascular Medicine – Phlebology*
*France*

**Keywords:**
venous duplex ultrasound, post-surgical varicose vein recurrence, venous reflux.

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## SUMMARY

Duplex ultrasonography has become the investigation of reference in patients presenting with recurrent varicose veins after surgery. It consists of three methods of investigation: B-mode ultrasound, pulsed duplex ultrasound, and color duplex scanning. A complete investigation of the different venous systems is necessary:

- the deep venous network, identifying any abnormalities that may be postthrombotic or a primary cause
- the superficial venous network and the perforator veins, identifying the origin(s) of the deep vein reflux to the superficial venous network, and also performing a complete, anatomical, and hemodynamic evaluation of the varicose vein network

Duplex ultrasonography enables precise anatomical and hemodynamic diagnosis of recurrent varicose veins. A precise description of different types of varicose vein recurrence, in particular, in the former saphenofemoral and popliteal junctions, is beyond the scope of this protocol.

Duplex ultrasound scanning has become the investigation of reference in patients presenting with post-surgical recurrent varicose veins. It consists of three methods of investigation: B-mode ultrasound, pulsed duplex ultrasound, and color duplex scanning.

A complete investigation of the different venous systems is necessary.

### A- Investigation of the deep venous network

The examination should investigate the totality of the deep vein network from the inferior vena cava to the calf veins in the lower one-third of the leg. Abnormalities of the deep veins can be postthrombotic[6] or primary in origin.[7] Postthrombotic abnormalities can be obstructive and responsible for venous reflux or combined obstruction + reflux. The examiner should first look for

anatomical anomalies using B-mode ultrasound, and then hemodynamic abnormalities.

1) A normal vein is totally compressible with the ultrasound transducer. Postthrombotic abnormalities are suggested by a non-compressible or partially compressible vein, hyperechoic parietal calcifications, or endoluminal fibrous formations (*Figure 1*). Special attention should be paid to the femoral vein (former superficial femoral vein) in the thigh which usually has several large branches, only one of which can be affected by postthrombotic abnormalities. Investigation of the calf veins requires careful adjustment of the different ultrasound parameters.

2) Hemodynamically, the absence of venous flow or the existence of diminished flow compared with the contralateral veins indicates an obstructive effect of postthrombotic abnormalities. Deep vein reflux of duration greater than 1 second (*Figure 2*) is considered significant.[8-11] Reflux of duration greater than 0.5 seconds is considered significant for the deep femoral vein and the calf veins, whether it involves the posterior tibial crural veins, the anterior tibial or fibular veins, the gastrocnemius calf veins (former triceps sural veins), or the soleus veins.[8-11]

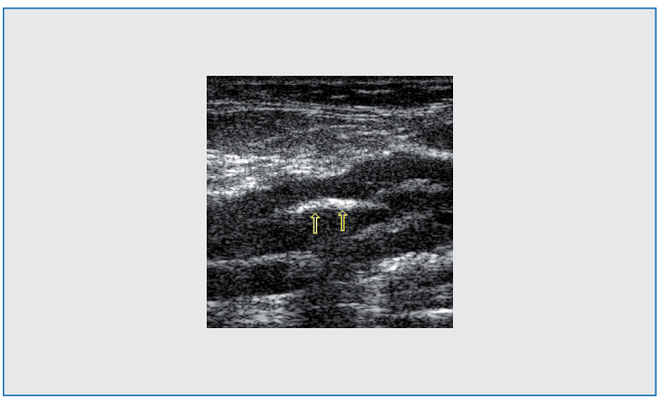

*Figure 1: Visualization with B-mode ultrasonography of post-thrombotic endoluminal fibrous formations.*

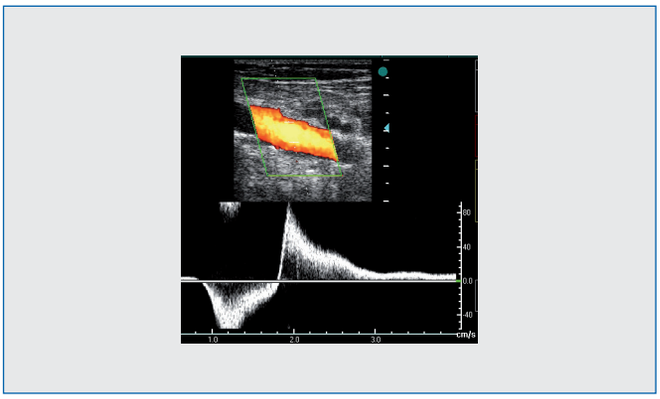

*Figure 2: Identification with color duplex ultrasound and pulsed duplex ultrasound of a reflux greater than 1 second in a popliteal vein.*

3) Methodology of the examination

The investigation consists of 3 phases:

1- Investigation of the inferior vena cava and the iliac veins is carried out with the patient supine. In a normal subject, gentle pressure exerted by the ultrasound transducer completely compresses the veins. In color and pulsed mode ultrasound scanning, the examiner checks the spontaneous blood flow, modulated by respiration and accelerated by manual compression of the thigh muscles. Then, with the Doppler transducer successively placed on the two common femoral veins, the examiner looks for blood flow modulated by respiration and by a Valsalva maneuver: abrupt stoppage of venous flow followed by its rapid resumption at the end of this maneuver is an important finding predictive of normal iliocaval patency.

With the patient still supine, the examiner looks for the following:

* reflux in the common femoral vein and in the deep femoral vein with a Valsalva maneuver

* postthrombotic anatomical anomalies with B-mode ultrasound in the common femoral, femoral (former superficial femoral) and deep femoral veins.

2- With the patient standing on a phlebology step stool, with weight-bearing on a handrail, knees slightly flexed and muscles relaxed, the examiner will look for reflux in the femoral and popliteal veins, and then in the calf veins, by exerting manual compression on the calf. Different investigation protocols have been proposed. Some teams investigate patients in the supine position, and in the Trendelenburg position.[12] But a consensus has emerged for the need to look for venous reflux in standing patients.[10] The use of a compression sleeve allows standardization of the method and enables a reproducible duration of reflux to be obtained.[10] However, manual compression is

unquestionably more suitable for everyday practice and several authors have emphasized that the sensitivity of this method is comparable to that of pneumatic compression.[8,10,13]

3- The patient is then examined seated on the edge of the examining table[8,9] with his feet resting on a stool. By compressing the base of the calf, the examiner will look for reflux in the various deep veins of the leg.

## B- Investigation of the superficial venous network and the perforator veins

Investigation of the superficial venous network in a patient presenting with a post-surgical recurrent varicose vein should be precise and thorough. The origin of deep vein reflux into the superficial venous network is identified, attentively examining the former saphenofemoral and saphenopopliteal junctions and the perforator veins, and a complete, anatomical and hemodynamic evaluation is made of the varicose vein network, using the new anatomical nomenclature to describe it.[14]

There is no consensus on quantification of venous reflux. We consider that a vein in the superficial venous network is varicose when its diameter, measured with B-mode ultrasound in transverse section, is greater than 3 mm, and when, after a compression test of the muscles upstream or a Valsalva maneuver, it presents reflux of duration greater than or equal to 0.5 seconds.[10] Like most authors, we consider that a perforator vein is incompetent when, after manual compression of the lower one-third of the thigh or of the leg, there is reflux of duration greater than or equal to 0.5 seconds[15], although a recent study has suggested that this figure can be decreased to 0.35 seconds.[10] As in the case of screening to detect deep vein reflux, the patient was examined standing[10] and then seated on the edge of the examining table with his feet resting on a stool, to investigate the perforator veins in the lower calf.

*1- At the former saphenofemoral junction*, incomplete or distant GSV stripping (crossectomy) should be differentiated from neovascularization. The description of the different types of neovascularization observed is beyond the scope of this protocol[4,16-19]. Duplex ultrasound investigates the *collateral veins*, which drain the abdominal wall and the pelvis (superficial epigastric vein, superficial external pudendal vein, deep pudendal vein, superficial iliac circumflex vein).

*2- In the thigh*, duplex ultrasound looks for leakage points in *the perineal veins*, which are often the origin of varicose vein recurrence in the medial aspect of the thigh, and *the perforator veins*, which can run along the entire length of the thigh, with special attention given to the perforators in the femoral canal (*Figure 3*). The perforator vein can be centered, but often the anatomy is more complicated.[20]

*3- In the popliteal fossa,* duplex ultrasound identifies the mechanism of the recurrence[4,9,21] whose description is also beyond the scope of this protocol.

*4- In the leg,* the different perforator veins (*Figure 4*) can be the origin of recurrent varicose veins.

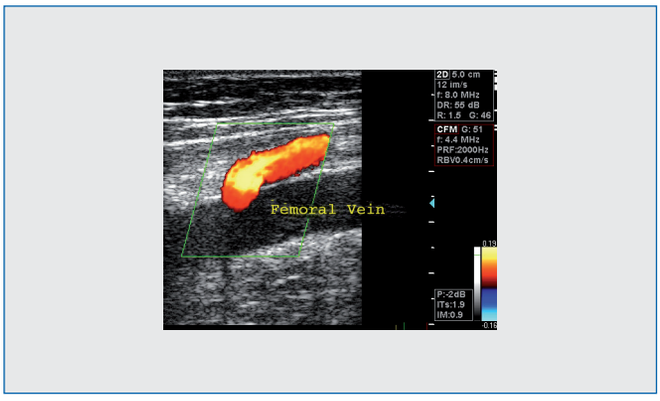


*Figure 3: Identification with color duplex ultrasound of an incontinent perforator vein in the femoral canal.*

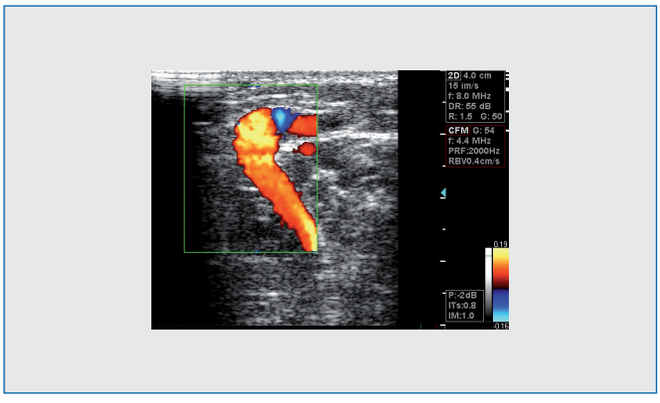

*Figure 4: Identification with color duplex ultrasound of an incontinent calf perforator vein.*

## CONCLUSION

Duplex ultrasound has become the investigation of reference in patients presenting with a recurrence of varicose veins after surgery. It should be thorough, investigate the totality of the different venous networks, and should be carried out following a strict protocol. It then allows a precise, anatomical, and hemodynamic diagnosis of post-surgical varicose vein recurrence.

**Address for correspondence**
Jean-Luc GILLET
Vascular Medicine - Phlebology
51 bis Avenue Professeur Tixier
38300 Bourgoin-Jallieu
France

E-mail: gilletjeanluc@aol.com

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# About new articles and books

Eklof B, Perrin M, Delis KT, Rutherford RB, Gloviczki P. the VEIN-Term Transatlantic Interdisciplinary Faculty: Updated terminology of chronic venous disorders: the VEIN-Term Transatlantic Interdisciplinary consensus document. *J Vasc Surg.* 2009;49:498-501.


Reasons to draw up this consensus document were legion. In the past ten years, huge efforts have been made to standardize practices and terminology, with the publication of the universally adopted Clinical, Etiological, Anatomical, Pathophysiological (CEAP) classification[1-3], together with vein nomenclature.[4-5] Despite this, many terms still create problems of interpretation, highlighting the need for a common international scientific language in the investigation and management of chronic venous disorders (CVDs).

The VEIN-Term Transatlantic Interdisciplinary Consensus Document reports recommendations for uniform usage of venous terms reached by consensus by a faculty of international experts.

A transatlantic interdisciplinary faculty of experts under the auspices of the European Venous Forum, the American Venous Forum, the International Union of Phlebology, the American College of Phlebology, the International Union of Angiology, and the Society for Vascular Surgery met in order to provide recommendations for fundamental venous terminology. A first meeting was held in the framework of *"Arctic Fjords Conference and Workshops on CVD"* aboard MS TrollFjord [2-6 October 2007], Hurtigruten, Norway, under the auspices of the European Venous Forum, the Societas Phlebologica Scandinavica, and the University of Tromsö, Norway. During this meeting, a list of problematic CVD terms was drawn up and a draft of provisional definitions was then circulated by open e-mail communications to the entire faculty for further refining comments. This process was repeated and three additional drafts were circulated prior to the second meeting held during the 20th Annual Meeting of the American Venous Forum, February 20-23, 2008, Charleston, South Carolina, USA, under the auspices of the American Venous Forum. This second face-to-face meeting produced further refinements in wording and document organization. These were incorporated into a final draft reflecting the consensus of the assembled faculty.

The consensus document was published in January 2009, meaning that the group managed to go from conception to publication in less than one year.

It includes thirty-three broadly used venous terms related to the management of CVD of the lower extremities, which were agreed to have variable applicability and interpretation in reports in the venous literature. The terms selected for inclusion in the VEIN-Term consensus document are stratified into three different groups: clinical, physiological, and descriptive.[6] It is worth noting that 13 terms had not to our knowledge been previously defined in the venous literature. They are: the acronym PREVAIT (PREsence of Varices (residual or recurrent) After InTervention), axial reflux, venous occlusion, venous obstruction, venous compression, recanalization, iliac vein obstruction syndrome, venous ablation, perforating vein interruption, perforating vein ligation, perforating vein ablation, mini-phlebectomy, sclerotherapy.

Some commonly used terms, as chronic venous disorders, chronic venous disease, chronic venous insufficiency, were clarified for the first time and given disease stage limits. For instance, a consensus was reached for "chronic venous insufficiency" as recovering patients from C3 to C6 of the CEAP classification, while some had until then used it for patients from C4 to C6 and others for all patients whatever the stage of CVD. The frequently encountered term "venous symptoms", which has had a very vague definition, is now given a description: "Complaints related to venous disease, which may include tingling, aching, burning, pain, muscle cramps, swelling, sensations of throbbing or heaviness, itching skin, restless legs, leg tiredness, and/or fatigue. Although not pathognomonic, these may be suggestive of chronic venous disease, particularly if they are exacerbated by heat or worsen during the course of the day, are relieved by leg rest and/or elevation."

The faculty managed to describe each of the 33 terms with restraint in words, capturing the essence of the definition, each word being weighed up and given a precise meaning. For instance, the term "postthrombotic syndrome" was defined as: chronic venous symptoms and/or signs secondary to deep vein thrombosis.

In conclusion, the VEIN-Term consensus document is intended to provide those involved in the management of CVD around the world, who may report their experiences in the English-language scientific literature, with clarifying refinements in venous terminology. It is to be hoped that it will result in a more precise use of venous terms in English-language articles on CVD in the future.

Nevertheless, one challenge remains, and that is to translate the English definitions as accurately as possible into other languages. This is not easy. This has already been done for the CEAP classification and the anatomic nomenclature.

Lastly, VEIN-Term has not covered all the imprecise terms in phlebology. Further meetings are needed to complete this work.

## CLINICAL VENOUS TERMS

**Chronic Venous Disorder**
This term includes the full spectrum of morphological and functional abnormalities of the venous system

**Chronic Venous Disease**
Morphological and functional abnormalities of the venous system of long duration manifested by symptoms and/or signs indicating the need for investigation and/or care

**Chronic Venous Insufficiency (C3*-C6)**
A term reserved for advanced CVD, which is applied to functional abnormalities of the venous system producing edema* skin changes or venous ulcers
[*C3*: moderate or severe edema as stratified by Rutherford et al*[7]]

**Venous Symptoms***
Complaints related to venous disease, which may include tingling, aching, burning, pain, muscle cramps, swelling, sensations of throbbing or heaviness, itching skin, restless legs, leg tiredness and/or fatigue. Although not pathognomonic, these may be suggestive of chronic venous disease, particularly if they are exacerbated by heat or dependency in the day's course, and relieved with leg rest and/or elevation

**Venous signs**
Visible manifestations of venous disorders, which include dilated veins (telangiectasiae, reticular veins, varicose veins), leg edema, skin changes, ulcers, as included in the CEAP classification[3]

**Recurrent varices**
Reappearance of varicose veins in an area previously treated successfully

**Residual varices**
Varicose veins remaining after treatment

**PREVAIT** ***(new acronym)***
This acronym means **PRE**sence of **V**arices (residual or recurrent) **A**fter **I**n**T**ervention

**Postthrombotic Syndrome**
Chronic venous symptoms and/or signs secondary to deep vein thrombosis

**Pelvic Congestion Syndrome**: Chronic symptoms, which may include pelvic pain, perineal heaviness, urgency of micturition and post-coital pain, caused by ovarian and/or pelvic vein reflux and/or obstruction, and which may be associated with vulvar, perineal, and/or lower extremity varices

**Varicocele**
Presence of scrotal varicose veins

**Venous aneurysm**
Localized saccular or fusiform dilatation of a venous segment with a caliber at least 50% greater than the normal trunk

*****<u>*Comment:*</u> *in the original article, the definition is followed by the sentence* **"**Existing venous signs and/or (noninvasive) laboratory evidence are crucial in associating these symptoms with CVD*" which conflicts with the acknowledged existence of the clinical CEAP category C0s En An Pn corresponding to those patients with leg complaints but presenting with no visible signs and without detectable pathophysiological abnormalities identifiable by routine investigations.*

*This is why we removed this paragraph in the present review.*

## PHYSIOLOGICAL VENOUS TERMS

**Venous Valvular Incompetence**
Venous valve dysfunction resulting in retrograde venous flow of abnormal duration

**Venous Reflux**
Retrograde venous flow of abnormal duration in any venous segment

**Primary:** caused by idiopathic venous valve dysfunction

**Secondary:** caused by thrombosis, trauma, or mechanical, thermal or chemical etiologies

**Congenital:** caused by the absence or abnormal development of venous valves

**Axial reflux**
Uninterrupted retrograde venous flow from the groin to the calf

**Superficial**: confined to the superficial venous system

**Deep**: confined to the deep venous system

**Combined**: involving any combination of the three venous systems (superficial, deep, perforating)

**Segmental reflux**
Localized retrograde flow in venous segments of any of the three venous systems (superficial, deep, perforating) in any combination in the thigh and/or the calf, but **NOT** in continuity from the groin to calf

**Perforator Incompetence**
Perforating veins with outward flow of abnormal duration

**Neovascularization**
Presence of multiple new, small tortuous veins in anatomic proximity to a previous venous intervention

**Venous Occlusion**
Total obliteration of the venous lumen

**Venous Obstruction**
Partial or total blockage to venous flow

**Venous Compression**
Narrowing or occlusion of the venous lumen as a result of extraluminal pressure

**Recanalization**
Development of a new lumen in a previously obstructed vein

**Iliac Vein Obstruction Syndrome**
Venous symptoms and signs caused by narrowing or occlusion of the common or external iliac vein

**May-Thurner Syndrome**
Venous symptoms and signs caused by obstruction of the left common iliac vein due to external compression at its crossing posterior to the right common iliac artery

## DESCRIPTIVE VENOUS TERMS

**High Ligation and Division**
Ligation and division of the great saphenous vein (GSV) at its confluence with the common femoral vein, including interruption of all upper GSV tributaries

**Stripping**
Removal of a long vein segment, usually most of the GSV or the small saphenous vein (SSV) by means of a device

**Venous ablation**
Removal or destruction of a vein by mechanical, thermal, or chemical means

**Perforating Vein Interruption**
Disconnection of a perforating vein by mechanical, chemical, or thermal means

**Perforating Vein Ligation**
Interruption of a perforating vein by mechanical means

**Perforating Vein Ablation**
Disconnection or destruction of a perforating vein by mechanical chemical or thermal means

**Mini-phlebectomy**
Removal of a vein segment through a small skin incision

**Sclerotherapy**
Obliteration of a vein by chemical introduction (liquid or foam)

**Endophlebectomy**
Removal of postthrombotic residue from the venous lumen

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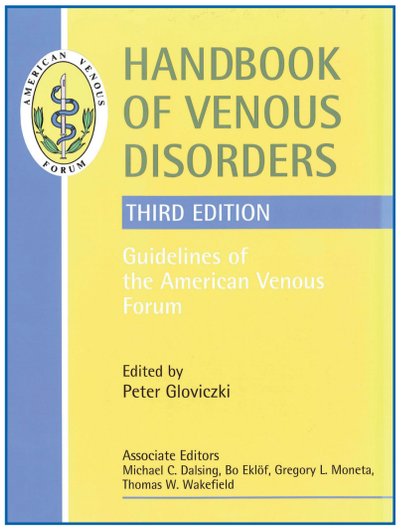



## HANDBOOK OF VENOUS DISORDERS.

Gloviczki P. Ed. *Guidelines of the American Venous Forum*. Third edition. London, UK. Hodder Arnold 2009. ISBN 978 0 340 938 805

***A review by Michel Perrin***

At first glance the third edition of the Handbook Of Venous Disorders looks like the previous ones: yellow cover, editor-in-chief Peter Gloviczki. But besides the blue on the page edges (in the previous edition it was green), the most important change is the increase in both chapters (57 to 65) and pages (from 557 to 744). As the book's subtitle is Guidelines of the American Venous Forum, most of the authors are North American, with one Australian, six Britons, and seven continental Europeans.

Following the foreword by R. Rutherford and the preface by P. Gloviczki, the first subchapter is entitled evidence-based guidelines. The reason is that in all the chapters dealing with chronic venous disorders management, that is to say investigation or treatment, recommendations from the College of Chest Physicians task force[1] bring real homogeneity to the various guidelines.

Before reading these recommendations, the reader needs to understand the important changes made in the methodology and how they were developed. The strength of the recommendation (1 : "We recommend", or 2 : "We suggest") no longer is based, as only a few years ago solely on the type and quality of available studies. It is a true judgement of the overall value of the balance between the benefits and risks incurred by following this recommendation, a judgement based on the expected benefits in terms of health, treatment-related risks, patients' values and preferences, but also on economic considerations and the allocation of resources.

Part 1 of the book is devoted to basic considerations in venous disorders. A special chapter on venous ulcer formation and healing has been added to the second edition's chapters on classification, pathogenesis, and epidemiology of chronic venous disorders.

Part 2 is devoted to diagnostic evaluations and venous imaging studies. All the ancillary and new investigations are reported in detail, including their advantages and pitfalls. Chapter 13, with its color duplex ultrasound scanning figures, and chapter 14, on computed tomography and magnetic resonance imaging, provide outstanding images of both acute and chronic venous disorders, including infrequent diseases.

Part 3 on acute management of acute thrombosis has been fully updated, and the chapter on treatment algorithms for acute deep vein thrombosis provides grade 1 recommendations to help physicians take decisions.

Part 4 is the longest—more than 200 pages—and deals with management of chronic venous disease, taking into account the most recent procedures: foam sclerotherapy, artificial valves, percutaneous ablation of perforating veins. The treatment algorithm for venous ulcer is particularly clear and informative. And two chapters are devoted to endovascular treatment of venous obstruction or occlusion. Part 4 also covers special venous problems including trauma, tumor, aneurysm, venous malformation, pelvic congestion syndrome, and superior vena cava syndrome.

**Address for correspondence**
Michel PERRIN
26, chemin de Décines
69 680 Chassieu
France

E-mail:
m.perrin.chir.vasc@wanadoo.fr

Part 5 covers lymphatic disorders: classification, investigation, noninterventional and interventional treatment.

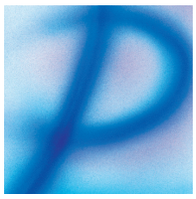
As in the previous edition, the last part covers issues such as outcome assessment in acute and chronic venous disease, but the most appealing chapter, already published in 2007[2] but now updated and more detailed, maps out the future.

Chapter 65 summarizes the American Venous Forum guidelines in a large table. It would be interesting to assess how physicians not only in the United states but all around the world comply with these guidelines in their daily practice.

The editor-in-chief and the four associate editors are all past Presidents of the American Venous Forum. They are to be congratulated for their immeasurable work in reviewing all the chapters that constitute the finest review of current knowledge on venous disease available between two hard covers.

## REFERENCES

1. Guyatt G, Gutterman D, Baumann MH, Adrizzio-Harris D, Hylek EM et al. Grading Strength of Recommendations and Quality of Evidence in Clinical Guidelines. Report from an American College of Chest Physicians Task Force *Chest.* 2006;129:174-181.
2. Meissner M, et al. *J Vasc Surg*. Proceedings of the International Summit of the 5th Pacific Vascular Symposium. 2007;46: Suppl S (1S-54S).



# Congress and conference calendar

| DATES | CONGRESS | COUNTRY | CITY |
|---|---|---|---|
| 31 August-4 September 2009 | XVI WORLD MEETING OF THE UNION INTERNATIONALE DE PHLEBOLOGIE (UIP) | Principality of Monaco | Monaco |
| 19-22 September 2009 | 18th EUROPEAN CHAPTER CONGRESS OF THE INTERNATIONAL UNION OF ANGIOLOGY Join with XIX ANNUAL MEETING OF THE MEDITERRANEAN LEAGUE OF ANGIOLOGY AND VASCULAR SURGERY | Italy | Palermo |
| 21-25 September 2009 | 22nd INTERNATIONAL CONGRESS OF LYMPHOLOGY | Australia | Sydney |
| 30 September - 3 October 2009 | XIII ITALIAN COLLEGE OF PHLEBOLOGY | Italy | Siena |
| 1-4 October 2009 | 3rd INTERNATIONAL COURSE / ULTRASOUND GUIDED ENDOVENOUS LASER / RF TREATMENT OF VARICOSE VEINS, SEMINAR AND HANDS-ON COURSE | Slovenia | Otoãec |
| 7-10 October 2009 | L NATIONAL ANGIOLOGY AND VASCULAR SURGERY CONGRESS | Mexico | Guanajuato |

| CONTACT | SECRETARIAT | WEB SITE |
|---|---|---|
| Eberhardt Rabe, PhD<br>*Chairman of scientific committee*<br>Jean-Jérôme Guex, MD<br>*Chairman of organizing committee* | Publi Créations – Partner of AIM<br>27, boulevard d'Italie<br>98000 Monaco<br>Phone no.: +377 9797 3555<br>Fax no.: +377 9797 3550<br>E-mail: uip2009@publicreations.com | www.aim-international-group.com/2009/uip |
| Claudio Allegra, PhD<br>Salvatore Novo, PhD<br>*Presidents of the 18th EUROCHAP Meeting*<br>Pier Luigi Antignani, PhD<br>Salvatore Novo, PhD<br>*Presidents of the XIX MLAVS 2009 Meeting* | AIM Group - AIM Congress<br>Via Flaminia, 1068<br>00189 Rome<br>Phone no.: +39 06 330531<br>Fax no.: +39 06 33053229<br>E-mail: eurochap2009@aimgroup.it | www.aimgroup.eu/2009/eurochap-mlavs |
| Neil Piller, PhD<br>*President* | E-mails: neil.piller@flinders.edu.au<br>lymphology@icmsaust.com.au | |
| Claudio Allegra, PhD<br>Genovese Giuseppe<br>Mancini Sergio | Servizio Congressi<br>Phone no: +39 0577-232129 | www.flebologiaitaliana.it |
| Andrej Šikovec MD, MSc | Andrej Šikovec MSc, MD<br>Phone no.: +386 7/ 30 75 107<br>Fax no.: +386 7/30 75 174<br>E-mail: avelana.pisarna@gmail.com | ww.avelana.si |
| Jacobo Nettel, MD | Jacobo Nettel<br>Phone no.: +442 1 92 30 63<br>E-mail: smacvv@prodigy.net.mx | |

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|---|---|---|---|
| 9-11 October 2009 | XI NATIONAL CONFERENCE OF BULGRIAN NATIONAL SOCIETY OF ANGIOLOGY AND VASCULAR SURGERY | Bulgaria | Zlatni piasatzi, Varna |
| 23-24 October 2009 | 2nd SCIENTIFIC MEETING OF THE D.R.I.M.S. MEDICAL ASSOCIATION ADRIATIC VASCULAR SUMMIT 2009 | Slovenia | Ljubljana |
| 23-25 October 2009 | II CONGRESS OF SERBIAN COLLEGE OF PHLEBOLOGY | Serbia | Belgrade |
| 5-7 November 2009 | VI LATIN AMERICAN VENOUS FORUM CONGRESS | Mexico | Guadalajara |
| 5-8 November 2009 | 23rd ANNUAL CONGRESS OF THE AMERICAN COLLEGE OF PHLEBOLOGY | USA | Palm Desert, CA |
| 25-28 November 2009 | XXXI ITALIAN SOCIETY FOR ANGIOLOGY AND VASCULAR MEDICINE (SIAPAV) | Italy | Rome |
| 21-25 April 2010 | XXIV WORLD CONGRESS OF THE INTERNATIONAL UNION OF ANGIOLOGY (IUA) | Argentina | Buenos Aires |

| CONTACT | SECRETARIAT | WEB SITE |
|---|---|---|
| Todor Zahariev, PhD<br>*President* | Veselin Petrov<br>Sofia<br>Phone no.: +359 888 358 752<br>Fax no.: +359 2 921 49 68 | |
| Igor D. Gregoriã, MD<br>Zvonimir Krajcer, MD<br>Michael Jacobs, MD PhD<br>*Program Directors* | Igor D. Gregoriã, MD<br>Phone no.: +386 1 832 355 6809<br>E-mail: igregoric@heart.thi.tmc.edu | www.adriatic-vascular summit.org |
| ARIA Conference & Events<br>*Management coordinator* | Phone no.:+381 11 3160 625<br>Fax.: +381 11 3160 862 | www.flebo.org.rs |
| Magdiel Trinidad MD<br>*drmagdiel@yahoo.com*<br>*Society President* | | |
| | Phone no.: +510 346 6800<br>Fax no.: +510 346 6808 | www.acpcongress.org |
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