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Prevention or reversal of deep venous insufficiency by aggressive treatment of superficial venous disease

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Abstract

Background: This study of patients who received either aggressive or less-aggressive treatment for superficial venous disease was undertaken to determine its effects on deep venous insufficiency (DVI).

Methods: From 1998 to 2004, we treated 1,500 consecutive patients with superficial venous disease at our outpatient care center. A total of 100 patients were available for the study; the remaining patients were not available for the complete follow-up duplex scans 6 months after therapy, irrespective of the therapeutic results. Sixty-four patients underwent aggressive therapy, which included high ligation with partial selective perforation-invagination (PIN) axial stripping of the greater saphenous vein, ambulatory stab phlebectomy of the varicose veins, and transdermal treatment of the spider veins. Thirty-six patients underwent less-aggressive treatment, which included high ligation with selective partial PIN axial stripping of the greater saphenous vein and ambulatory phlebectomy of varicose vein clusters but no spider vein treatment.

Results: Follow-up duplex scanning after aggressive treatment of superficial venous disease showed improvement or complete reversal of DVI in the majority of patients. This improvement was defined as a marked decrease in the size of the deep veins in 80% of patients and a decrease of the reflux closure time of the deep venous valves in 83% of patients. Only 28% of patients receiving less-aggressive treatment without transdermal laser therapy of the spider veins showed improvement in their reflux valve closure time; the remaining 72% were unchanged or deteriorated.

Conclusions: Aggressive treatment of superficial venous disease can prevent or even eliminate deep vein insufficiency (DVI).

Keywords: Deep venous insufficiency; Venous telangiectasias; Varicose veins; Superficial venous disease

Chronic venous diseases of the lower extremity are prevalent in United States and Europe. In the population at large, 70% of women and 40% of men suffer from these diseases in one form or another [1,2]. Venous insufficiency can manifest in the superficial or deep veins of the legs and is often neglected. This may also be associated with incompetence of the major named perforator veins or minor unnamed feeding veins most commonly associated with clusters of venous telangiectasias (spider veins) [3–9]. At least 60% of patients with spider veins have significant symptoms that may include pain, itching, burning, heaviness of legs, cramps in calves, spontaneous bruising of skin, or bruising with minor trauma [10].

Association of superficial venous disease (varicose veins and/or venous telangiectasias) with deep venous valvular insufficiency is well documented [11–15]. The incidence of deep venous insufficiency (DVI) associated with superficial venous disease is reported to be 20% to 60% [16,17].

Making a decision to treat venous lesions, varicose veins, and spider veins depends on the association of superficial and deep venous insufficiency with these lesions, and, if not totally eliminated, DVI may not improve and may even deteriorate over time. Patients with superficial and deep venous disease are at high risk of developing chronic leg ulcers and deep venous thrombosis. The lifetime incidence of superficial thrombophlebitis in patients with untreated varicose veins has been estimated at 20% to 50%. Venous duplex scanning of the legs is usually performed with the patient either in a horizontal position or in a slightly reversed Trendelenberg position, often masking the presence

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Table 1
Clinical signs and symptoms of superficial venous disease

Signs and symptoms	Percent
Venous telangiectasis	98
Pain	97
Swelling	93
Edema	92
Heaviness	74
Varicose veins	61
Itching	53
Burning	43
Cramping	38
Pigmentation	8
Healed ulceration	2
Bleeding	1

of DVI. When the duplex scan is performed with the patient in a standing or extreme reverse Trendelenberg position, the incidence of DVI is much higher.

Unrecognized deep vein thrombosis is present in up to 45% of patients with what appears to be a purely superficial thrombophlebitis. The risk of deep venous thrombosis has been reported to be 3 times higher in patients with superficial varicosities [16]. Approximately 1 million people in the United States have ulceration because of superficial venous disease, and about 1 of 10 are functionally disabled [18].

This retrospective study was undertaken to examine the results of treatment for superficial venous disease of the legs and the effects of such treatment on DVI. This report will show an improvement in DVI after complete aggressive treatment of superficial venous insufficiency, as clinically manifested by varicose and spider veins.

Methods

Over a period of 6 years (1998–2004), 1,500 patients with superficial venous disease were treated at an outpatient care center (Hamilton, NJ). A total of 100 patients were available for a complete follow-up. Only those patients who were symptomatic and had follow-up duplex scans were included in this study; the remaining 1,400 patients were unavailable for complete follow-up. The majority of the patients were women between the ages of 30 and 40 years. Most of the patients were overweight, and their daily activity level was low. The clinical symptoms of the patients and their physical findings are shown in Table 1. The majority of the patients had varying degrees of pain and swelling of the legs; the most common clinical findings were venous telangiectasias and edema. Each patient was categorized according to the clinical, etiological, anatomic, and pathophysiological (CEAP) classification system as shown in Table 2.

All of the patients were examined for superficial venous disease using color duplex scanning (ATL 300; Philips Medical Systems, Bothell, WA) and (Sonoline Elegra; Siemens Medical Systems Inc, Issaquah, WA) with the patient

Table 2
CEAP classification and categorization of patients

CEAP class	Description	No. of patients (n = 100)
0	No signs of venous disease	0
1	Telangiectasias	10
2	Varicose veins	10
3	Edema	80
4	Skin changes	4
5	Healed venous ulcer	2
6	Active ulceration	0

in an upright position. A complete venous study was performed, including the greater and the lesser saphenous veins, common and superficial femoral veins, and popliteal and perforator veins. The venous study documented reflux in the veins by color duplex examination, with measurements of valve closure time and the size of the veins before the treatment began. All of the patients had reflux in both the superficial and the deep veins. Once the diagnosis of the symptomatic superficial venous insufficiency was documented by clinical symptoms, physical findings, and confirmed by duplex scanning, treatment with surgical intervention began.

Sixty-four patients underwent high ligation with partial perforation-invagination (PIN) stripping, ambulatory phlebectomy, and treatment of their spider veins with transdermal laser therapy. These patients were classified as receiving complete aggressive treatment of superficial venous disease. Thirty-six patients did not receive transdermal laser therapy of their spider veins and were classified as receiving less-aggressive treatment, despite the fact that they had high ligation with partial selective stripping of the saphenous vein and, in some cases, ambulatory phlebectomy.

All of the patients had saphenous vein insufficiency and underwent high ligation and partial selective stripping of the vein up to the next competent valve assessed at surgery. The operation was performed under local anesthesia with intravenous sedation. Clusters of varicose veins were treated with ambulatory phlebectomy using very small skin incisions made with a Beaver blade knife, phlebo dissectors, and Oesch vein hooks. Multiple clusters of varicose veins were treated in separate sessions.

The aggressive-treatment group underwent therapy of spider veins and reticular veins in addition to high ligation and partial PIN stripping of the long saphenous veins. The therapy used was intense pulse light and/or Nd:YAG (1,064 nm) laser in multiple sessions 2 to 4 weeks apart. The majority of clusters of spider veins treated responded in 2 or 3 sessions. Some of the areas of very dense proliferated telangiectasias required more than 3 sessions of treatment. Every attempt was made to avoid interruptions between sessions.

Both groups of patients were required to wear groin high stockings with 20 to 30 mm Hg compression. A few patients who presented with advanced venous insufficiency (CEAP

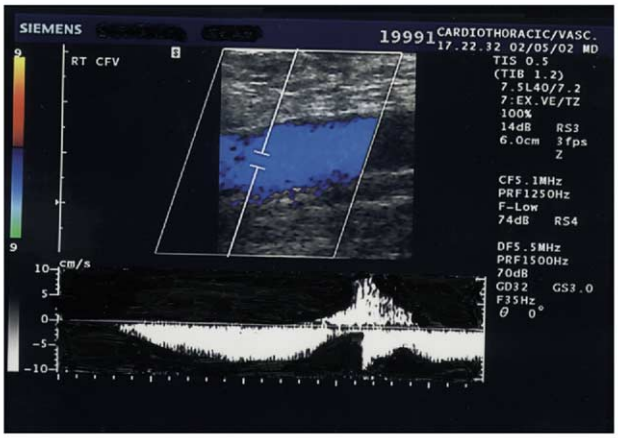

Fig. 1. Example of a duplex scan in 1 patient (**A**) before therapy (vein size = 19 mm, reflux = 1 second) and (**B**) after therapy (vein size = 11 mm, reflux = 0.3 seconds).

= 4, 5, or 6) were prescribed elastic compression of 30 to 40 mm Hg. All of the patients were provided with detailed instructions on exercises to build up their calf muscles.

Results

Complications of both treatments were few, including superficial thrombophlebitis in 7 patients, deep venous thrombosis in 1 patient, and hyperpigmentation of the skin in 2 patients. After completion of therapy, both groups of patients underwent a 6-month clinical follow-up that included duplex scanning of the common femoral, superficial femoral, and popliteal veins. The size and the reflux valve closure time of these veins were compared with preoperative studies. An example is shown in Figure 1.

The aggressive-treatment group, which included treatment for varicose and spider veins (Figs. 2 and 3) in addition to saphenous vein reflux, showed significant improvement in the size of the deep veins (80% of patients) and the reflux closure time of the deep vein valves (83% of patients). The patients who received less-aggressive treatment showed less improvement in the size (47% of patients) and reflux closure time of the deep veins (28% of patients). Table 3 shows the results of both groups of patients.

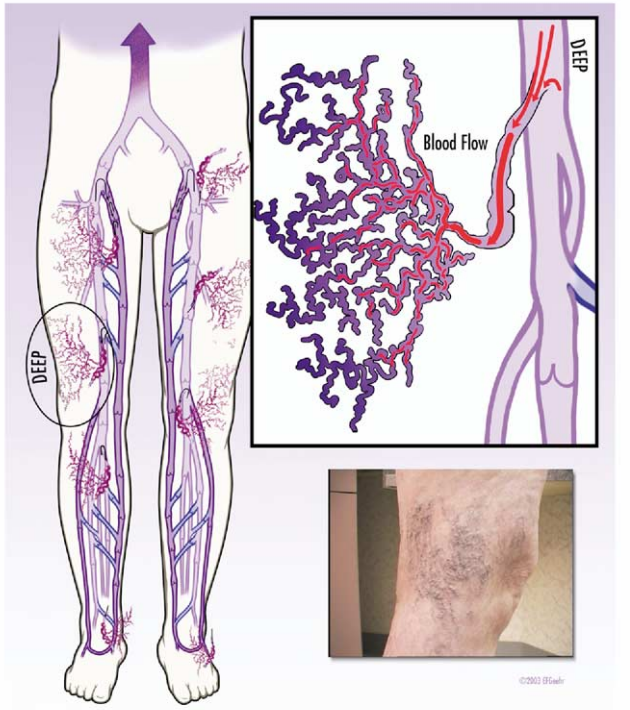

Fig. 2. Spider veins and their connection to the deep veins.

The highest number of our patients were classified as being in the earlier stages of chronic venous disease (CEAP = 1, 2, or 3) as compared with later stages of the disease (CEAP = 4, 5, or 6) (Table 2).

Comments

Many recent studies have shown that superficial venous insufficiency causes DVI because of reflux overflow of blood into the deep veins of the legs. This causes gradual distention of these veins over time, and the venous valves become incompetent [11–15,19]. It has also been shown that the treatment of superficial venous insufficiency leads to recovery of the deep venous insufficiency in more than 30% of the patients [11,12]. Our study has shown improvement in at least 80% of the patients when aggressive treatment of the superficial lesions was undertaken along with the proper management of the superficial venous reflux. We also found the majority of our patients showing improvement were also treated at earlier stages of the chronic venous disease (CEAP = 1, 2, or 3) as compared with later stages of the disease (CEAP = 4, 5, or 6) (Table 2). This

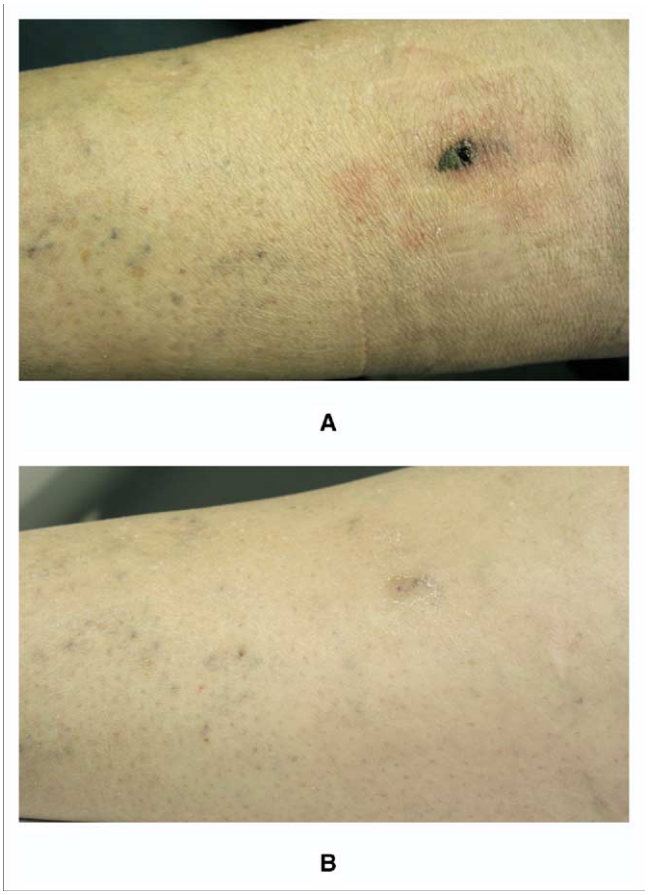

Fig. 3. This is a 50-year-old woman who was treated in the emergency room with bleeding from spider veins controlled with compression for 24-hours and subsequently treated with an Nd:YAG laser. (**A**) Before laser treatment. (**B**) Two weeks after 1 laser treatment.

Fig. 4. Example of spider hemangioma.

signifies the importance of early and aggressive treatment of both varicose and spider veins.

The clusters of spider veins are usually associated with one or more incompetent perforator veins connected to either the saphenous vein system or the deep veins of the legs (Figs. 2 and 3) [3,4,8,9]. These incompetent perforators cause significant reverse flow of blood into the clusters of spider veins, and, if there are multiple large clusters of these spider veins, the DVI will continue to worsen as time passes. This is then bound to cause the appearance of new spider vein clusters in other locations on the legs, thus perpetuating DVI and its sequele. Many recent studies have clearly shown that reflux overflow of blood from superficial venous insufficiency causes continued stress on the deep venous system, perpetuating and deteriorating the deep venous insufficiency [11,12].

Table 3
Treatment results for the study groups

	Aggressive treatment		Less-aggressive treatment	
	Size of deep veins (%)	Reflux of deep veins (%)	Size of deep veins (%)	Reflux of deep veins (%)
Decreased	51 (80)	53 (83)	17 (47)	10 (28)
Increased	1 (1)	4 (6)	5 (14)	11 (31)
Unchanged	12 (19)	7 (11)	14 (39)	15 (41)

The destruction of spider veins results in the obliteration of incompetent perforators, thus eliminating a major source of reflux overflow of blood into the deep veins. Over time, this could decrease the size of the deep veins and result in improvement of valvular function. It is, therefore, very important to treat venous insufficiency at all levels. When any one of these sources of venous incompetence is left untreated, it will perpetuate the condition of DVI and expose the patient to the risks of superficial thrombophlebitis, DVT, and eventual postphlebitic syndrome. Hospitalized patients with these problems are especially at very high risk of developing DVT and pulmonary embolism.

The terminology spider vein is misleading when used to describe these lesions affecting the legs. Most patients and even physicians have confused this term, associating it with spider veins of the face and upper body but not realizing that the spider veins of the legs are reflective of a serious underlying pathology. It has been well documented in the literature that the clusters of spider veins in the legs are usually associated with one or more perforating veins connect-

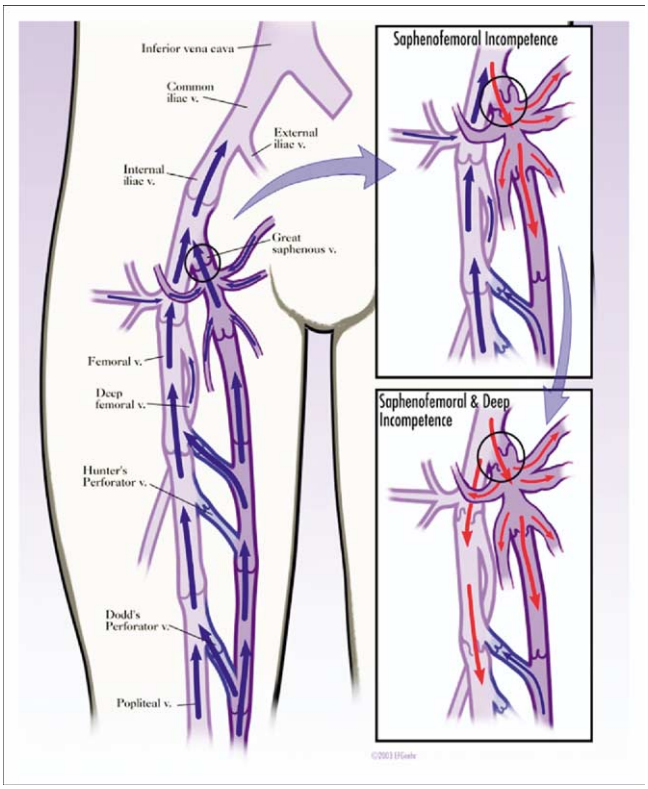

Fig. 5. Stages of venous insufficiency showing incompetence in deep vein.

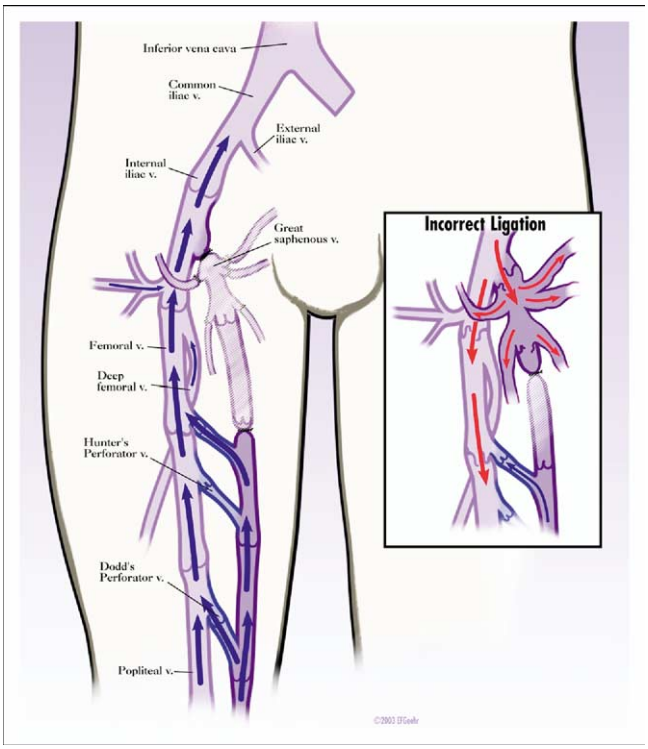

Fig. 6. Correct method of ligation of the saphenous vein. (Inset shows incorrect method.)

ing them with incompetent saphenous or deep veins [3,4,8,9]. Chronic venous insufficiency is a serious condition because of the associated high risk of superficial and deep venous thrombophlebitis [16]. We recommend that the spider veins of the lower extremities, especially when very dense and associated with venous insufficiency, be called spider hemangiomas or acquired venous hemangiomas (Fig. 4). The reason is because the spider veins of the legs are caused by the proliferation of new vessels in the skin and subcutaneous tissues caused by the release of endothelial growth factors under the influence of chronic venous hypertension [20,21]. This phenomenon has also been observed after sclerotherapy and local tissue trauma, leading to the appearance of small capillary blemishes sometimes referred to as postsclerotherapy matting, easily treatable with intense pulse light or laser therapy [22].

There are many well-established methods of treating venous conditions. Each method has pros and cons. The treatment methods we chose have excellent results. We do not use sclerotherapy because of the unacceptably high incidence of complications [22–26]. For the management of venous reflux, our preferred method of treatment is high ligation and partial selective stripping of the greater saphenous vein up to the next competent valve using Oesch's inversion PIN stripping technique (Figs. 5 and 6). To minimize the possibility of neovascularization at the groin, minimal dissection is carried out, and ligation is performed flush with the common femoral vein to avoid leaving behind any possible stump or the tributaries of the greater saphenous vein. Performing endovenous saphenous vein ablation with laser or radiofrequency and not performing the high flush ligation is not appropriate. Leaving behind the most proximal part of the saphenous vein along with its tributaries is likely to cause recurrence because of neovascularization at the groin, as has been reported in several recent studies [27–31]. A few recent studies have reported good results after percutaneous obliteration of the greater saphenous vein with laser or radiofrequency, without high ligation [32,33]. However, the follow-up of patients treated in these reports is rather short to recommend this therapy over the well established high ligation with partial selective stripping of the greater saphenous vein.

We have recently introduced into our practice endovenous laser treatment of the greater saphenous vein with high flush ligation, including excision of the tributaries. We prefer to introduce the laser tip with the delivery system from groin downward. This technique helps to locate the next competent valve, thus avoiding ablation of a normal segment of the saphenous vein. It is well established that greater saphenous vein incompetence below the knee level is very rare. We have found in majority of our patients that the valves below the middle of the thigh are competent. The number of patients treated with this technique is not enough to make any recommendation yet, but the procedure is less traumatic than PIN stripping.

Our study shows minimal or no improvement of DVI in patients who were treated only for superficial venous insufficiency and not for spider veins, especially when spider veins were extensive. On the other hand, the patients who

received aggressive treatment, including therapy for saphenous vein reflux, varicose veins, and aggressive elimination of spider veins showed marked improvement not only of the symptoms and edema but also of DVI. Several patients have shown 100% improvement of DVI, as documented by results of duplex scanning. We, therefore, strongly recommend that the patients with varicose and spider veins receive treatment not only of the venous reflux but also a complete elimination of both the varicose and spider veins of the legs to achieve improvement or the complete reversal of DVI.

Complete and aggressive treatment of superficial venous lesions accomplishes better results as has been shown by higher numbers of patients with improvement of reflux and size of the deep veins as compared with the patients in whom the spider veins are left untreated after treatment of saphenous venous incompetence. For the safety of the patients, it is highly recommended that the spider veins, when associated with venous insufficiency, should be treated aggressively along with saphenous venous reflux therapy.

References

[1] Alexander CJ. The epidemiology of varicose veins. Med J Aust 1972;1:215–8.

[2] Schultz-Ehrenbuegh U, Weindorf N, von Uslar D, Hirsch H. Prospektic epidemiologische studie uber die entstehunsweise der krampfadern bei kindern und jegendichen (Bochumer Studie I und II). Phlebol Proktol 1989;18:10–25.

[3] Bohler-Sommeregger K, Karnel F, Schuller-Petrovic SS, Sautler R. Do telangiectasias communicate with the deep venous system? J Dermatol Surg Oncol 1992;18:403–6.

[4] Tretbar LL. The origin of reflux in incompetent blue reticular telangiectatic veins. In: Davy A, Stemmer R, eds. *Phlebology 89.* Montrogue, France: John Libbey Eurotext; 1989:95.

[5] Bjordal RI. Circulation patterns in incompetent perforating veins of the calf in venous dysfunction. In: May B, Partsch H, Staubesant J, eds. *Perforating Veins.* Munich: Urban and Schwarzenberg; 1981: 71–88.

[6] Thompson H. The surgical anatomy and perforating veins of the lower limbs—A consensus conference. Vasa 1995;24:313–8.

[7] Arnoldi CC. The aetiology of primary varicose veins. Dan Med Bull 1957;4:102–7.

[8] Faria JL, Moraes IN. Histopathology of the telangiectasias associated with varicose veins. Dermatologica 1963;127:321–9.

[9] Moraes IN, Puech-Leao LE, Simone JC, et al. Microangiographic study of telangiectasia. J Cardiovasc Surg 1962;3:415–9.

[10] Weiss RA, Weiss MA. Resolution of pain associated with varicose and telangiectatic leg veins after compression sclerotherapy. J Dermatol Surg Oncol 1990;16:333–6.

[11] Walsh JC, Bergan JJ, Beeman S, Comes TP. Femoral venous reflux abolished by greater saphenous vein stripping. Ann Vasc Surg 1994; 8:566–70.

[12] Sales CM, Bilof ML, Petrillo KA, Luka NL. Correction of lower extremity deep venous incompetence by ablation of superficial venous reflux. Ann Vasc Surg 1996;10:186–9.

[13] Padberg FT Jr, Pappas PL, Araki CT, et al. Hemodynamic and clinical improvement after superficial vein ablation in primary combined venous insufficiency with ulceration. J Vasc Surg 1996;24: 711–8.

[14] Labropoulos N, Tassiopoulos AK, Kang SS, et al. Prevalence of deep venous reflux in patients with primary superficial vein incompetence. J Vasc Surg 2000;32:663–8.

[15] Yamaki T, Nozaki M, Fujiwara O, Yoshida E. Comparative evaluation of duplex-derived parameters in patients with chronic venous insufficiency: correlation with clinical manifestations. J Am Coll Surg 2002;195:822–30.

[16] Nicolaides AN, Kakkar VV, Field ES, Fish P. Venous stasis and deep-vein thrombosis. Br J Surg 1972;59:713–7.

[17] Kaplan RM, Criqui MH, Denenberg JO, et al. Quality of life in patients with chronic venous disease: San Diego population study. J Vasc Surg 2003;37:1047–53.

[18] Coon WW, Willis PW, Keller JB. Venous thromboembolism and other venous disease in Tecumseh community health study. Circulation 1973;48:839–46.

[19] Stonebridge PA, Chalmers N, Beggs I, et al. Recurrent varicose veins: a varicographic analysis leading to a new practical classification. Br J Surg 1995;82:60–2.

[20] Bodian EL. Techniques of sclerotherapy for sunburst venous blemishes. J Dermatol Surg Oncol 1985;11:696–704.

[21] Burnard KG, Whimster I, Clemenson G, et al. The relationship between the number of capillaries in the skin of the venous ulcer-bearing area of the lower leg and the fall in foot vein pressure during exercise. Br J Surg 1981;68:297–300.

[22] Davis LT, Duffy DM. Determination of incidence and risk factors for post-sclerotherapy telangiectatic matting of the lower extremity: a retrospective analysis. J Dermatol Surg Oncol 1990;16:327–30.

[23] Avramovic A, Avramovic M. Complications of sclerotherapy: A statistical study. In: Raymond-Martimbeau P, Presscott R, Zummo M, eds. *Phlebologie '92.* Paris: John Libbey Eurotext; 1992.

[24] Upton J, Mulliken JB, Murray JE. Major extravasation injuries. Am J Surg 1979;137:489–506.

[25] Biegeleisen K, Neilsen RD, O'Shaughnessy A. Inadvertent intra-arterial injection complicating ordinary and ultrasound-guided sclerotherapy. J Dermatol Surg Oncol 1993;19:953–8.

[26] Feied CF. Deep vein thrombosis: the risk of sclerotherapy in hypercoagulable states. Semin Dermatol 1993;12:135–49.

[27] Stucker M, Netz K, Breuckmann F, et al. Histomorphologic classification of recurrent saphenofemoral reflux. J Vasc Surg 2004;39:816–22.

[28] Nyamekye I, Shephard NA, Davies B, et al. Clinicopathological evidence that neovascularisation is a cause of recurrent varicose veins. Eur J Vasc Endovasc Surg 1998;15:412–5.

[29] Glass GM. Neovascularization in recurrence of the varicose great saphenous veins following transaction. Phlebology 1987;2:81–91.

[30] Fischer R, Chandler JG, De Maesener MG, et al. The unresolved problem of recurrent saphenofemoral reflux. J Am Coll Surg 2002; 195:80–94.

[31] Chandler JG, Pichot O, Sessa C, et al. Defining the role of extended saphenofemoral junction ligation: a prospective comparative study. J Vasc Surg 2000;32:941–53.

[32] Rautio T, Ohinmaa A, Perala J, et al. Endovenous obliteration versus conventional stripping of primary varicose veins: a randomized controlled trial with comparison of the costs. J Vasc Surg 2002;35:958–65.

[33] Pichot O, Kabnick LS, Creton D, et al. Duplex ultrasound scan findings two years after great saphenous vein radiofrequency endovenous obliteration. J Vasc Surg 2004;39:189–95.